The Australian Craniofacial Unit

1975 – 1996

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January 1997

Dedication

Dedicated to my family for their love patience and support.

CONTENTS

Abstract

This thesis sets out the principles of craniofacial surgery and how they have been utilised to form the Australian Craniofacial Unit. Progress of the organisation is mapped over twenty one years using selected published papers in which the author has in some way contributed to the development of teaching, research and service in craniofacial surgery. The papers are grouped so as to show the progress made in the areas of Trauma, The Craniosynostoses, Rare Craniofacial Clefts, Frontal Ethmoidal Meningoencephaloceles, Craniofacial Tumours, as well as Research and Development.

The central theme of the development of this new surgical discipline is that progress has been from the technically possible to an understanding of the pathology, pathogenesis and natural history of the disease processes dealt with by the craniofacial team. In reciprocal fashion research inspired by clinical experience validates the choice of technical procedures and defines their range of application.

The thesis reaffirms the need for the multi-disciplinary setting, properly administered and politically supported, which must be closely aligned with research and development. The author's efforts have been directed towards establishing an infrastructure in which this could happen and developing tools by which it could be achieved.

The thesis concludes with some comments about the future of craniofacial surgery in the light of the progress made since 1975.

Declaration

Declaration regarding thesis

I, David John David

Head

of The Australian Craniofacial Unit, Department of Plastic and Reconstructive Surgery, Royal Adelaide Hospital and the Women's & Children's Hospital, Adelaide, Clinical Senior Lecturer, Department of Surgery, University of Adelaide, solemnly and sincerely declare that the thesis entitled

The Australian Craniofacial Unit

has been composed by myself (with the provisions set out below), and has not previously been submitted in whole or in part for any other degree or diploma.

And I make this solemn declaration conscientiously believing the same to be true and by virtue of the Oaths and Declarations Act 1957.

Declared by: Witnessed by:

Dated: 14 January 1999 Dated: 14 Jan 1999

As is usually the case in research work, I cannot claim to be uniquely and wholly responsible for the content of the consequent publications. Indeed, the multi-author nature of a great many of the articles incorporated in this thesis would give the lie to such a claim. It nevertheless appears to me that I have been the most significant author, in the majority of the work here submitted. That is, I have in each case been primarily responsible for identifying the problem to be studied, in devising or selecting the investigatory techniques, and in making deductions from the results. Where this

is not the case I have identified the particular significance of my role in the publication and its relevance to the development of craniofacial surgery in the context of this thesis. I wish to record my appreciation of the physical and intellectual assistance given to me by all my collaborators in these various pieces of work.

The work embodied in the publications that form this thesis was undertaken between 1975 and 1996. During that period of time the successive appointments I have held:

1974 –	Senior Visiting Plastic Surgeon, Royal Adelaide Hospital
1974 –	Senior Visiting Plastic Surgeon, Women's & Children's Hospital, Adelaide
1975 –	Head, The South Australian Craniofacial Unit (to become in 1988 the Australian Craniofacial Unit).
1985 –	Head, The Department of Plastic & Reconstructive Surgery, Royal Adelaide Hospital.
1989 – 1994	Chief of Surgery, Adelaide Children's Hospital (to become the Women's & Children's Hospital)
1991 –	Head, The Department of Plastic & Reconstructive Surgery, Adelaide Children's Hospital (to become the Women's & Children's Hospital).

Acknowledgement

I would like to express my gratitude to all my teachers past and present; to all members of the Australian Craniofacial Unit and its associated organisations, the Australian Cranio-Maxillo Facial Foundation and the Institute of Craniofacial Studies; and to those in administration, government and the general community who have supported the Australian Craniofacial Unit and its work.

Introduction

In nature there's no blemish but the mind;
None can be call'd deform'd but the unkind

Shakespeare — Twelfth Night

Introduction

In 1971 Paul Tessier, a French plastic surgeon, presented his views on the current state of, future development of, and training in craniofacial surgery[1]. This presentation was made at the International Society of Plastic Surgeons meeting, held in Melbourne Australia. Notwithstanding the usual progress in surgery being a gradual one, as others had crept up on the problems (Converse)[2], or flirted with them (Gillies)[3], there was the usual excitement, anger, rejection, and slow acceptance of Tessier's views. From that time to this there has been the development of a new surgical discipline, the goals and practice of which have been set down by the International Society of Craniofacial Surgeons since 1982.

New disciplines evolve in response to problems that are not being adequately addressed by the current order. The development of plastic surgery after the First World War is an example of this process. The problem being the very visible presence in all affected nations of millions of injured young soldiers, who by other advances in trauma care were not destined for early death. The traditional modus operandi of surgery and surgeons had been extended to deal with this problem. So Gillies in Britain and its empire, Lexer in Germany, and Barrett-Brown in the USA, established the organisation that is modern western plastic and reconstructive surgery.

What was the nature of the problem facing surgery in the 1960's, that was addressed by Tessier and his followers?

There existed a group of patients with dramatic deformities of the face and skull which were unable to be treated by the available surgical techniques. The epicentre of the deformity is in the area of the orbits, cranial base, and indeed often in that key stone of the skull, the sphenoid bone. This was a truly difficult problem, the site being small, encompassing the brain, sight, appearance, as well as

the airway. At least three different well established disciplines of surgery deal with some aspects of, but not the whole problem.

These children were often diagnosed as nonviable and when they did survive were confined to institutions where they became retarded through lack of treatment, neglect or both. The absence of a technical solution to the problem seemed to retard efforts to investigate and more fully understand the aetiology and pathogenesis of these conditions.

The prospect of a satisfactory outcome is often a stimulus to further understanding.

With a little imagination it became obvious that understanding the bizarre anatomy of the craniofacial skeleton in syndromal craniosynostosis and rare craniofacial clefts would help in managing trauma and tumours of the same region. Patients with these more common problems were falling between the disciplinary fires resulting in inadequate management.

A paraphrase of Tessier's proposition in 1971 states that:
The technical problem of the surgical approach to the cranial base and orbito-cranial complex, for the purpose of reorganising that area to achieve functional and cosmetic correction in congenital deformity, involved two components:

1. **Teamwork** — working hand in hand in the operating theatre with a neurosurgeon, thus redefining the strictly individualistic culture enshrined for almost half a century.

2. **To describe a safe approach** to the orbito-cranial complex via the coronal scalp flap dissected in the sub periosteal plane to within ten millimetres of the orbital apex preserving the exiting cranial nerves.

By combining with the neurosurgeon who exposes the anterior

cranial fossa, the stage was set for craniofacial surgery. The possibility of successful treatment in turn provided a further stimulus to investigate the aetiology and pathogenesis of the conditions being treated (See Chapter 3).

The author's promotion of this process began in 1972 with a series of visits to Paul Tessier in Paris and consequential involvement with him and craniofacial surgery from that time to this.

The time period over which the substance of this thesis extends is 1975 (the formal founding of the then South Australian Craniofacial Unit), to the present day.

The purpose of this thesis is to describe the process of development of the Australian Craniofacial Unit, the author's contribution to that development, and that of the discipline in general over twenty years, using selected published material to support that description.

It is important to understand that many of the innovations made in the mid seventies are now considered commonplace. Some, whilst widely acknowledged are not widely practised for a variety of reasons, some political, some psychological.

Although Tessier promulgated the idea of the multidisciplinary team, he himself did not implement it fully. It was left to his disciples to form the wider assessment team comprising all the necessary specialists, who see each patient, meet and discuss management and implement treatment. Several papers were written on this and other aspects of establishing a craniofacial unit (Munro[4], McCarthy[5], David[6]).

The author's additional contribution at this stage was to understand the necessity to establish the craniofacial unit as an administrative entity, with secure political backing. Under these circumstances a craniofacial unit is able to function as a trans-

institutional body utilising resources of hospitals, universities, and institutes functioning in Australia and overseas.

At this early stage the South Australian Craniofacial Unit was an unique organisation in the world.

From Tessier's first articles to the present time it has been agreed that a craniofacial team must serve a critical population in order to see enough serious cases to develop and maintain the skills of the clinicians and to study the disease processes. For those practising the discipline seriously this is a painfully self evident truth, sadly often becoming relevant in retrospect. The author has vehemently held this view, primarily for the direct benefit of the individual patient, but also because such units are responsible for investigating the pathology and pathogenesis of the diseases that have been the interesting object of technical advance. This view underpins the author's philosophy on the management of craniofacial deformity and is one of the central strategies of the Australian Craniofacial Unit.

To apply the philosophies and implement the strategies, the organisation needed further development. Small but unique funding was supplied for research by the State Government.

In addition a charitable fund raising arm was created to act as a liaison with the general community as well as to raise funds for research and support of the Unit's overseas service and teaching commitments.

The post graduate teaching activities expanded rapidly and an additional structure was needed to administer this area of work and deal with any of the fruits of the research with commercial potential when and if necessary.

With these structures in place patients could be treated according to protocols which were continually revised as a result of

outcome monitoring. Tools of investigation could be developed and used in an integrated way to investigate the disease processes. Teaching could be conducted in Australia and overseas, intellectual property and other products of research could be marketed.

In 1988 the organisation was designated the Australian Craniofacial Unit (ACFU), and a Centre of Excellence by the Australian Health Ministers' Advisory Committee.

The ACFU has an unique structure which was designed to maximise the multidisciplinary approach, as well as integrate teaching, research and delivery of health care. This thesis will show the achievements to date with respect to various disease entities, and conclude with some natural projections which follow from what has been achieved.

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Chapter 1
Development and Organisation

Miracles do not happen
Mathew Arnold

1. Development and Organisation

Craniofacial Surgery: The Team Approach, 1977[1] describes the need for a wider craniofacial team. The arguments for multidisciplinary care, critical patient mass, and centralisation were very topical at the time and remain so at the present. This paper describes the ideals behind the organisation set up to serve Australia in the mid 70's.

A decade later the Editorial Comment: Craniofacial Surgery: The Interface, 1989[2] in The Australian and New Zealand Journal of Surgery addresses the tensions and dilemmas that accompanied the delivery of complex high technology health care. At this stage the author's experience showed that the problems of congenital and acquired craniofacial deformity could be successfully managed and the challenge was to establish multidisciplinary units that were medically and economically efficient to deliver healthcare, teaching and research.

Clinics of Australia — The Australian Craniofacial Unit, 1994[3] in the Australian Journal of Otolaryngology sets out the structure of the ACFU as it has developed. Information about the range and intensity of the workload is presented together with a sketch of the scientific activities. This review paper shows the development of the idea and the physical structure from the situation reported in 1977.

It has long been the author's view that craniofacial surgery would become a separate surgical specialty, the essence of which is multidisciplinary care.

Craniomaxillofacial Injuries: The Wider View, 1995[4] shows how such ideas are applied to the trauma setting as well as dealing with vexed questions of training in craniofacial surgery and the nature and funding of trauma research.

These four papers represent the progress from the initial concepts and early structures of a craniofacial service to the recent adaptation of the ideas to form a model of service, teaching and research for the commonest of craniofacial problems, namely trauma.

■

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■

Craniofacial Surgery: The Team Approach

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Craniofacial surgery offers a new hope to some grossly deformed people. This complex surgery, which is based on a multidisciplinary team approach, needs to be carefully rationalised and regionalized to facilitate investigation, to improve planning, to reduce the number of complications, and to conserve financial resources. To date the Cranio-Facial Clinic at the Adelaide Children's Hospital and the Royal Adelaide Hospital has reviewed 37 cases and operated upon 13 of these. This work is presented together with a review of the team approach.

The principles and practice of craniofacial surgery are now well established. This service is widely offered in a number of centres around the world to patients suffering from severe facial deformities. Paul Tessier (1967) has developed and perfected these techniques over the last decade, demonstrating that wide and adequate exposure of the craniofacial skeleton can be obtained by subperiosteal stripping of bones, cutting and repositioning the skeleton, and reconstructing the deficiencies by bone grafts. He pioneered the team approach to these problems by combining with his neurosurgical colleagues to expose the base of the anterior cranial fossa, which is so often the keystone in the correction of these deformities.

Because this kind of surgery is associated with a high risk of morbidity and mortality, there is a special need for the multidisciplinary approach. The team has been expanded to include all of the specialities necessary for the complete assessment and follow-up of these patients. Those units in which the surgery has been successfully pioneered and advanced have made their advances on this basis.

The organisation that has been established in South Australia will be described. The patients who have been assessed and those who have been operated upon will be discussed, together with the implications of organising and maintaining such a service.

The Team

The composition of the team is shown in Table 1. With the approach to surgery of a team of this magnitude, the risks can be kept to a minimum, but also, and more importantly, the results can be adequately measured so that the continuance of the concept of craniofacial surgery can be justified. The team must of necessity be large. In the preoperative investigation it is responsible for data collection, diagnosis, and operative design, and the responsibilities of its members are as follows.

TABLE 1

Craniofacial Team

Plastic surgeon	Orthodontist
Neurosurgeon	Oral surgeon
Anaesthetist	Prosthodontist
Ophthalmologist	Psychiatrist
E.N.T. surgeon	Social worker
	Photographer

(a) Plastic surgeon.—The plastic surgeon is the head of the team and as such assumes responsibility for patient care. Special training in neurosurgery, orthopaedic surgery, and maxillofacial surgery is recommended. It has been suggested that craniofacial surgery should become a separate speciality (Munro, 1975). This is probably an extreme point of view, but the plastic surgeon must not only be capable of performing the bony surgery, but also be able to correct the many difficult soft tissue problems that are always associated with craniofacial deformities.

(b) Neurosurgeon.— It is important that the neurosurgeon's role be seen as an extension of the one already played in the correction of skull deformities resulting from craniosynostosis. The neurosurgeon is responsible for the preoperative neurological assessment of the patient and, when the transcranial approach is used, for the exposure of the anterior cranial fossa. A high degree of cooperation is required throughout these procedures, with a blending of the techniques of plastic surgery and neurosurgery.

(c) Anaesthetist.— The importance of the anaesthetist's role is highlighted by the fact that craniofacial operations are frequently in excess of 12 hours' duration and are performed on patients whose ages range from five to 40 years. Large blood replacements are invariably necessary.

(d) Orthodontist and oral surgeon.— These dental disciplines are involved in the planning procedures *(vide infra)*. Many patients need presurgical or postsurgical orthodontic treatment.

TABLE 2

Routine Preoperative Skiagrams

Routine skull and facial views
Anteroposterior and lateral skull (cephalometric)
Anteroposterior and lateral tomography of orbits
Anteroposterior and lateral chest

(e) Ophthalmologist. — The concept of craniofacial surgery is based on the fact that the orbits can be safely moved in three dimensions, therefore a detailed preoperative examination by a neuro-ophthalmologist is essential. A close examination of the following is of the greatest importance: interorbital distance interpupillary distance; lateral canthal width; palpebral fissure width; exophthalmometry; visual acuity, visual fields; eye position; muscle function; and fundoscopy.

(f) Radiologist.— The radiologist is responsible for taking and interpreting the skiagrams. The routine views taken are shown in Table 2. In addition, cerebral arteriograms, air studies and brain scans are often needed. The standard lateral and anteroposterior skull films, taken on a cephalostat, are used for the operative planning and to plot growth up to the time of surgery, and after operation until stability has been achieved.

(g) Speech pathologist.— Surgery for deformity of the mid-face frequently involves sagittal displacement of the bones with a possible change in the

relationship of the components of the velopharyangeal sphincter. Preoperative assessment involves an interview, speech recording and nasendoscopy. These procedures are repeated after operation.

(h) Otorhinolaryngologist.— Each patient is checked for middle and inner ear problems. Midfacial stenosis frequently creates secondary nasal and paranasal sinus problems which need to be assessed and managed by the ear, nose and throat surgeon.

TABLE 3

Craniofacial Deformities Assessed in South Australia	
Crouzon's syndrome	7
Apert's syndrome	2
1st and 2nd bronchial arch syndrome	5
Cleft lip and palate with maxillary hypoplasia	15
Faciostenosis	2
Hypertelorism	2
Orbital dystopia	2
Uncorrected trauma	1
Others	1

(i) Social worker.— Throughout time, people have associated mental retardation with a deformed face. An assessment of the patient's intelligence should be made before operation. A thorough knowledge of the patient and his environment is needed for assessment as an indication for surgery, and as a base line for subsequent follow-up. The true worth of these surgical achievements can only be measured in terms of patient satisfaction, and this measurement must of necessity be made by the psychosocial team. Patients and their families also need preoperative adjustment to the realities of the surgery. Often the severely deformed face cannot be made completely normal. In other patients the sudden drastic change makes them unrecognisable to their friends. Such patients will almost certainly need help in adjustment.

TABLE 4

Osteotomies Performed
Transcranial correction of hypertelorism
Fronto-orbital advancement
High Le Fort I osteotomy x 4
Le Fort II osteotomy
Le Fort II osteotomy + mandibular osteotomy
Le Fort III osteotomy, subcranial
Le Fort III osteotomy + mandibular osteotomy
Le Fort III osteotomy, transcranial

Patients

In the past year the South Australian craniofacial team has assessed 37 people with craniofacial anomalies, and operated on I3 of these (Table 3). From the complex range of osteotomies available. we have undertaken the management of the types shown in Table 4.

Designing the Operation

The operative design cannot be done on an *ad hoc* basis in the operating theatre. It is necessary to have appropriate cephalometric skiagrams of the skull. The

outline is traced and the standard lines and angles drawn. An accurately superimposable transparent photograph is taken as well. A tracing can be cut up and the angles repositioned into acceptable dimensions.

The skiagram and the transparent photograph can be cut out and changed to give a suitable postsurgical prediction of appearance. The methods used have been based on those of Henderson (I974), with some modification by the members of the Department of Oral Pathology and Surgery at the University of Adelaide (Figure 1) . In this way the exact size of the defects to be created by the osteotomies and hence the size of the bone grafts can be predicted. Reference to a wide range of "normals" is essential. In one case it was necessary to have access to the normal range of measurements for a female aborigine of a particular tribe before it was possible to correct her untreated craniofacial fracture.

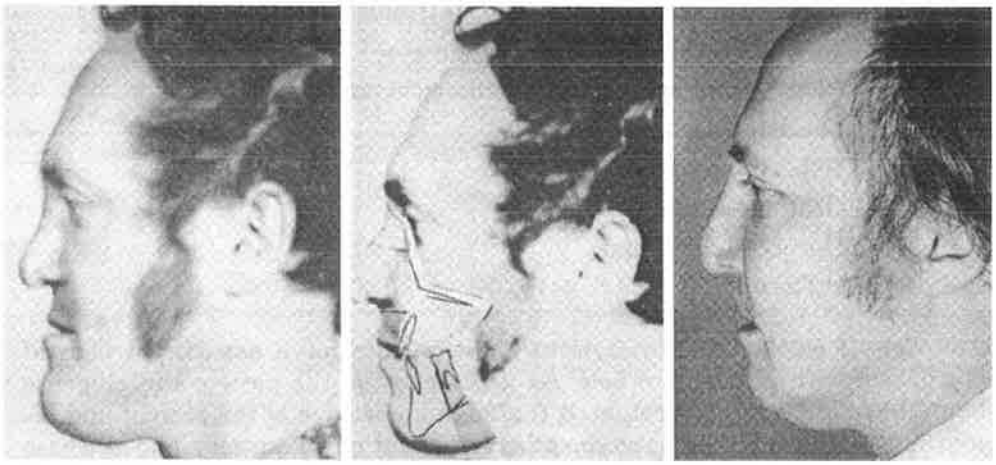

FIG. 1. *(left) preoperative lateral transparent photograph; (centre) the photograph is sectioned along the proposed osteotomy lines to produce the postoperative appearance; (right) postoperative appearance after a mid-face advancement and mandibular recession.*

Many procedures will of necessity involve a simultaneous correction of the mandible as well as the orbitocranial skeleton. These corrections revolve around a correct occlusion, and for this purpose dental study models are cut and mounted on an articulator (Figure 2).

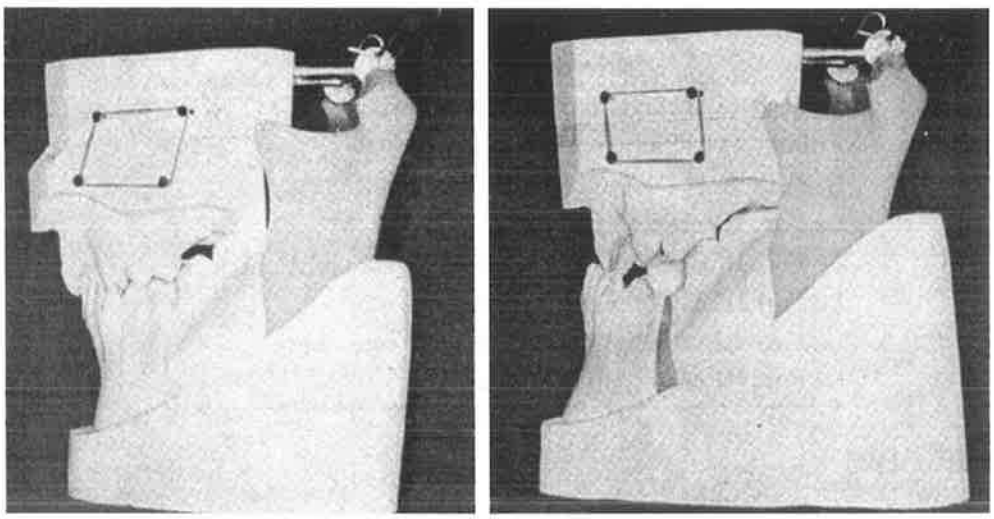

FIG. 2. *Model plan to demonstrate the proposed osteotomy.*

In those cases where the middle third of the face is being repositioned we have used the craniomandibular fixation with external rods fixed at a predetermined distance, so that when the maxilla is moved into occlusion with the mandible, the gaps at the osteotomy sites that must be filled with bone become apparent.

The anaesthetic team bases its care on a complete biochemical and haematological work-up with central venous pressure, cardiac, and body temperature monitoring, a spinal catheter also being inserted when a transcranial

approach is made. Blood replacement is titrated against the central venous pressure level. In those cases where the upper airway is threatened, a tracheostomy is routinely performed.

Tessier (1971) established that the useful orbit can be displaced in the three spatial directions. This phenomenon is the basis of many patterns of osteotomies now available to the craniofacial surgeon. Sagittal displacement of the orbits will be applicable to retrusions of the face or frontal bone related to craniosynostosis or faciostenosis. Transverse movement is applicable in the correction of hypertelorism. Vertical displacement will correct orbital malposition secondary to trauma or orbitofacial clefts. A combination of all the movements produces a rotational effect which can: (i) increase one or both orbital diameters; (ii) correct maxillary open bite; and (iii) lengthen a short face (Figure 3).

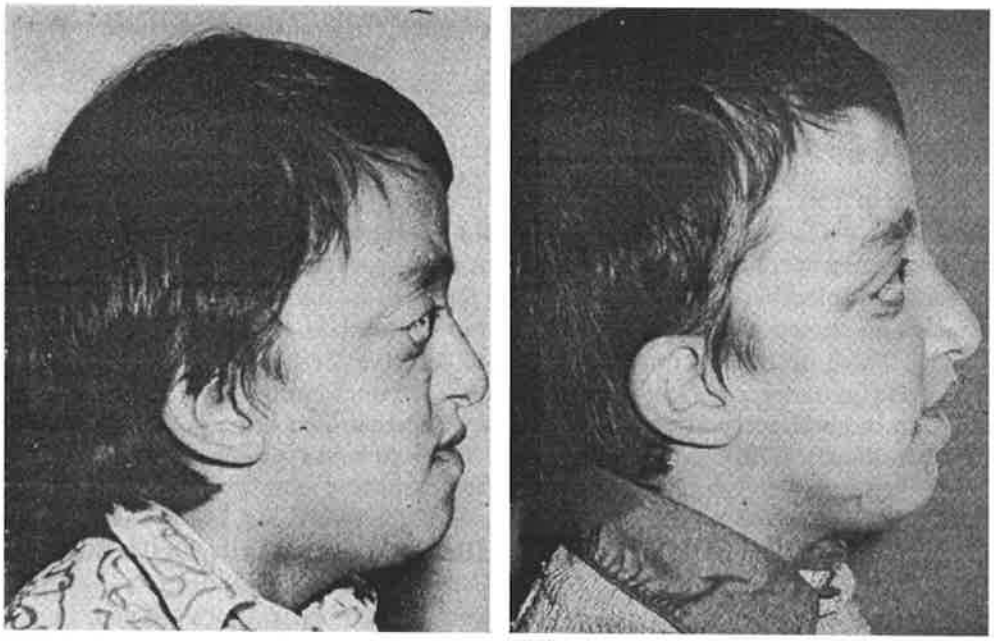

FIG. 3. *Preoperative and postoperative photographs of boy with Crouzon's disease. The middle third of this face was advanced and lengthened.*

The principles of surgery are essentially simple. They are:

1. Careful dissection of periorbitum and pericranium permits every osteotomy and every displacement without major risk to optic, oculomotor, palpebral, or lachrymal functions.

2. It is possible to perform a one-stage correction of all displacements, malformations, and bony defects, as well as correction of the canthi and lachrymal systems.

3. For the orbital roof, the cranial route is the simplest and safest. The dura mater can be preserved or reinforced if necessary.

4. The orbitocranial and maxillofacial problems can be dealt with together as they are essentially inseparable.

In the view of Paul Tessier, the abnormal anatomy associated with craniofacial dysostosis makes each dissection an adventure in itself. The distorted relationship between the orbital walls, and the anterior and the middle cranial fossae, presents obvious hazards (Figure 4). Adequate functional results can only be obtained by painstakingly careful dissection and reconstruction with as much as possible being done at the one time because experience has taught that operating for a second time on the orbits is always exceptionally difficult. So gross is the distortion of the anatomy encountered, especially in cases of craniofacial dysostosis, that no animal skull or even human cadaver can be used as a source of experimentation.

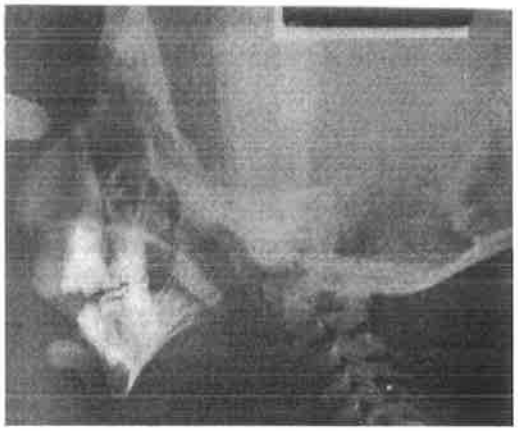

FIG. 4. *Lateral skiagram of skull, showing the distorted relationship between the cranial base and the face in craniofacial dysostosis.*

Incidence and Epidemiology

Table 5 gives a list of the diseases that could be treated by the craniofacial team. The frequency of these deformities in the Australian community is uncertain. If we take a projection against the figures for Britain and the United States of America as shown on this table against our population of approximately 13½ million, which produces approximately 240,000 live births per year, then some idea of the occurrence rate of these deformities in our community can be gained, although in precise terms this remains very obscure. The exact population that the craniofacial team should serve is obviously to some extent arbitrary. Paul Tessier has suggested that one team per 10 to 20 million people would be suitable, if the team performed only craniofacial surgery. On this basis. Ian Munro (1975) has suggested that Canada and the United States be served by seven craniofacial centres. It is a well-known fact, from experience gained by kidney transplant programmes, that any centre performing less than 25 transplants per year has a considerably higher mortality than a centre performing more than 100 (Scribner, 1973). This fact, combined with the rapid rise in medical costs, would indicate that regionalization and rationalization of such a service are almost an economic as well as a medical necessity.

TABLE 5

Types	Incidence (live births)	New cases per year in Australia
Craniofacial dysostosis	1:170,00	≈ 2
1st, 2nd bronchial arch syndromes	1: 6,000	40
Treacher-Collins syndrome	1:10,000	24
Hypertelorism	?	?
Faciostenosis	?	?
Rare facial and orbital clefts	?	?
Uncorrected trauma	?	?
Severe cleft lip and palate	?	?

Conclusions

1. There is an obvious need for accurate diagnosis to enable records to be compiled to facilitate the epidemiological studies of craniofacial deformities.

2. Accurate preoperative and postoperative measurements with access to standard records and routine follow-up to assess the late technical results are obviously necessary.

3. Because this surgery is difficult technically and carries a high morbidity and mortality rate, the craniofacial team must be skilled and experienced.

4. Accurate assessment of the psychosocial status of the patient and the subsequent follow-up results determine the true value of such surgery in the long term, namely the effect on the patient himself and his family.

5. Accumulation of skill in a central unit enables the development of the discipline and makes it possible for the teaching of a new generation of craniofacial surgeons.

Acknowledgements

I wish to acknowledge the contribution made by the other members of the South Australian Cranio-Facial Team, namely: Mr D. Simpson, neurosurgeon; Dr G. Keith, ophthalmologist; Dr T. Allen, anaesthetist; Dr R. Edwards, anaesthetist; Mr J. Rice, otorhinolaryngologist; Dr M. Nugent, orthodontist; Dr J. Herd, oral surgeon; Dr K. Moore, prosthodontist; Mrs J. A. Barritt, social worker; Miss C. Shepherd, social worker; Mrs A. Bagnall, speech pathologist; and members of the nursing staff and Photographic Departments of the Royal Adelaide Hospital and the Adelaide Children's Hospitals.

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Editorial Comment

CRANIOFACIAL SURGERY: THE INTERFACE

It is now 21 years since Paul Tessier first enunciated the concept of the craniofacial team, combining plastic surgical and neurosurgical approaches to expose the facial skeleton below and the cranial base above, so that the orbitocranial complex could be mobilized and moved in three dimensions.[1,2] The expansion of the indications for these techniques has resulted in the evolution of craniofacial surgery into a now well-established discipline, and it is an appropriate time to review the way in which it interfaces with its parent specialties.

The surgical challenge of the correction of severe craniofacial deformity was largely unanswered until Gillies and Harrison performed the first successful Le Fort III advancement osteotomy for the correction of midface retrusion in a patient with Crouzon's Syndrome in 1948.[3] Between then and the late 1960s, numerous authors attempted the correction of deformities of the orbitocranial complex by onlay bone grafting and superficial cosmetic surgery of the soft tissues of the head and neck.

The quintessential advance, however, was the demonstration by Tessier in 1967 of the two-stage, combined craniofacial approach to the orbitocranial complex: extensive subperiosteal dissection of the craniofacial skeleton exposing the anterior and parts of the middle cranial fossae above, and the orbits and midface below, allowing the safe section and movement of the face, orbits and anterior cranial complex in three dimensions. [1,2]

More importantly, the recognition that patients with a craniofacial deformity undergo a lifetime of change in terms of the evolution of their craniofacial 'make-up', and cannot be managed solely by 'an operation', was an early and fundamental development. Patients with these rare and complex facial deformities can present at any stage in the progression of their disorder, thus demanding an understanding of the pathology of the deformity in order to permit an integrated holistic approach to management.[4]

Based on the well-tried model of the 'cleft lip and palate clinic', with the multispecialty input of plastic surgeons, speech pathologists, dentists, orthodontists, etc., the development of major craniofacial units was a natural extension.[5–8] Such units have evolved similarly as complex multidisciplinary organisations capable of performing all the surgery necessary to correct the spectrum of craniofacial deformity, but doing so within the context of the overall management of the deformity as it affects both the patient and the family.

Essential to the composition of the craniofacial team is a coexistent research and scientific section. The very recondite nature of the craniofacial deformities demands close clinical examination, and extensive photographic, radiological and operative documentation of each patient. Only with this approach can we expand, complete or alter the descriptions of the anatomy of the deformity, trace its progression with time and document and evaluate the changes induced by intervention.

Thus, newly described disorders, such as Binder's syndrome, follow the elucidation of those consistent features of nasomaxillary hypoplasia, and allows an understanding of where it lies in the overall spectrum of chondrodysplasia punctata.[9,10] Similarly, accurate documentation has allowed the proposal of a new classification of hemifacial microsomia, that not only orders patients according to the severity of their skeletal and soft tissue deformity but also speculates on a planned programme of management. The variable presentation

of this disorder demands diverse approaches, such as non-surgical orthodontics for minor occlusal disturbances, multistaged surgical procedures correcting orbital dystopia, bimaxillary osteotomies to level the occlusal plane and microvascular free tissue transfer to augment the soft tissue and bony deficits.[11,12]

In Treacher-Collins syndrome, the combination of high quality, computerized tomographic (CT) scanning with three-dimensional reformats, and wide surgical exposure, has demonstrated the complete anatomy of the orbital deformity. Exposing the three-dimensional relationship of the periorbita, infratemporal fossa and pterygoid fossa has produced the understanding necessary to evolve a blepharo-orbital correction using pedicled calvarial bone, and local hyocutaneous transposition flaps.[13]

Adopting the same philosophy with the rare craniofacial clefts has extended the three-dimensional documentation of these diverse but recondite disorders, and confirmed the anatomical basis for their distinction from the previously included frontonaso-ethmoidal meningo-encephaloceles.[4,14-16] As importantly, appreciation of the extreme soft tissue and skeletal deficiencies which accompany these rare clefts has exposed the reconstructive demands. Indeed, here, more than anywhere, the early promise of a somewhat 'magical' one-off surgical intervention has not been fulfilled, but the potential for good results has followed the recognition of the effect of the fourth dimension, and the provision for adequate sequential correction of the skeletal and soft tissue deficiencies.

Apart from these disorders of intrinsic growth, where the template for future craniofacial development is deficient in its basic make-up, one also identifies those conditions where the intrinsic structural outline is normal, but deforming forces influence subsequent growth. Thus, in the craniosynostoses, the early radical release of the synostosed sutures of the cranial vault and base arose from the demonstration that cranial remodelling and facial growth were optimized by such early intervention.[4,17] Similarly, in the frontonasoethmoidal meningo-encephaloceles, early removal of the space-occupying lesion and relocation of the otherwise normal, but displaced, facial skeleton restores the potential for relatively normal craniofacial growth.[14]

Indeed such evolution in surgical approach can only occur after the considered longitudinal review of treatment, with an honest appraisal of the real results and the complete recording of complications.

Nowhere has it been more important than in craniomaxillofacial surgery that surgical manipulations are planned and executed with such precision, stability and long-term predictability of outcome. The advent of CT radiographic assessment, and in particular the addition of three-dimensional reformatting, has provided an exciting graphic, but qualitative depiction of the skeletal deformities.[16,18] As the facility to quantitate the three-dimensional documentation evolves, so its role in surgical planning and follow-up will expand.

The concomitant development of surgical approaches such as primary bone grafting and rigid internal osteosynthesis of the craniofacial skeleton are facets of the improving accuracy and predictability of surgical outcome.[19,20]

The application of microvascular free tissue transfer, and the techniques of tissue expansion have similarly broadened the options, and improved the quality of soft tissue reconstruction in the head and neck.[12,21,22] This spectrum of technical advances represents but a small aspect of the interface between this superspeciality and its parent, plastic and reconstructive surgery.

The principles established in the treatment of congenital craniofacial deformity have been applied widely in the acute management of severe craniofacial trauma. Employing the techniques of wide craniofacial exposure, elective primary bone grafting of the orbit, midface and nose in combination

with rigid internal fixation, has produced primary predictable correction of deformity, reduced the period of hospitalisation by 50% and minimized the requirements for surgery for uncorrected deformity.[4] The accompanying facility of rigid miniplate and miniscrew fixation ensures stability and diminishes the requirement for prolonged periods of intermaxillary fixation.[20] However, questions remain: how long should plates remain and should they be removed routinely?

The exposure, extirpation and reconstruction of tumours involving the previously surgically inaccessible regions of the orbit, cranial base and infratemporal fossa is now possible. The confidence in, and understanding of, techniques of both craniofacial exposure and approaches to the disassembly of the craniofacial skeleton, permits access to, and the potential feasibility of curative resection of, such tumours while minimising disabling disfigurement.[22]

The logical extrapolation of these treatment philosophies is into the realms of aesthetic surgery. No longer must one organ, for example, nose, chin, etc., be considered in isolation, and neither is it appropriate to assume that superficial cutaneous surgery will reproducibly correct the aesthetic problems or ageing changes of the face. The face must be viewed as a whole, with an extension of pictorial representations into objective quantitation, both clinically and radiologically, to the relationships between profile and occlusion, and, as importantly, into the area of the psyche. Of necessity, then, complete and rigorous assessment demands again a multidisciplinary approach.

The extra dimension to the surgical management of aesthetic problems follows an understanding of the changes induced by skeletal shifts, muscle plication and soft tissue augmentation, which are so overtly manifest in the craniofacial patient. Thus, no longer are changes solely produced by superficial surgery stretching the skin over an abnormal basal skeleton, but, instead, the facade of the face can now be draped over the remodelled bony and cartilaginous skeletal framework pushing forward from behind.

What has then emerged in the last 21 years is a transformation from what was originally technically demanding surgery to a now complete multidisciplinary assessment and management team functioning across the state and national boundaries.

Such larger parent units then depend upon regional subunits for the referral of those patients with the more complex craniofacial deformities. The surgical and scientific lessons learned in their assessment and management are then disseminated via scientific literature, the training of plastic surgeons and neurosurgeons, and the regular visits of the members of the parent unit to the referring subunits. The culmination is a medically and economically efficient three-tiered relationship involving the central movement of patients, the sending of multidisciplinary teams to the peripheral regional subunits, and the provision for the dissemination of ideas through surgical training and scientific endeavour.

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THE AUSTRALIAN CRANIO-FACIAL UNIT WOMEN'S AND CHILDREN'S HOSPITAL ADELAIDE

(associated with the Royal Adelaide Hospital)

The Adelaide Children's Hospital was founded in 1876 by Dr Alan Campbell, being funded and built with donations from the South Australian public. It became South Australia's specialist paediatric hospital, accommodating 13 wards and 230 beds.

On March 15th, 1989, the Queen Victoria Hospital, formerly the Queen's Home, and the Adelaide Children's Hospital were amalgamated to become the Women's and Children's Hospital, the vision being to enhance and promote the health of women, children and young people. While the Adelaide Children's Hospital and the Queen Victoria Hospital presently continue to operate at their respective, separate sites, progress is currently being made to locate both hospitals at the North Adelaide site of the Adelaide Children's Hospital.

Present Adelaide Children's Hospital, North Adelaide

Proposed redevelopment to form the Women's and Children's Hospital

The Australian Cranio-Facial Unit commenced as the Maxillo-Facial Clinic, grafted onto the established Cleft Palate Clinic at the Adelaide Children's Hospital. This subsequently became the South Australian Cranio-Facial Unit which was given national unit status by the Federal Government in 1988. It is at the forefront of this form of surgery in the southern hemisphere and sustains regular clinics in Indonesia, Malaysia, Singapore, Hong Kong, Thailand, New Zealand, Oman and Kuwait.

THE AUSTRALIAN CRANIO-FACIAL UNIT 1974-1994

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This paper addresses the beginnings of the Australian Cranio–Facial Unit in the early 1970's and traces its evolution over the next twenty years. The structural developments are seen against the background of the state of knowledge at the time and the real and perceived needs of patients and communities for this form of health care. The development of the unit can be examined not only by reference to its size and workload but also by reference to parallel developments in the diagnosis, investigation and treatment of craniofacial conditions.

An appropriate political and administrative climate is necessary for the establishment of a structure that can deliver this type of health care to a community. Once that has been achieved, progress can be made on the clinical and wider scientific front. In addition to reviewing the factors influencing the Australian Cranio-Facial Unit's establishment and how it was achieved, selected cases are reviewed that are representative of the milestones in the progress to the present position. These milestones are organisational, surgical, technical, in data collection, and philosophical, and deal with access to, and relationships with, other specialists and other communities.

The Australian Cranio-Facial Unit resulted from a combination of the desire to set up such an organisation by a group of people, an ability to set it up and achieve the results, the possibility that it could be done, and a need for it to be done. The desire was that of individuals who chose to work together and had the ability to deliver the care. The possibility was largely presented by a community in South Australia, and more widely in Australia, that facilitated the exercise in a political and administrative sense and ultimately had the need for such services and/or their sequelae or products.

By the beginning of the seventies, the work of Tessier, Converse, and others had been widely presented to the world. The presentation of Paul Tessier's article on The scope and principles — dangers and limitations — and the need for special training — in orbito-cranial surgery (Tessier 1971) at the Melbourne meeting of the 5th International Congress of Plastic and Reconstructive Surgery had a particular significance in setting the scene. Not only was his paper spectacular from the clinical point of view, it set out a clear definition of the entity of orbitocranial surgery, an historical summary and specific guidelines for the setting up of Cranio-Facial Units and the training and maintenance of the team specialized in orbito-cranial surgery.

The situation that existed in Australia at that time was that patients with a need for cranio-facial surgery could be treated by visiting surgeons (Wood-Smith and Dey 1974), by travelling to Europe or the USA, by inferior techniques, or not treated at all. Accordingly, at that time there existed in Australia the need for a Unit to pursue this work in a way set out by Tessier, in the same way that these developments were contemporaneously taking place in France, the United Kingdom, the United States of America, Canada and Central America. Coinciding with this need, there existed in South Australia a number of people who had interest and experience of the work. They covered the disciplines of Neurosurgery, Plastic Surgery, Ophthalmology, Anaesthesia, Ear Nose and Throat Surgery and Dentistry. These people were appropriately enthusiastic. They were granted administrative approval by the hospital administrations who adjusted hospital policies to accommodate a multidisciplinary team and a transhospital service between adult and children's institutions via joint appointments and ease of access

to these institutions. Government approval for the management of interstate and overseas patients with appropriate support followed shortly afterwards.

The mechanics of establishing such a unit involved its being grafted onto the well established Cleft Palate Clinic at the Adelaide Children's Hospital. For a short time, the grafted segment, was called the Maxillo-Facial Clinic. This then became the South Australian Cranio-Facial Unit following appropriate government approvals. In 1988 the South Australian Cranio-Facial Unit was given national unit status by the federal government. The arguments and philosophies then stated were well set out by Munro (1975) and David (1977).

Team development

Taking into account the principles enunciated by Tessier together with the need for such an organization in the region, the Australian Cranio-Facial Unit was established on the dual bases of teamwork and technical expertise. Concepts of the team were developed initially from Tessier's collaboration of plastic surgeon and neurosurgeon. These were expanded in his 1971 presentation and later by many others. This enlarged multidisciplinary approach has developed in our Unit and now extends beyond the medical and paramedical specialists, involving the wider community which, of necessity, must play a supporting role and must be influenced to have a more humane approach to the facially deformed. Multidisciplinary centralised organization needed then, as it does now, to establish relationships with satellite units. Such an organization depends on the appropriate infrastructure being developed in order to connect the various existing health units in different parts of the same city and in different states and countries.

For each of the specialities involved in craniofacial surgery, the symbiotic relationships that have developed have enabled new techniques to emerge and these have been fed back into the professions. The relationship between the Cranio-Facial surgeon and the Neurosurgeon has profoundly affected the way of thinking and operating for both of these groups. The initial strength of this relationship in the Australian group was augmented by the ophthalmological and orthodontic commitment to the project. The latter involvement brought a specialized knowledge of normal and abnormal craniofacial growth. This knowledge influenced surgical decisions as much as the surgical possibilities extended the horizons of the dental specialities. Focusing on individual patients, of necessity, involves each speciality in forming networks across institutional, state, and national boundaries to cater for their needs, eg. an orthodontic treatment programme is designed as part of the general treatment plan at the central unit and implemented by an orthodontist in the patient's home town in collaboration with the unit's specialist.

International relationships

In the first instance, a small community of 1.5 million people was able to provide a sufficient pool of severe craniofacial cases in order to support the Cranio-Facial Unit because of the backlog of serious untreated cases. Arguably, the patients in a country of 16 million people should be able to support one unit treating serious transcranial congenital cases but whether this is true or not remains, to this day, one of the contentious issues facing the delivery of Cranio-Facial services to a community. In 1977, the Australian Cranio-Facial Unit established a relationship with the Ministry of Health in Malaysia through the South Australian Government. The relationship involved the treatment in Australia of fifteen patients per year suffering from severe craniofacial deformity. This also involved lecturing and demonstrating techniques of basic reconstructive surgery with reference to the head and neck region in an integrated progressive programme, treating patients in the region with cleft lip and palate and related anomalies thus helping with the enormous backlog of these cases, bringing surgeons and associated health professionals from the region to Australia for specific training

in relevant aspects of management of craniofacial deformity, and the establishment of regional clinics for these teaching and review activities so that new patients could be assessed, and established patients properly reviewed. Similar relationships have been entered into with other countries subsequent to Malaysia, namely Singapore, China, Hong Kong, Thailand, Indonesia, Brunei, Fiji, New Zealand and some of the Middle-East Gulf States. It is fundamental to these relationships that they are bilateral and are based on the concept of advancing teaching, research and service. As teams have been established in the region, the nature of the relationship with the central unit has changed so that as the proportion of work done in the region increases, only the more complex problems continue to be treated in Adelaide.

Technical advances

The influence on Cranio-Facial Units in general, and on the Australian Unit in particular, of the availability of certain technical equipment for the diagnosis and treatment of gross facial abnormalities has been significant. In the early 1970's, it was desirable to have state of the art anaesthetic equipment and Intensive Care facilities available. As far as the technical surgical aspect was concerned, the most desirable equipment was, and is, an appropriate set of power tools for cutting the bones of the face and skull with precision. Since that time there have been many suggestions and some real advances in the paraphernalia associated with this surgery and the techniques that have been developed. In the Australian Unit, two technical aspects stand out as being more important than any other during this period, namely the developments in organ imaging and changes in the methods of bony fixation.

Developments in radiological examination
Plain skull radiographs have always been indispensable and in many cases will make the diagnosis eg. coronal synostosis. Their application in the form of cephalometry represents the commonest and to date the most effective way to follow the progress of craniofacial deformities whether treated operatively or not (David et al 1982a). When more complex studies were necessary to understand the potentially dangerous anatomy of the anterior cranial fossa in conditions such as Crouzon syndrome, tomograms were obtained in the coronal and parasagittal planes. These studies were useful to show the optic canals and, in craniofacial fractures, to help plan the surgical approach. Fortunately this cumbersome and radiation intense form of investigation had a short life and was replaced by computerized tomography. This field expanded rapidly and the information imparted to the surgeons about the anatomy of craniofacial deformity was immense. Data acquired from this source has continued to change surgical practice eg. visualisation of the medial orbital walls after trauma enables the surgeon to determine the need to operate on this area, thus obviating subsequent enophthalmos, the cause of which for many years remained mysterious.

In the early 1980's, the Australian Unit worked closely with Professor D.C. Hemmy of Milwaukee and Professor Gabor Herman of Philadelphia in order to advance the development of three dimensional imaging obtained from CT scan information (Hemmy et al 1983a, 1983b, Hemmy and David 1985, David et al 1987c, Herman and Abbott 1989). This tool was used almost immediately to enhance our understanding of the rare craniofacial clefts (David et al 1990). The obvious scientific challenge that needed to be met was to bring the same three dimensional mathematical validity to those images as existed with three dimensional analyses obtained from biplanar cephalometry and stereophotography. This challenge was met by Dr. A. Abbott and her colleagues in the Australian Cranio-Facial Scientific Unit, opening the way to a truly scientific appraisal of craniofacial deformity, its biology and therapeutics (Abbott et al 1990a, 1990b). The current situation is well illustrated by a case of Crouzon syndrome analysed prior to surgery with a surgical plan and post surgery (Figure la-c).

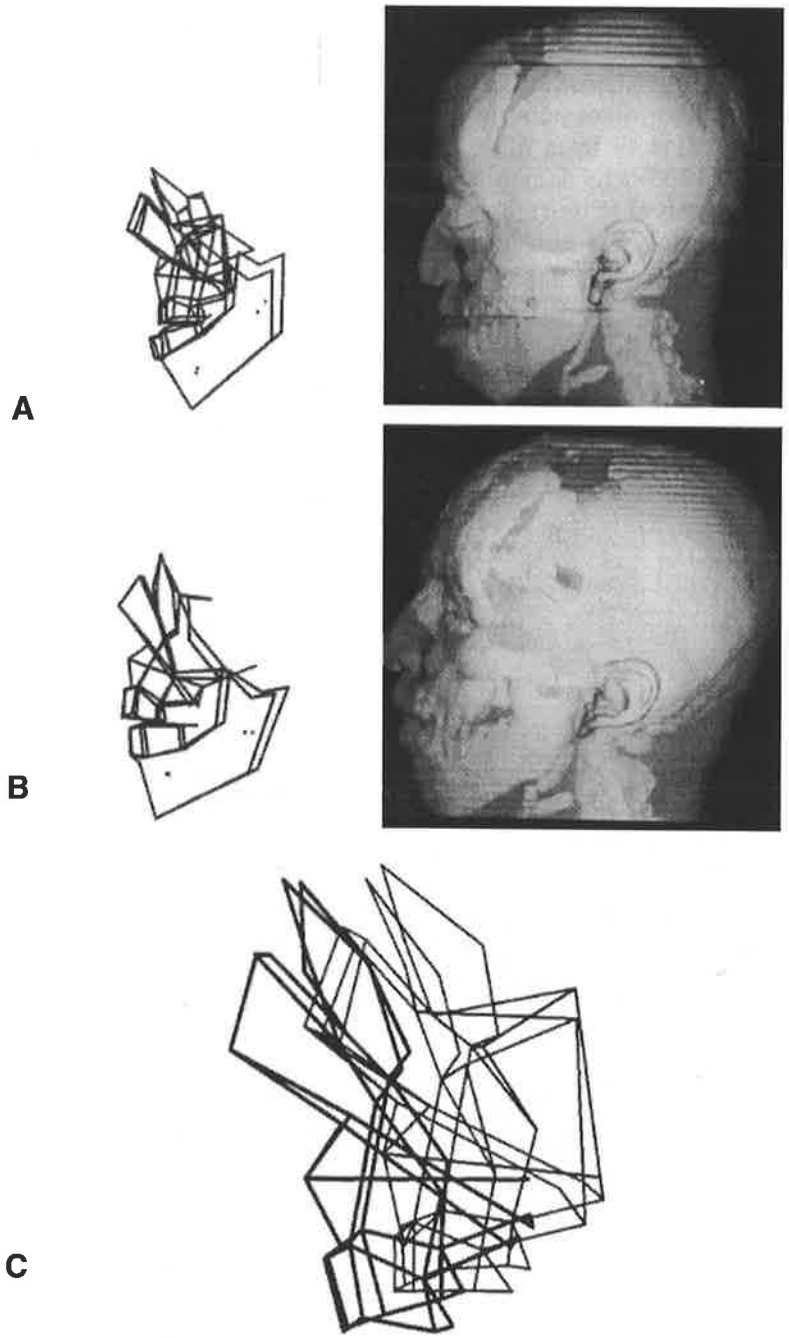

FIG. 1. ***A*** *Pre-operative and* ***B*** *post-operative analysis of a patient with Crouzon syndrome, showing 3D CT reconstruction, and a wireframe model of the maxilla, orbital rims, zygomas and mandible.* ***C*** *Pre-and post-operative wire-frame models, superimposed by alignment on cranial base landmarks, showing the surgical advance of the maxilla, orbital rims and zygomas (thin lines denote pre-op and thick lines denote post-op).*

Methods of bony fixation

The technological advances in the production and use of mini and micro plating systems for interosseous fixation are well known. Such systems are crafted from stainless steel, vitalium or titanium. Efforts to successfully use biodegradable substances have not gained wide usage and self stabilising osteotomies seem to always require the addition of rigid bony fixation (Trots and David 1986). The craniofacial surgical approach provides wide access to the pathology, whether congenital or acquired. Modern imaging allows us to detail the pathology before surgery, and plating systems enable bone and bone grafts to be fixed rigidly under direct vision. The Australian Unit appreciated the need for such systems from the beginning, particularly in the management of trauma where the teamwork and surgical approach endured a much more efficient and cost effective

management program (David 1984). When such an approach is combined with the possibility of rigid internal fixation which interferes minimally with CT and MRI images, not only are the results better but patient comfort and the time in hospital is reduced. The latter point is well illustrated by Gruss et al (1990) with reference to restoration of form after craniofacial fracture treatment, and by Phillips and Rahn (1990) who demonstrated that the rigid fixation of bone grafts maximised their survival (Figure 2a and b) (David and Moore 1989a). To this end, the Australian Unit has developed its own titanium plating system which allows increased ease of contouring while maintaining strength (Ausystems®).

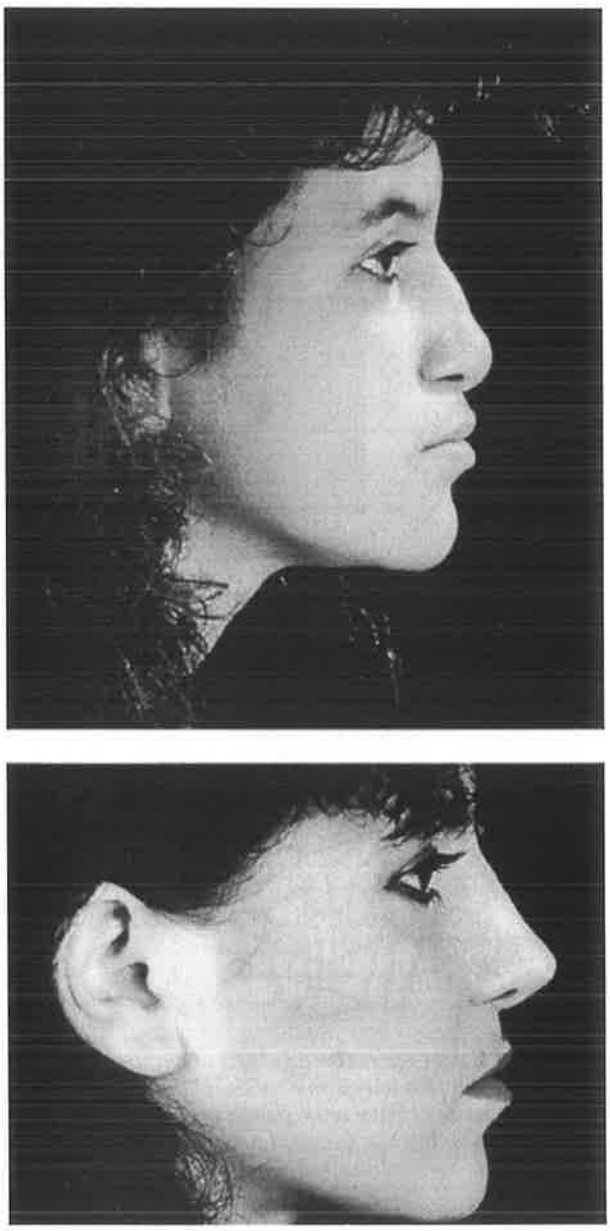

FIG 2 ***A*** *and* ***B*** *Nasomaxillary hypoplasia of Binder before and after surgery. Surgery involved maxillary advancement with rigid fixation, bone grafts in the perialar and nasal region fixed rigidly with plates and screws.*

Modus operandi of the Unit

Processing of patients

The sources of patient referral vary greatly. Patients may be referred from general practitioners or from other specialists in the community, or they may come after referral has been initially made to various individual members of the team in their capacity as experts in their own disciplines. In all cases, initial contact is followed by an interview with the plastic surgeon and a decision is made

concerning the suitability of the patient for assessment by the Cranio-Facial Unit. Once this decision has been made, the patient is then seen by the various members of the team in such sequence as will allow each discipline the maximum amount of information.

Assessment meeting
An assessment meeting is held at regular intervals in the Cranio-Facial Unit. A special room has been set aside in which there is a large table where members of the team can sit. Radiological and video tape facilities are available. The format of the meeting follows a regular pattern. The Unit Registrar reads out a short history and summarises the physical examination of the patient, then each member of the team presents his individual report, after which a certain amount of discussion follows. The patient then comes into the meeting and any relevant matters are discussed with the patient. After the patient has left, final decisions are made concerning a definitive treatment plan.

Planning meeting
In addition to the assessment meeting, there is a planning meeting for the designing of surgical procedures. Once the decision for surgery has been made, the relevant members of the surgical team meet to design the surgical procedures. They have with them information gained from the examination of the patient, transparent photographs, radiographs, cephalograms, and dental casts. The Unit maxillo-facial technician is also present at these meetings in order to help with the technicalities of splint design and manufacture. An exact operation protocol is planned for each patient and this is sent to all relevant people, including the operating theatre staff, before operation.

Unit Statistics (July 1974-April 1994)

Since its inception in 1974, the Australian Cranio-Facial Unit (formerly the South Australian Cranio-Facial Unit) has grown rapidly. The unit has managed 3,892 patients. Table 1 shows a breakdown according to state and/or country of origin. Table 2 shows a breakdown of the overseas patients for the similar period. This is represented graphically.

After referral and preliminary assessment by the multidisciplinary team members, suitable patients attend a Cranio-Facial Clinic meeting, which are held twice per month. The overseas patients are harder to fit into this regular format. Their numbers are quite low and an ad hoc meeting is convened on most occasions for them. Surgical planning meetings occur twice per week on a regular basis but are also held on an ad hoc basis when necessary. Since 1974, 3316 major operations have been performed (Table 3). Of these, 633 were transcranial and 921 were major subcranial operations requiring wide orbital dissection.

TABLE 1

The breakdown for state and country of origin.

Total Australian Cranio-Facial Unit Patients Seen
July 1974 - April 1994

SOUTH AUSTRALIA	2810
INTERSTATE	660
OVERSEAS	422
TOTAL	**3892**

TABLE 2

Overseas patients presenting to the unit. (not a full year)*

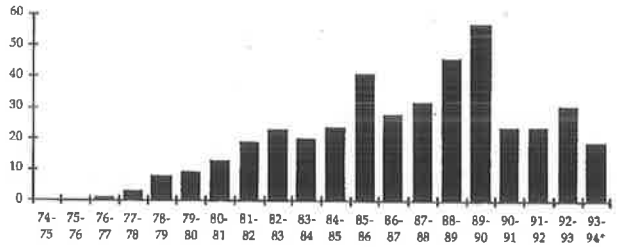

TABLE 3

Major operations performed since 1975.

OPERATIONS

	Transcranial	Subcranial peri-orbital	Subcrainal non-orbital	Total
1975–79	20	20	52	**92**
1980	22	14	37	**73**
1981	26	9	24	**59**
1982	36	1	37	**74**
1983	23	16	43	**82**
1984	43	10	28	**81**
1985	26	28	83	**137**
1986	45	19	139	**203**
1987	55	74	146	**275**
1988	60	60	190	**310**
1989	64	100	180	**344**
1990	67	143	204	**414**
1991	51	147	210	**408**
1992	47	139	192	**378**
1993	48	141	197	**386**
Total	**633**	**921**	**1762**	**3316**

Research

Clinical research has proceeded as a matter of course since the beginning of the unit, reporting treatment protocols (David and Sheen 1990, David 1985), complications (David and Cooter 1987), and operative techniques (David et al 1982b) amongst other publications.

The establishment in 1983 of a formal Cranio-Facial Research Unit with its own budget and director meant that there was a structure into which the unit's clinical aims could be fitted. The current team consists of a director who is an anthropologist, a deputy director and two research scientists. The initial task of this research unit was to develop tools of investigation and measurement for the assessment of growth in craniofacial deformity and use these tools to measure the long term results of treatment. The first part has been achieved with the work of Abbott (1989), and Brown and colleagues working with three dimensional data from biplanar x-rays (Brown and Abbott 1989). This group is affiliated with the University of Adelaide and takes students for post-graduate degrees using the unit's material. Working in the same group, Cooter (1990) produced a thesis on craniofacial fractures as well as a computer based coding of fractures in the

craniofacial region (Cooler and David 1989). His further work on the mechanism of causation of fractures in motorcyclists wearing helmets with chin bars was published in Lancet, 1988 (Cooler et al 1988).

The Royal Australasian College of Surgeons has indicated that a prerequisite for entry into plastic surgical training is post-graduate scientific experience, and to date the unit has been involved with four students preparing for Master of Surgery degrees using the unit's tools, and supervised by the scientific members of the unit as well as the clinicians. Their work has included analyses of Crouzon syndrome, Apert syndrome, craniofacial fractures with respect to bony fixation and the contrasting growth features of fronto-ethmoidal meningoencephaloceles, and rare craniofacial clefts.

A parallel research programme co-exists in the area of speech (David et al 1982, Bagnall and David 1988, David and Bagnall 1990). In order to build on this work, a speech dynamics laboratory has been established with the objectives of recording normal speech in the Australian population, assessing abnormalities in the craniofacial patient population, and then recommending changes in therapy.

Relationships exist with other research bodies in Australia. Use has been made of the marmoset colony at the Commonwealth Scientific and Industrial Research Organization (Goss et al 1983) in collaboration with the Genetics Department in the attempt to establish the locus of the Treacher Collins gene (Dixon et al 1991), and with the Road Traffic Research Unit in the University of Adelaide.

The research unit has made it possible to assess the long term success of surgical intervention and has provided a basis for testing existing practices and introducing new ones. The value of collaborative research in identifying causal factors of damage and disfigurement with a view to their elimination cannot be overemphasized.

Education

In addition to the educational implications of the Unit's overseas activities and the research programme, there is a commitment to medical and dental undergraduate education through the University of Adelaide. There is also formal teaching of speech pathology students through Flinders University, Adelaide. The post-graduate masters and doctoral programmes have been mentioned above.

For many years, a Fellowship programme has existed for trained surgeons who wish to acquire further experience in craniofacial surgery. Ideally this is a one year attachment with an option for further interaction with the Australian Unit. During the year, the Fellow sees a wide range of clinical material, is involved in a clinical research project, is trained in the philosophy and administrative techniques of delivering this type of health care as well as acquiring the basic surgical techniques necessary to begin a career under supervision, such attachments not being sufficient in their own right to "train" the surgeon in craniofacial surgery. Recently, the International Society of Cranio-Facial Surgeons has established a matching programme to assist in coordinating units offering fellowships. Along with this, the Society is drafting its thoughts and conclusions on the training of craniofacial surgeons and the Australian Unit is actively involved in this trading in ideas. The views emanating from the Australian Unit on this subject can be encapsulated as follows: training of surgeons for this discipline should be comprehensive and should be tested.

Peer group education has always had a high priority. Two very successful and innovative international workshops were held in Adelaide, South Australia by the Australian Cranio-Facial Unit. The first, in 1986, on the subject of rare craniofacial clefts attracted wide participation and introduced the idea of audience

participation, using telemedicine techniques, in a series of operations performed by experts. The second workshop following the same format was conducted in 1988 on the subject of craniofacial trauma. This "in-depth" approach to a particular area of practice involving many of the experts in the field proved to be both profitable and enjoyable for all participants and points the way to more efficient methods of conducting scientific meetings.

Clinical development

The approach that has been adopted by the Australian team to structure our clinical development has been one of formulating a thesis about the aetiology and pathogenesis of each disease entity and of developing our management protocols consistent with that thesis, modifying them as our experience and the experience of others brings to light new information.

This approach may be illustrated by considering a few conditions with different aetiologies and demonstrating some of the changing emphases.

The Craniosynostoses

A clear understanding has developed that cases of simple craniosynostosis, if treated early during the rapid brain growth phase, will maintain their correction. David and Simpson (David et al 1982) and others recognised the challenge to make an early diagnosis of plagiocephaly and to select those cases where the cause is craniosynostosis. Figure 3 illustrates quite severe frontal plagiocephaly due to unicoronal synostosis which was treated with fronto-orbital advance. The patient is shown as an infant and at ten years with a near normal face.

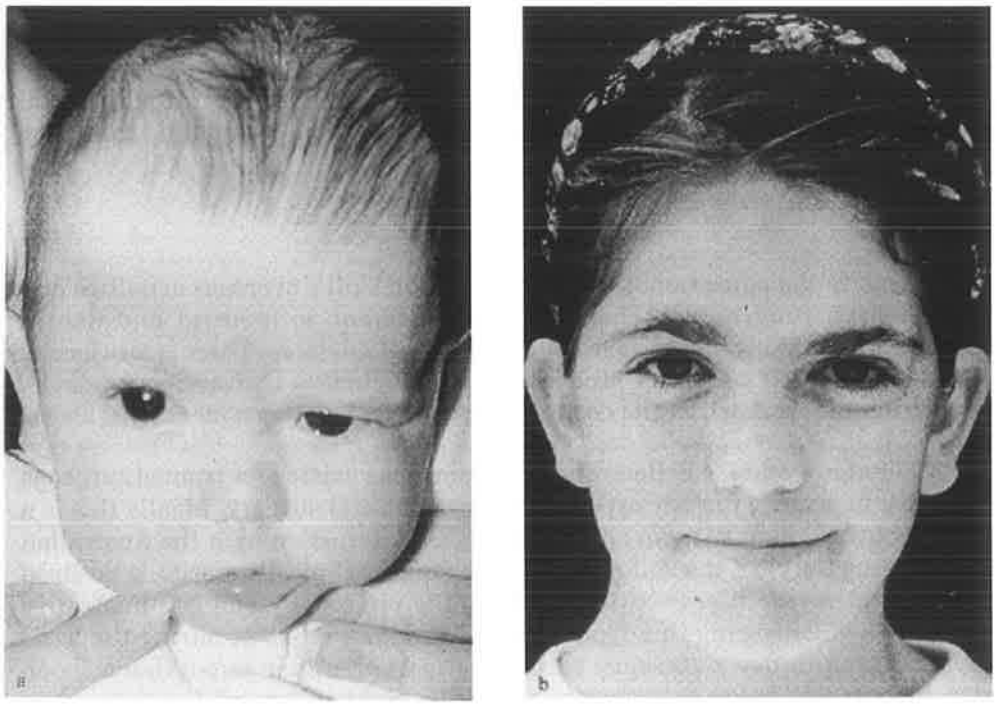

FIG. 3. *(at right).* ***(a)*** *Showing severe frontal plagiocephaly due to unicoronal synostosis, corrected at three months of age and* ***(b)*** *the ten year follow-up showing complete restoration of craniofacial form.*

The craniofacial syndromes of Crouzon, Apert and Pfeiffer are rarely easy to treat. They often require many operations between birth and full development and certainly need meticulous care of the brain, airway and eyes (Trots 1985). The Adelaide Unit has recognised the rare problems of the upper cervical spine in Crouzon syndrome and more importantly the early and often severe airway problems in Crouzon, Apert and Pfeiffer syndromes. As we and others have recognised, infant craniofacial advancement is a dangerous procedure. Techniques of palatal resection together with tonsillectomy and adenoidectomy have been applied to obviate the airway obstruction emanating from severe faciostenosis, leaving infant facial advancement as a last resort (Figure 4). When these

syndromes combine with significant hydrocephalus, the cloverleaf (Kleeblattschadel) skull deformity may follow. The Australian Unit's experience with these cases indicates that the prognosis is guarded (Figure 5a and b).

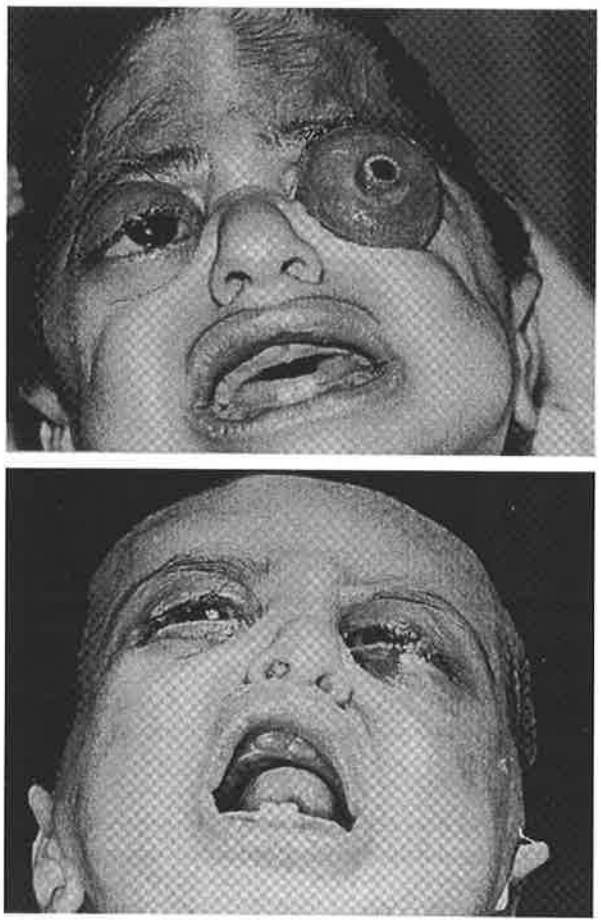

FIG. 4. *Showing fronto-facial advancement in an infant with severe craniostenosis, orbitostenosis and faciostenosis .*

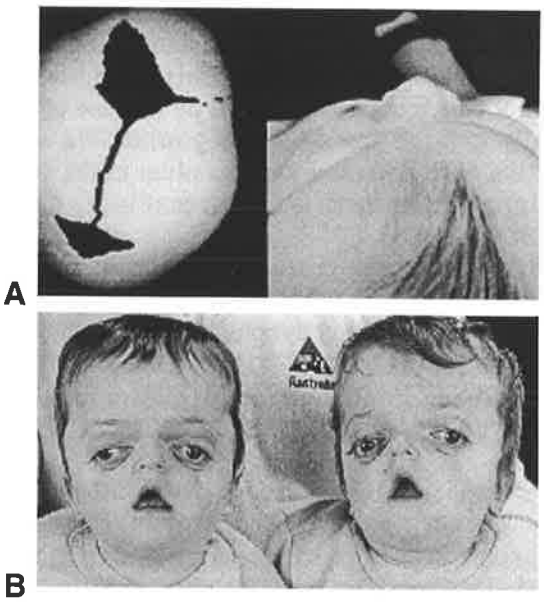

FIG. 5. ***A*** *Twins with cloverleaf skull deformity diagnosed in utero and* ***B*** *aged 5 years of age with craniostenosis, orbitostenosis and faciostenosis under control but far from having a definitive result produced*

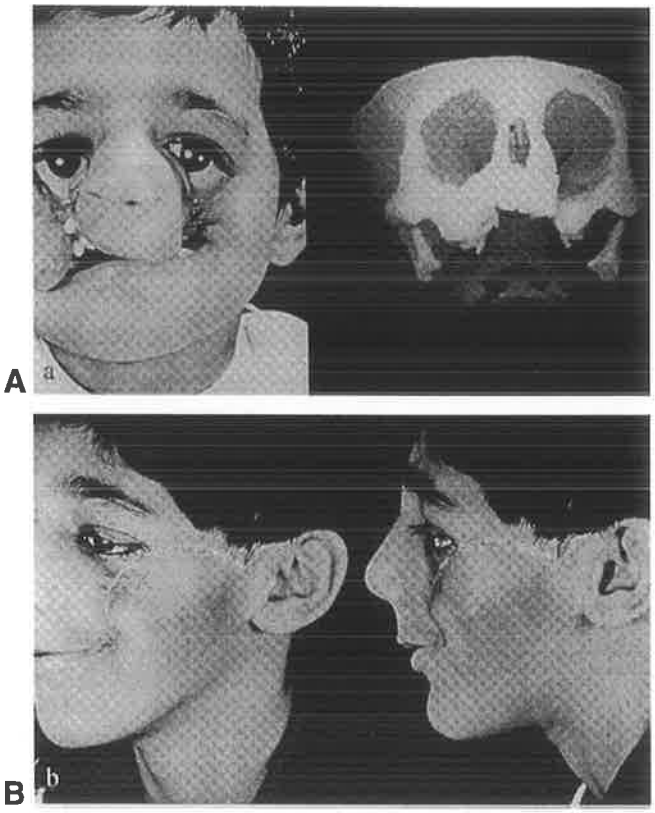

FIG. 6. ***A*** *A Malaysian child with severe Tessier 4 and 5 oro-ocular clefts and* ***B*** *5 years later after bone grafting and tissue expansion of the integument.*

Craniofacial clefts

The basis for understanding and management of these deformities has been that the clefting process manifests itself throughout development so that a definitive surgical result cannot be obtained before growth is complete. The Australian Cranio-Facial Unit's management protocols have been developed taking this into account (David et al 1989). In addition to the concepts of orthodontics involved in the management of these cases, there is the use of tissue expansion to enlarge the soft tissue capsule. The thoughts of the Australian group on this subject were presented at the International Society of Cranio-Facial Surgeons meeting in Santiago de Compostela, Spain, 1991. At the same meeting, the idea of bone lengthening of the jaws was introduced by McCarthy and we have been able to validate the potential of this technique as well as the use of intraorbital tissue expansion to gradually enlarge the developing orbit. We are also using this technique in conjunction with osteotomy in the older child. Management of the rare clefts must continue until growth is complete (Figure 6a and b).

In contrast with these rare craniofacial clefts, the fronto-ethmoidal meningoencephaloceles demand an early release of the growing skeleton from the pressure applied by the protruding dysplastic brain (David et al 1983, David et al 1984, Sheffield 1984, Simpson et al 1984, Hemmy and David 1985, David and Simpson 1987, David and Proudman 1989) (Figure 7a-d).

Trauma

In the early 1970s, transcranial fractures were most often managed by neurosurgeons and plastic surgeons acting separately. The development of team work saw the development of protocols for the integrated management of these cases (David 1984). Modern imaging has shown us exactly what is wrong, and the team can plan and operate as one in a craniofacial fashion using wide exposure, primary bone grafting and modern methods of fixation to achieve the stabilisation of all elements of the cranio-facial skeleton. This approach has been set out by Gruss 1991 (Gruss et al 1990), and has been in use in the Australian Cranio-Facial Unit for the last nine years (Figure 8a and b).

The "spin off" of philosophy, techniques and technology to the wider field of surgery is no better illustrated than in the case of trauma. However, it applies equally to other areas, such as aesthetic surgery and base of skull tumour surgery.

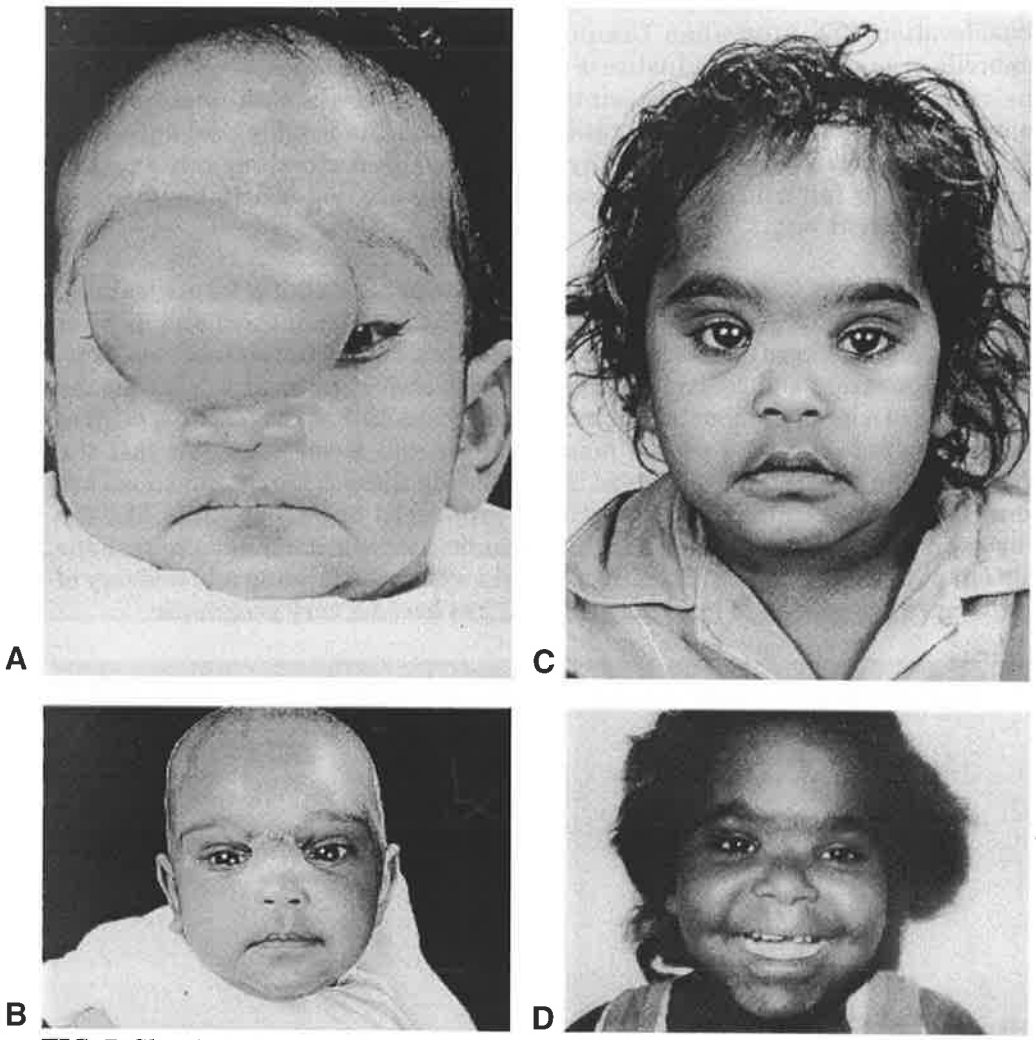

FIG. 7. *Showing a fronto-ethmoidal meningoencephalocele. The same child in infancy* ***A*** *before and* ***B*** *after surgery,* ***C*** *at 5 years and* ***D*** *8 years.*

Conclusion

This paper contains an abbreviated account of the structure, philosophies, and functions of the Australian Cranio-Facial Unit 1974-1994. A considerable number of clinical topics have been left out of the discussion and much could be said, from the Unit's experience, about the newly described disorders such as Binder syndrome or the current variation in protocols for the management of Craniofacial Microsomia (David et al 1987a, David et al 1987b). Cranio-Facial Units also offer an extra dimension to the surgical management of aesthetic problems. This follows an understanding of the changes induced by skeletal shifts, muscle plication, and soft tissue augmentation which are so manifest in the Cranio-Facial patient. No longer are changes only produced by superficial surgery stretching the integument over an abnormal skeleton but, instead, the facade of the face can be draped over a remodelled skeletal frame work (David and Moore 1989b).

The preceding analysis of the Australian Cranio-Facial Unit begs many serious questions. The rationale for the existence of larger central units is that, while depending upon regional sub-units for the referral of those patients with the more complex deformities, they provide quality care, teaching and research (David 1987). The surgical and scientific lessons learned in assessment and management are then disseminated via literature and training to the world

community. This is a sophisticated model involving economic efficiency and a "something for everybody" concept in the development of an aspect of health care. Is such a model viable? Given that change and development must be constant, what direction should change in this area take? After careful consideration, the Australian Cranio-Facial Unit has decided to develop an umbrella organisation — an Institute of Cranio-Facial Studies — embracing all the components that already exist, viz the government service, the private practice component, the clinical unit, the research unit and the teaching organisation. This separately incorporated entity offers, in the Australian context, the opportunity for full utilization of government, private and charitable funds in the most efficient way.

Another significant question "begged" by the paper is that of future training and accreditation of Cranio-Facial Surgeons. Who is to train them and in what accredited units? These questions presuppose suitable units and suitable teachers. What organisation and control, if any, is to be exercised in order to prevent the production of a large number of half trained surgeons so that the teaching centres themselves fade as more people manage fewer and fewer cases. At last the philosophies and strategies involved in addressing these different questions are being discussed. Future directions will be influenced in part by units like the Australian Cranio-Facial Unit but this organisation will itself have to reshape and adapt to the needs and changes of a world where the funding and delivery of health care has ceased to be ideological and has become very pragmatic.

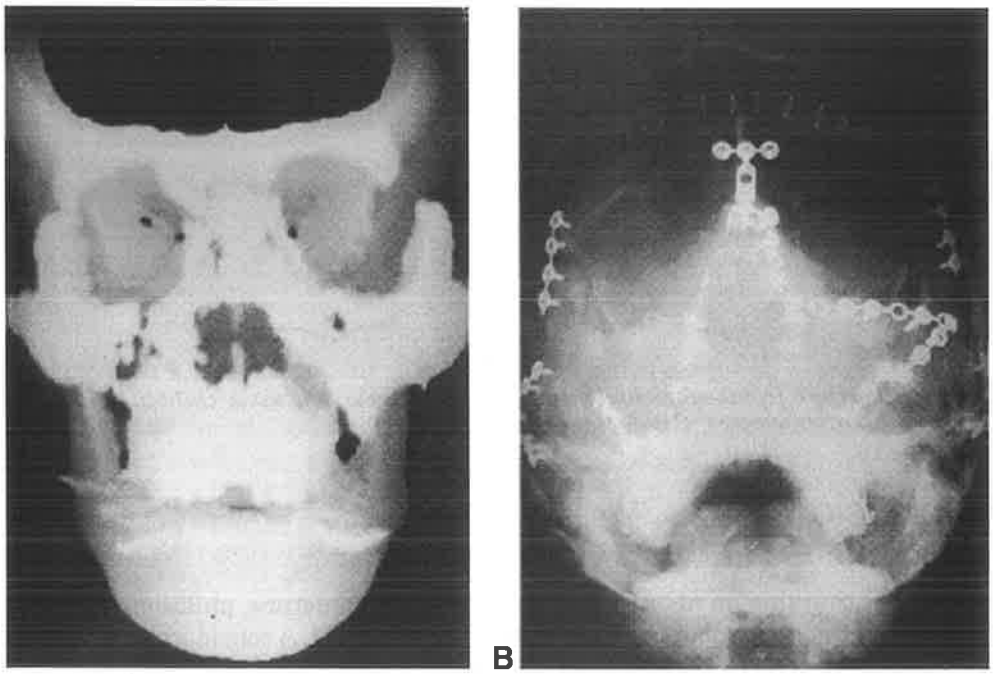

FIG. 8. *Radiograph of severe facial fracture* ***A*** *before surgery and* ***B*** *after reduction and plating.*

Summary

The developmental history of the Australian Cranio-Facial Unit is traced from 1974 to 1994. The structure of this multi-disciplinary organization, which includes scientific and teaching arms, is described. Patients come mainly from Australasia and South-East Asia, and trainees are accepted from Australasia, South-East Asia, Europe and The Americas. The influence of technical changes is illustrated with reference to organ imaging and methods of interosseous fixation. Examples of the developmental changes of treatment protocols are given with particular reference to the craniosynostoses, rare Craniofacial clefts and trauma.

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ENT SURGEONS:	Mr J. Tomich, Mr M. Jay, Mr D. Matison,
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RADIOLOGISTS:	Dr B.E. Clark,
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MICROBIOLOGIST:	Dr D. Hansman,
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Craniomaxillofacial injuries: the wider view

D. J. David T. Brown

Introduction

This book sets out a system of management for injuries in the craniomaxillofacial (CMF) region. These often complex injuries have been discussed from the viewpoint of a specialist multidisciplinary craniofacial unit, but we have tried to keep in mind the very different perspective of the general surgeon who must often deal with cases of severe CMF trauma without the resources of a modern Level 1 trauma centre. If we have failed to do full justice to the problems of such surgeons, it is because in our environment the development of long range communications and air retrieval has been very successful in extending the range of the trauma centre (p. 222). We know from discussions with colleagues working elsewhere, and especially colleagues in various developing countries, that it is possible to achieve excellent results in CMF trauma management by methods that to us seem less than ideal.

The concept of the integrated regional trauma service is now generally accepted (p. 26), at least as an ideal. A craniofacial unit providing multidisciplinary treatment for CMF injuries is readily integrated in such a service. If this is done, three closely related themes demand consideration: the organisational basis of the CMF trauma unit, its roles in research, and the educational challenges inherent in the multidisciplinary management of injuries in the CMF region.

Organization

This book has team-work as its central theme. The organisation of craniofacial teams is based on certain fundamentals. These include:

- the relationship of the team to a wider trauma service designed to provide life-saving emergency care and specialist care for extracranial injuries
- the inflow of sufficient diagnostic challenges to maintain the skills needed in multidisciplinary assessment
- the availability of all of the disciplines necessary to treat the patient in the short and long term.

The key elements are the clinical team and the clinical focus. The obvious focus for clinicians, especially surgeons, is clinical action: what can be done for the injured person. Particularly attractive are the operative aspects of management, and especially those that involve recent technical advances that can produce even better results for the patient and job satisfaction for the treating team. Conservative policies are never so appealing; the clinical team should therefore have members temperamentally inclined to argue for older techniques or for non-operative management plans when this is appropriate. Improved imaging methods such as three-dimensional computed tomography (3D CT) and magnetic resonance imaging (MRI), wide exposure of the craniofacial skeleton, power saws, titanium mini – and microplates, and microvascular tissue transfer have been discussed in more or less detail in this book. But for those people and communities looking to establish a service for the victims of CMF injuries, wider issues must be considered. How is all this expertise to be made part of a health care system?

As this book is being written (1993/4), new and old models of health care delivery are under review and revision in almost every country in the world. Devotees of the absolutes at both ends of the spectrum – the centralised socialist system at one end and the capitalist free market system at the other – are both trying to assume the best elements of the other system and moving towards the centre; most are driven by desire to control cost and increase efficiency.

Developing countries often find it hard to adopt a suitable model, especially when models evolved in the developed countries are changing so rapidly; we have seen health authorities adopting a system that has been recently rejected or greatly modified by the country in which it was first implemented. This can lead to much saddening waste of scanty resources: for example, to the purchase of complex items of equipment for use in an environment where medical and nursing skills and other support systems do not allow for their effective use. Another example is the focus by health planners on the repair of injuries from vehicular accidents in a rapidly motorising society without putting in place proven preventive measures such as seat belt legislation or effective alcohol control (p. 89) or providing adequate primary care and transport from the accident site. These issues are well explored in relation to injuries in outback Ghana by Mock et al (1993) and in New Delhi by Colohan et al (1989).

Costs of CMF trauma

In presenting experiences that illustrate the complexity of the restoration of facial integrity after trauma, this book and other similar studies give testimony to the huge cost in human misery, the cost in loss of function and the subsequent cost to the community from permanent disability. This is most obvious in judicial awards that endeavour to cost the lifetime care needed for a child with frontal lobe damage resulting from a vehicular accident, in which payments may have to be made on the assumption of permanent dependency for the term of the child's natural life expectancy: The ongoing cost of dentofacial restoration after complex jaw fractures is also well known. Given that a structured community service is necessary to the delivery of this type of health care, how can it be provided in a cost-effective manner?

Deployment of services for CMF trauma management

The question of population ratio per special unit has always been a vexed one since Tessier (1971) argued that a craniofacial unit treating complex congenital deformities should serve approximately 20 million people. At that time, he saw trauma management as an important but secondary role for such a unit. Munro (1988) considered the appropriate workload for units treating major craniofacial trauma as well as trauma in other parts of the body, on the basis of his long experience in Canada and the USA. He suggested that a regional trauma centre could handle such cases arising from a population of about 3 million people; the implication of this may be that such a regional team needs a case load of this magnitude to be competent and thus, in managing major trauma well, to reduce the incidence of late traumatic deformities. The concept of centralised multidisciplinary craniofacial units for complex cases serving distinct populations with a guaranteed work load has been endorsed in principle over and over again (David 1977, Munro 1975, McCarthy 1976). But while the principle remains unchanged, and indeed is central to many health care delivery systems, it is often very hard to achieve. Moreover, a different set of numbers is applicable to units which provide primary treatment of CMF trauma as well as elective treatment of congenital and acquired deformities. The number of units and their interactions should be the subject of constant review. That organisation is necessary is a truism, but how it should be achieved in a government system is

more difficult.

In our community, it can be hypothesised that a metropolitan area of 2 million people should be served by two to four major hospitals with all specialist disciplines, and that one of these should house a superspecialized craniofacial unit which does the more complex secondary work and takes the lead in teaching and research. This superspecialized central unit should relate to other major hospitals; in such relationships, unifying structures are necessary and these may be:

- Agreed treatment protocols
- Agreed guidelines for transferral of patients between institutions
- Unified medical and paramedical appointments
- Joint undergraduate and postgraduate teaching programmes
- Specialist medical and surgical college or board facilitation of joint rotating training programmes and recognition of these programmes rather than appointments to individual institutions.

Such an ideal is hard enough to achieve in the Australian context, but becomes even more difficult in some emerging communities. Nevertheless, we believe that this ideal is realisable, at least in part, and we have seen impressive moves in this direction in many parts of the world.

Rehabilitation and community support

Considerations of the clinical management of CMF trauma cannot be separated from rehabilitation and its organizational basis. Rehabilitation implies long-term follow-up of patients and measurement of outcomes. The complexity and difficulties in rehabilitation of patients with frontal lobe damage, diminished or absent vision, loss of jaw and tooth function, compromised nasal airway, and changed appearance are discussed in Chapter 22. That clinical and social systems are necessary to support the long term rehabilitation and care of people thus affected may seem obvious, but the difficulties of meeting these needs are enormous, even in countries with well-established social services.

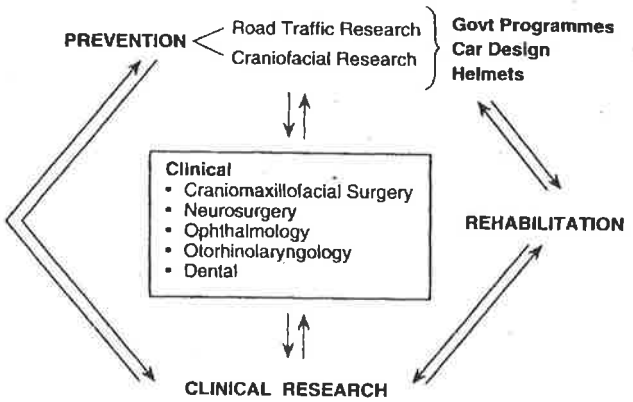

FIG. 23.1 *A craniofacial unit providing multidisciplinary treatment for craniomaxillofacial injuries can deal with treatment, research and teaching and be integrated into a wider regional trauma service.*

The person permanently disabled by brain damage has many needs, and the social help available varies greatly; even in affluent nations, health and welfare services may be unable to do much for the victim of traumatic brain injury or

gross disfigurement. Murphy et al (1990) concluded that, in London, the vast majority of severely head-injured patients were discharged home to the care of relatives who were then left to cope alone. Family care is seen by most authorities as the ideal, but places an often intolerable burden on parents or spouses; family support groups have been formed in many communities, with variable success. Institutional state care is expensive and often of niggardly quality; it is resented and seems inappropriate except in cases of very severe brain damage. Other alternatives depend on the structure and resources of the society; community care by volunteers is being assessed in many countries.

The patient with significant jaw deformity and subsequent reconstruction may be rehabilitated by surgery, dental prosthetic restoration, and physiotherapy, only to be plunged into a new state of crisis and need for support with the onset of old age and loss of bone and teeth. It may then be that the supporting systems once available in hospital are no longer available. In some developing countries, patients may undergo sophisticated reconstructive treatment with no prospect of effective long-term treatment to accommodate for the ageing process or for changing of dental or facial prostheses: In this setting, consideration must be given to implementing other forms of primary treatment that do not require the long-term social support that will not be available. In planning a community trauma service, rehabilitation and social support cannot be overlooked.

Research in CMF Trauma

Research in a trauma unit

The clinical application of knowledge derived from basic and applied research has been an important factor in the development of CMF surgery to its present high status. Craniofacial biology, for example, is a rapidly expanding field of research covering many topics with direct relevance in the diagnosis, immediate care and postoperative evaluation of victims of CMF trauma. These include the longitudinal evaluation of the short – and long – term consequences of trauma and of its management, the investigation of craniofacial morphology and growth, the responses of soft and hard tissues to trauma, tissue responses to implant materials and the quantification of medical images in two and three dimensions. The physical and social sciences also have much to offer: important fields for collaborative research include the design and manufacture of products used in CMF surgery, the application of computer techriology to the production of custom prostheses, the improvement of imaging techniques, the causes and prevention of CMF trauma and the social aspects of CMF trauma from the personal, family and community points of view.

Members of a CMF trauma unit should be aware of current research findings in the fields applicable to their work, and able to evaluate them. Ideally, some members of the unit should be involved in research either independently or as a partner in a research team, even though the time available for such activities may be limited. In some instances it may be possible for a CMF unit to engage personnel whose primary duties are in research, although the costs for staff and facilities are likely to be high.

The type of research conducted in a CMF trauma unit will be determined by the backgrounds and expertise of those involved as well as by thc facilitics available. In some, radiology and the computerisation of medical images may form an important research thrust; this is so in our unit. Other units have been more involved in implant design, bioengineering or other aspects of the biological or behavioural sciences. The evaluation and reporting of surgical procedures and their effectiveness should always form a major area of research.

Collaboration

Trauma units usually have affiliations with institutions outside the parent hospitals; fruitful research collaboration can often be established with universities and other tertiary institutions, other local and national hospitals, government research organisations, industry and privately endowed research units. With the ever-increasing cost of research, the trend in funding is towards the successful and productive research team rather than the individual. Important and well-argued proposals may attract adequate funding for a clinical unit to employ a qualified person to pursue a particular research goal; however, research is highly dependent on the expertise of the members involved, and in a small unit the loss of a key research worker may lead to termination of a promising line of enquiry. Consequently it makes good sense to liaise with the research staff of outside bodies to establish a true multidisciplinary approach to specific research challenges. By sharing staff and facilities, this approach is likely to be cost-effective and beneficial to all concerned.

Graduate and postdoctoral programmes in medical, surgical or dental disciplines that include a research component are usually a source of effective assistance, even though the candidates may have a limited time commitment. These candidates, who work under the guidance of an experienced supervisor should be encouraged to present their research progress at regular seminars attended by all members of the CMF trauma unit.

Research staff

Research workers, whether clinical or laboratory, need to establish, refresh and maintain contacts with colleagues working in similar or related fields of interest. Regular presentation of personal research findings at local, national and international meetings, where peer comment is available, is an essential component of good research. From time to time the opportunity to visit or work in other research units, preferably in foreign countries, will arise; these opportunities are very rewarding particularly for the younger researcher. The spread of international computer communications and the widespread use of electronic mail services have simplified the task of maintaining rapid and regular contact with colleagues located in other institutions. Personal computers operated with a modem from home are now in everyday use by researchers to simplify the exchange of information and data.

Research in prevention of CMF injury

It is a truism that prevention of injury is more rewarding than trauma treatment. Road traffic research has generated legislation on traffic speed, alcohol consumption, restraining devices for front seat and rear seat occupants in vehicles, restraining devices for children, helmets for motor and pedal cyclists and the development of various forms of motor car design aimed at injury prevention or mitigation. The enactment of such legislation has been constantly driven by the results of research coupled with public awareness campaigns, which are necessary to publicise the enactments and obtain the cooperation of the community.

In Australia and many other countries, such campaigns have been generally mounted by government-sponsored health promotion agencies, most obviously in the area of road traffic accidents, for example 'belt up' campaigns. In the USA, grass roots community feeling, exemplified in bodies such as MADD (Mothers Against Drunk Driving) has been a dynamic force, and appears to be changing social patterns in alcohol consumption (Evans 1991). Public education has also been undertaken in other areas where people may be damaged, for example 'safety in the home' campaigns, child protection campaigns, and industrial safety education. Some aspects of these endeavours to prevent injury have been reviewed in Chapter 3; here, it is sufficient to direct attention to the steady fall in the numbers and rates of road deaths in the USA, Australia and other developed countries in recent years (Fig. 3.2).

The technical research behind these advances in injury prevention is complex and beyond the expertise of most members of the multidisciplinary team. Nevertheless, there should be a close relation between clinicians who see trauma and the research workers and designers who may prevent it. Historically, surgeons have been among the leaders in accident prevention; in Chapter 1, the achievements of Hugh Cairns and Elisha Gurdjian (p. 25) were briefly discussed, and they have had many less well known successors. There has been a constant trade-off between the marketability of motor vehicles in respect to speed and design against the ability to make them safe for drivers and pedestrians; this is part of the tension between the safe and the profitable. Helmet design is another area of controversy that involves clinicians, designers and users in a complex debate that is yet to be resolved (p. 111). It is — or should be — the role of clinicians to give warning of changes in accident patterns; the rise in CMF injuries from assault exemplifies this (Levy et al 1993), and it seems likely that the sociology of crime will be an area for multidisciplinary research in the near future (p. 93).

Research and audit in clinical practice

Whatever the framework of practice, the CMF team should feed back to research workers in the following areas:

1. The measurement of outcomes, both physical and psychological
2. The development of therapeutic techniques.

In measuring and evaluating the outcomes of treatment, properly conducted audits are essential. Where possible, controlled trials should be used, though it is by no means easy to do this in testing new surgical and medical techniques for the treatment of CMF injury (Miller & Teasdale 1985). For reliable auditing, it is desirable to have one or more team members with training in research and ready to call on a statistician for advice on the design of audits and trials. Auditing the results of treatment and awareness of the outcomes have received new significance in the need for clinicians to respond to governmental demands for specific practice parameters. Throughout the world, health administrators are using case-mix systems as analytical tools in controlling costs and monitoring quality of care. The Diagnosis Related Groups (DRG) system, developed in the Yale School of Management, has been widely used for these purposes in the USA and elsewhere. Difficulties have arisen in applying the system in some surgical specialties (Sanderson et al 1989). The relevance of a case-mix costing system should be studied in the community in which it is to be applied (McKee & Petticrew 1993), and clinical audits should be used for this purpose. Habal (1993) points out the need for craniofacial surgeons to be involved in establishing proper coding systems in measuring outcomes in the multidisciplinary setting.

It is the area of applied research that has had greatest impact on the management of CMF trauma. In our own practice, this has been evident in two fields: most dramatically in the development of 3D CT, and more slowly in the evolution of titanium in facial reconstruction.

Herman & Liu (1979) first described the display of 3D information derived from CT scanning, and the method was introduced to us in the context of craniofacial deformity by David Hemmy in 1980 (Hemmy et al 1983, David et al 1991); it was soon used also in trauma. From this have come extensions in the numerical evaluation of deformity and in the production of prostheses by computer-aided design/computer-aided manufacture (CAD/ CAM) reconstruction, and in other technical advances described in this book.

This interplay between technological advance and clinical exploitation is also evident in our experience of titanium in CMF surgery. Titanium plates were used in cranioplasty in 1961 at the recommendation of a local industrialist, but

the methods of shaping and contouring remained primitive until 1977 when the Belfast method of moulding in a high pressure chamber was adopted (Gordon & Blair 1974). In the 1980s titanium miniplates were made for fracture fixation (p. 270) and with increasing experience, more complex implants are being fabricated by casting (p. 554). These developments are enticing for the surgeon, who is offered the use of more and more beautifully crafted titanium plates and screws; there are related implantable devices for securing dentures and other prostheses both intra – and extraoral, metallurgical triumphs which are now part of the craniofacial surgeon's armamentarium. Particularly impressive are the dimensionally accurate castings made from 3D hard models generated by computer from CT scans.

Basic research offers other possibilities; even transplant surgery may be just around the corner. Growth factors (p. 121) have great promise in the promotion of wound healing; neuroprotective drugs may have roles in promoting recovery after brain injury. But technical advances and therapeutic interventions frequently proceed well in advance of the creation of organisational systems in which they are used and before their ethical implications and cost-benefit ratios have been worked out.

Research and delivery of its benefits

Notwithstanding the exciting achievements that have been made and that will be made in the not too distant future, the exploitation of the technical possibilities remains limited by inability to deliver therapeutic applications to the people in need, and this often expresses deficiencies in organisational structures. With the best will in the world, institutions and governments still struggle with the concept of the integrated multidisciplinary delivery of this type of health care. Moreover, individualistic surgeons are often most reluctant to embrace it, let alone have it imposed on them.

Funding research and development

Research is intended to make new contributions to the body of scientific knowledge; development is the application of established knowledge to improve clinical treatment and to assess its results. Innovative and productive research in CMF trauma requires adequate funding to cover salaries, equipment, materials, publications and other costs involved. Unless privately endowed, a research unit must rely on its parent institution, usually a hospital or university, as well as on external funding bodies to support its activities.

In most countries, government departments concerned with health, education or science offer publicly funded research grants for open competition. Many forms are available: these include project grants for a specific research proposal, large-scale grants to establish and fund research centres, setting-up grants for young researchers, contract grants for a government-determined research priority and training fellowships for graduate and postdoctoral programmes. Competition for such grants is usually intense.

Privately endowed funding bodies will often be interested in proposals for research projects in CMF trauma. It is essential for the leaders of a craniofacial unit or a trauma service to know what sources are available, and how approaches should be made; even if research is not seen as a major commitment, the need to develop new technology will often call for more funding than can be met from normal departmental budgets. It is usually profitable to maintain a list of the many sources of funds available together with their terms and conditions. The art of 'grantsmanship' is a skill that the successful researcher must master, and several texts have been published to assist in reaching this goal (e.g. Margolin 1983).

Today, in the more affluent countries, trauma research receives financial support from many sources. These include:

- National institutions committed specifically to support research, such as the National Institutes of Health and Centres for Disease Control in the USA and the Medical Research Council in the UK
- Military establishments
- Health agencies: hospitals and similar institutions committed to patient care, and government departments concerned with trauma in various ways
- Universities and other tertiary educational or professional bodies with research commitments
- Commercial firms — especially the pharmaceutical industry and surgical instrument manufacturers
- Private foundations, trusts and similar endowed bodies.

National research institutions dispose of large sums; applications are usually assessed by very well qualified committees, and success in an application confers prestige that will often generate further funds from less exalted sources. It has been said that research in trauma management tends to have low priority in national health planning. In the USA, it was recently stated that the annual Federal research expenditures relating to trauma care are approximately 5% of those for cancer, heart disease or AIDS (Third National Injury Conference 1992). In the UK, the Medical Research Council (1991/ 92) allotted to trauma research in the broadest sense the large sum of £2 017 000, but this represented only about 1.0% of the annual expenditure of the Council (V. Chandy, personal communication). In Australia, the analogous body is the National Health and Medical Research Council (NH&MRC); in 1992 it funded trauma projects and a trauma research unit with substantial grants. (The epidemiological and neuropathological research reported in Chapters 3-5 was in part funded by the NH&MRC and other governmental agencies.) But as in the UK, these grants represented only about 1% of the year's budget.

Nevertheless, there is reason to believe that this attitude towards trauma research is changing. In the USA, the Centres for Disease Control are playing an important part in funding research in trauma and related fields, and other US government agencies are also active. It has to be said, however, that grants from such bodies are usually made to research workers with good track records, and laboratory studies tend to be favoured, while projects aimed at developing a new technology ordinarily receive funding only if embodied in a valid research project. It is never easy to do this, and still less easy to convince the vigilant assessors that the research is valid.

We have no experience of research funded by the armed forces, but much excellent trauma research has been funded in this way, especially with respect to missile injury. The fields of military interest are necessarily specialized, and at times may raise ethical issues. Nor have we much experience of research or development funded by commercial institutions, though again much excellent research has been done in clinical trials of new pharmaceuticals and similar collaborative projects. In the development of 3D scanning, our work has benefited very substantially from support by Silicon Graphics, General Electric and Toshiba.

Much clinical and laboratory research is done in university departments, and this is surely appropriate. This is one among many reasons which make it very desirable that the Level I CMF Trauma Centre should be located in a teaching hospital and closely affiliated with university medical, dental, engineering and other faculties. It is unfortunate that, in times of economic recession, university research budgets are often cut.

Finally, something must be said of the roles of private research foundations. These may have been endowed to promote research in a particular age group, or for victims of specific categories of diseases, or to support a particular hospital or clinic. There are sometimes very specific requirements regarding the age of the applicant, the proposed research and its objectives, and the time frame envisaged. Trauma research and development will often benefit when such a foundation

develops a relationship with a clinical group: funds may be made available at relatively short notice, or given for a development project that is not a research project in the strictest sense. Much of our work has been fostered by two such bodies, both founded initially by surgeons concerned by the difficulties encountered in beginning research activities in a new discipline without academic affiliations. For a successful partnership between foundation and research group, the leadership of the research team must have skills in public relations, or must be able to call on such skills; it is also essential for the financial basis of the collaboration to be impeccable and for the activities of the research group to be monitored by an independent research committee responsible to the foundation, and through it to the donors. If these requisites can be provided, and if the research work attracts community support, the outcome can be rewarding both to donors and to research workers.

Training and Qualification

Career structures

It goes without saying that the specialists involved in the management of CMF trauma should have some special training in the area. Each medical and dental discipline provides its own specialty training in which training in trauma is included and upon which further, more specialized training can be superimposed. But specialised training must have a realizable goal. Individual clinicians must always have a special interest in their area of work, but this interest can only be channelled into productive growth if there is an appropriate career path and an employment structure that offers at least adequate remuneration and sufficient job satisfaction. The craniofacial unit and the associated units in neurosurgery and other disciplines should develop such employment structures. Trauma is not an attractive surgical field, and those committed to it should be offered the prospect of a satisfying career.

The craniofacial surgeon

The craniofacial surgeon has a central role in the CMF team. The term 'craniofacial' now forms part of the description of a surgical service in many countries, and substance should be given in the description of this service and the related training programmes. Training for this role has been a controversial issue since the introduction of the craniofacial concept by Tessier in the late 1960s (Tessier 1971). In France, Tessier and his disciples grew out of the tradition of general surgery and its speciality of plastic and reconstructive surgery (p. 26). There were at that time other models for the new subspecialty that emerged from Tessier's concept. In Germany there were already maxillofacial surgeons, trained both in surgery and dentistry, and this development owed much to the leadership of Martin Wassmund (1892-1956). In some other European countries, there were stomatologists, medical specialists with dental training. In the UK, there were plastic surgeons with no formal training in dentistry who performed maxillofacial procedures, and oral surgeons some with only a dental qualification and some with both dental and medical degrees; from this situation, British maxillofacial surgery evolved into a complex surgical discipline with a strong dental background (Poswillo 1977).

In the USA, there were plastic surgeons who performed maxillofacial surgery and have latterly engaged in craniofacial surgery, and there were maxillofacial surgeons with both dental and medical degrees. Today, US craniofacial surgery exemplifies these traditions: some craniofacial work is done from a dental background, other maxillofacial surgeons proceed from a medical and dental background, and others from a purely surgical background. In all three, the scope of practice is subject to the checks and balances that prevail in countries with a lively medicolegal arena. Wolfe (1993) has given a historical view of US experience. Maxillofacial surgery began informally in that country in

1923, when an American Association of Oral and Plastic Surgeons was formed. Both dentistry and medicine were recognized in training and in qualification. Wolfe suggested that training in craniofacial surgery should include special dental components of instruction, formally organized by dental schools. This view has long been held by many in Australasia, and would provide an excellent link between dentistry and medicine, parallel with the current provision of training in medicine for dentally based oral surgeons. In both approaches, the aim would be to bring dental and medical specialties closer together.

In the past 20 years, much time and effort has been expended in designing training courses for craniofacial surgeons and maxillofacial surgeons. These efforts continue as we write this book. The tendency to train one surgeon to do all things has been replaced by training a particular type of surgeon who works within a team of other people who do other things.

This conceptual change is very relevant in the organization of CMF trauma services. Craniofacial surgery as a plastic surgical discipline arose from the elective surgery of malformations and secondarily entered further into the field of trauma. A similar sequence took place in neurotrauma management: neurosurgery began with tumours and only secondarily and reluctantly entered trauma. There has been a different course in oral surgery which entered trauma very early and has moved on to the correction of dentofacial and craniofacial aesthetic deformities.

Each discipline has its own traditions, and these have to be taken into account in the formulation of programmes for craniofacial surgical training by colleges, certifying boards, and specialist societies. These bodies have to match their qualifications with the problems needing to be solved in an area that has expanded very rapidly.

There is an outstanding dilemma for the non-medically qualified oral surgeon who may be called upon to manage a seriously ill patient and feels that he is not armed with the necessary medical background to fill this role. In several leading countries in continental Europe, this dilemma is resolved: both dental and medical qualifications can lead through a general surgical training to a specialised maxillofacial qualification which may now be extended to encompass craniofacial surgery, although to our knowledge that specific add-on is not yet formally established. The European Union now recognises maxillofacial surgery as a distinct surgical specialty (Wolfe 1993). In the British system (now closely resembling that of other European countries), two of the Royal Colleges have established fellowships in maxillofacial surgery for surgeons who have a dental degree; in these, the emphasis is on surgical training and in future both oral and maxillofacial surgeons will have degrees in medicine and dentistry.

An alternative training in parts of Europe, the UK and the USA is exemplified by the medically trained plastic surgeon who trains according to the guidelines of the International Society of Craniofacial Surgeons as promulgated in 1981. This career was adopted by most of the founding and subsequent members of this organization — now the International Society of Craniofacial Surgery.

At the time of writing, a number of propositions concerning the role and training of craniofacial surgeons are before the American Society of Maxillofacial Surgeons, the American Society of Craniofacial Surgeons and the International Society of Craniofacial Surgery.

It is now recommended that in future the craniofacial surgeon should be broadly competent and should experience an extra 2 years' training in craniofacial surgery on top of Board certified plastic surgical or maxillofacial training. These proposals raise contentious issues. One is the argument that entry into craniofacial surgery should be only from plastic surgery. Another is the argument that craniofacial surgery should be separated from maxillofacial surgery in higher

training, on the basis that it entails transcranial procedures. Yet more contention exists on the definition of an adequate and relevant workload for a training programme.

From these fruitful debates have emerged some agreed views that we see as crucial to the role of the craniofacial surgeon in a CMF trauma service:

- additional training is necessary for a craniofacial surgeon
- the emphasis in training should be on the team approach
- the craniofacial surgeon should be trained as a potential team leader
- an adequate dental background is essential
- recognition that craniofacial surgery is intrinsically transcranial but with considerable overlap between craniofacial and maxillofacial surgery
- recognition (Tessier 1971) that trauma is an integral part of the training regimen for a craniofacial surgeon.

The problem of demarcation has bedevilled the delivery of health care since antiquity. This problem appears to have its origin in the concept of exclusivity. Throughout the world, there are today massive problems in the management of CMF trauma. If we accept the principles of team management set out in this book, then the question of who should be the deliverers and what they should know can be addressed, and what the deliverers should do and know will determine who they are. We need to develop some very broad policies that avoid named disciplines and established institutions. A tentative effort to do this is now outlined.

The craniofacial surgeon should be able to manage the facially traumatized patient. He should be able to perform the necessary major surgical procedures on the craniofacial skeleton, but within a team of skilled peers. He should be sufficiently knowledgeable and skilled to have the respect of other team members. He should have a knowledge of the scientific method, and also be aware of the need for quality assurance and quality control (Habal 1993). He should have sufficient administrative skills to be able to organize his team. Common in all this are medical skills, but the superadded expertise necessary to achieve the stated ends is not necessarily embedded in any traditional discipline, whether medical or dental. Therefore, the future craniofacial surgeon could enter the training program from many origins — from maxillofacial surgery, from plastic and reconstructive surgery, from ear, nose and throat surgery, from ophthalmology or neurosurgery — always providing that the medical background is adequate.

Poswillo's seminal paper on the relationship between oral and plastic surgery (Poswillo 1977) gave an excellent historical and philosophical analysis of this complex relationship. He spoke of the dynamism that would produce rapid change and development and in this he was prophetic: that the specialty of plastic surgical and the dental specialties have changed their grounds is now a matter of history. Poswillo quoted Claude Bernard: 'a science that is halted in a system remains stationary, and is isolated, because systematisation is really scientific encystment, and every encysted part of an organism ceases to take part in the organism's general life.' Poswillo warned against those who might attempt to prevent development by oversystematization and restriction, rather than to develop tolerance, intellectual freedom and the skills necessary to work in a team situation.

Trainee orthodontists and maxillofacial surgeons should also have the opportunity to work for a period as members of a craniofacial team and follow all procedures closely from initial examination through the unit's planning meetings to the operative procedure and the postoperative follow-up.

Fellowships in craniofacial surgery

One popular way to gain proficiency in craniofacial surgery is a fellowship, an attachment of an apprenticeship type to a unit which performs an adequate amount of craniofacial surgery. Recent efforts have been made by the International Society of Craniofacial Surgery to recognize such fellowships and it is acknowledged that they should fulfil certain standards. Fellowships range from 6 months to 2 years, the most popular being 1 year fellowships. This method of training may well be a step on the road to the formalisation of training in craniofacial surgery (see above) but at the moment there is no way to measure the quality of outcome of the training offered in such programmes. It seems sensible at this stage:

- to define an entry point, namely that the candidate entering such a fellowship should have an adequate surgical background, not necessarily restricted to one discipline
- to accept that the fellowship should be for a sufficient period (we see one year as the minimum)
- to ensure that the content of the training should be identifiable and adequate
- to measure the outcome in terms of some form of assessment and possibly ultimately by examination.

CMF surgery in undergraduate and postgraduate courses

We believe that some contact with modern craniofacial surgery and trauma management should be included in the undergraduate curricula of most health sciences, and especially those of medicine and dentistry. Dental students benefit from the experience of watching members of a craniofacial team working together, particularly if the dentition or jaws are involved and there are disturbances of dental occlusion and normal oral function. Medical students benefit from seeing the work of a very broadly based multidisciplinary team, and especially from hearing the interplay of patient and clinical counsellor.

Trainee surgeons should be aware of the breadth of modern dental materials and restorative procedures and the scope of maxillofacial surgery, orthodontics and other specialties of dentistry by secondment for a period to units specialising these fields.

A craniofacial unit should be well placed to teach at all levels of the health care curriculum — at the graduate and postgraduate levels and at the various levels of scientific education through programmes for masters and doctoral study. A craniofacial unit with international linkages may be able to design education packages to suit the particular needs of developing communities with respect to varying aspects of the managing of facial trauma. Those who argue that plastic surgery should be the only entry point for craniofacial surgery should consider the diversity of medical structures in the world today. In several developing countries known to us, craniofacial and maxillofacial trauma are major problems. In countries struggling (often with admirable success) to provide health services of all kinds, it could be very damaging if a prestigious national or international organisation should demand that only plastic surgeons should enter into this field of expertise; indeed, craniofacial surgeons working in developed countries should be given every encouragement to provide training in their discipline that is appropriate to the needs and wishes of colleagues working in countries where medico-economic conditions are different. The delivery of health care in relation to CMF trauma can be very difficult in developing countries, where such injuries are only one in a wide range of demanding priorities. Specialist resources may be sparse, and training programmes should be structured to produce surgeons with the skills requisite in the real environment. Under such circumstances, we believe that entry into craniofacial surgery can well come from various specialties, given that training is adequate and that the multidisciplinary concept is kept in mind. Collaboration with colleagues in several neighbouring countries has taught us that this is not impossible.

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Chapter 2
Craniofacial Trauma

Physicians of the utmost fame
Were called at once, but when they came
They answered, as they took their fees,
'There is no cure for this disease'.

Hilaire Belloc

■

2. Craniofacial Trauma

The first paper in this chapter, Exploration of the Orbital Floor Through a Conjunctival Approach, 1974[1] concerns the application of a technique for the surgical approach to the orbital floor, used by Tessier in congenital cases, to the management of everyday trauma. It was at the time an unusual thing to do. It is now commonplace and has been "extended" both physically and in its usage. This otherwise insignificant paper represents an example of what often happens in surgery where the use of a technique has focused attention so that the right questions can be asked about the real problems of trauma, one of which was cosmetically acceptable exploration of the orbit.

Trauma was and remains the everyday task for the Craniofacial Unit. The second paper in this Chapter, published in the Annals of the Royal College of Surgeons of England, New Perspectives in the Management of Severe Craniofacial Deformity, 1984[2] is a "cut down for publication" version of the author's Hunterian oration given the year before. Part of the paper details the new perspective that the craniofacial approach has given to the management of trauma of the face and skull, namely teamwork, up to date imaging, wide surgical exposure and primary bone grafting.

Computer-Based Coding of Fractures in the Craniofacial Region, 1989[3] presents the need to provide an objective coding system for craniofacial fractures as a baseline for future clinical research and outcome measurement.

The next group of papers in this Chapter, Oblique Craniofacial Fractures in Children, 1990[4]; Fractures of the Anterior Cranial Fossa — the Craniofacial Approach, 1990[5]; Fractures of the Forehead and Anterior Cranial Base, 1990[6]; Associated Injuries in Facial Fractures: Review of 839 Patients, 1993[7] and Sports Related

■

Facial Fractures — a Review of 137 Patients, 1993[8] represent examples of clinical reporting, auditing and research.

The next two papers The Relationship Between Fracture Severity and Complication Rate in Miniplate Osteosynthesis of Mandibular Fractures, 1994[9] and Comparative Study of Miniplates used in the Treatment of Mandibular Fractures, 1996[10] propound the author's view that the unit should have a role in the development and testing of new technology. The value of the computer based coding system when undertaking such studies is underlined.

Papers 11 – 18 are Chapters from Craniomaxillofacial Trauma, David DJ & Simpson DA, Churchill Livingstone, 1995. This work was produced in its entirety by members of the ACFU and advances their up to date views on the subject.

The Chapters included are those to which the author had a significant input. They concern historical perspectives; functional anatomy; pathology of injury and repair; emergency management; definitive management, principles, priorities and basic techniques; facial fractures; massive tissue loss; and deformities.

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Papers

1. David DJ 1974 Exploration of the Orbital Floor Through a Conjunctival Approach. Aust. N.Z. J. Surg 44:25–27

2. David DJ 1984 New Perspectives in the Management of Severe Craniofacial Deformity. Anls R.C. Surg. Eng 66:270–279

3. Cooter RD, David DJ 1989 Computer-Based Coding of Fractures in the Craniofacial Region. Br J Plast Surg. 42(1):17–26

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16. Trott JA, Moore MH, David DJ — contributing author: Jay M 1995 Facial Fractures — Chapter 11. Craniomaxillofacial Trauma 263–342

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■

Exploration of the Orbital Floor Through a Conjunctival Approach

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An approach made through the conjunctiva provides an excellent exposure of the orbital rim and floor. Fourteen occasions on which exploration was performed through this incision are reported. The method is described, and is diagrammatically illustrated in Figure 1.

Acute awareness of the sequelae of orbital floor damage has resulted in a greater number of explorations and reconstructions of this region. The orbital floor is involved in almost every fracture of the zygomatic complex, but in the majority of cases the periorbital periosteum remains intact, and the orbital contents do not herniate into the antrum. The pure "blow-out" fracture occurs when the orbital contents are driven through the thin floor without disruption of the orbital rim. When entrapment of orbital contents is suspected in either pattern of injury, it is necessary to explore the orbital floor.

The usual approach is through an infraorbital incision. The exact site can be varied and may be under the eyelashes, in a convenient natural skin line of the lower lid, or at the junction of cheek and eyelid skin. The latter tends to produce lower lid oedema (Hopkins, 1971). Usually these scars are satisfactory, but occasionally they are quite obvious (Figure 2, left).

Clinical Material

In this study the cases of 14 patients are presented in whom the exploration was performed through a conjunctival incision (Tessier, 1973). They presented with facial fractures at the Royal Adelaide Hospital between March and July, 1973. Six patients were explored for suspected entrapment associated with an isolated fractured malar bone. Silastic sheet was inserted in one of them, and lower border interosseous wiring was performed in two. Four patients were explored for pure "blowout" fractures, and Silastic sheet was inserted in each case. In the remaining four patients, the disrupted zygomatic complex was associated with a Le Fort II fracture of the middle third of the face. Three of them were reconstructed with the use of lower-border interosseous wiring to reconstitute the rim and Silastic sheet to cover the disrupted orbital floor and support the orbital contents. In one case Silastic sheet only was required.

Exploration of Orbital Floor

Technique

The lower eyelid is drawn out with two skin hooks and the conjunctiva incised two to three millimetres below the tarsal plate (see Figure 2). A stay suture is placed in the distal edge which, when retracted, will expose the plane of dissection

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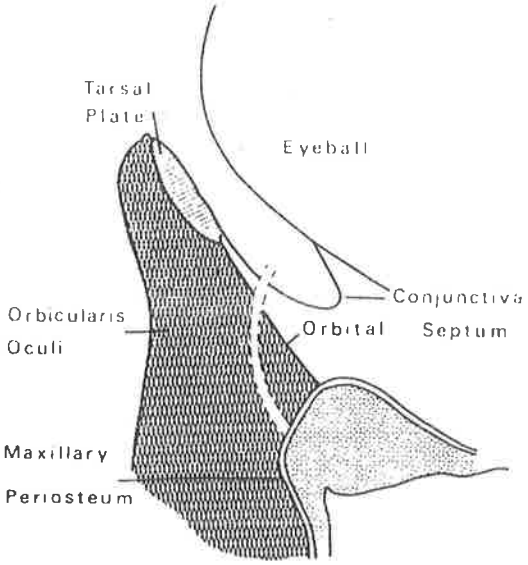

FIG. 1. *Sagittal section showing the perconjunctival approach to the orbital floor*

as well as protect the cornea. This plane is developed with a pair of curved iris scissors between the orbital septum and the orbicularis muscle. As the orbital rim is approached, a malleable retractor can be used to support the orbital contents. Two small curved retractors replace the skin hooks to hold the lid. The whole lower orbital rim is exposed, and the periosteum is incised on the maxillary surface, not on the crest of the margin, thus preventing fat herniation into the wound. In ten patients so explored, there had been disruption of the rim, and orbital fat was encountered. The periosteum of the orbital floor is easily raised with the flat end of a Howarth elevator. Excellent exposure of the orbital floor is obtained, and it is easy to insert sheet Silastic or to reconstruct the orbital margin by fine wiring (Figure 2, centre). This latter procedure is facilitated by the use of a contra angle dental drill. Closure of the wound is unnecessary, but can be achieved with a few fine catgut sutures in the conjunctival layer only.

Discussion

Tessier described this approach to the orbital floor and maxilla for late corrections of traumatic defects and congenital malformations. It has proven very satisfactory for the more commonplace situation of exploration and reconstruction of the orbital floor following recent trauma. Fine wiring of a disrupted infraorbital rim has not proven difficult, and the insertion of sheet Silastic beneath the floor periosteum is a simple matter.

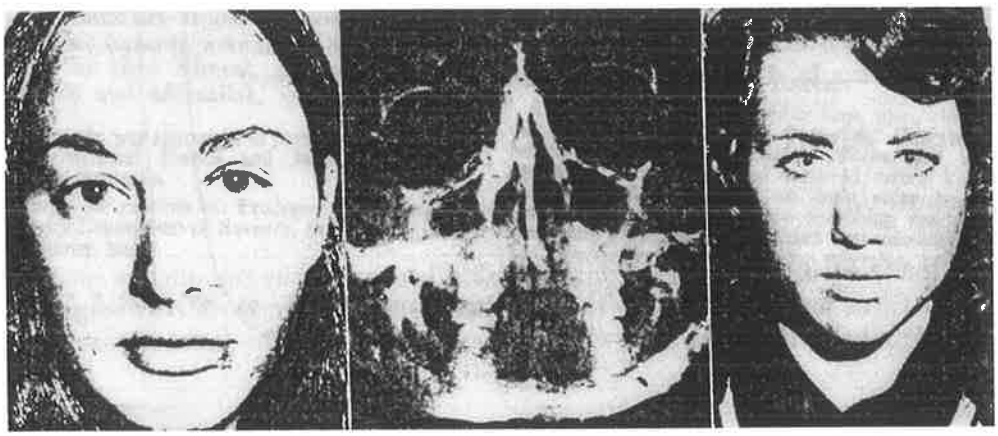

FIG. 2. *(left) a prominent scar following a lower eyelid incision; (centre) skiagram of Patient No. 3 showing a lower border interosseous wire; (right) Patient No. 4, showing the*

TABLE 1

Serial Number	Pattern of Fracture	Treatment	Complication of Incision
1	Middle third and zygoma	Silastic sheet	Lower lid entropion
2	Middle third and zygoma	Silastic sheet and interosseous wire	Nil
3	Zygomatic complex	Interosseous wire	Nil
4	Zygomatic complex	Exploration only	Nil
5	Pure "blow-out"	Silastic sheet	Lower lid entropion
6	Zygomatic complex	Interosseous wire	Nil
7	Pure "blow-out"	Silastic sheet	Chemosis
8	Zygomatic complex	Silastic sheet	Nil
9	Zygomatic complex	Exploration only	Nil
10	Middle third and zygoma	Silastic sheet and interosseous wire	Nil
11	Zygomatic complex	Exploration only	Nil
12	Pure "blow-out"	Silastic sheet	Nil
13	Pure "blow-out"	Silastic sheet	Chemosis
14	Middle third and zygoma	Silastic sheet and interosseous wire	Nil

After operation there is usually very little chemosis or lid swelling. However, in two cases of "blow-out" fracture, lower conjunctival chemosis was me severe and took several days to settle. In two other patients there was slight entropion of the lower lid due to inaccurate suturing of the maxillary periosteum and the orbital septum. In both cases this improved spontaneously. The technique of protecting the cornea with the distal conjunctival flap prevented any corneal abrasion.

Conclusion

The approach to the orbital floor through the lower fornix of the conjunctival sac provides excellent access for inspection and reconstruction. The absence of a visible scar is an additional advantage (Figure 2, right).

Acknowledgments

I wish to thank Mr D. N. Robinson for access to his patients and for his helpful advice and Mr J. Smith for his photography.

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New perspectives in the management of severe cranio-facial deformity *

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Key words: *Craniosynostosis; Fronto-Ethmoidal Meningoencephalocele; Cranio-Facial Trauma; Cranio-Facial Clefts; Cranio-Facial Surgery*

Summary

It is postulated that craniosynostosis is due to a growth abnormality in all or part of the cranial capsule. Release of the stenosed part in the first months of life will re-establish the balance between the rapidly growing brain and eye, and the cranial capsule. three periods for operative treatment are described: early, intermediate and late. Only in the early period can operative treatment restore normal growth dynamics; in the late period the aim is correction of an established deformity.

The relationship between cranial clefts and frontonasal encephaloceles is explored. If the space-occupying encephalocele is removed early, the distorted facial bones adopt a more normal position, whereas cranial clefts do not respond to early operation by remoulding.

The treatment of the acquired deformities of acute cranio-facial trauma have taken on new perspectives with the application of the multi-disciplinary approach and surgical techniques developed in the treatment of congenital deformities resulting in considerable reduction in the period of hospitalisation.

Introduction

'Monstrosities contribute to rectify our opinions... From wrong construction of parts arises unnatural action, which by studying we may discover the natural action.' John Hunter [1].

John Hunter introduced a scientific attitude towards surgery that has produced a more logical pattern of approach. However, his capacity to advance the practice of surgery was distinctly limited by the inadequacies of the techniques of his day (the tools of surgery changed very little from Roman times until the mid-19th century). Hunter's ideas of inductive and deductive thought have been combined with modern technology to the utmost effect in solving the problems of severe cranio-facial deformity. The most significant advance in this area was taken by Paul Tessier[2] in 1967 when he developed a two-stage combined cranio-facial approach to the orbito-cranio complex. It is this approach which has allowed cranio-facial surgery to develop to the sophisticated multi-disciplinary level practised in a number of units around the world today.

Such a unit has been functioning in South Australia since 1975. Centred at the Adelaide Children's Hospital and also operating at the Royal Adelaide Hospital, this Unit incorporates staff from several major hospitals and institutions in South Australia and since 1977 the Unit has established a liaison with New Zealand, Hong Kong, Malaysia, Singapore, Papua-New Guinea and Fiji. Tessier

* *Based on a Hunterian Lecture given at Royal College of Surgeons on May 26th 1983*

indicated that a unit should serve about 30 000 000 people. Munro extrapolated this concept to 7 units in North America. The size of the area will vary with the skills of the general plastic surgeons. As cranio-facial techniques pass into general use, fewer cases will need to be referred to a cranio-facial unit, but the need for such units will remain to deal with the extreme deformity, or the failure of conventional treatment. Since 1975 the South Australian Cranio-Facial Unit has been referred 496 patients; 301 operations were performed, 104 of which were transcranial and 197 subcranial. The aim of this paper is to present new approaches in the management of three categories of severe cranio-facial deformity:

1. The craniosynostoses.
2. Fronto-ethmoidal meningoencephaloceles and their relationship to cranio-facial clefts of the fronto-nasal region.
3. Acute cranio-facial injuries.

Management of the craniosynostoses

Craniosynostosis is premature fusion of the cranial sutures and is frequently associated with skull deformity. Rudolph Virchow[3] noted the relationship in 1852 and explained that premature fusion of many of the sutures could reduce the cranial capacity, the concept of cranio-stenosis, or narrowing of the skull.

Virchow noted that premature fusion of one suture results in cessation of growth in the direction perpendicular to that suture, and the compensatory over-growth across other sutures results in various cranial deformities.

As clinical interest in malformation of the skull intensified, it became evident that some of the more severe cases of craniosynostosis were associated with other birth defects as seen in Crouzon, Apert and Carpenter Syndromes, etc. In many cases there were serious associated facial malformations. Surgery designed to decompress the brain could not and did not improve these and new techniques were needed together with a new surgical philosophy. In contrast to Virchow's purely mechanical theory, Moss[4] has argued that premature sutural fusion is secondary to more fundamental dysplasia of the skull base. In his view, synostosis is a symptom not a cause of deformity.

This view is not wholly acceptable. For surgeons, the most compelling evidence of the role of the sutures is the response to adequate resection of fused sutures. The re-appearance of a previously obliterated suture after resection surely suggests that regional cranial growth has been released by operation allowing the brain to expand in a more normal way. It seems logical therefore to give some role to the fused sutures in the production of skull deformity.

It appears likely that the individual deformities associated with craniosynostosis represent dyscephalies due to distorted growth of the entire cerebral capsule; including cranial base, vault, pericranium and dura as well as bone. The hypothesis thus expressed that craniosynostosis is a regional failure of skeletal growth affecting a number of tissues is little more than a descriptive hypothesis. It still does not shed any more real light on the fundamental cause except in the minority of cases that are genetically determined.

When craniosynostosis is extreme there may be restriction of growth of the cerebral capsule producing raised cranial pressure and threatening vision and intellectual capacity. This situation is described as craniostenosis.

In the same way one can speak of orbitostenosis. The orbits are the incomplete bony capsule of the expanding eyeball and orbital sutural synostosis has been described. The orbital cavity becomes shallow and wide and the eyeball is extruded forwards.

Delaire *et al.*[5] in 1963 used the analogous term 'faciostenosis' to describe the midface hypoplasia of Crouzon syndrome. It is uncertain whether the maxillary hypoplasia of Crouzon or Apert syndromes represents an intrinsic local growth failure associated with premature sutural fusion or whether it is secondary to a primary dysplasia of the skull base acting both on the growth of the vault and the facial skeleton.

In summary, premature sutural fusion is seen as an important local manifestation of an underlying defect in the growth of the skull. This defect may be regional as in the simple calvarial deformities, or it may be generalised. Examples of the generalised disorders of skull growth are seen in the metabolic craniosynostoses and in the more severe types of complex cranio-facial deformity.

Thus far, the concept is in accordance with Moss' argument that craniosynostosis is not a primary disease process. There is evidence that premature calvarial sutural fusion has very real significance in the dynamics of abnormal craniocerebral growth. It results in a relatively unyielding cerebral capsule which fails to respond normally to the forces exerted by the expanding brain. There is now experimental[6] as well as surgical evidence to support the classical concept of the role of the sutures in determining to some extent the nature and severity of the deformity of the skull vault. So a concept of regional skeletal growth failure emerges. It is tempting to take it a little further when one reviews X-ray evidence that suggests that regional growth failure in the vault seems to be exerting upward tension on the developing orbito-frontal region, perhaps through dural tension lines. This would be a reversal of Moss' concept of the base distorting the calvarium by dural attachment. It is not yet possible to say whether premature fusion of the facial sutures has similar autonomous influence in determining the character of facial deformities; even if future research shows that this is not so, we believe that the deformities also represent the outcome of regional skeletal growth.

The principles of treatment are therefore based on a knowledge of the pathological process.

The surgical relevance of maturity varies in different types of craniosynostosis and this can be relevant in choosing the time to operate. We have therefore found it important to consider treatment in three somewhat arbitrary epochs, as described by David, Poswillo and Simpson[7].

1. The early stage — the first 12 months of life.
2. The intermediate stage — up to 9 years.
3. The late stage — 10 years or more.

The Early Stage

Operation may be performed:

(a) for the release of existing pathology, eg exorbitism or raised intracranial pressure, or

(b) prevention or minimising of future cranial and/or facial deformity.

Analysis of the more complex synostoses have led to some new perspectives.

Frontal plagiocephaly, where the deformity results from a fusion of one coronal suture and its basal extensions, or, in the more severe hemicranial plagiocephaly, from premature fusion of coronal, squamosal and lambdoid sutures.

The operation of linear craniectomy has been superseded by a modification of a logical and very elegant operation devised in Toronto[8]. This represents an application of Tessier's original concept of fronto-orbital advancement (Fig. 1a and b). The procedure can be used as a bilateral fronto-orbital advancement in cases of turricephaly[9].

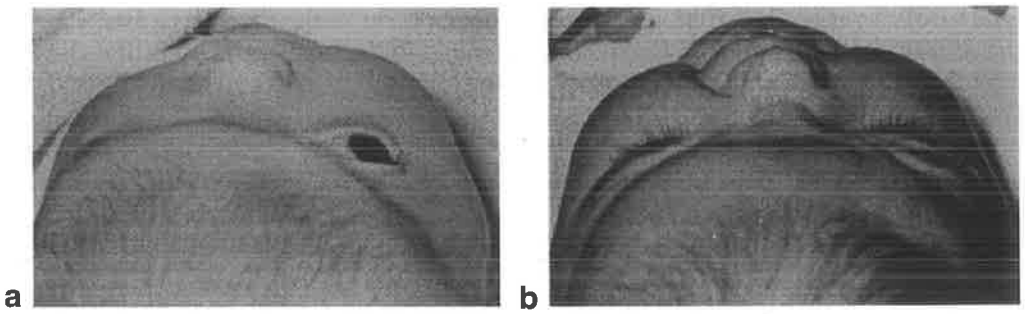

FIG. 1. *Infant with frontal plagiocephaly associated with unilateral coronal synostosis,* **a.** *before and* **b.** *after unilateral advancement of the fronto-orbital complex and frontal bone.*

In these forms of craniosynostosis the facial component of the deformity is considered secondary, yet where the primary defect is left uncorrected the secondary facial problems may be severe. The effects of early craniectomy on reducing secondary facial deformity are encouraging but as yet unsubstantiated.

Cranio-facial syndromes Decompressive operations for craniostenosis are frequently necessary. The first stage is a coronal craniectomy and fronto-orbital advancement with subtemporal craniectomy. The second stage, 2–3 weeks later, consists of parasagittal and lambdoid craniectomies. These operations have relieved the raised intra-cranial pressure well.

The Intermediate Stage

Operations at this stage are performed for

(a) threats to function, the most common of which are the effects of extrusion of the globes and/or recurrent raised intracranial pressure,

(b) psycho-social distress.

Considering this period between 1 and 9 years separately is justified because the child is now too old to reap the prophylactic benefits of early treatment, yet too young to achieve a definitive cosmetic and functional result. A range of procedures is performed during this time, intermediate in the total treatment plan. In the younger child a fronto-orbital advancement may be sufficient, whereas in the older child the more radical operation of fronto-orbital advancement and anterior maxillary shift may be indicated.

The Late Stage

These are invariably for psycho-social and cosmetic indications but have associated functional advantages.

(a) Correction of exorbitism resulting from orbito-stenosis.

(b) Release of the upper airway constriction resulting from faciostenosis.

(c) Improved occlusion.

In this period the surgical options are more complex. It is now possible to move parts of the cranial and facial skeleton in three dimensions in order to correct all deformities associated with craniosynostosis. In simple plagiocephaly, fronto-orbital advancement of one or both sides may be adequate. Frontal depression and asymmetry of the mid and lower face may be corrected by adding facial osteotomies to the fronto-orbital advancement. The osteotomies of the midfacial skeleton are conveniently categorised in terms of Le Fort's classifications of facial fractures, Le Fort I, II and III. All three sections enter the region of the pterygomaxillary fissure, where the maxilla and palatine bone must be detached from the pterygoid process. Of course the osteotomy lines do not exactly duplicate Le Fort's fractures, but the comparison gives a useful shorthand identification of these complex mid-facial procedures. The following procedures have been used in the late stage.

(a) Sub-cranial Le Fort III osteotomy and facial advancement, done when the anatomy of the cranial base is relatively normal and no calvarial reconstruction is needed.

(b) Transcranial Le Fort III osteotomy and facial advancement when the anatomy of the cranial base is such that the temporal lobes or the contents of the cribriform fossae are at risk during osteotomy. The procedure may be combined with a fronto-orbital advancement (Fig. 2a and b). Combined facial osteotomies may be appropriate, eg Le Fort III and Le Fort I sections. In cases with exophthalmos and naso-maxillary retrusion but normal dental occlusion the upper maxillary complex can be advanced independently of the palate and dentition. This is termed Le Fort III minus Le Fort I section. Le Fort III and I sections can be combined when the upper jaw has to be moved independently of the orbito-nasal complex and it is expected that correction by onlay bone grafts would be unsatisfactory. When there is hypertelorism as well as maxillary hypoplasia, Le Fort III and Le Fort I sections can be combined with transcranial paramedian naso-ethmoidal resections and orbital shifts.

(c) Transcranial orbital translocations are now standard procedures for the correction of hypertelorism and orbital dystopia. They may be combined with frontal reshaping. They have been performed for the hypertelorism of Cohen syndrome and severe frontal plagiocephaly (Fig. 3a and b).

From the study of the 'unnatural action' produced by the deformity has emerged an hypothesis on which is based a rational approach to surgery.

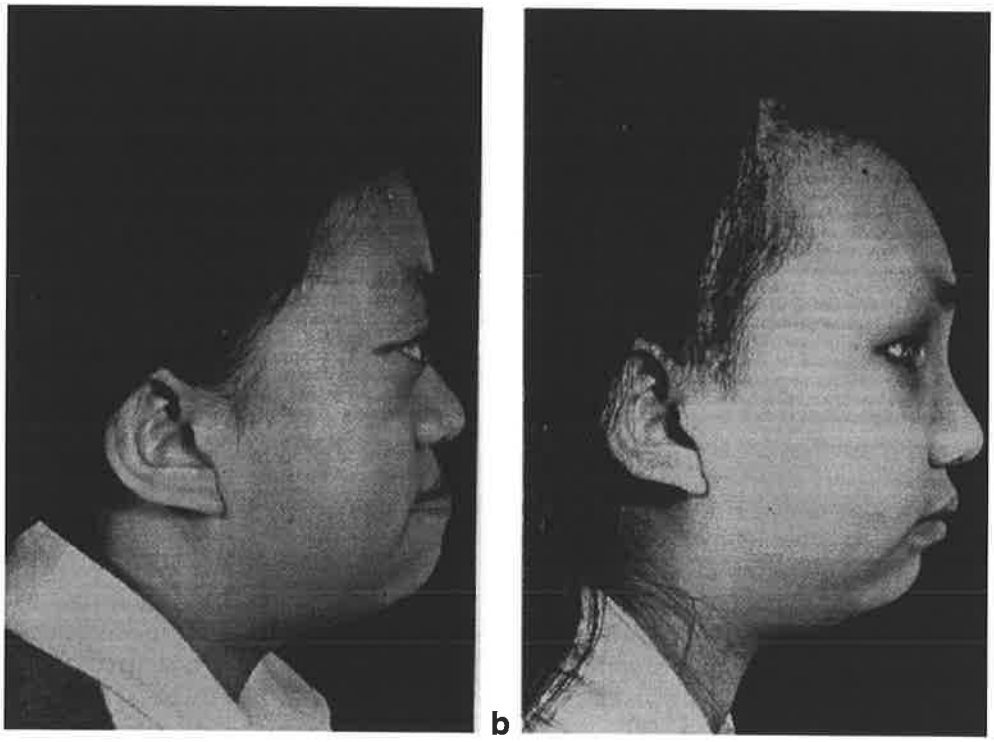

FIG. 2. *Chinese girl with Crouzon syndrome,* **a.** *before and* **b.** *after transcranial Le Fort III osteotomy and facial advancement combined with fronto-orbital advancement.*

Relationships between fronto-ethmoidal meningoencephaloceles and cranio-facial clefts

Many attempts have been made to classify clefts of the cranio-facial region. Tessier has made an anatomical classification which describes clefts arranged around the orbit, numbered 0 to 14. 'This attempt at classification of clefts does not prevent a more sophisticated or detailed explanation, but rather it provides an immediate reference to the exact location and character of what is being described'[10].

Mazzolla[11] has produced a morphological classification of malformations to the fronto-nasal area based on embryological studies. Mazzolla uses the term 'fronto-nasal dysraphia' to include median clefts of nose, possibly equivalent to Tessier's clefts 0, 14 and 1, 13. Fronto-nasal dysraphia also includes cysts and fistulae of the fronto-nasal region. He includes fronto-ethmoidal meningoencephaloceles in the median cleft nose group. Difficulties with classification of defects in this area provide a challenge to the use of the Hunterian techniques of observation, induction of principles, and the use of these principles in the further management of problems.

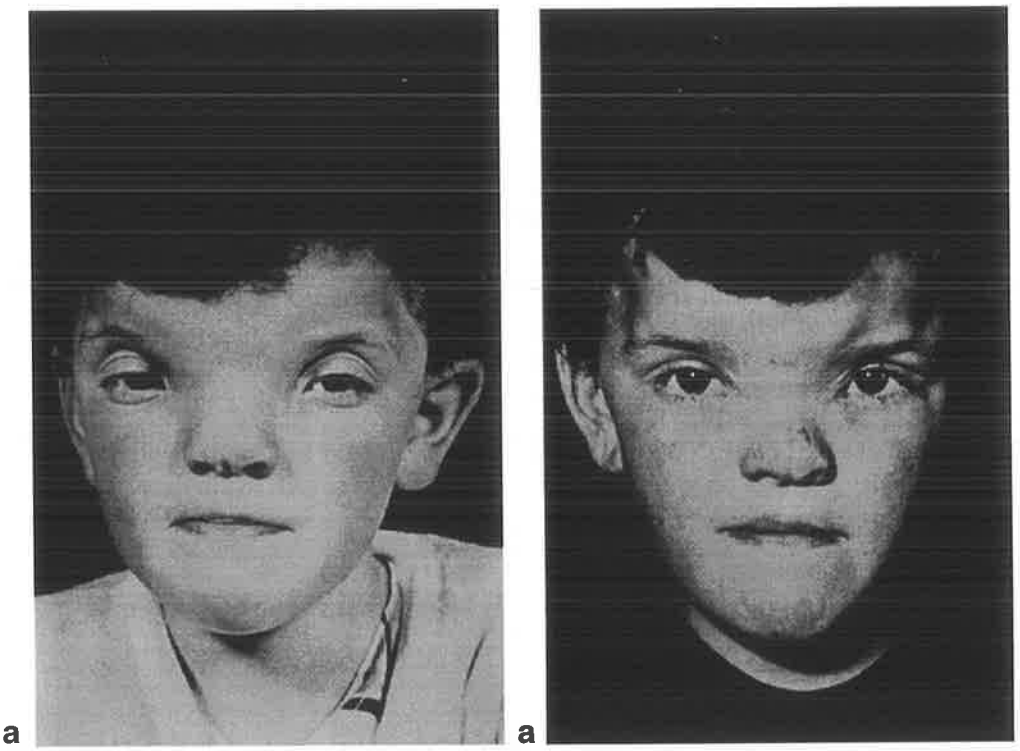

FIG. 3. *Boy with craniofrontonasal dysplasia (Cohen II syndrome) and resultant hypertelorism* **a.** *before and* **b.** *after transcranial orbital translocations and nasal reconstruction.*

Case material

Seventeen cases of fronto-ethmoidal meningoencephalocele were compared with 13 cases of midline facial clefts of the median and paramedian cleft nose variety. Cephalocele appears to be the appropriate generic name for a congenital herniation of brain through a skull defect; the alternative name is craniumbifidum, in the attractive but unproven assumption that these conditions are the cephalic equivalent of spina bifida. The contents of the herniae are usually meningoencephaloceles which can be classified in four main groups:

(i) Occipital

(ii) Parietal.

(iii) Basal.

(iv) Sincipital.

The cincipital group have been further classified by Suwanwela[12] basing his classification on that of Von Meyer[13] into fronto-ethmoidal meningoencephaloceles:

(i) Naso-frontal.

(ii) Naso-ethmoidal.

(iii) Naso-orbital.
Interfrontal encephaloceles.
Cranio-facial clefts.

The description 'fronto-ethmoidal' is most appropriate because it describes the site of the cranial end of the defect which is always through the position of the foramen caecum at the junction of the frontal (membranous) and ethmoidal (cartilaginous) bones. The crista galli is at the posterior margin of the defect.

Morphology

Cranial End of the Bony Defect

The exit hole from the anterior fossa was always at the junction of the frontal bone and the ethmoidal bone at the site of the foramen caecum (Fig. 4). Behind this, that is at the posterior margin of the cranial defect, was the cribriform plate with the crista galli at its anterior end. The cribriform plate was tilted downwards, deepening the central portion of the anterior cranial fossa. The anterior end of the cribriform plate lies much lower than the posterior end; the cribriform plate thus forms an angle of 45-50 degrees with the horizontal. The defect varied in size and shape; all naso-frontal defects were round and central; all naso-orbital defects were bilobed (2 patients from this group were previously operated and one side of the exit holes had been obliterated by metal mesh); 2 of the naso-ethmoidal type were bilobed, in 1 case the defect was lozenge shaped and central and the remainder were round.

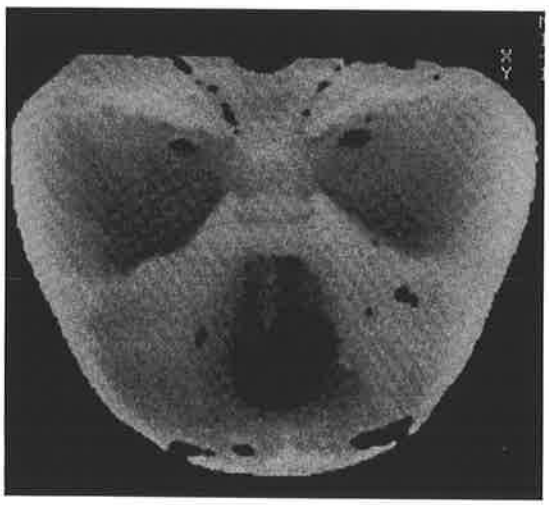

FIG. 4. *Three-dimensional computer reconstruction of a CAT scan of a child with fronto-ethmoidal meningoencephalocele. The rotational view shows the anterior and middle fossae from above, with the exit hole of the meningoencephalocele at the junction of the frontal bone and ethmoidal bone.*

The Facial End of the Bony Defect

Naso-frontal The defect lies at the junction of the frontal and nasal bones with the nasal bones attached to its inferior margin. The defect varies in shape (Fig. 5a and b).

Naso-ethmoidal The facial defects lie between the nasal bones and the nasal cartilages. The nasal bones are deformed, often broadened with crimped margins. The fronto-nasal angle is obliterated, producing the appearance of an overhanging ledge (Fig. 6a and b).

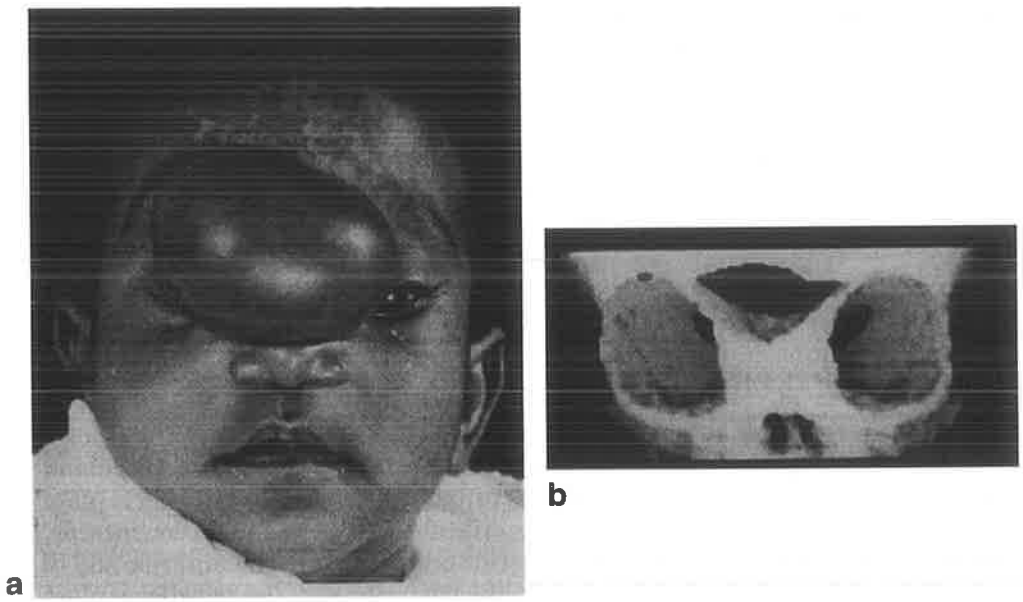

FIG. 5. a. *Aboriginal infant with naso-frontal variety of frontoethmoidal meningoencephalocele. The facial defect lies at the junction of frontal and nasal bones, with the nasal bones attached to the inferior margin.* **b.** *Three-dimensional reconstruction of CAT scan*

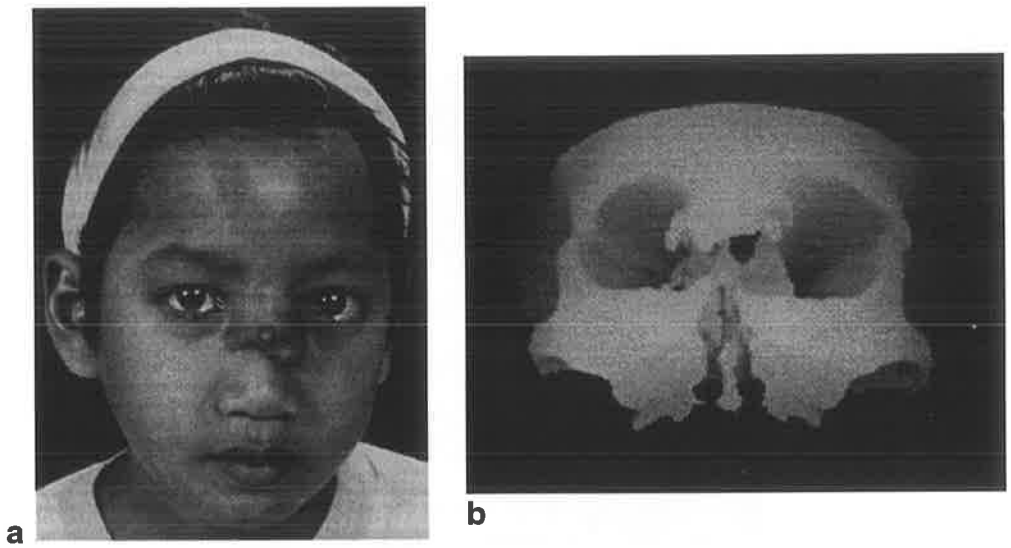

FIG. 6. a. *Malaysian boy with naso-ethmoidal variety of frontoethmoidal meningoencephalocele. The facial defect lies between the nasal bones and nasal cartilages, with erosion of the anterior margins of the medial orbital walls.* **b.** *Three-dimensional reconstruction of CAT scan.*

If the facial defect is confined to the nasal pyramid it is small and oval, and the medial walls of the orbit are not involved; if, however, the meningoencephalocele is larger and the facial defect extends more laterally, then the anterior margins of the medial orbital walls are eroded and become crescent shaped.

Naso-orbital type presents on the face through defects in the medial orbital walls situated in the frontal process of the maxilla and the lacrimal bones (Fig. 7a, b and c). The bony tract is long and shaped like an inverted 'Y'. The inverted 'Y' may be symmetrical. The meningoencephalocele comes through a frontal process of the maxilla onto the face leaving the nasal bone intact anteriorly and the lacrimal bone and lateral plate of the ethmoid intact posteriorly. However, during its passage through the substance of the ethmoid the lateral plate is pushed laterally forming a bony tunnel by the extruded cranial contents. The orbital soft tissue mass may extend over the medial orbital rim onto the face producing a groove or flattening of the inferior orbital margin medially.

General Features of the Bony Facial Skeleton

In all cases the face appears to be longer than normal. The pyriform aperture with nasal cartilages is compressed from above and displaced inferiorly. There is telecanthus. This is usually not severe compared with clefts, and is of the Tessier second degree variety, with normal lateral canthal distance. Six patients had malocclusions which may be related to the deformity in that the vertical plate of the ethmoid bone being attached to a tilted cribriform plate is itself retrodisplaced and this may result in some maxillary hypoplasia. The naso-ethmoidal type of encephalocele has a direct effect on the nasal septal cartilage pushing it downwards and backwards. It is as though the encephalocele has blown out onto the face through the weakened junction of the frontal and ethmoidal bones and displaced the otherwise normal orbits and nasal capsule widening the orbits and lengthening the face.

In contrast to clefts which have a deficiency of tissue at their margins, the defects of the fronto-ethmoidal meningoencephaloceles are like tunnels or 'blow-holes', and they are lined by normal tissues.

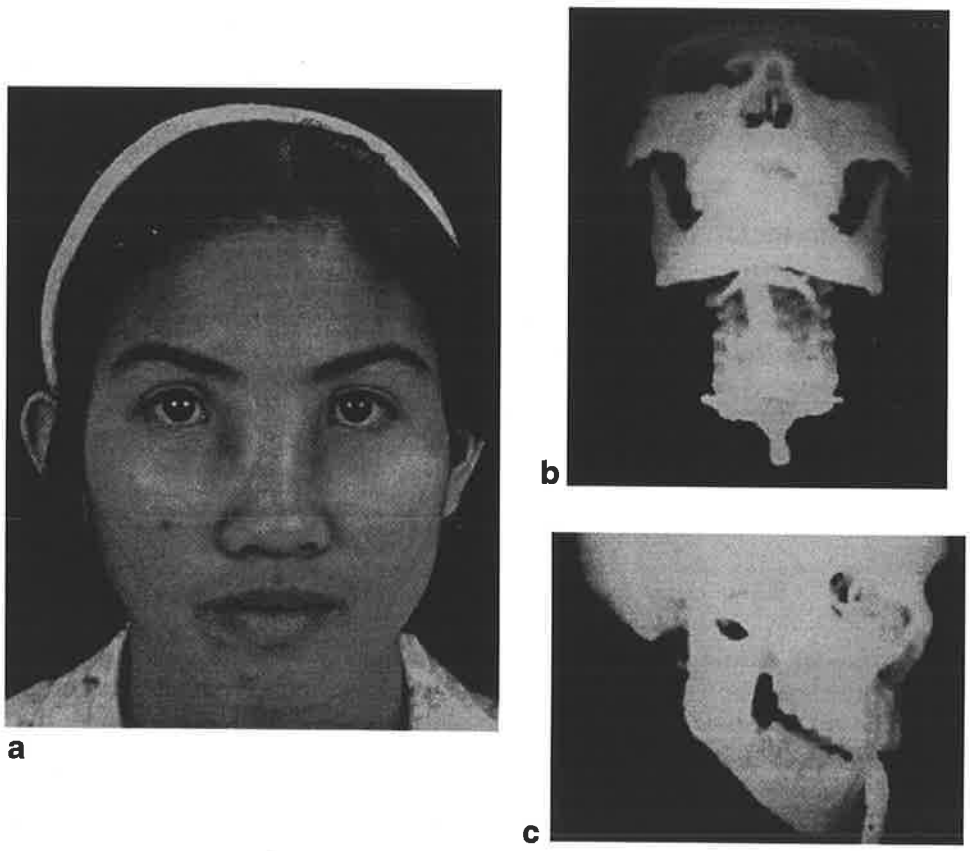

FIG. 7. a. *Malaysian woman with naso-orbital variety of frontoethmoidal meningoencephalocele. The encephalocele presents on the face through defects in the anterior medial orbital walls. The nasal bone is intact anteriorly.* **b, c.** *Three-dimensional reconstruction of CAT scan. Endotracheal tube in place for anaesthetic.*

Soft Tissue

Pathological constituents of the encephalocele vary. No patients had extension of the ventricular system into the defect. Some patients had a small amount of normal-looking brain in the neck of the encephalocele but usually the tissue looked atrophic. Nine patients had previous surgery, 7 had intercranial operations only and 2 had intracranial and facial operations.

Those cases where the neck of the encephalocele has been divided at previous surgery did not show significant spontaneous atrophy of the facial extension suggested by Naim-Ur-Rahman[14]. This tissue appears to remain in enough bulk to produce the significant distortion of the face.

Soft tissue mass of the meningoencephalocele may extend into the orbits and fuse with the peri-orbitum, but may flow over the infra-orbital rim which becomes indented and depressed and onto the face to be associated with abnormal skin which is discoloured and may be scarred from previous ulceration and healing. The skin is often thickened and crusty.

Ocular Problems

Four patients had decreased visual acuity, 3 patients had squints, 8 presented with lacrimal drainage disfunction and 3 with a degree of orbital dystopia. Fig. 8 shows the increased intercanthal distance, increased interpupillary distance and relatively normal lateral canthal distance in these patients.

Neurological Problems

Three patients had a degree of mental retardation, 5 had hydrocephalus and 2 had epilepsy.

The interfrontal type of sincipital meningoencephalocele referred to by Suwanwela has not been seen by us. However, such a case is presented and described in Tessier's article on facial clefts[10]. It appears from the description to be indeed a midline facial cleft where the interfrontal meningoencephalocele is secondary to the clefting which continues down the midline of the nose into the maxilla and has produced a short, wide face with severe hypertelorism.

Median clefts of the nose (Tessier 0,14 and 1,13)

Thirteen such cases have been studied including 2 cases of syncephalic twin. In all of these cases the facial height is decreased, the interpupillary distance, intercanthal distance and lateral canthal distance is much increased compared with the fronto-ethmoidal meningoencephalocele. There is a deficiency of both bone and soft tissue adjacent to the clefts and the degree of hypertelorism is greater than with the fronto-ethmoidal meningoencephaloceles.

Thesis

Fronto-ethmoidal meningoencephaloceles are fundamentally different in origin from the midline clefts. The deformity is related to the space-occupying extruded brain and is not intrinsic to the bones. Early complete surgery should allow the developing brain and eyes to mould the skeleton. The forces generated by the nasal airway, speech and mastication will remodel the facial deformity. The midline clefts of the nose have a primary deficiency of tissue. The abnormality is intrinsic to the tissues themselves and early surgery will not be expected to reduce the final deformity.

Treatment

All operations were performed by the cranio-facial approach through a bicoronal scalp flap. Sub-periosteal dissection of the orbits was performed. Where indicated the skin over the midline soft tissue deformity was excised. Lower facial osteotomies were performed before the bifrontal craniotomy. The neck of the meningoencephalocele was isolated by a combined intradural and extradural approach. Brain was inspected and as much as possible was conserved. The neck of the encephalocele was transected and the dural defect repaired. The remaining orbital cuts were then made. The medial orbital walls are often defective in the naso-ethmoidal type and the angle of the cribriform plate is so steep that the translocated orbits come to overlay the cribriform plate. Canthopexies and nasal bone grafts are performed as necessary.

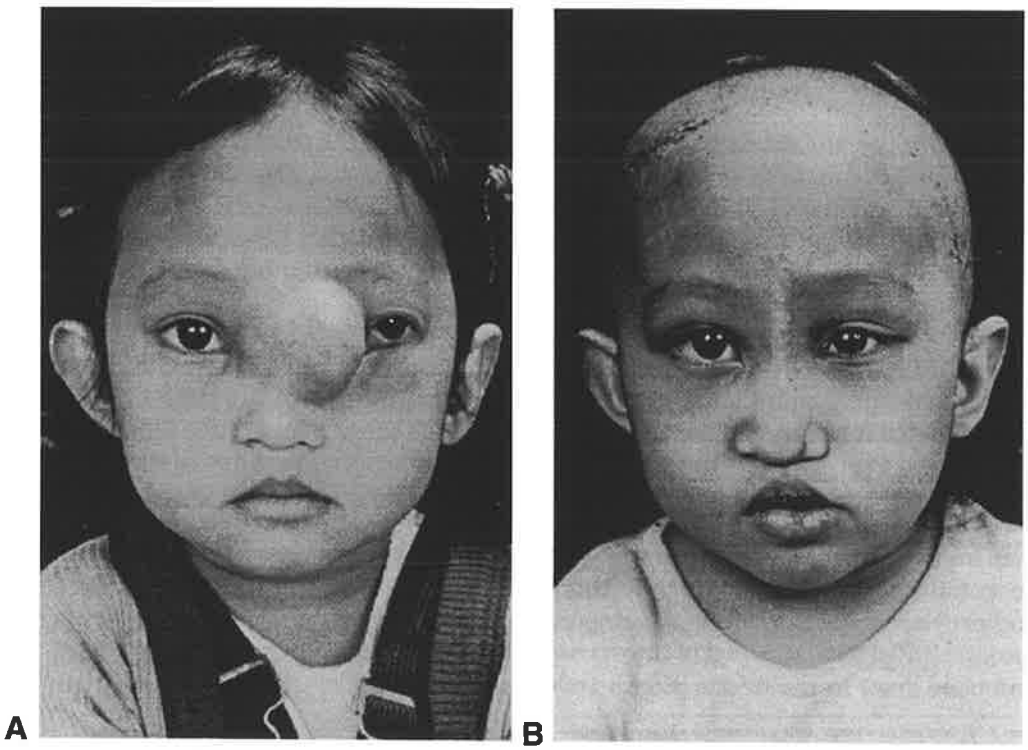

FIG. 8. *Naso-frontal variety of fronto-ethmoidal meningoencephalocele (a) before and (b) after transcranial translocation of both orbits, and nasal reconstruction.*

Eleven patients had both orbits moved (Fig. 8a and b). Two patients had one orbit moved and 4 patients had medial orbital wall osteotomies plus canthopexies, this being the operation of choice in the first year of life (Fig. 9a and b).

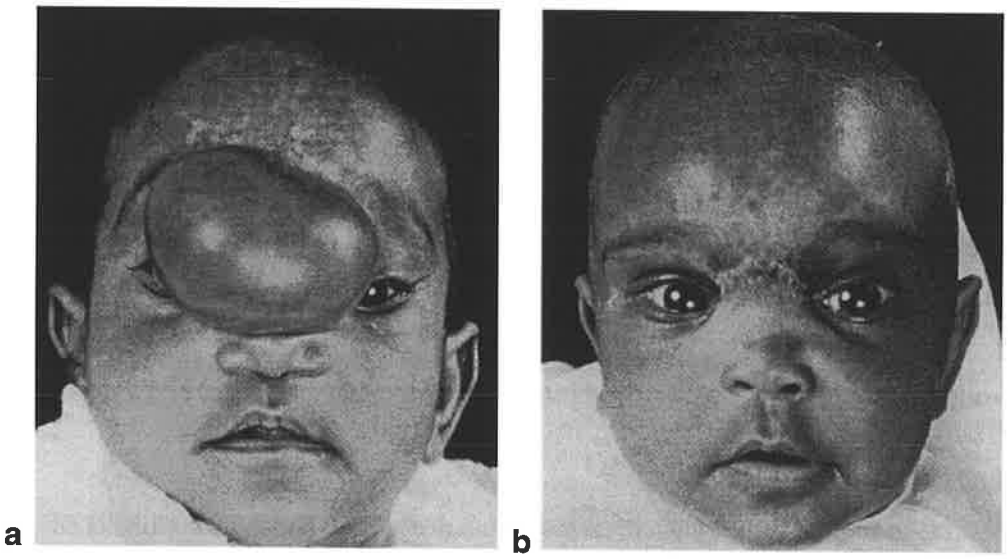

FIG. 9. *Naso-frontal variety of fronto-ethmoidal meningoencephalocele* **a.** *before and* **b.** *after transcranial reduction of encephalocele and medial orbital wall osteotomies, canthopexies and nasal reconstruction.*

Discussion

The logical classification of sincipital encephaloceles has been suggested, establishing a relationship between clefts of the nose and hypertelorism and short faces and frontoethmoidal meningoencephaloceles with its three sub-groups.

It has been previously suggested that sincipital meningoencephaloceles are one of the neural tube defects to be considered with anencephaly and myelomeningocele as a varying expression of a single developmental aberration.

However, the sincipital meningoencephaloceles of the frontoethmoidal type pose some difficulties to this interpretation. They do not have the circumstantial supporting evidence of sibling affectation present in the other neural tube defects. They also show quite a different geographical distribution affecting people in Malaysia, Thailand, Burma, Pakistan and Southern Russia but being rare in Europe and North America.

Thus in their epidemiology the sincipital frontal-ethmoidal meningoencephaloceles show remarkable peculiarities and in the present state of knowledge it seems unwise to lump them in with other neural tube defects. It may indeed express some unknown environmental agents, perhaps dietetic.

There are obvious functional differences between the meningoencephaloceles and the clefts; mere division of the neck of the encephalocele is not enough to wither the distal extent of the extruded tissue and prevent distortion of the developing facial skeleton. Cranio-facial surgery is recommended with removal of the extruded brain and the appropriate osteotomies and bone graft in the first 3 months of life if possible.

Cranio-facial fractures

Hunter stated that *'the principles of our art are not less necessary to be understood than the principles of other sciences ...'*. The principles established from studies of some congenital deformities have been effectively applied in the treatment of severe cranio-facial trauma. I wish to trace the evolution of the *techniques of treatment* in the South Australian CranioFacial Unit and outline the current management.

Material

Forty cases with severe cranio-facial fractures are analysed spanning the period of change from one approach to the other. Seventeen patients had CSF rhinorrhoea and were treated conservatively, that is, by reduction and treatment of facial component of the fracture only.

In each of these cases the CSF rhinorrhoea ceased postoperatively. The second group of 23 patients required both a neurosurgical procedure and a plastic surgical procedure.

Classification

In addition to the Le Fort classification which deals with the middle third of the face, we have come to consider the pattern of the cranial component of the fractures as:

Trans-frontal
Trans-orbital
Trans-basal: trans-ethmoidal
trans-sphenoidal

Traditional Treatment

In the early 1970s the treatment of such fractures in our Institution represented a compromise between two extreme views. At one end of the spectrum was a neurosurgical view[15] that patients with persistent CSF rhinorrhoea due to a fracture of the cranial base retained a high risk of meningitis even if the leak

ceased spontaneously. At the other end of the spectrum was the plastic surgical view that if CSF rhinorrhoea stopped with reduction of the facial fracture the risk of meningitis was negligible.

The situation existed where neurosurgeons whose experience was largely derived from cranial fractures which extended into the base of the skull rarely took the pattern of the facial fracture into account. The plastic surgeons rarely considered the problems of brain, dura and scalp.

Phase of Developing Co-operation

Initial co-ordination of disciplines was brought about by a joint assessment of the patient and radiographs in the first week after injury. Conventional tomography was initially used to determine the pattern of the naso-ethmoidal component of the fracture. Our neurosurgical colleagues were principally interested in whether the fracture could be seen crossing the floor of the anterior cranial fossa. It soon became clear that the tomograms shed more light on all components of the fracture. With an atmosphere of co-operation and the information provided by the tomography an individual plan for treatment of a particular patient would be made. During these years of inter-disciplinary treatment, the correction of the facial skeleton was usually performed first, unless urgent neurosurgery was necessary. The neurosurgical correction was then left for a number of weeks and done as a second individual procedure.

Modern Techniques

With increased influence of the Cranio-Facial Unit, the early combined neurosurgical and plastic surgical approach was developed. Of 23 cases requiring both neurosurgical and plastic surgical intervention, 10 have been treated by a single cranio-facial operation between the first and sixteenth day post-accident. In cases treated before 1979 separate plastic and neurosurgical operations were performed from 1 to 14 weeks apart.

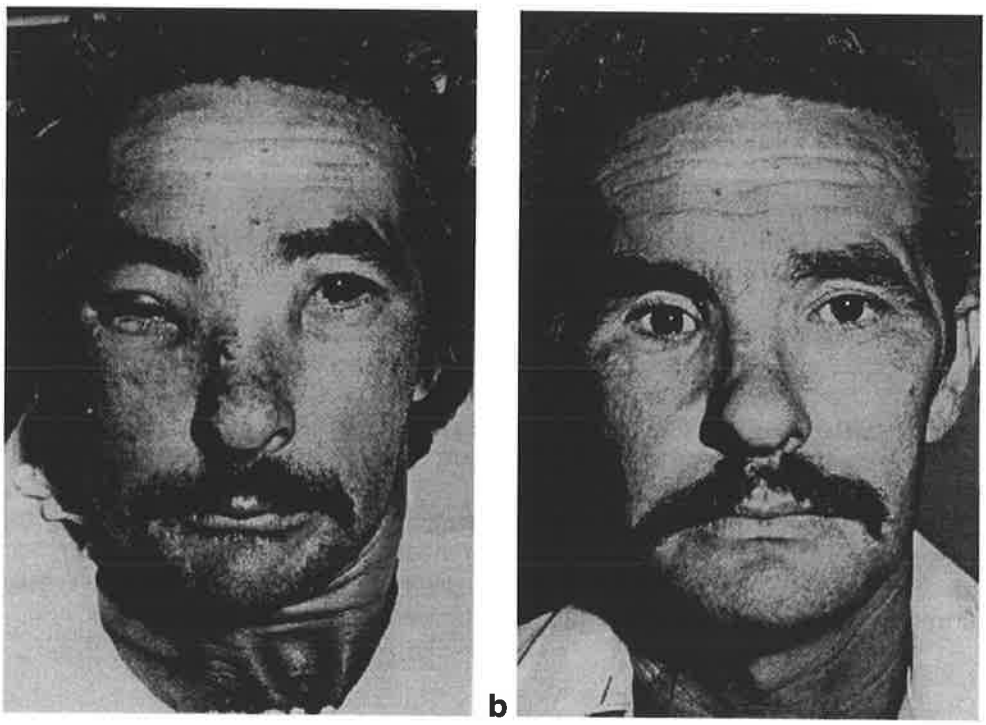

FIG. 10. a. *Man with extensive cranio-facial fractures on presentation after accident.* **b.** *After recovery from combined neurosurgical and cranio-facial primary repair.*

The indications for combined early treatment became clear.

(i) Cranio-facial fractures with intra-cranial air or intracranial blood requiring urgent neurosurgical intervention.

(ii) X-ray evidence of significant disruption of the frontal bone, orbital roofs or base of skull.

The conventional tomography of the 1970s has been supplanted by CAT scanning with varying degrees of sophisticated reconstruction. At the present time we are able to produce three-dimensional reconstruction of considerable detail.

Techniques

A bicoronal scalp flap is turned down to expose the superior orbital margin. The frontal zygomatic and naso-ethmoid regions are dissected. Neurosurgical repair of the dura through a frontal craniotomy takes precedence followed by reduction and fixation of all fractures. Fine wiring of the fronto-orbital components is necessary and is often combined with external fixation. The latter has to be placed above the craniotomy.

Because such surgery exposes the dura and CSF spaces to the nasal airway, particular care is taken about sepsis. Preoperative and postoperative antibiotic cover is used.

There have been no serious postoperative complications in any of these patients since the implementation of the combined surgical approach. Uncorrected cranio-facial fractures produce hideous deformities. The early combined cranio-facial approach has been proved an effective way to restore these people to normality (Fig. 10a and b). The outstanding result from this approach is that the period of hospitalisation is reduced by 50%.

Conclusion

The study of abnormal growth pattern in craniosynostosis syndromes during the post-natal period has led to a working hypothesis of the pathogenesis of the deformity. This has led in turn to a rational approach to the timing of surgery.

The concept of a fundamental difference in the genesis of the deformity associated with fronto-ethmoidal meningoencephaloceles and cranio-facial clefts determine the timing and type of surgery.

From such studies a system of management has been effectively applied to the early treatment of patients with cranio-facial fractures.

From the study of the 'wrong construction of parts' acquired cranio-facial deformity can be managed more effectively.

It is to John Hunter that we are indebted for applying the inductive system of observation and experiment to the study of disease, producing general principles which we are then able to apply to solve further problems, allowing us to reach out and extend our surgical care into the future.

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* Based on a Hunterian Lecture given at Royal College of Surgeons on May 26th 1983

The Editor would welcome any comments on this paper by readers

Computer-based coding of fractures in the craniofacial region

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Summary

A systematic approach to the coding of fractures in the craniofacial region is presented. An alpha-numeric code is formulated from an alpha component depicting the anatomical region of interest and a number which reflects the degree of disruption.

The complex shapes of the bones forming the craniofacial skeleton have made the classification of fractures difficult. As a consequence, many attempts at fracture classification have been restricted to one bone or a small region of anatomy. However, the intimate association of each bone with its neighbours, and their intricate connections at serpiginous sutures, provides a structural design susceptible to multiple bone fracturing with a single impact. Such multiplicity of bony derangement often taxes the versatility of existing fracture classifications, many of which employ eponymous nomenclature and may be subject to misinterpretation.

In view of the above, the objectives of the present study were:

(i) to develop a fracture coding system which was simple, reproducible and meaningful to both clinicians and other personnel who require craniofacial fracture data suitable for computer analysis;

(ii) to devise a numerical scoring system to document the overall degree of craniofacial bony disruption in any patient.

An alpha-numeric system is presented which offers a method of coding all bony elements of the craniofacial skeleton. In this system the craniofacial region is considered as 10 bilateral *major* anatomical zones, each composed of a series of *minor* zones and an alphabetic code has been assigned to each zone. The numerical component of the coding system is used to indicate the degree of fracture in each *minor* zone, where the number 1 represents an undisplaced fracture, through to a number 3 which describes fracture with comminution. The numerical score of disruption in each *major* zone is the sum of that region's ipsilateral minor zone scores (to a maximum numerical value of 5).

In this way, the alpha-numeric coding system offers a method of describing not only the fracture location but also the severity of the traumatised area and its relationship to unaffected bones. Furthermore, the major zones contribute to an overall numerical score of craniofacial bony disruption.

Alpha map

In this system the craniofacial region was divided into 10 bilateral major anatomical zones. To facilitate documentation, an "alpha map" of the craniofacial skeleton was produced and each of these 10 major zones was assigned a simple alpha code derived from the bone in that region:

Cranial zones	—	Frontal	F
		Parietal	P
		Sphenoidal	S
		Temporal	T
		Occipital	OC

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Facial zones	—	Naso-ethmoidal	NE
		Zygomatic	Z
		Orbital	O
		Maxillary	MX
		Mandibular	MD

With due consideration for the fracture patterns within each major zone, further anatomical subdivisions were delineated. Each subdivision, or minor zone, was represented by an expanded alpha code (Tables 1A and 1 B) which, in the majority of minor zones, was simply an extension of the code assigned to the parent major zone. These subdivisions also included the commonly diastased bony junctions. For example, the zygomatic major zone Z was considered to have four subdivisions of relevance to fracturing. These four minor zones were: the zygomatic arch ZA, the zygomatic body ZB, the zygomatico-frontal suture Z:F, and the zygomatico-maxillary suture Z:MX.

In order to maintain uniformity of the anatomical alpha coding system, calvarial sutures were coded according to their adjacent bones:

Coronal suture (between Frontal and Parietal)	F:P
Sagittal suture (between each Parietal)	P:P
Squamosal suture (between Parietal and Temporal)	P:T
Lambdoid suture (between Parietal and Occipital)	P:OC

TABLE 1A

Alpha coding of cranial bones

Major zone	Major code	Minor zone	Minor code
Frontal	F	calvarial	FC
		frontal sinus anterior	FSA
		frontal sinus posterior	FSP
		anterior fossa	FA
		cribriform plate	FCP
		coronal suture	F:P
Parietal	P	calvarial	PC
		sagittal suture	P:P
		squamosal suture	P:T
		lambdoid suture	P:OC
Sphenoidal	S	lesser wing	SL
		greater wing	SG
		sph-frontal sature	S:F
		basal	SB
		sph-occ synchond.	S:OC
Temporal	T	calvarial	TC
		basal	TB
		petrous	TP
Occipital	OC	calvarial	OCC
		basal	OCB

TABLE 1B

Alpha coding of facial bones

Major zone	Major code	Minor zone	Minor code
Naso-ethmoidal	NE	nasal bone	N
		naso-fontal suture	N:F
		maxill. frontal process	NMX
		ant ethmoid	EA
		post. ethmoid	EP
Zygomatic	Z	arch	ZA
		body	ZB
		zyg.-frontal suture	Z:F
		zyg.-maxill. Suture	Z:MX
Orbital	O roof OR	medial wall	OM
		lateral wall	OL
		floor	OF
		inferior rim	OI
		superior rim	OS
Maxillary	MX	ant.wall	MXA
		buttress	MXB
		palate	MXP
		dento-alveolar	MXD
		pterygoid	MXT
Mandibular	MD	condyle	MDC
		coronoid process	MDP
		ramus	MDR
		angle	MDA
		body	MDB
		symphyseal	MDS
		dento-alveolar	MDD

Numerical coding

A numerical score of bony disruption was used to code the fracture severity, or the degree of sutural diastasis, in each minor zone according to the following gradation:

0 = no fracture
1 = undisplaced fracture
2 = obviously displaced fracture
3 = comminuted and/or compound fracture

For each major zone a numerical score was derived from the sum of the minor zone scores (to a maximum value of 5). For example, fracturing of the left zygoma which included undisplaced fractures of the zygomatic arch and the body of zygoma together with obvious displacement at the zygomatico-frontal and zygomatico-maxillary sutures would be coded as follows:

Minor zone scores:		
zygomatic arch	ZA	1
zygomatic body	ZB	1
zygomatico-frontal suture	Z:F	2
zygomatico-maxillary suture	Z:MX	2
Major zone score: Z score =		5

Thus, although the summation of this zygoma's minor zone scores is actually 6, a maximum limit of 5 has been assigned to each major zone: this facilitates the calculation of an overall numerical score of bony disruption *(vide infra)*.

Coding forms

Two coding forms were developed which graphically illustrate the component zones of the cranio-facial alpha map and also provide diagrams onto which the fracture pattern can be drawn (Fig. 1A, B). To complete each form the encoder allocates the numerical coding of fracture severity (from 0-3) to each minor zone. The scores for the major zone (from 0 to 5) can then be readily calculated.

Alternatively, the provision exists for a more rapid coding process in which an experienced observer can allocate a numerical score directly to a major zone (from 0-5).

Craniofacial disruption score

The sum of the 20 major zone scores therefore provides an overall score which can be directly expressed as a percentage. This is referred to as the Craniofacial Disruption Score (CDS) (Fig. 2).

Discussion

In the past, numerous methods for the grading and classification of facial fractures have been described. Although each of these methods has its particular merits, there is no universally accepted single system (Langdon and Rapidis, 1982; Beaumont *et al.,* 1985). Even the time-honoured Le Fort (1901) levels of midfacial fracture have attracted criticism from clinicians who have observed many fracture patterns at variance with those originally described (Wassmund, 1927; Merville, 1974; Manson *et al.,* 1980; Matras and Kuderna, 1980). Furthermore, many fracture patterns have been associated with eponymous labels which offer little inherent meaning to people seeking objective fracture data. Such nomenclature may be confusing unless one has a thorough understanding of all synonyms; for example, each Le Fort fracture level may be equated with alternatives: Le Fort 1 fracture = Guerin fracture; Le Fort 11 fracture = Wassmund 11 fracture; Le Fort 111 fracture = Wassmund IV fracture.

Many existing fracture classifications do not provide any indication of fracture severity. Also the closely packed bony anatomy in some regions of the craniofacial skeleton has led to indistinct boundary definition for some fracture categories; this is exemplified by the naso-ethmoid region (Stranc and Robertson, 1979), trauma in which has generated several variants of terminology such as a "fracture of the ethmoids" (Dawson and Fordyce, 1953), "naso-orbital fracture" (Converse and Smith, 1963), "fracture of the naso-ethmoid" (Stranc, 1970), "naso-ethmoid-orbital fracture" (Luce, 1984; Gruss, 1985) and "naso-frontal-ethmoidal complex fractures" (Williamson *et al.,* 1981).

The establishment of multidisciplinary craniofacial units with a large volume of trauma patients has expanded the general understanding of fractures in anatomical regions which were previously managed by separate surgical units (Jones *et al.,* 1977). Recent years have also witnessed the introduction of high-resolution CT scanning which provides accurate delineation of fractures in the craniofacial region (Cooper *et al.,* 1983; Gentry *et al.,* 1983a, b). Also the facility for generating three-dimensional image reconstructions from CT data has further broadened our knowledge of disrupted craniofacial form (Hemmy *et al.,* 1983). Such improved methods of diagnostic imaging and the wide surgical exposure currently employed for fracture repair have both provided a clearer insight into the variability and extent of traumatic craniofacial disruption.

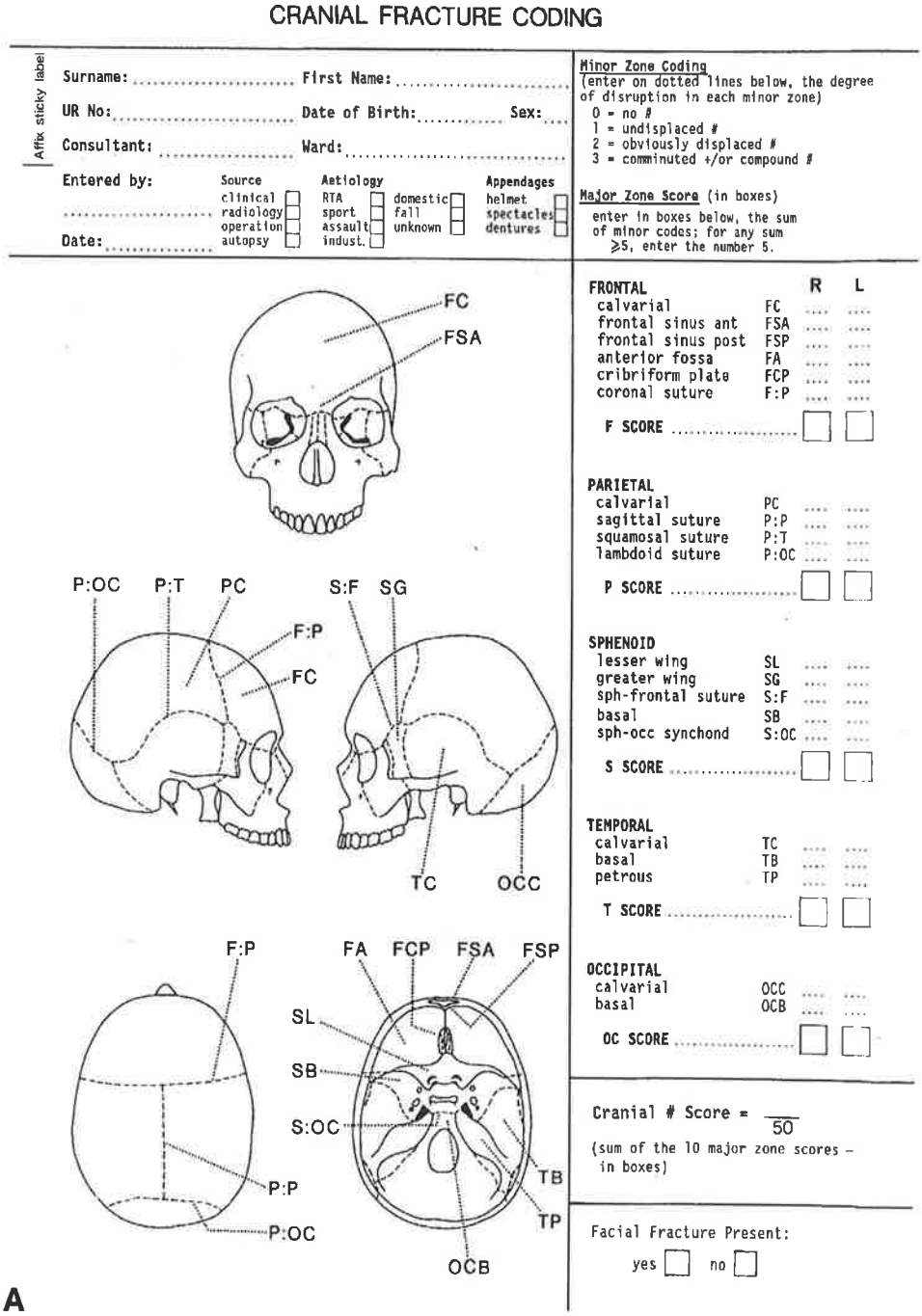


FIG. 1. A. *Cranial and* **B.** *Facial fracture coding forms.*

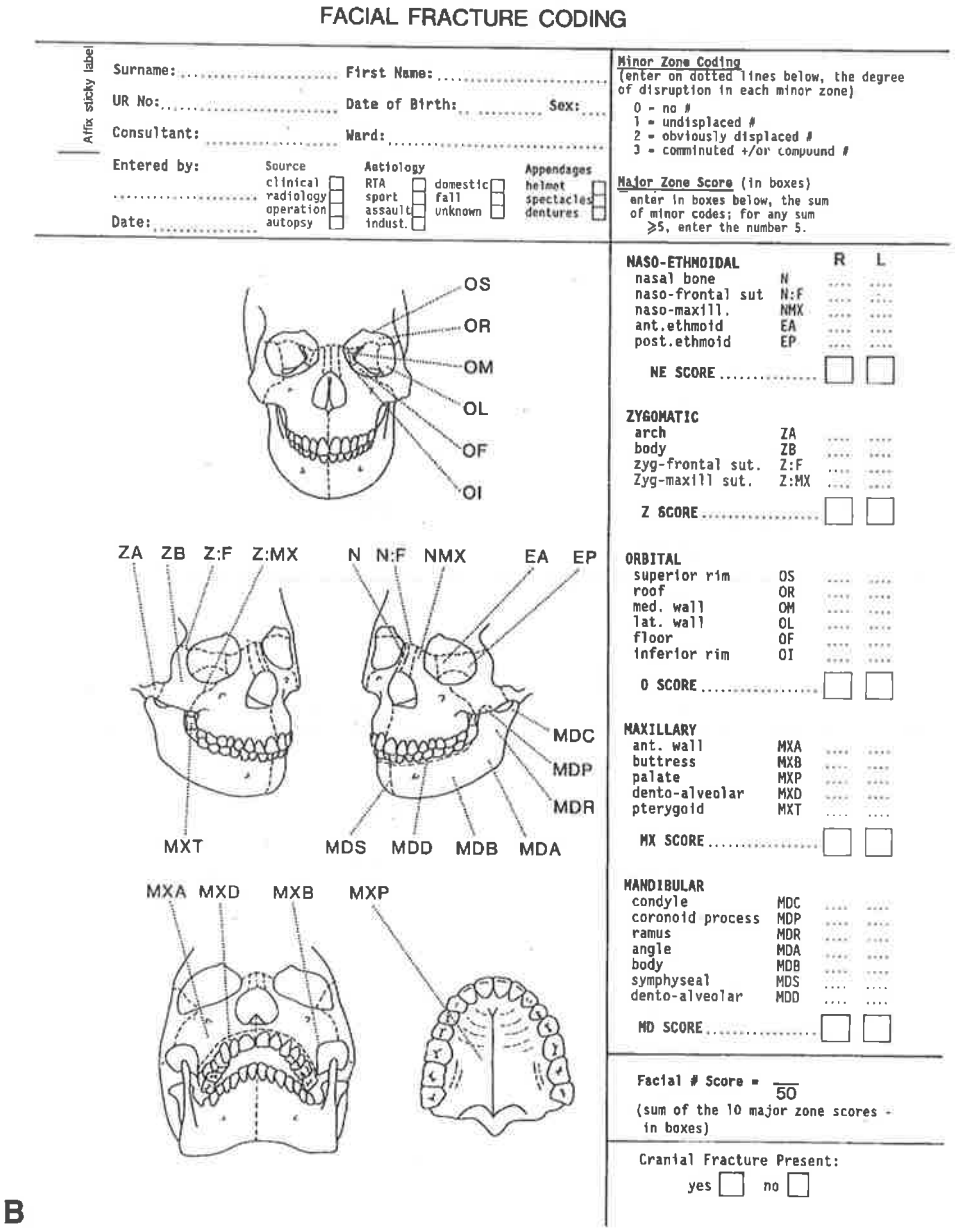


FIG. 1. A. *Cranial and* **B.** *Facial fracture coding forms.*

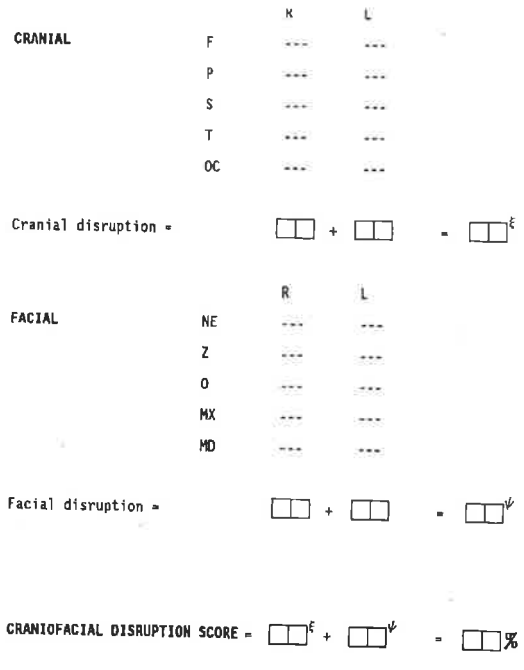


FIG. 2. — *The Cranio-facial Disruption Score is the sum of the 20 major zone scores.*

From the above it was evident that the need existed for a system of fracture coding which could incorporate both cranial and facial regions because fractures may traverse both of these territories. An additional requirement for the documentation of fractures was a method of clearly identifying the stable bony elements to which flail segments could be secured. Furthermore, it seemed prudent to produce a coding method with inherent value to surgeons, radiologists and other clinicians involved in the interdisciplinary approach to fracture management, as well as being of benefit to research personnel who may require objective data about injury to the craniofacial region.

With this in mind, the alpha-numeric fracture coding system was developed in order to provide an accurate appraisal of the degree of bony disruption. The alpha-numeric system is considered to be complementary to some existing classifications. The Le Fort system, for example, is now accepted for the description of some osteotomy patterns and this will reinforce its continued use for *verbal communication* relating to fractures. However, the alpha-numeric coding system offers a succinct, but comprehensive, method of objective fracture *documentation.* In addition, the alphanumeric system requires that all bones be coded, thus ensuring that an observer makes a critical assessment of the total craniofacial skeleton. In this way, alpha-numeric coding provides an evaluation of both fracture location and fracture severity.

The anatomical zones included in this coding system were selected to provide a comprehensive representation of the craniofacial region with due regard for the common sites of fracture or diastasis. A few minor zones are represented twice; in particular, the orbital roof (OR) is contained within the anterior cranial fossa portion of the frontal bone (FA), the orbit's medial wall (OM) is formed by the anterior and posterior ethmoid air cells (EA, EP), and the orbit's lateral wall (OL) is duplicated within the body of zygoma (ZB) and the zygomatico-frontal suture (Z:F). Such overlap is deliberate as it provides an appropriate "weighting" to the bones forming the orbit as well as acting as an in-built double determinant of an encoder's consistency.

The naso-ethmoid zone includes the anterior and posterior ethmoid air cells, which have been labelled EA and EP respectively; such labelling is at variance with other minor zone codes which all incorporate their major zone's alphabetic label. This is to allow for isolated ethmoidal fracture, as in a medial orbital wall "blow-out" in the absence of nasal bone fracturing. The pterygoid plates, although actually part of the spheroid bone, are included with the maxilla,

as MXT, because they are most commonly fractured with maxillary trauma. Similarly, the frontal process of the maxilla is often fractured by impacts to the nasal region and it is therefore coded as NMX in the nasoethmoidal zone. Also the cribriform plate of the ethmoid which lies between the two orbital plates of the frontal bone has been included in the frontal zone.

Components of the skull base have been included in the four major zones: F, S, T and OC. The frontal zone contains the floor of the anterior cranial fossa, FA, on each side with the cribriform plate, FCP, intervening. The spheroidal zone contributes to the skull base with its lesser wing of spheroid, SL, and the basal portion of the greater wing, SB. As the junction of the spheroid and occipital bones, the spheno-occipital synchrondrosis is represented as S:OC. In the floor of the middle cranial fossa, in addition to SB, are two portions of the temporal bone including the basal squama, TB, and the petrous part, TP. The occipital region, OC, has been considered as the two minor zones OCC and OCB which represent the calvarial squamous and the basilar parts respectively. OCB is that part of the occipital bone which extends forwards and upwards from the foramen magnum.

An example is presented to highlight the difficulty of fracture description with existing classifications. Although the alpha-numeric coding system is equally suited to the coding of fractures of isolated bones, a case of complex fracturing was chosen to demonstrate this coding system's versatility:

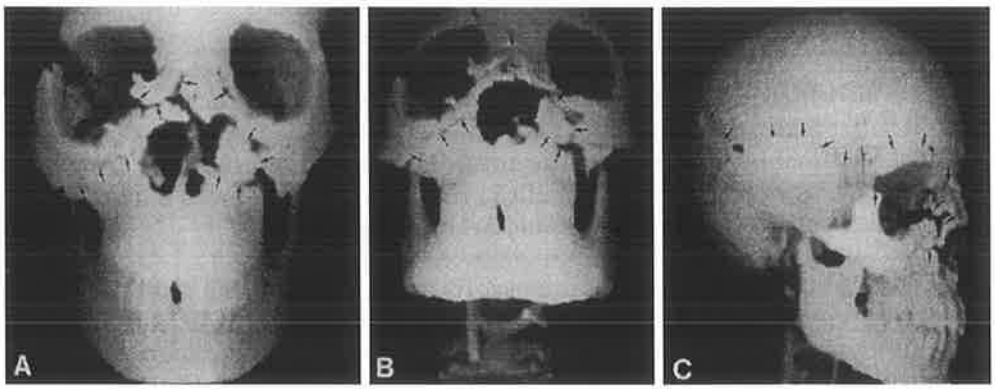


FIG. 3. *Three-dimensional CT reconstructions of a patient who sustained multiple fractures of the craniofacial region in a car crash.*

Case report

The left front seat passenger in a car that collided with a heavy vehicle sustained multiple fractures of the craniofacial region (Fig. 3).

Cranium

A fracture extended from the posterior aspect of the right parietal bone to the right supra-orbital rim. The squamosal and spheno-frontal sutures were diastased and the anterior cranial fossa was traversed by an obviously displaced fracture which radiated into the right lesser wing of spheroid.

Existing classification systems may label the anterior component of this cranial fracture:

*Escher Types III and IV

*Fain Type VI

*Tvete Type 4

Face

Comminution of the entire naso-ethmoid region was associated with craniofacial disjunction on the right with marked inferior displacement of the right zygoma

and a pyramidal-type extension on the left. There was a high horizontal fracture across the maxilla at the mid-level of the piriform aperture. An undisplaced mandibular symphyseal fracture extended downwards from an avulsed lower right incisor.

Existing fracture classifications may yield a series of diagnoses for these facial fractures:

*Le Fort I

*Hemi Le Fort II on left

*Hemi Le Fort III on right (strictly speaking, Le Fort described only bilateral fractures).

*Wassmund type IV

*Gruss naso-ethmoid-orbital type 3, plus type 4b on right side

*Stranc and Robertson plane 3 nasal injury

*Knight and North group V type zygomatic fracture on right side

The alpha-numeric coding of this patient's fractures is illustrated in Figure 4. The simple addition of the Cranial # Score (= 11/50) and the Facial # Score (= 38/50) produces this patient's Craniofacial Disruption Score of 49%. From the individual major zone numerical scores it can be readily appreciated that the principal contributing zones to this high level of craniofacial disruption include: NE, 0, and MX (Fig. 5).

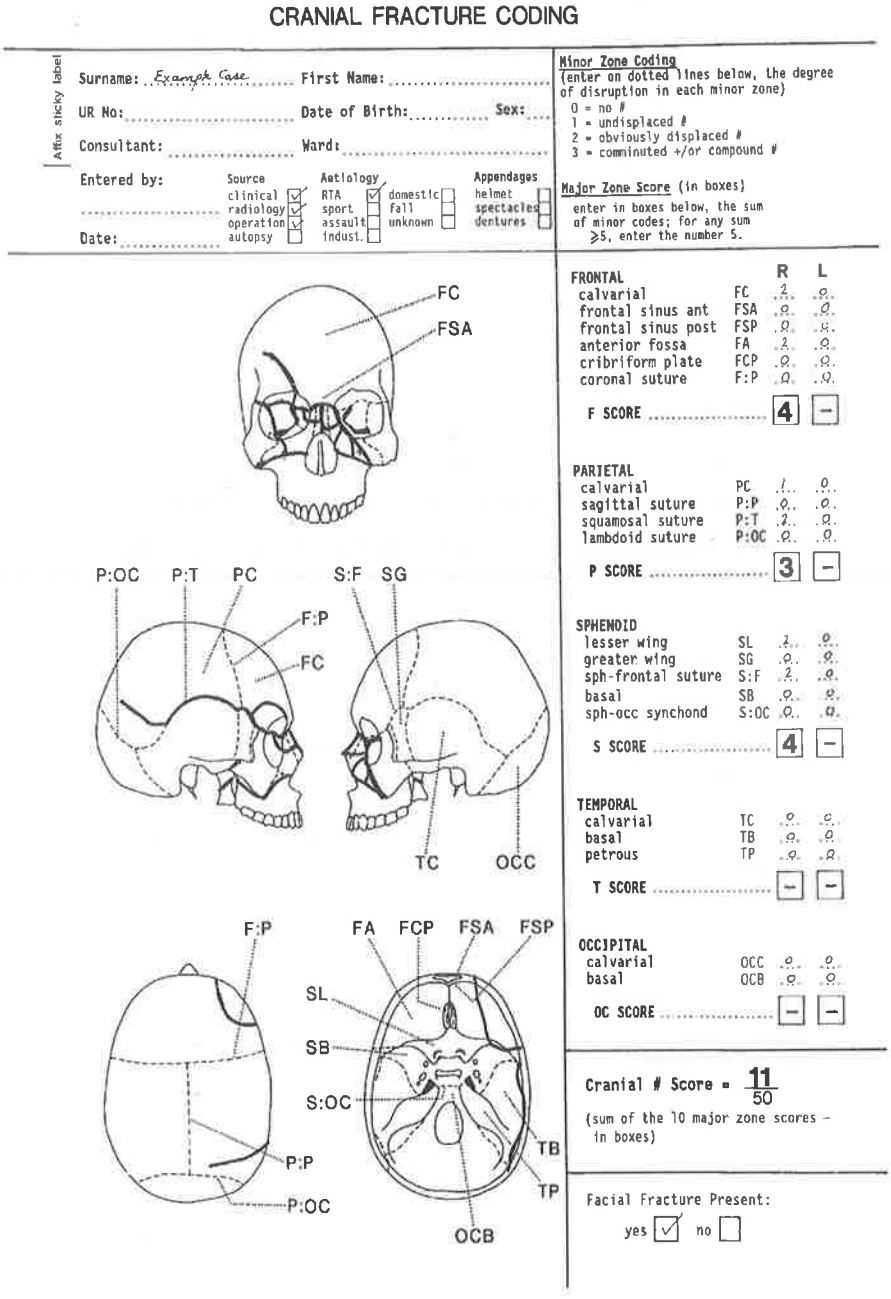
It is intended that the alpha-numeric coding of craniofacial bony disruption will contribute to existing methods of trauma assessment by providing simple and reproducible numerical data which are suitable for computer analysis.

Conclusion

The alpha-numeric coding technique is simple and provides objective data for both clinicians and research personnel and also offers an overall craniofacial disruption score (CDS) which is expressed as a percentage.

Acknowledgments

Financial assistance for this project was provided by a Royal Australasian College of Surgeons Foundation "Johnson and Johnson" Research Scholarship, the Neurosurgical Research Foundation of South Australia Inc., and the Australian Cranio–Maxillo–Facial Foundation.



A

FIG. 4. A. *Cranial and* **B.** *Facial fracture coding of the fractures seen in Figure 3.*

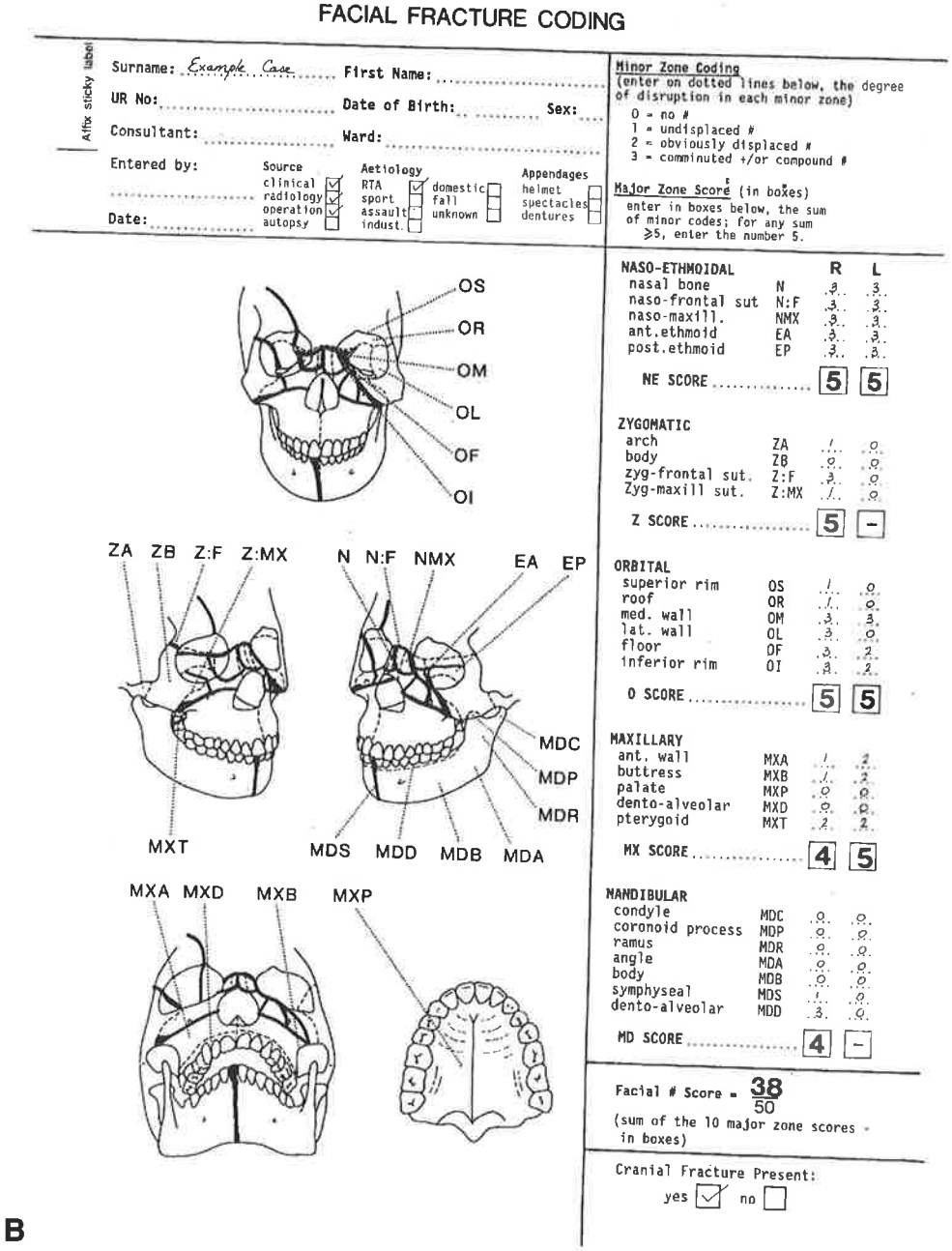


FIG. 4. A. *Cranial and* **B.** *Facial fracture coding of the fractures seen in Figure 3.*

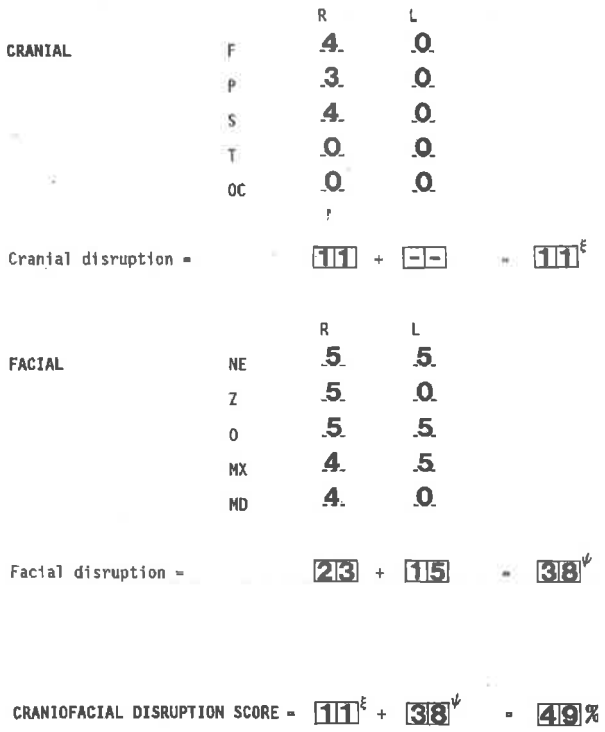


FIG. 5. *The Craniofacial Disruption Score calculated for the example in Figure 3.*

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Oblique Craniofacial Fractures in Children

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The protected childhood environment and the anatomy of the craniofacial skeleton largely protect children from experiencing facial fractures. However, when major trauma to the head and face is sustained, an oblique pattern of fractures has been observed, distinct from those common in adults and explicable in terms of the anatomic differences between the child's and the adult's head and face. This difference in pattern of facial fracturing is relevant in terms of the examination, investigation and treatment of the primary injury, and prevention of any subsequent facial growth disturbances.

Key Words: *Pediatric fractures, facial injuries, facial growth, cranial fractures*

Children account for less than 10 percent of the total number of facial fractures.[1,3,7] The protected childhood environment and specific anatomic features of the pediatric craniofacial skeleton combine to minimise the potential for such trauma in a child.

Although there are recognised anatomic mechanical differences between the child and the adult face and consequently, in the pattern of facial fractures, most authors do not take these into account. They treat both children and adults in the same way, thus not making the necessary alterations to fixation techniques in order to prevent disturbances of facial growth.

We present a series of severe pediatric craniofacial injuries that manifested patterns of fracturing at variance to those observed in adults. It is the objective of this review to examine the potential contributors to the patterns of facial fracturing observed in children in an endeavour to provide a more rational basis for their management.

METHOD

During the 3 year period of May 1985 to April 1988, 10 children between 2 and 12 years of age were treated for severe craniofacial fracturing in the South Australian Craniofacial Unit at the Adelaide Children's Hospital. Children who sustain severe fractures, involving more than one facial bone, routinely undergo computerised tomographic (CT) examination of the craniofacial skeleton, with the generation of three dimensional reformats, as part of their preoperative assessment. In children under the age of 7 years and those who are cerebrally irritated, the CT scan is performed under general anaesthesia. The preoperative clinical and radiographic examinations and the subsequent operative findings were documented with a computer-based fracture coding system.[2] This numerical coding system summates individual subregions of injury to produce a facial disruption index out of 50.

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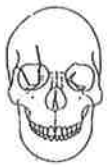
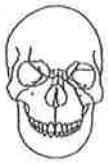
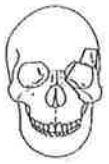
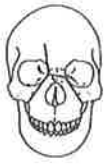
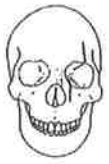
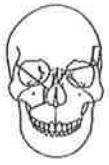
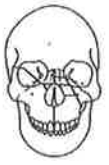
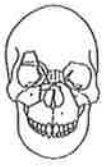
TABLE 1
Pediatric Craniofacial Fractures 1985-1988

Case	Age (yrs)	Mechanism	Facial Fracture Score	Fracture Pattern
1	2	MVA	7	
2	2	MVA	29	
3	3	CRUSH	30	
4	3	MVA	27	
5	3	MVA	18	
6	5	MVA	34	
7	6	Fall	9	
8	9	MVA	30	
9	12	MVA	31	
10	12	MVA	37	

MVA = Motor vehicle accident

RESULTS

The patterns of craniofacial fracturing in our patient group are recorded in Table 1.

Almost all of these pediatric craniofacial injuries were sustained in high velocity accidents, frequently involving the projection of the child from the confines of a motor vehicle. One child died from her extensive craniofacial and primary intracerebral injuries.

An obliquity of these fractures was consistently observed. Typically the fracture extended obliquely across the frontal bone, with a radiation to a variable extent into the posterior calvarium (Fig. 1). Inferior extensions of the frontal fracture involved an irregular disruption of the orbital roof and walls, often with extensions back to the orbital apex and greater and lesser wings of sphenoid (Fig.2).

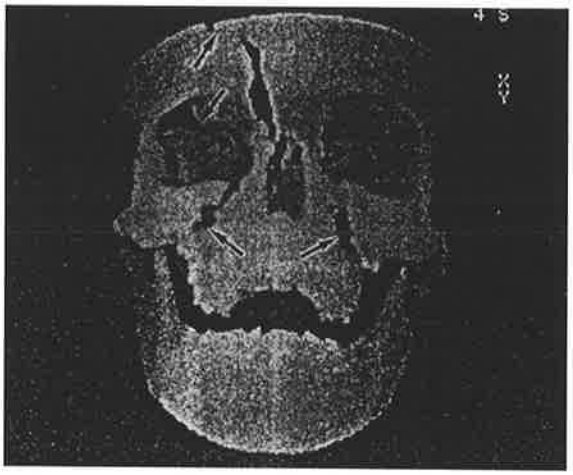


FIG. 1. *Extensive linear frontal fracture with superior extension toward the coronal suture and temporoparietal region, and inferior radiation onto the midface (see Table 1, Case 6)*

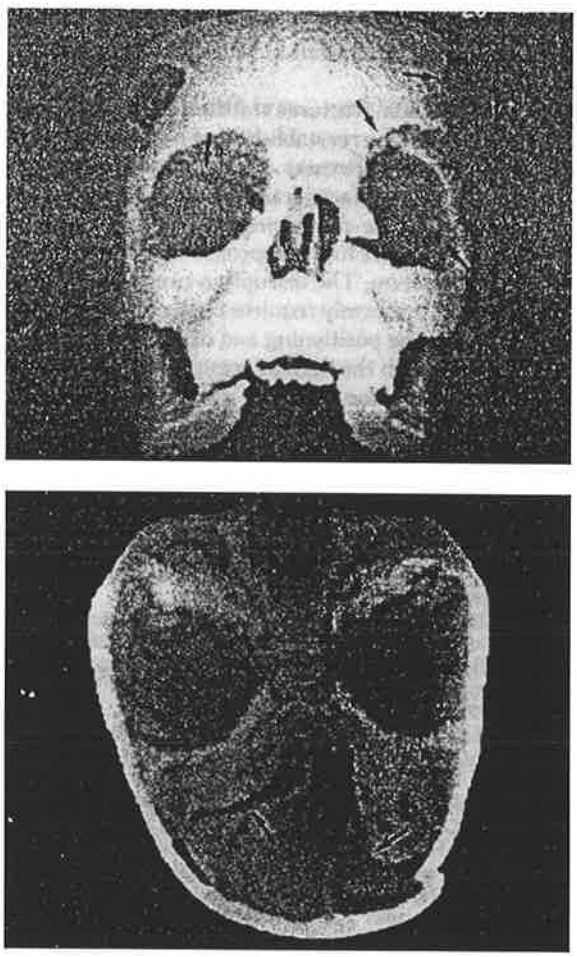


FIG. 2. Top, *Cross disruption of all the bony margins of the left orbit with a linear extension across the roof of the right orbit.* Bottom, *View of the anterior cranial fossa demonstrating the disruption to the roof of both orbits (see Table 1, Case 3).*

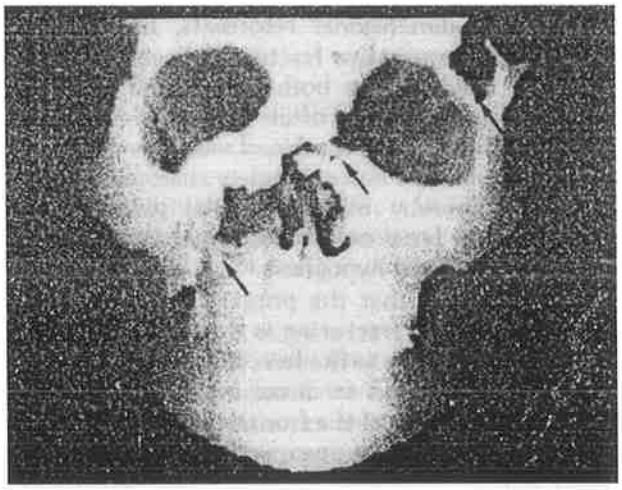


FIG. 3. *Compound comminuted oblique fracturing pattern from supraorbital margins above, converging on the nasal cavity and radiating below into the right midface (see Table 1, Case 8).*

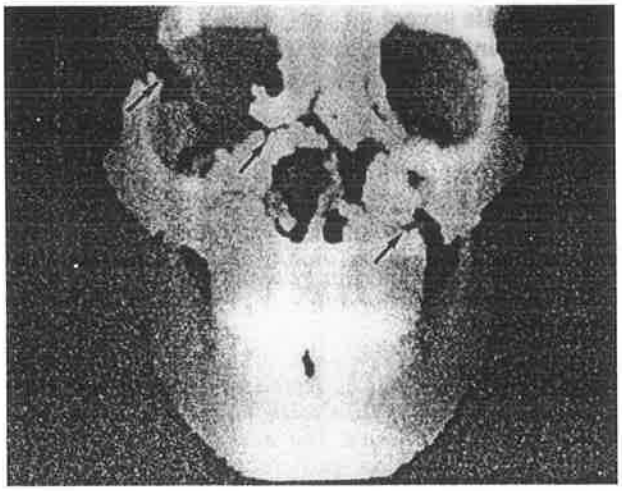


FIG. 4. *Inferior tilting and dislocation of the midface associated with oblique craniofacial fracturing (see Table 1, Case 9).*

The facial component of these fractures also displayed an obliquity across the orbit, orbital floor, medial orbital wall, and midface, with an infrequent extension to the mandible (Fig. 3).

Grossly comminuted fractures traversing the paranasal and medial orbital regions produced tilting of the midface and orbital floor, with an inferior prolapse or orbital contents, which led to enophthalmos and vertical orbital dystopia (Fig. 4). It was uncommon for the pattern of midfacial fracturing to follow the classical descriptions of Le Fort.[4]

Discussion

Due to the infrequent nature of pediatric facial fractures the assessment, documentation and treatment of such injuries has evolved in a relatively undisciplined manner. Consequently, despite the fact that the distinction between the pediatric and adult facial skeleton is well recorded and the alterations in surgical intervention are appropriately understood, approaches to fracture treatment in children continue to assume adult-like patterns of facial fracturing.[1,3,7]

The close clinical, radiologic, and operative examination of the severely traumatised pediatric craniofacial skeleton has demonstrated a consistent pattern of oblique fracturing that traverses the frontal bone, orbit, and midline nasal structures with radiation to the contralateral midface, but with relative sparing of the mandible. The application of CT scanning techniques including three-dimensional reformats, has allowed more detailed preoperative fracture delineation in the traumatised child, where both clinical and standard radiologic examination are often notoriously difficult. The improved radiology, combined

with a wide operative exposure, makes a more complete anatomic restitution possible thereby minimising the potential for persistent or late facial deformity, in particular orbital dystopia and midfacial hypoplasia.[8]

It is postulated that the primary determinant of this pattern of facial fracturing is the anatomy of the pediatric face.[1] Relative to the face, the cranium is more prominent and exposed to direct injury in the child. Thus, the involvement of the frontal bone by linear and depressed fractures is not unexpected.

A child's facial skeleton has relatively more cancellous and less cortical bone than an adult's and is less mineralised. This, in combination with less paranasal sinus pneumatisation, mixed dentition, and unerupted teeth, makes the midface more elastic, stable, and thus resistant to fracturing. More particularly, the structural pillars or buttresses that determine the classical patterns of adult midfacial fracturing (eg., disjunction of the midface in a horizontal plane) have not yet fully developed in the child.[6] Furthermore, the maxilla is not weakened by the extensive pneumatisation seen in the adult.

This may explain why the observed pattern of midfacial fractures in this pediatric group radiated in an oblique fashion from the supraorbital rim above, through the soft tissue filled orbit, to the relatively weakened nasomaxillary region and hence into the bony pyriform margin of the nose — the primary airfilled structure in the pediatric midface. Variable extension into the adjacent maxilla, and further into the mandible, occurred in those cases in which the impact force was quite severe.

Documenting the pattern and extent of these severe craniofacial fractures provides a basis for their accurate and complete primary anatomic restitution and fixation with the minimisation of late facial growth disturbance.[8]

In adult midfacial fractures stabilisation techniques are directed toward the reestablishment of the vertically orientated pillars or buttresses. By contrast the oblique pattern of pediatric midfacial fracturing, in concert with the prominence of the developing dentition, demands another level of finesse in the approach to the mechanics of internal fixation. The disruption caused by these fractures almost uniformly requires open reduction and internal fixation. The disruption caused by these fractures almost uniformly requires open reduction and internal fixation, the positioning and orientation of this being tailored to both the need to preserve the unerupted permanent dentition and the obliquely dislocated craniofacial skeleton.

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Fractures of the Anterior Cranial Fossa: The Craniofacial Approach

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During the years 1954-1988, the neurosurgical staff of the Royal Adelaide and Adelaide Children's Hospitals treated 238 cases of anterior fossa fractures involving the accessory need sinuses. Complications included cerebrospinal fluid (CSF) rhinorrhoea (47.5%), aerocele (25.2%), meningitis (13.1%) and brain abscess (2.5%). Strategies of management changed over this period from an initial policy of routine subfrontal exploration in all suspected cerebrospinal fluid fistula, to a selective policy of exploration in cases of prolonged leakage, fractures with bony separation, or intracranial infection. This selective policy demands precise radiographic examination of the skull base. There were associated fractures of the facial skeleton in 89 patients (37.4%). Initially these facial fractures were treated separately; during the last 12 years, the development of craniofacial surgery has permitted combined one stage repairs, usually carried out electively as soon as the cerebral swelling has subsided.

These patients often have serious long-term disabilities, and may need interdisciplinary rehabilitation.

KEY WORDS: *skull fractures—facial injuries—brain injuries—meningitis—brain abscess—cerebrospinal rhinorrhea*

Road crashes often injure the forehead and face. In head-on car crashes, the impacting object is often the steering wheel, dashboard, windscreen, or roof pillar.

Motor cyclists may run head-on or face-on into a vehicle or some other rigid structure, and their helmets may not save them. Both in civil and military conflicts, and in suicidal attempts, the face and forehead may be the target of a bullet, and industrial accidents may also throw solid objects into the face.

Central frontal impacts may shatter the strong frontal bone, and may also buckle and fracture the much thinner bones of the anterior cranial fossa in the midline (Fig. 1). Off-centre frontal impacts may have similar effects on the side of impact, buckling the orbital roof and sending fracture lines radiating across the anterior fossa. When the impact point is lower, on the nose or cheek bones, the facial skeleton may suffer, and a Le Fort type II or III fracture may absorb much of the impact energy, but there can nevertheless be involvement of the anterior fossa in the ethmoid region (Fig. 2). Gunshots, whether suicidal, criminal, or accidental, may disrupt the anterior cranial fossa in ways that are quite unpredictable (Fig. 3).

These injuries may have serious consequences. Damage to the frontal lobes may be lethal, or may cause permanent psychiatric disabilities. Damage to the optic nerve in the optic foramen, or to the globe of the eye, may cause blindness. Damage to the olfactory bulb or tract may cause anosmia. Damage to the carotid artery within the cavernous sinus may result in a carotid cavernous fistula, which causes proptosis and a bruit, and may lead to blindness. Finally, fractures of the

anterior cranial fossa may tear the dura mater and open a communication into the frontal, ethmoid or spheroid air sinuses, or into the nasopharynx. These internally compound fractures may establish a cranionasal fistula. The most obvious signs of such a fistula are the escape of CSF from the nose, or the entry of air into the cranial cavity, whether into the subdural or subarachnoid space, or into the brain itself, presumably into an area of damaged brain, through a valvular track, to expand as an aerocele. More seriously, a cranionasal fistula may result in meningitis or brain abscess[1]. To prevent these very dangerous complications, neurosurgeons have, since the pioneering work of Dandy in 1944[2], devised standardised plans of management. These plans of management have been reviewed in the light of neurosurgical and craniofacial experience in Adelaide, South Australia.

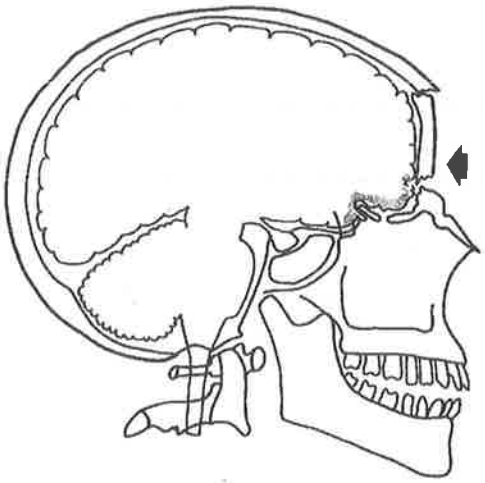

FIG. 1. *Disruption of anterior fossa by a central frontal impact*

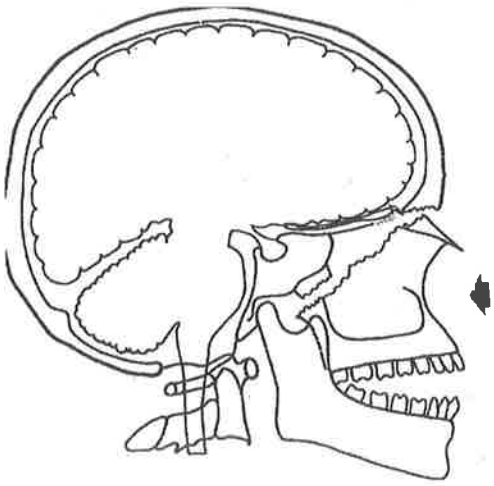

FIG. 2. *Facial impact causing Le Fort II or III fracture and damage to the ethmoid bone.*

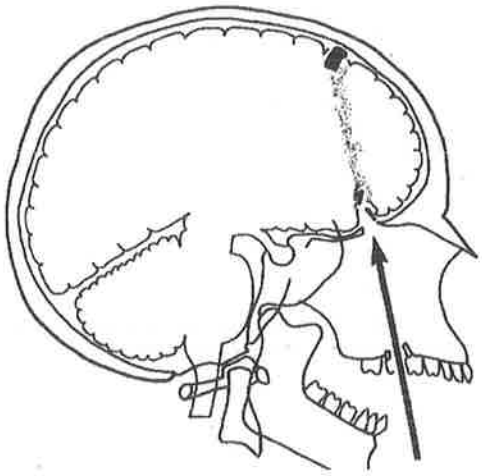

FIG. 3. *Gunshot wound involving anterior cranial fossa.*

Patients and methods

During the period 1954-1988 the neurosurgical units of the Royal Adelaide and Adelaide Children's Hospital treated 238 cases of fractures of the anterior cranial fossa involving the accessory nasal air sinuses. Most of these were patients referred for neurosurgical management soon after injury; however, 19 patients presented with late complications of injuries sustained from 6 weeks to 27 years earlier. Case summaries were examined, with special attention to the complications of the injury and the outcome. Operation notes were studied to determine the strategies of management as these evolved over the 35 years covered by the survey.

Results

Age and Sex

The age incidence is set out in Fig. 4. There were 204 males and 34 females.

Cranial Fractures

There were 32 compound frontal fractures associated with external skin wounds; in most of these, the frontal air sinuses were directly involved. In 114 cases, there were fractures of the skull base (frontobasal fractures), involving the ethmoid and/or other paranasal air sinuses, without external wounds. In 73 cases, there were both external wounds and frontobasal fractures involving the paranasal sinuses. There were also 10 cases of gunshot or other missile wounds involving the anterior cranial fossa. In the remaining 9 cases, full classification of the cranial injury was not possible.

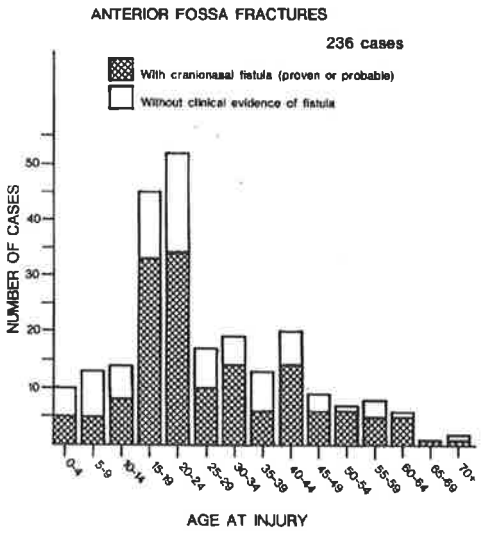

FIG. 4. *Age incidence of anterior fossa fractures with and without clinical evidence of cranionasal fistula.*

Facial Fractures

In 89 (37.4%) cases, there were associated fractures of the facial skeleton (Table 1). The data were inadequate in many cases seen before 1974, and it has not been possible to classify the facial fractures exactly: however, most of the 50 maxillary fractures were of the Le Fort type, chiefly types II and/or III.

TABLE 1

Facial fractures associated with anterior fractures in 238 cases. Figures in brackets indicate cases listed as another fracture type

Principal facial fractures	Number of patients
Naso-ethmoidal	4
Zygomatic	10
Orbital	
Floor (blow-out type)	3 (3)
Other	13
Maxillary (Le Fort I-III etc.)	50
Mandibular	2 (12)
Other	7
Total	89

Cranionasal Fistulae

In 158 (66.4%) cases, there was evidence of a cranionasal fistula at some period, as witnessed by CSF rhinorrhea, X-ray evidence of aerocele, intracranial infection, the escape of brain tissue through the nose, or some combination of these signs (Table 2).

The sites of the fistulae were not always ascertained: even at operation, it was not always certain whether the fistula entered the posterior part of the frontal sinus, or an anterior ethmoid air cell. Table 3 sets out the locations as determined from operation notes in 117 cases: in the cases classed as having multiple fistula sites, the ethmoid air cells were thought to have been involved in all cases, and the spheroid sinus in 6. The single case of fistula into a maxillary air sinus was a gunshot wound involving also the middle cranial fossa.

TABLE 2

Manifestations of cranionasal fistulae in 238 cases of anterior fossa fractures

	Number of patients	As % series
CSF rhinorrhoea		
Alone	81	34.0
With aerocele	28	11.8
With extruded brain	4	1.7
Total no. of patients	113	47.5
Intradural aerocele		
Alone	32	13.4
With CSF rhinorrhoea	28	11.8
Total no. of patients	60	25.2
Brain tissue in nostrils		
Alone	2	0.8
With CSF rhinorrhoea	4	1.7
Total no. of patients	6	2.5
Meningitis and/or brain abscess		
Alone	11	4.6
With CSF rhinorrhoea and/or aerocele or extruded brain	26	11.0
Total no. of patients	37	15.6

TABLE 3

Sites of cranionasal fistulae, as determined from operation, autopsy, or X-ray findings

Air sinus	Number of patients	
	Definite	Possible
Ethmoid alone	58	8
Frontal alone	17	3
Sphenoid alone	5	4
Maxillary	1	—
Multiple sites (chiefly ethmoid)	21	—
Total	**102**	**15**

Intracranial Infections

The types of intracranial infection are set out in Table 4, with the infecting organisms. The time of onset of infection varied: in 9 cases it occurred within 4 weeks of injury (one child developed fulminating meningitis within 24 hours), in 2 the interval is uncertain, and in 25 infections developed at later periods. One woman injured in the Second World War suffered 3 attacks of pneumococcal meningitis 10, 14, and 15 years later[3]. There were 2 deaths from meningitis and one from brain abscess; at least one patient suffered additional brain damage from pneumococcal meningitis.

TABLE 4

Meningitis and bran abscess complicating anterior fossa fractures

Organism	Meningitis	Meningitis abscess	Cerebral abscess
Streptococcus pneumoniae	22 (1 died)	1	—
Other streptococci	2	—	—
Neisseria meningitidis	1	—	—
Haemophilus influenzae	3	—	—
Staphylococcus aureus	—	—	2
Streptococcus + bacteroides	—	—	1
Pseudomonas species	1	—	1
Enterobacter	1	—	—
"Mixed"	—	—	1 (died)
Unknown	1 (died)	—	—
Total	31	1	5

Operations

Twenty-six patients underwent no definitive neurosurgical operations, either because there was no indication, or because of poor condition. Five of these died, 3 from complications of cranionasal fistulae. Dural repair was carried out in 168 cases, with or without other procedures such as closure of an externally compound fracture; 33 cases underwent only closure of externally compound fractures, and the remainder underwent various neurosurgical procedures appropriate to other complications (chiefly intracranial haemorrhage). There were 2 postoperative deaths. Recurrent CSF leaks were reported in 7 patients (3.5% operated cases) supposed to have undergone adequate operative repair, one of whom refused further operation and later died of fulminating pneumococcal meningitis. Two other operated patients also later developed serious intracranial infections, and there were 12 cases of sinusitis, osteomyelitis and other local infections.

Outcome

This was graded by the Glasgow outcome score[4]: there were 99 (41.6%) good recoveries, 38 (16.0%) recoveries with moderate disability, 17 (7.1%) recoveries with severe disability, and 7 (2.9%) early deaths. In 77 (32.4%) cases, disability was not accurately graded. Among the cases with moderate or severe disabilities, there were 21 with visual impairment, 4 being totally blind.

Discussion

Age and Sex

Cranionasal fistulae are less common in childhood (Fig. 4). Young children and infants are less likely to suffer impacts to the face and forehead than adults. Moreover, the development of the paranasal air sinuses is a progressive post-natal process; at birth, only the nasopharynx is pneumatised, and although the ethmoid air cells develop rapidly during the first 2 years of life, the frontal air sinuses appear somewhat later, and the sphenoid sinuses later still, expansion continuing at least until puberty[5]. Nevertheless, this relative immunity of the young should not be exaggerated: there were 37 cases (15.6%) of fractures involving the paranasal sinuses in the age group 0-14. The infant's ethmoids are well aerated by 3 months, and we have had to operate for CSF leaks sustained at the ages of 14 months and 2 years—one from a steel drill poked up the nose, and the other from a blunt frontal impact by a horse's hoof.

The high male-female ratio (6:1) presumably expresses the greater liability of men, as car-drivers, and in various dangerous occupations, to suffer frontal or facial impacts. However, women tend to have smaller air sinuses relative to the size of the skull, and this may also be a factor.

Management of Externally Compound Fractures

There is little controversy over the management of externally compound injuries involving the frontal air sinuses. Operation is undertaken as soon as resuscitation is complete. The fracture is explored, either through the wound or through a concealed coronal scalp incision. The dural tear is exposed and the brain wound is explored: foreign bodies, clot and necrotic brain are removed as gently as possible, and the dural tear is closed, often with a pericranial graft. The frontal sinus injury is inspected: if the sinus is shattered irreparably, we remove the posterior wall and erase the sinus mucosa. We have not inserted a tube into the frontonasal duct, as advocated by Raveh et al, 1984[6]: indeed, if the exenteration is complete, we have sometimes plugged the duct with muscle. We believe that this management will usually result in obliteration of the frontal sinus, and in this series we have seen only one case of delayed frontal mucocele in a patient treated thus. It must however be admitted that some cases of delayed extradural

infection in our series may have resulted from poor sinus drainage. The anterior wall of the frontal sinus is preserved; loose bone fragments are put into normal position and held there by wires or small plates.

Management of Inernally Compound Frontobasal Fractures

The management of these fractures is more debatable. There is no question that frontobasal fractures may be complicated by intracranial infection, early or late (Table 2). These infections result from incomplete healing of a meningocerebral hernia through a dural tear into an air sinus: the thin bone over the air sinuses heals poorly, and infection can develop many years after the causative injury. Sir Hugh Cairns and his colleagues in Oxford saw many cases of delayed intracranial infection during and after the Second World War: they emphasised the importance of CSF rhinorrhoea as a warning sign of a cranionasal fistula, and advised transcranial exploration and operative repair in all cases of CSF leaks, however transient. Lewin, 1966[7], reported on 55 operated cases, with one delayed infection and no deaths, compared with 26 unoperated cases of whom 6 developed delayed infection and 4 died. This philosophy of preventive exploration after CSF rhinorrhoea was widely accepted, and was the basis of management in Adelaide for many years. Our indications for operation have, however, been modified significantly by experience, both our own and that reported from other centres.

When we first reported on experiences in Adelaide[3], we gave equal importance to CSF rhinorrhoea and aerocele as indications for anterior fossa repair[8], and we also suggested that there might be radiological indications for such operations even in cases without rhinorrhoea or aerocele, namely fractures involving an air sinus with wide separation, bone defect, or a tilted bone spicule. There is now much evidence to suggest that the significance of transient CSF rhinorrhoea has been overemphasised. The evidence has been well reviewed by Loew et al, 1984[9], who conclude that post-traumatic CSF rhinorrhoea can be treated conservatively if it ceases spontaneously in one week, if there is no visible fracture in the anterior fossa, or only a small linear fracture, and no anosmia. We endorse this view, and indeed would not give decisive importance to the presence or absence of olfaction though this should always be noted. Operative treatment is mandatory if CSF leakage persists for more than one week, or recurs thereafter, or if there is a large aerocele. Operative treatment is certainly indicated if there is a previous history of meningitis, likely to be due to a cranionasal fistula, and is also indicated if there is radiological evidence of a fracture with separation of bone edges and/or a tilted bone spicule. If these criteria had been always respected, some of the intracranial infections recorded in our series might have been avoided.

Conservative treatment, in our view, should include antibiotics. There have been at least 5 published trials of the value of antibiotics in preventing infection in skull base fractures, and none is entirely satisfactory: however, the Glasgow study by Leech, 1974[10], did appear to show a protective benefit. The choice of antibiotic is obviously important: we favour Septrin, a trimethoprim sulphonamide combination, which crosses the blood-CSF barrier, and ampicillin or amoxycillin, which do not, but will penetrate to the nasal mucosa. Medication is continued for 7-10 days after injury. It has been argued that the routine use of antibiotics will promote the appearance of resistant organisms[11]. Review of our own experience does not suggest that this is a serious problem: our series includes 3 cases of infection with gram negative organisms where previous chemotherapy may have been responsible, but all responded well to appropriate treatment with specific antibiotics.

Although the broad indications for operation are now widely accepted, there are still problems in individual cases. There may be doubt as to whether a nasal leakage really is CSF. A profuse drip of watery fluid is unmistakeable. Chemical tests for glucose mean very little, since serum and nasal mucus[12] may react

positively. Immunochemical tests for proteins specific to CSF, (beta-trace-protein and transferrin) may be more useful[13], and we are at present studying the value of transferrin estimation. Intrathecal injections of radioactive agents can be used to determine whether a leak is indeed CSF, and if so where is the fistula. This is sometimes most useful, as in a case of ours with CSF rhinorrhoea from a fistula into the middle ear[14]. But if the leak temporarily ceases, then the test is useless. The mainstay of pre-operative diagnosis is radiology: plain radiographs and tomograms to visualise the skull base, supplemented in selected cases by intrathecal injection of metrizamide to visualise the basal cisterns and perhaps the fistula itself[15].

The timing of operation is important. When the brain is swollen, subfrontal exploration is difficult, and may damage the already traumatised brain. Loew et al, 1984[9], reported the remarkably high mortality of 25% after operations in the first few days, with a fall to 3% when operations were postponed for about 3 weeks. In selected cases, and with good anaesthesia, earlier operation is often possible, but we have seen very few cases of meningitis when operation has been electively postponed.

Most neurosurgeons use a bicoronal scalp flap, and a unilateral or bilateral bone flap, depending on the need for bilateral exploration[7,9]. If the frontal bone is already fractured, then the bone flap is modified accordingly. With a slack brain, a small dural incision is adequate, and it is rarely necessary to divide the falx. Olfaction is preserved wherever possible. Adhesions, which are often very vascular, are dissected away from the floor of the anterior fossa, and the fistula is located—there are often several possible sites. The fistula is packed with muscle, or with a piece of bone if the defect is very large. The whole area is then covered with a large sheet of temporalis fascia or pericranium; occasionally, fascia lata is needed. We secure the graft with a few silk or absorbable sutures; some surgeons prefer fibrin glue and others rely on the post-operative expansion of the brain to hold the fascial graft in place. The dura is closed in watertight fashion, and the bone flap is replaced. Burrholes and small bone defects are filled with bone dust. Sometimes there needs to be a final reconstitution of a shattered or deformed orbital margin, with wires, plates, or bone grafts.

In general, transcranial duraplasty is effective. We have seen recurrent CSF leaks in 7 cases and recurrent intracranial infections in 3. Of the latter, one may have resulted from an extradural abscess spreading internally, rather than from an inadequate repair. Recurrent leaks have been due chiefly to failure to locate the chief fistula: contributory causes included failure to explore bilaterally and undertaking repair when intracranial pressure was elevated. In the 3 cases where recurrent infection occurred, there was a time delay of 4 years: a long follow-up is needed in the evaluation of treatment policies.

We feel in general satisfied with the transcranial repair of ethmoid and frontal fistulae, which constitute the great majority in our series (Table 3). Sphenoid fistulae behind the tuberculum sellae are, however, less easily exposed, and placing a graft under the chiasma may risk damage to the pituitary stalk. It is for such fistulae that the trans-sphenoidal approach has merits. Loew et al, 1984[9], advocate this approach, packing the sinus with muscle and bone, and maintaining a low CSF pressure post-operatively by lumbar drainage. One case in our series was successfully treated in this way: the leakage was well visualised and has not recurred since operation. This approach seems appropriate if the fistula has been shown definitely to be into the spheroid sinus by X-ray, or if no fistula was found by transcranial exploration.

Management of Facial Injuries

For patients with associated anterior fossa and facial injuries (Table 1), the multidisciplinary approach of the modern craniofacial unit has been especially valuable[16]. When our series was begun, many maxillofacial surgeons, influenced

by the fine work of Rowe and Killey, 1955[17], immobilised maxillary fractures by interdental wiring and by external splints which precluded early neurosurgical intervention. It was believed that such immobilisation would in itself promote healing of anterior fossa fractures, despite assertions to the contrary by neurosurgeons. In Adelaide, during the period 1955-1975, it was usual for the initial maxillofacial fracture management to be undertaken by plastic surgeons, using the techniques of fixation then available: when the fractures were considered stable, a transcranial repair was done. In a typical case, this meant primary closure of an externally compound wound on day 1, maxillary fixation on day 6, and transcranial repair on day 24 or even later. As has been indicated, there was in this period an increasing readiness by neurosurgeons to accept that transient CSF leakages do not necessarily require dural repair, but there was also an awareness by maxillofacial surgeons that even the most rigid fixation of a Le Fort III fracture will not guarantee dural healing; our series includes several cases where leakage recurred after internal fixation.

The merits of the craniofacial approach have been evaluated in Adelaide in 30 combined operations. Multidisciplinary assessments give a far more accurate evaluation of the priorities. In a typical case, operation is performed 10-14 days after injury, when the facial fractures are still mobile and the cerebral swelling is subsiding. A preliminary tracheostomy is necessary when the jaws must be wired together, though the use of miniplates makes long-term intermaxillary wiring a rare event. The operation begins with alignment of the lower fractures by dental arch bars and a wafer, giving temporary intermaxillary fixation. The maxillary fractures are exposed by transbuccal and transconjunctival incisions. Then a standard coronal scalp flap is turned down and the orbits are exposed from above subpericranial dissection. The coronal scalp flap gives access to the nasal and zygomatic bones, and much of the maxilla. The anterior fossa is explored by a frontal craniotomy, and repaired appropriately: this is ideally done after the dissection of the facial skeleton but before the fixation, to ensure that exposure of the brain is as brief as possible. The facial fractures are reduced and definitively fixed with titanium miniplates—titanium is preferred because it does not interfere with subsequent CT scanning (Fig. 5). Bone grafts are often used, for orbital repair, rhinoplasty, or onlay reconstruction. If bone grafting is needed then calvarial bone can be obtained by splitting the bone flap.

These combined operations are time consuming. They demand skilled team work, including multidisciplinary planning, exact radiography, good anaesthesia and control of intracranial pressure. But they have some outstanding advantages: better aesthetic correction of facial deformities, shorter hospital stay, and easy interchange of ideas between specialists, making possible changes of operative strategy in the light of what is found.

An interesting alternative to this strategy has been reported by Raveh et al, 1984[6]. In their preferred protocol, patients with CSF rhinorrhoea undergo unilateral or bilateral transethmoid exploration, through the frontal, ethmoid and anterior spheroid air sinuses. Dural tears are located and patched with fascia lata applied externally, after the mid-face has been fixed by maxillomandibular splints and transconjunctival fixation of the orbital floor. Transcranial exploration is reserved for those cases with upward dislocation of bone fragments or intracranial haematomas needing evacuation. The merit of this strategy of repair by transethmoid exposure is the capacity to explore early, even when there is still cerebral swelling: the chief drawback appears to us to be the limited exposure and the need to repair extradurally. Nevertheless, it is clear that the method exemplifies the increased range of options made available by the multidisciplinary craniofacial team.

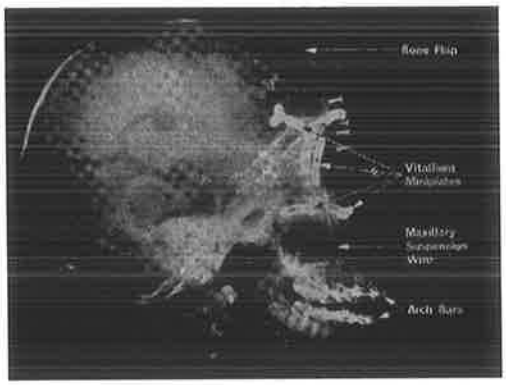

FIG. 5. *Radiography showing fixation after a combined craniofacial procedure for anterior fossa and facial fractures. The bone flap permitted dural repair; a temporal bone flap was also needed for an associated extradural clot. The facial fractures were fixed with small plates, arch bars and suspension wires: with increasing experience, suspension wires have been found unnecessary.*

Rehabilitation

The value of an integrated multidisciplinary team is also evident in the field of rehabilitation. The victims of craniofacial injuries often have very grave disabilities. Many of our cases have major changes in intellect and personality, reflecting frontal and less often temporal lobe damage. Visual loss may result from damage to the optic nerves or eyes—especially from shattered windscreens before the advent of safer glass. Skilled neurological and visual rehabilitation, combined with good aesthetic recovery from facial disfigurement, offers these unfortunate people, and their families, a better chance of social recovery.

Acknowledgement

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Fractures of the Forehead and Anterior Cranial Base

David J. David, F.R.C.S., F.R.A.C.S, and Mark H. Moore, F.R.A.C.S.

Despite modern trends toward improved safety consciousness and the evolution of means of protection of the craniofacial region, the upper third of the face and forehead remain uniquely exposed to high velocity impact. Advances in the resuscitation of such victims of complex acute trauma have produced significant improvements in immediate patient survival. Consequently, greater numbers of patients are now presenting for the assessment and management of complex combined injuries of the cranium and face. These patients require the application of the multidisciplinary team approach of reconstructive craniofacial surgery to the management of their injuries.[1-3]

Developed initially for the treatment of established craniofacial deformity, in all its manifestations, the improved techniques of clinical diagnosis, investigation, and surgical exposure, fracture reduction, fixation, and reconstruction are ideally applicable to the complex injuries of the forehead and anterior cranial base — this watershed area.

Pathogenesis/Classification

The interface between forehead, cranial base and midface is formed by the frontal, ethmoid, and spheroid bones centrally, and by the frontal, zygomatic, and spheroid bones laterally.

In the pediatric population the cranium is more prominent and exposed to direct injury, compared with the adult. Additionally, the bony architecture is less mineralised and has relatively more cancellous and less compact bone. Less extensive paranasal sinus pneumatization and the presence of unerupted teeth in the midface make the child's face more elastic, stable, and resistant to fracturing. Hence, when subjected to major impacts, the pattern of fracturing does not follow the classic descriptions of adult-type fractures, (Le Fort I, II, and III) but rather tends to produce oblique fracture patterns involving the forehead with extensions radiating into the orbits and midface[4] (Fig. 1).

By contrast, the adult craniofacial skeleton is characterised by the development of paranasal sinuses and the attendant production of regions of relative bony weakness and adjacent skeletal strength (buttresses).[5] Understanding these anatomic characteristics in concert with recognition of the velocity, direction, and transmission of impact to the craniofacial region establishes reproducible patterns of forehead, cranial base, and upper facial fracturing (Fig. 2).

Subclassification of these fractures is then possible according to the involvement of the central forehead, lateral forehead, or combined complex central and lateral forehead regions.[3,6,7] Logically, the latter occur in association with a more severe impact, with frequent extension posteriorly into the frontobasal region and inferiority into the lower face.

Although all such grading or classification according to skeletal area of involvement is preferable to a variety of eponymous labels, no provision for assessment of skeletal disruption is provided.[8] The advent of an alpha-numeric coding system of fractures of the craniofacial skeleton produces localisation of

fracturing, a level of quantification of bony disruption, and hence a means of accessing requirements for reconstruction.

Centrally directed impacts over the frontonasal region produce fractures involving the frontonaso maxillary and frontoethmoidal vomerine buttresses with extension into the frontal and ethmoid sinuses. The latter are associated with comminution and displacement of the medial orbital walls (Fig. 3).

The presence of well-pneumatized frontal sinuses, just as in the midface, absorbs much of the impact, with comminution of the anterior wall and the potential for preservation of an intact posterior wall. When the frontal sinus is small or undeveloped, as in the podiatric age group, impacted oblique linear and segmental fractures, with extension through the supraorbital rim to the orbital roof, occur more frequently.[4] The latter being a site of potential dural disruption, in addition to dural injury at the posterior wall of the frontal sinus.

Lateral impact at the level of the cranial base produces fracturing of the frontozygomaticomaxillary buttress with extension into the greater wing of spheroid, and when more severe, to the adjacent parietal and temporal bones (Fig. 1) Intracranial complications are less frequent, with the principal symptoms and signs relating to the inferior and lateral displacement of the orbital floor and lateral orbital wall (Fig. 4).

Extreme impacts may less frequently produce grossly comminuted fractures of both the central and lateral elements with an associated higher incidence of intracranial injury and dislocation of the face on the cranial base.

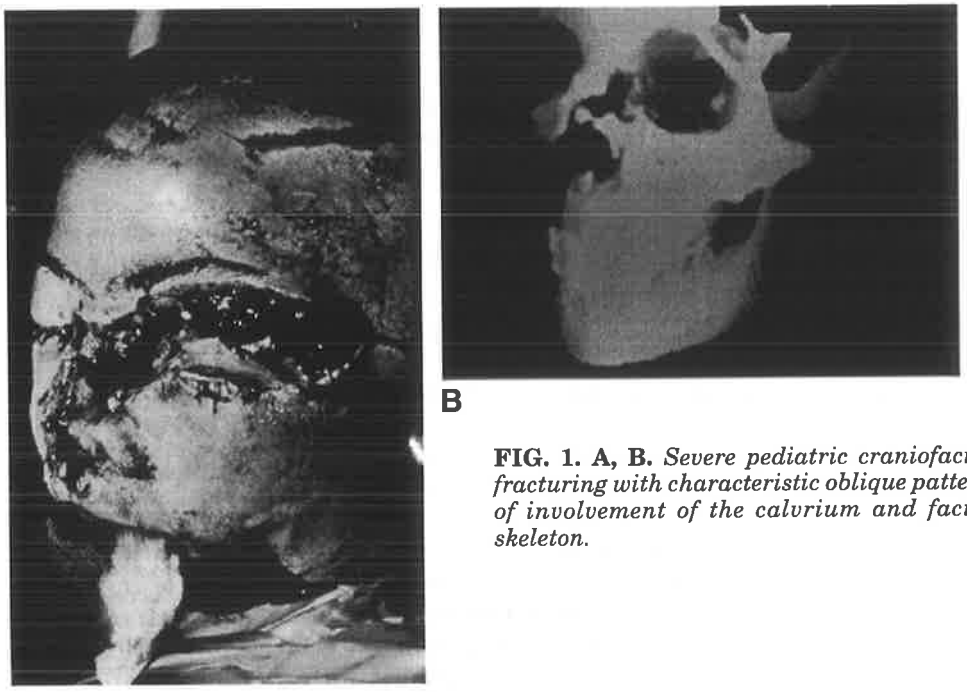


FIG. 1. A, B. *Severe pediatric craniofacial fracturing with characteristic oblique pattern of involvement of the calvrium and facial skeleton.*

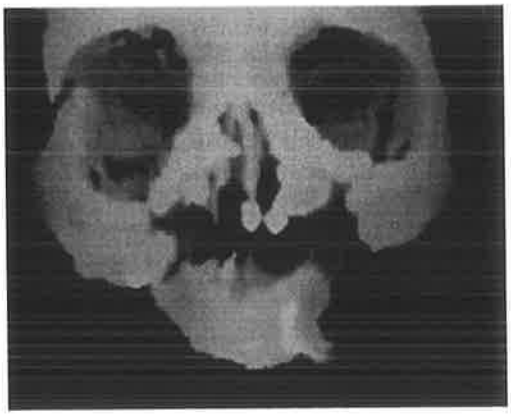


FIG. 2. *Major craniofacial fracture in adult, involving combinations of Le Fort I, II, and III*

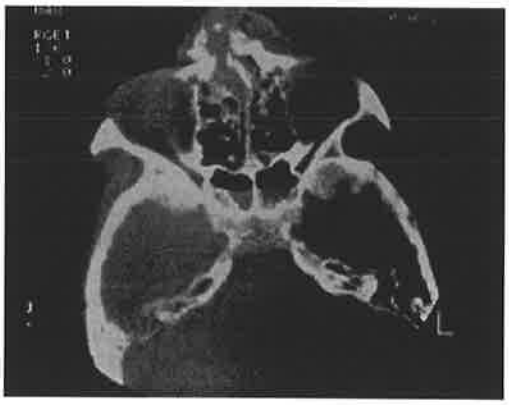
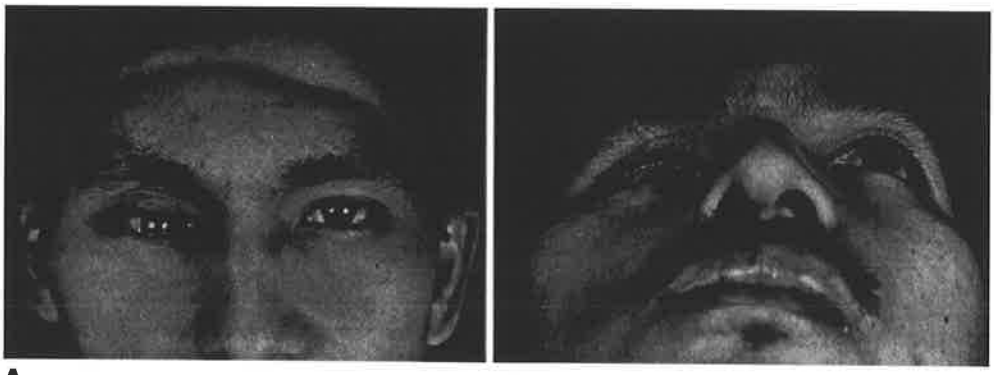
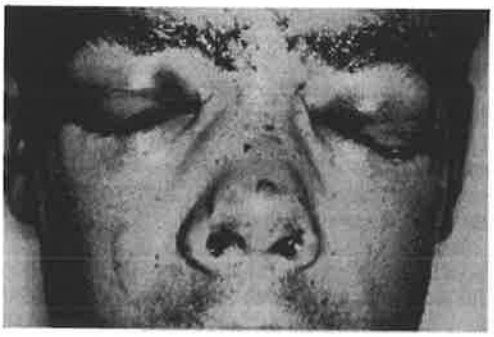


FIG. 3. *Central impact with disruption of the frontonasoethmoid region extending to involve the medial orbital wall.*



A B

FIG. 4. A, B. *Old inadequately treated major fractures of the right forehead and orbit with persistent forehead flattening, orbital dystopia, enophthalmos, and retropositioning of the zygoma*



A

B

C

FIG. 5. A. B. C: *Patterns of periorbital bruising, subconjunctival hemorrhages, and facial edema denote severe fractures of the anterior cranial base and periorbital region.*

Diagnosis

A complete and thorough clinical examination is mandatory. Included in these observations should be the patterns of facial bruising, position of the canthal ligaments, subconjunctival hemorrhages, eye movements, and the presence or absence of diplopia (Fig. 5).

Although the rapid onset of facial edema can obscure the clinical signs, careful palpation can outline the bony skeleton of the frontonaso-orbital region. Documenting sensory disturbances on the forehead and face, alterations in lacrimal drainage, nasal airway disturbance, and the presence of cerebrospinal fluid rhinorrhea completes the local clinical examination.

Since the degree of force associated with fractures of the forehead and anterior cranial base is significant, a thorough general clinical examination is essential, including a complete neurologic assessment.

Detailed radiologic assessment displays both the pattern and degree of displacement of fractures, often not appreciated by clinical examination in the presence of rapidly developing facial edema.

Routine plain radiographs provide an overview and may detail the pattern of linear fractures of the forehead (Caldwell view) or demonstrate any pneumocephalus overlying the frontal lobes (lateral views). However, little information is provided about injuries to and degrees of displacement of the inner table, posterior wall of the frontal sinus, and extensions into the orbital roof and cranial base.

Computerized tomography (CT) dramatically elevated the volume of information about patterns and displacement of frontal fractures.[7] Axial CT scans ideally demonstrate fractures of the anterior and posterior walls of the frontal sinus, taking care to scan sufficiently inferiorly to include views of the nasofrontal duct (Fig. 6). Direct coronal CT, or reformatted coronal and sagittal scans, provided detailed documentation of orbital roof and floor fractures, cribriform plate, and skull base disruptions. Three–dimensional CT reconstructions provide a qualitative visual overview, which aids in treatment planning and helps in the operating room environment to give a rapid graphic interpretation of fracture patterns and disruption (Fig. 2).

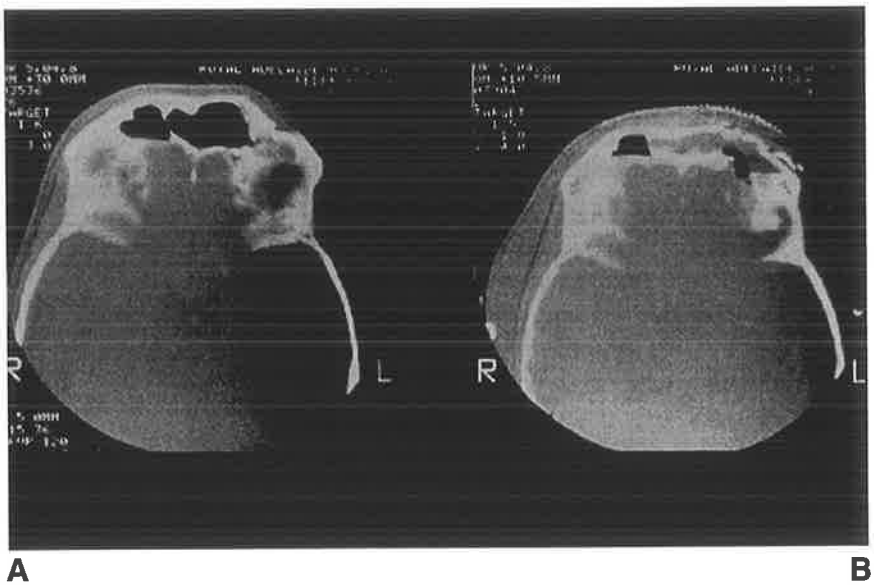


FIG. 6. A. *Axial computed tomography scans delineate frontal fracture with depression of supraorbital ridge.* **B.** *Postoperative appearance after elevation and internal fixation*

Management

General Philosophy

Technical advances in surgical management have kept pace with the rapid improvement in diagnostic, and in particular radiographic, techniques, so that the major step forward is more in terms of the general philosophy of treatment. The forehead and cranial base lying as they do at the junction between cranium and face, demand a coordinated, interactive and efficient approach by a number of surgical disciplines. Adequate pretreatment assessment should enable the surgical correction of the fractures and their associated sequelae, both intracranially and in the face, in a single major one-step surgical procedure. A coordinated plastic, maxillofacial, and neurosurgical team permits repair of dural, skeletal, and soft tissue disruptions in a stable patient in a single stage.

Such an approach both minimises the need for the technically difficult and complicated secondary or repeat dissections and explorations, and shortens the duration of hospitalisation. This complete multidisciplinary approach should also reduce the incidence of the late and frequently troublesome secondary sequelae that attend inadequate initial assessment and treatment (such as malunion, enophthalmos) (Fig. 4).

Inherent in the successful application of this approach are the technical facility to expose the involved regions widely, reduce adequately, and provide stable fixation of the identified fractures and appropriately reconstruct primarily those severely comminuted areas. These surgical techniques are now widely practiced and applied.

Surgical Techniques

The initial management of the patient presenting with frontal and cranial base fracturing involves routine clinical assessment and stabilisation. Specific investigation targeted at the involved area are instituted, enabling elective one-stage surgical correction of their injuries within 5 to 7 days of presentation. Earlier intervention is often difficult because of the attendant facial swelling, whereas delay beyond 2 weeks produces increased tissue adherence and a difficult dissection.

Surgical exposure is through a bicoronal scalp flap, taking care to protect the frontal branches of the facial nerve during flap elevation, and also being selective in the dissection of the temporalis muscle where lateral fractures are to be exposed. Temporalis atrophy from denervation and/or devascularization, after excessive dissection, can be a troublesome sequelae of the exposure.

Although the bicoronal exposure provides access to the forehead, upper orbits, and medial and lateral orbital walls, a range of lower eyelid, subciliary, and conjuctival incisions expose the lower orbit. Where the pattern of fracturing extends into the midface, this can be defined via an intraoral upper vestibular incision

Once the subperiosteal exposure of the fractures is completed, a frontal bone flap can be elevated to facilitate the required neurosurgical procedures. In the presence of midfacial fractures mobile at the Le Fort III level, placing the frontal bone flap more superiorly preserves a stable frontal bar onto which the midface is fixed.

After completion of the neurosurgical procedures, the identified fractures are reduced and internally fixed with miniplates (Fig. 7) The sequence of fixation being individual to each case and based on anatomic skeletal reconstruction built from the visible stable elements or buttresses.

In general, when cranial injury is minimal and facial injury severe, the facial skeleton can be built on the reconstituted cranial vault. When the cranial bony injury is major, initially the face is rebuilt in isolation, then reattached to the remaining stable cranium before it to is reconstituted.

When there are bony defects or severe comminution in either the face or cranium, rigidly fixed primary bone grafts provide the ideal means of repair. Split calvarium, either inner or outer table, split rib graft, and iliac bone provide a ready source of bone graft for reconstruction. Local pedicled soft tissue flaps (temporo-parietal fascial or galeal frontalis flaps) are available as a means of providing vascularized soft tissue to separate the cranial and nasal cavities (Fig. 8).

Certain aspects of frontal and cranial base fractures require separate discussion.

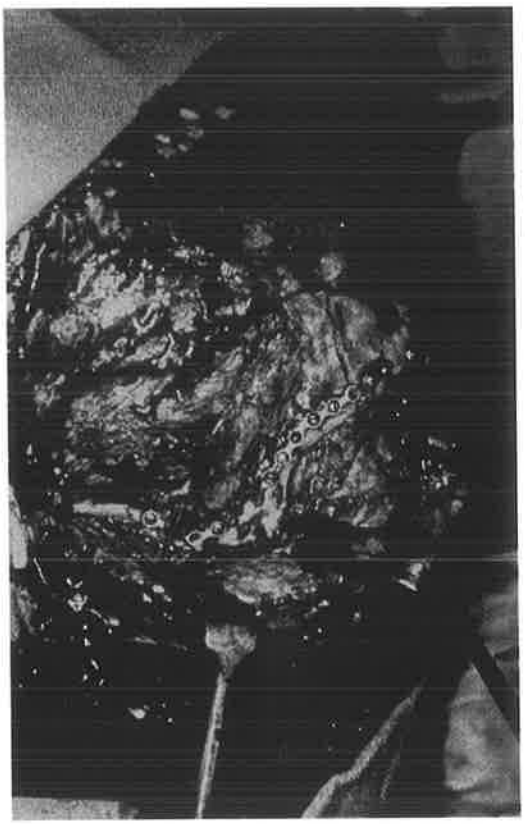


FIG. 7. *Bicoronal flap exposure of complex comminuted right fronto-orbital fractures after reduction and internal fixation of supraorbital ridge, frontozygomatic and zygomatic arch with miniplates.*

Frontal Sinus Fractures

The approach to treatment of fractures involving the frontal sinus depends on their position and degree of displacement, whether the fracture is simple or compound, and finally the presence of an associated cerebrospinal fluid leak.[3,7,9]

Anterior wall fractures, when not displaced, closed, and not involving the region of the nasofrontal duct, do not require surgical intervention. Open or displaced anterior wall fractures require exploration, debridement of damaged mucosa, and appropriate bony reconstitution.

Associated undisplaced posterior wall fractures do not require treatment, and only the anterior wall is reconstructed.

Gross comminution of the posterior wall is commonly attended by dural injury and here the posterior wall is removed to aid neurosurgical access, and bony reconstruction is confined to the anterior wall.

When the pattern of frontal sinus fracture extends inferiorly, the risk of nasofrontal duct obstruction, mucocoele, and sinusitis is increased. To minimise this and obliterate the potential for ascending infection from the nose, the frontal sinus and cranial cavity are ideally physically separated from the nasal cavity. Stripping the retained sinus mucosa, bone grafts, and vascularized galeal frontalis flap, in concert with cranializing the sinus (removing the posterior wall), most consistently accomplishes the separation of nose and cranial cavity.

Medial Orbital Wall Fractures

When fracturing of the cranial base is accompanied by marked posterior displacement of the facial skeleton, there is frequent involvement of the medial orbital wall with blowout into the adjacent ethmoid sinuses. In the acute injury this is often asymptomatic, but untreated may be a cause of significant late post-traumatic enophthalmos.

Diagnosis is difficult on conventional films and may be frequently missed. Axial and coronal CT scans will by contrast visualize this area extremely well and should be performed when there is the slightest suspicion.

Surgical correction involves exposure via a bicoronal incision, reduction of the fractures, and almost universal primary bone grafting of the medial orbital wall. Split outer table of calvarium can be conveniently harvested through the same exposure and contours well to the medial orbital wall.

Frontonasoethmoid Fractures

Although conventional open reduction and miniplate fixation achieves close to anatomic restoration in most areas of the craniofacial skeleton, fractures of the frontonasal region are challenging. Severe comminution of the nasal bridge is common, with concomitant marked nasal septal buckling. The latter requires a septoplasty as a late secondary procedure. Premorbid nasal projection is not restored by simple reduction and miniplate fixation, and primary augmentation of the nasal dorsum is indicated with bone fixed as a cantilever graft or with cartilage.[10] When projection is maintained, an appropriate bridgeline may be refined and when not ideal in terms of projection primary grafting serves to stretch and expand the nasal soft tissue envelope, minimising the contracture, which is so difficult to overcome as a secondary procedure.

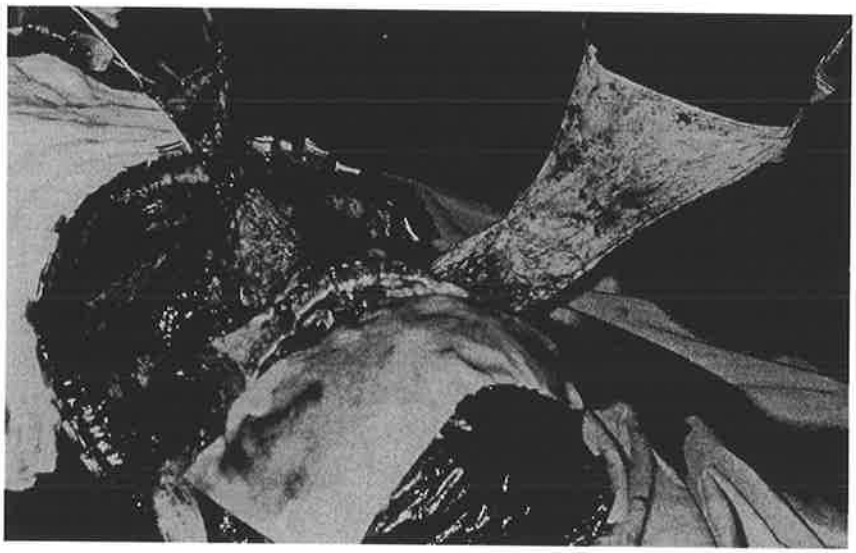


FIG. 8. *Temporoparietal fascial and galeal frontalis flaps provide vascularized barriers to separate the cranial and nasal cavities.*

Summary

The advent of the multidisciplinary approach to the clinical assessment, diagnosis, and management of fractures of the forehead and junction of cranium and faces has unveiled the opportunity for single–stage correction of these very complex injuries. Detailed preoperation assessment, wide fracture exposure, open reduction with stable internal fixation, and primary bone grafting will restore both form and function at the frontonaso-orbital interface with a minimum of long-term sequelae.

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Associated injuries in facial fractures: review of 839 patients

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SUMMARY

Patients with facial trauma may have associated injuries requiring immediate or specialised attention. This paper reports the incidence and nature of significant associated neurosurgical, ocular, spinal, torso and extremity injuries in facial fracture patients treated by the Department of Plastic and Reconstructive Surgery from June 1989 to June 1992. Of 839 patients treated during the period, 95 patients (11.3%) sustained significant concomitant injuries outside the facial skeleton. There were 45 (5.4%) patients with associated neurosurgical injuries, 33 (3.9%) with ocular injuries, 8 (0.9%) with spinal injuries, 16 (1.9%) with injuries of the torso, and 62 (7.4%) with injuries of the extremities. The spectrum of the injuries is presented. Most neurosurgical injuries are a result of focal impact and the intervention required is related mainly to local fracture management and the repair of dural tears. The risk of significant ocular injury is highest when the fracture involves the orbit. Injuries of the spine, torso (chest, abdomen, pelvis), and limbs were seen mainly in road trauma patients.

Facial injury crosses the anatomical boundaries of several surgical subspecialities. Localised injury to the face may involve not only the soft tissue and the facial skeleton but also by local extension the brain, the eyes, the sinuses and the dentition. Where trauma occurs in more diffuse high velocity impacts, concomitant injuries that can be more immediately life-threatening than the facial injury have been frequently reported.[1,2] This paper details the incidence and spectrum of significant associated injuries in a prospectively studied group of adult patients with facial fractures evaluated and treated in our department.

Materials and methods

All patients with facial fractures presenting to the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital between June 1989 to June 1992 have had standardised data forms completed on hospital discharge. Included are full patient personal details, history of injury, physical examination findings, radiological findings, diagnosis and treatment instituted. The findings on formal ophthalmological evaluation were noted for all patients with orbitozygomatic, frontal nasoethmoid, midfacial and complex facial fractures. Review by other specialties was instituted as indicated by the findings on screening evaluation. From this large series of facial injuries, a smaller subgroup was identified in whom there were significant associated injuries both locally in the craniofacial region and at a distance. The pattern of associated injury has been analysed and their relation to individual facial fractures identified.

Results

839 patients with facial fractures were evaluated and treated by the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital over the 3-year period between June 1989 to June 1992, either a primary consultation or on referral from another department. The aetiological factors in this series are

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detailed in Table 1. There were 678 males (80.9%) and 161 females (19.1%) and the age range was 15-88 years, with the peak incidence in the third decade of life.

A total of 95 (11.3%) patients sustained concomitant injuries to other parts of the body. The incidence of the injuries according to the various sites is shown in Table 2.

TABLE 1

Aetiological factors in 839 patients with facial fractures

Aetiology	No. of patients (%)	
Road traffic accident	158	(18.8)
Assault	430	(51.2)
Sports-related	137	(16.3)
Falls	81	(9.7)
Gunshot	6	(0.7)
Work-related	13	(1.6)
Others	14	(1.7)
Total	**839**	**(100.0)**

TABLE 2

Associated injuries in 839 patients with facial fractures

Type of injury	No. of patients (%)	
Neurosurgical	45	(5.4)
Ocular	33	(3.9)
Spinal	8	(0.9)
Torso (chest, abdomen, pelvis)	16	(1.9)
Extremities	62	(7.4)

TABLE 3

Computerised tomography findings in 45 patients with moderate to severe head injury.

CT findings	No. of patients
Intracranial haemorrhage	
Subdural haematoma	5
Extradural haematoma	2
Contusional	9
Fractures: vault	
Single	17
Multiple	3
Fractures: base	
single	18
Multiple	3
Diffuse cerebral oedema	4
Pneumoencephaly	6
Within normal limits	3
Miscellaneous	1

TABLE 4

Aetiology of facial fractures in the 45 patients with associated head injury

Aetiology	No. of patients (%)	
Road traffic accidents	33	(73)
Assault	4	(9)
Falls	4	(9)
Work-related	3	(7)
sport	1	(2)
Total	45	(100)

TABLE 5

Associated ocular injuries

Type of injury	No. of patients
Anterior segment	
Corneoscleral abrasions/lacerations	4
Hyphaema	10
Severe chemosis with infection	1
Increased intraocular pressure	1
Posterior segment	
Vitreous haemorrhage	3
Retinal oedema/tear/haemorrhage	10
Commotio retinae	3
Choroidal haemorrhage/rupture	1
Optic nerve damage	5
Penetrating globe injury (followed by enucleation)	3

TABLE 6

Aetiology of facial fractures in the 33 patients with associated ocular injury

Aetiology	No. of patients (%)	
Road traffic accidents	13	(39)
Assault	14	(43)
Falls	1	(3)
Sports	3	(9)
Others	2	(6)
Total	33	(100)

TABLE 7

Sites of fracture in the patients with associated ocular injury

Site of facial fracture	No. of patients (%)	
Orbit	6	(18)
Zygoma	11	(33)
Nasal and related fractures	2	(6)
LeFort, panfacial and other complex fractures	14	(43)
Total	33	(100)

TABLE 8

Aetiology and associated injuries of the spine, torso and extremities in 839 patients with facial fractures

Aetiology/ injury	RTA	Assault	Falls	Sport	Others
Torso:					
Chest	6	4	-	1	-
Abdomen	2	-	-	-	-
Pelvis	3	-	-	-	-
Spine:					
Cervical	2	-	-	-	-
Thoracic	1	-	-	-	-
Lumbar	4	-	-	-	-
Sacral	1	-	-	-	-
Extremities:					
Single fracture	32	6	1	-	1
Multiple fractures	18	1	-	-	-
Others:					
Brachial plexus	2	-	-	-	-
Compartment syndrome	1	-	-	-	-

Of the neurosurgical group, only moderate to severe head injuries were included, these being injuries severe enough to warrant neurosurgical consultation as well as computerised tomography of the brain. Excluded were patients managed by the general surgeons with mild closed head injury and temporary loss of consciousness who recovered without any neurological sequelae. Also excluded were patients referred for neurosurgical assessment but who did not require CT scanning and recovered without further intervention. There were 45 patients who fulfilled these criteria and their various CT findings are displayed in Table 3. The patient classified under 'miscellaneous' had a carotico-cavernous fistula and a fractured mandible as a result of a fall. Table 4 shows the aetiology in the head injured group. The majority of these patients sustained their injuries in high velocity road traffic impacts. The facial fracture pattern was complex *(i.e.* LeFort, panfacial, or complex) in 33 patients (73%). The other 12 patients sustained fractures of the zygoma (4), mandible (6) and supraorbital ridge (2). Neurosurgical intervention was required in 13 patients (29%) and of these, seven patients required dural repair, four patients underwent elevation of depressed skull fractures, one needed a cranioplasty for a severely comminuted frontal fracture and one required drainage of a subdural haematoma. The carotico-cavernous fistula was embolised.

The spectrum of ocular injuries is shown in Table 5, whilst the aetiology and site of facial fracture in these patients are recorded in Tables 6 and 7. Zygomatic fractures affecting the orbital floor were classified under the zygoma. Excluded from this group were patients with eyelid or adnexal injuries, nonvisually threatening injuries such as transient subconjunctival haemorrhages, and socket-related problems such as enophthalmos and telecanthus which were included in a previous report.[3]

The range of other associated injuries according to the aetiology is displayed in Table 8. The vast majority of these occurred where the energy of impact was high.

Discussion

Patients with facial fractures can present with a combination of associated injuries of varying complexity and severity. Gwyn *et al.*[1] reviewed 567 patients with facial fractures and found that 51.6% had associated injuries and 28.9% had pre-existing systemic disease. Luce *et al.*[2] stressed the role of a capable primary surgeon in making a proper overall assessment and organising management priorities. They reported a 14% incidence of associated injuries in the group injured in a low-velocity circumstance *(e.g.* assault or fall) and 64% in the high-velocity group *(e.g.* road traffic accident). Both these series had a relatively high proportion of road traffic accidents 35% and 65% respectively. In keeping with recent epidemiological trends, assault was the major aetiological factor (51.2%) in our series of 839 patients, with 18.8% being due to road trauma (Table 1). It is this changing pattern of aetiology, the change from high velocity to low velocity impact, that would explain the lower incidence (11.3%) of associated injuries in our series. However, the nature of these injuries continues to be significant and the well recognised need for a multidisciplinary approach to the management of facial trauma remains evident.

Davidoff *et al.*[4] reported that 55% of 200 patients with facial fractures had closed head injury (CHI), which was defined as documented evidence of loss of consciousness and/or post-traumatic amnesia from reviews of ambulance and police reports, and interviews with patients and witnesses. More than 90% of their cases had minor CHI. Our overall incidence of moderate to severe head injury is 5.4%. Apart from a different selection criteria, this lower incidence is also attributed to a different aetiological profile in the other series. 52-71% were due to road traffic accidents in previous series.[4,5] Although it is recognised that mild CHI is not inconsequential, we chose to focus on the group with moderate to severe head injury the better to evaluate the spectrum of head injuries in our patients with their pattern of facial fractures. In this series, the majority of the injuries were skull fractures resulting from the focal traumatic impact, with a smaller proportion having parenchymal injury. Primary brain injury was thus very uncommon, supporting the theory of the air-filled impact-absorbing facial bones serving as a cushion protecting the brain.[5] In line with this, the neurosurgical intervention required in 29% of these patients was related mainly to the repair of aural tears and local fracture management. Although five patients demonstrated evidence of a subdural haematoma on CT scanning, only one of them was large enough to require drainage. It is, however, recognised that the presence of intracranial haemorrhage is associated with a poorer survival rate.[6]

The incidence of associated ocular injury varies from 2.7% to 67% in retrospective series.[2,3,7] In a recent prospective series, 12% of the facial fracture patients had vision-threatening ocular injuries.[8] Our incidence of vision-threatening ocular injuries is 3.9%. This could partly be explained by the sizable percentage (36%) of isolated mandibular fractures in our 839 patients where the risk of an associated ocular injury is much lower. The risk is highest when the fracture involves the orbit.[8] Aetiology is varied in this group, occurring almost equally with high energy diffuse impacts (RTA) and the more localised but lower energy impact of an assault. Although the incidence is low, associated ocular injury is significant in its nature and severity. The practice of a preoperative ophthalmological assessment in high risk patients is useful not only because of the possibility of fracture treatment-related complications, but also because successful management of such injuries requires early recognition and prompt intervention by the appropriate ophthalmic specialist. Injuries of the torso, spine and extremities were seen mainly in road traffic accidents. The more brutal assaults resulted in rib and limb fractures which, although not infrequent, were seldom life-threatening in the acute period nor associated with significant long-term functional disturbance. Previous series have also found associated spine and torso injuries to be uncommon.[1,2]

In summary, it is apparent that the incidence of associated injuries is altered with changing patterns and aetiology of injury. However, it remains a characteristic feature of facial trauma that the anatomical boundaries of various disciplines are frequently crossed, on the face as well as in the rest of the body. It should not only be viewed as an isolated injury of the facial skeleton but as part of a spectrum of potentially disabling and sometimes life-threatening injuries which requires a coordinated team approach.

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Sports-Related Facial Fractures: A Review of 137 Patients

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One hundred and thirty-seven patients with sports-related facial fractures were reviewed. These made up 16.3% of 839 patients with facial fractures seen at the Department of Plastic and Reconstructive Surgery, Royal Adelaide Hospital, between June 1989 and June 1992. Males made up 93.4% of patients and 89.1% were aged below 35 years. There was an intent to injure in 11%. Australian Rules football was the causative sport in 52.6%, all the injuries being the result of human contact. Orbitozygomatic fractures were the most frequently observed overall (62%) as well as in Australian Football (58.3%). Cricket contributed to 14.6%, the ball being the agent of injury in all but one of the patients. Horse-riding injuries were the most severe. 89.1% of the patients required surgery and hospital stays ranged from 0 to 18 days with an average stay of 4.7 days. Sports activities, although a significant source of enjoyment, are a significant cause of facial fractures with their attendant morbidity.

KEY WORDS: *facial fractures, sports.*

Introduction

Sports activities, in general, provide healthy recreation and considerable gratification for the participant but can at the same time cause injuries. In recent decades, with greater overall participation in sports, a wider range of sports and a greater emphasis on physical fitness, sports-related injuries have become a topic of interest. The adverse sequelae apply not only to the injured player but also to the sport itself, which can easily fall into disrepute if the risks of major injury become unacceptably high. Most sports injuries involve the lower extremities[1-3] and, in the head and neck region, sports-related eye injuries have been well discussed.[4-6] However apart from dental injuries, fractures of the facial skeleton as a result of sports injuries so far have been addressed to a small extent only.[7] The aim of this study was to review the extent of the problem, analyse the spectrum of facial fractures sustained in sports in South Australia, discuss the risk factors involved and suggest possible preventive measures.

Methods

A total of 839 consecutive patients with facial fractures were evaluated and treated at the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital from June 1989 to June 1992. A standardised form was completed for each patient on discharge. All patients with sports-related fractures were included in this study and data recorded in these forms and obtained from a review of the radiology were analysed. The areas of interest were age and sex of the patients, the type of sport, the presence of an intent to injure, the mechanism of injury if known, the pattern of fracture sustained, the treatment required and the length of hospital stay. It should be noted that nasal fractures are treated by three

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departments in the Hospital (ENT, Plastic Surgery and Casualty) and occlusal fractures are treated by two departments (Plastic Surgery and Oral Surgery); hence there is likely be an underestimate of these two groups of fractures in the statistics presented here.

Results

Of the 839 patients with facial fractures seen at the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital, 137 patients sustained facial fractures as a result of sporting activities. The ages ranged from 15 to 65 years with 122 patients (89.1%) aged below 35 years. One hundred and twenty-eight (93.4%) were males. The types of sporting activities causing the facial fractures are summarized in Tables 1 and 2. There was an identifiable intent to injure in 15 patients (11%).

Australian Rules football was observed to cause the highest rate of facial fractures corresponding to the high participation rate in contact sports as compared to other types of sports. The injuries were sustained in a variety of ways: a clash of heads, collision with the opponent during tackles or being struck by the knee, elbow, shoulder or foot of the opponent during a struggle for the ball (Fig. 1). Injuries as a result of human contact were also seen in soccer and rugby. In cricket, however, 19 out of the 20 patients were injured by the ball. Eight of the nine horse-related injuries occurred as a result of the patients being kicked by the horse after falling off the animal that they were riding.

The injuries in hockey, golf, tennis, lacrosse and baseball were all implemental, being due to the hockey stick, golf club or the balls, respectively.

Figure 2 demonstrates the sites of facial fracture seen in the 137 patients with relation to each sport. In general, these fractures were localized injuries due to isolated blows to the facial skeleton. The most common were fractures of the zygoma, seen in 66 patients (48.2%). Figure 3 demonstrates the pre- and postoperative radiographs of a patient who had been struck by a cricket ball, sustaining a fractured zygoma. Fractures of the mandible occurred in 40 patients (29.2%) with fractures of the orbit, nose and more complex patterns making up the rest. Fractures of the orbit included isolated fractures of the rim, floor or walls of the orbit and excluded those that were related to zygomatic fractures.

Grouped together, orbitozygomatic fractures were observed in 62% of the patients overall and in 58.3% of the patients who played Australian Rules football. Complex facial fractures were those where there were two or more fracture sites (e.g. fractures of both the zygoma and the mandible) and included panfacial and LeFort fractures. These were seen primarily in the horse-riding injuries. An example is shown in Fig. 4. Table 3 shows a further analysis of the type of facial fractures sustained in Australian Rules football. As with the overall picture, the zygoma was the most frequently fractured bone. The mandible was the next most frequently injured. Fig. 5 shows the pre- and postoperative radiographs of a patient with a fractured mandible sustained during a football tackle.

One hundred and twenty-two patients (89.1%) of the patients required surgery with the remainder receiving conservative treatment because the fractures were either undisplaced or minimally displaced. Duration of hospital stay ranged from 0 to 18 days with an average stay of 4.7 days.

TABLE 1

Sporting activities in 137 facial fracture patients

Sport	Patients	
	n	(%)
Australian Rules football	72	(52.6)
Cricket	20	(14.6)
Rugby	8	(5.8)
Soccer	7	(5.1)
Horse-related	9	(6.6)
Others*	21	(15.3)
Total	137	(100)

*See Table 2.

TABLE 2

Examples of other causative sports in sports-related facial fractures

Hockey
Golf
Tennis
Lacrosse
Baseball
Karate, martial arts, kickboxing
Water sports: diving, skiing
Basketball

FIG. 1. *Australian Rules football—the most common cause of injury in this series (photograph courtesy of The Advertiser).*

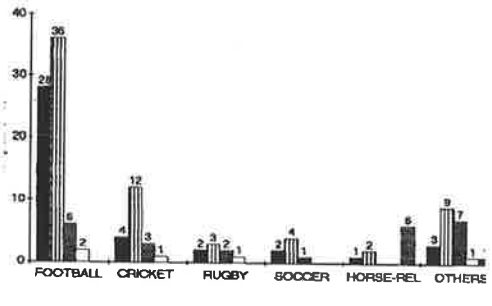

FIG. 2. *Fracture sites of sports-related facial fractures. (■) Mandible; (▥) zygoma; (▨) orbit; (□) nose; (⊞) complex (combination of two or more fracture sites).*

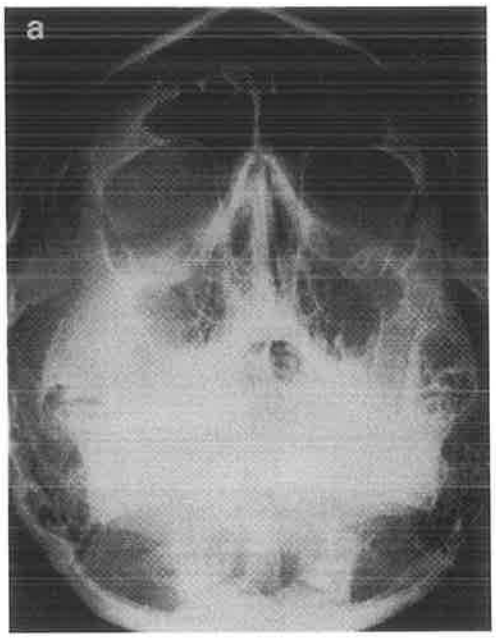

FIG. 3a. *Pre-operative radiograph of a patient injured by a cricket ball resulting in a fractured right zygoma.*

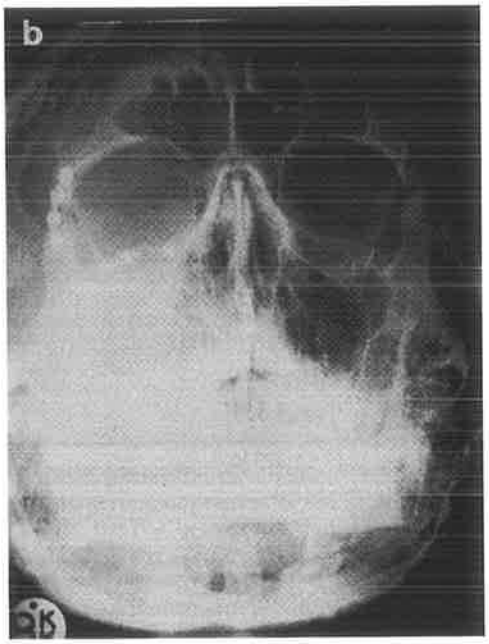

FIG.3b. *Postoperative radiograph of the patient in Fig. 3a.*

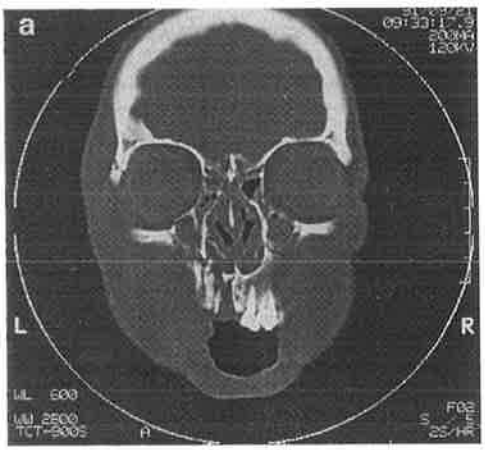

FIG. 4a. *Coronal CT scan of a complex facial fracture sustained in horse-riding accident.*

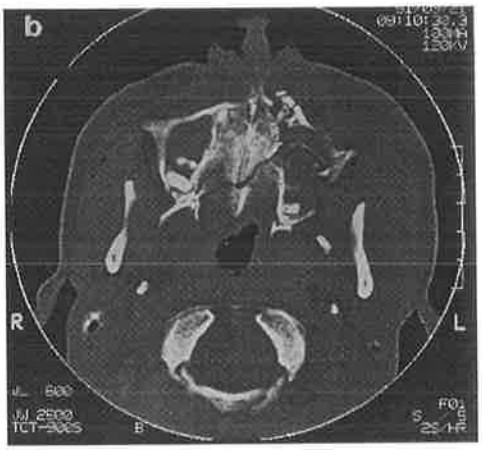

FIG. 4b. *Axial CT scan of the patient in Fig. 4a.*

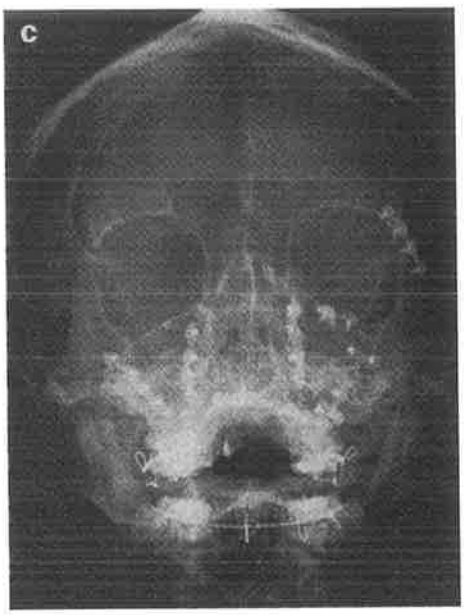

FIG. 4c. *Postoperative radiograph of patient in 4a demonstrating reduction and internal fixation.*

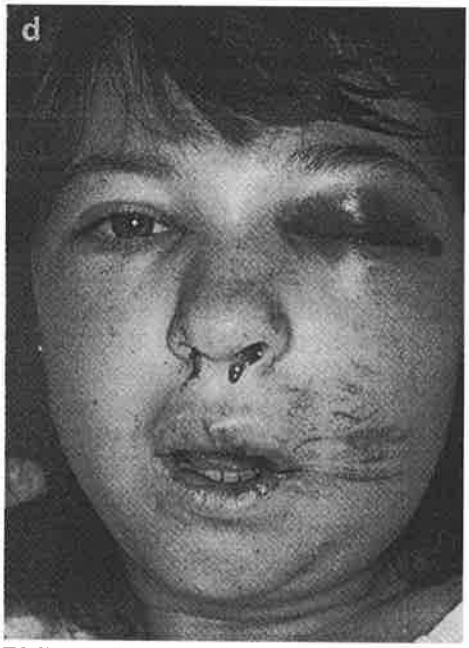

FIG. 4d. *Clinical appearance of the same patient immediately following injury.*

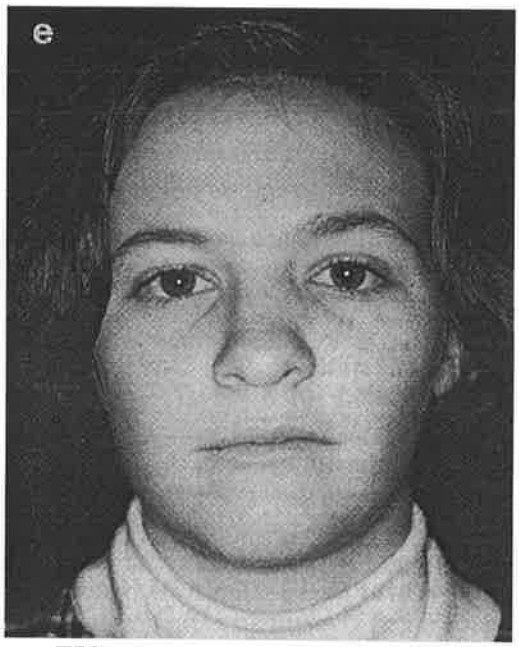

FIG. 4e. *Clinical appearance of the same patient three months after repair.*

Discussion

With the popularity of various sports increasing over the past few decades, it is not surprising to see a rising number of sports-related injuries. Epidemiological studies show that most sports injuries involve the lower extremities.[1-3] Although injuries above the clavicle make up a small proportion of sports injuries as a whole, the seriousness of the injuries in this area cannot be overemphasised. Serious cervical cord injury during rugby and ocular injuries in sports are well-recognized[4-6,8,9] and chronic head injuries from repetitive blows have been fully discussed recently.[10] Awareness of a problem and accumulation of data on it is the beginning of a solution, as is well-illustrated by the establishment of a National Football Head and Neck Injury Registry in the USA where observations led to a dramatic decrease in the occurrence of cervical quadriplegic from football.

Brook and Wood showed the number of sports-related facial fractures increased from 1965 to 1980.[11] Sports-related facial fractures made up 16.3% of the total number of facial fractures in the present series (L. H. Lim and D. J. David unpubl. data), 17.4% in a Swedish report,[12] 9% in an American study[13] and 1-10% in other series.

Sports injuries are often considered to be a necessary payment for the physical amusement and psychological satisfaction derived from the sporting activity. However recognition of patterns of injuries helps to identify inherent risks in sports and hence the preventable injuries. Lehmann and Ravich classified contact, competitive and individual sports into high and low risk groups with particular reference to neurologic injury.[10] Representative higher risk sports were boxing, American football, 'amateur' wrestling, ice hockey, martial arts and rugby, while examples of lower risk individual sports were dancing, cheer leading, archery, jogging, bicycling and horse-riding. Pickham and Kohn discussed the risk factors as extrinsic and intrinsic.[14] Extrinsic risk factors were those potential predictors of injury that are independent of the individual for example, the risk inherent in the type of activity (jogging and stress injuries), climatic conditions, status of equipment or quality of playing surface. Intrinsic factors were predisposing characteristics present in the individual sports participant, for example, biologic and psychosocial characteristics such as age, gender, temperament and body size. The vast majority of the patients in this series were males. This supports the role of gender as a potential risk factor for sports injury, indicating the inclination of males towards more vigorous and probably more

aggressive sports. Patients below 35 years of age made up 89.1% of cases seen in this study, indicating that the young are more likely to engage in sports. Age as a risk factor is, however, difficult to define. Youthful vigour and temperament may contribute to playing styles predisposing to injury. Conversely, advancing age resulting in slower co-ordination and degenerative changes in bone and ligaments may increase the chances of injury.

Australian Rules football, a very popular sport, is categorized as a high-risk contact sport. Not surprisingly, it was the causative sport in more than half of the present patients. The face is frequently at risk of injury when a player is 'burrowing' for the ball, where he may be struck by the opponent's knee or involved in a head-on collision. The face may also be injured when a player attempts to 'spoil' the ball by hitting it off its course and hits another player instead or at any time during the frenzied scurry for the ball. Eleven per cent of the patients could identify a definite intent to injure, while a large proportion were unable to tell. Several of these intentional injuries amounted to on-field assaults. Injury prevention should commence with strong discouragement of aggressive behaviour, either during training or during play, to decrease the incidence of intentional injury. Where persuasion fails, rules may need to be more strictly enforced or even changed to ensure a safer sport. Immediate penalties during the game rather than after should be more effective at regulating behaviour. Of the patients in the present study, nearly 60% sustained orbitozygomatic fractures and almost 40% had mandibular fractures. Protective headgear may be found to be beneficial and these fracture patterns should be taken into consideration in their design.

Rugby and soccer, the other two contact sports in this study group, made smaller contributions being played less frequently than Australian Rules football. Cricket, baseball and other small-ball games such as lacrosse and hockey have all been known to cause facial fractures. A fast-flying or bouncing ball, rather than the bat, was the predominant cause of injury in the cricket players. In hockey and golf, however, the stick or club may be just as likely to injure. Horse-riding although classified with dancing and cheer leading as a low-risk individual sporty caused the most severe injuries in this group of patients. These injuries were mainly a result of falls followed by kicks, resulting in panfacial, LeFort or other complex facial fractures, patterns seen more often in victims of high-speed road accidents. Wearing of helmets may decrease the severity of these injuries.

In conclusion, facial fractures, although not usually catastrophic injuries, can result in longterm morbidity and deformity, and at the least may require initial hospitalisation and surgery. As it primarily affects the young, it also results in loss of work-hours. Injuries due to sports should not be accepted as a necessary consequence of participation and enjoyment but should be looked upon as largely preventable. Appropriate measures should be taken to maintain sports activities in their rightful place as a healthy source of recreation allowing a pursuit of excellence, and to stop them degenerating into treacherous physical violence. More data on sports-related facial trauma should be accumulated, not only to further define the magnitude of the problem but also to offer solutions.

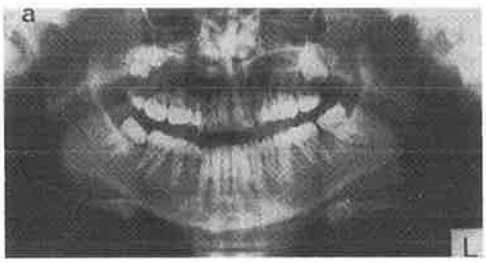

FIG. 5a. *Pre-operative radiograph of a football injury resulting in a fractured mandible*

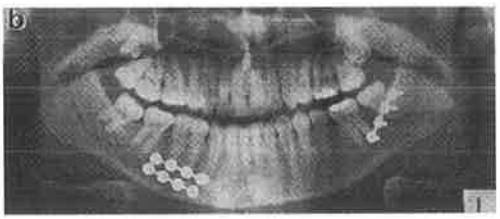

FIG.5b. *Postoperative radiograph of the patient in Fig. 5a.*

TABLE 3

Facial fractures sustained in Australian Rules football

Site of Fracture	Patients	
	n	(%)
Mandible		
Condyle	5	(6.9)
Angle	7	(9.7)
Body	3	(4.2)
Double fractures	13	(18.1)
Zygoma		
Zygomatic complex	30	(41.7)
Isolated arch	6	(8.3)
Orbit		
Floor, walls (excluding zygomatic fractures)	6	(8.3)
Nose	2	(2.8)
Total	72	(100)

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The relationship between fracture severity and complication rate in miniplate osteosynthesis of mandibular fractures

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SUMMARY

There are many factors influencing the outcome of mandibular fracture management; however, the relationship between fracture severity and complication rate has only been recognised intuitively due to the absence of an accepted system of classification of the severity of these fractures. In 1989 Cooter and David described the alpha numeric system of computer based coding for craniofacial fractures.[1] Using this system, a prospective sample of 324 patients with mandibular fractures presenting to the Royal Adelaide Hospital was coded for fracture severity and their progress followed with respect to complication rate. A strong correlation between complication rate and fracture severity was established.

Mandibular fractures are common, and in order to achieve satisfactory cosmesis and occlusion, open reduction and internal fixation is often necessary. This is associated with a significant morbidity, including infection, malocclusion, non union, plate fracture, and the need for removal of plates as a second procedure in some cases.[2-4] This study was designed to investigate the relationship between the severity of the fracture being treated, and the incidence of complications that develop following surgery.

Method

The patients included in this study included all patients with a facial fracture presenting to the Department of Plastic and Reconstructive Surgery at The Royal Adelaide Hospital during the 3-year period from 1 July 1989 up to and including 30 June 1992. Prior to this, members of the Department of Plastic and Reconstructive Surgery met and designed a form known as the "Trauma Form". This form remained with the patient's case notes for the duration of his or her inpatient and outpatient treatment and details of management were entered as they occurred, thereby eliminating the need for retrospective case note analysis. In particular, the operative description was completed by the surgeon who performed the surgery, and the outpatient details were entered at the time of the examination by the clinician conducting the outpatient examination.

Alpha-numeric code and complication rate

All mandibular fractures were coded according to the alpha numeric system of computer based coding for craniofacial fractures as described by Cooter and David.[1] This system divides the craniofacial region into 10 bilateral major anatomical zones, each of which is composed of minor zones. An alphabetic code is assigned to each zone. The fracture is then assigned a numerical value where an undisplaced fracture is scored 1, a displaced fracture 2, and a comminuted fracture 3 points.

British Journal of Plastic Surgery (1994), 47, 310-311

The ten major zones are: Cranial-frontal, parietal, spheroidal, temporal, occipital; Facial-nasoethmoidal, zygomatic, orbital, maxillary, mandibular.

Each of the major zones is divided into a number of minor zones. For the mandible these zones are; condyle, ramus, body, dentoalveolar, coronoid process, angle, symphyseal.

In the usual situation, the maximum score allowable for a major ipsilateral zone is 5, thus the total points for the ten bilateral zones is 100. This enables the total fracture score to be expressed as a percentage.

For the purposes of this study the total mandibular fracture score was considered, regardless of whether it exceeded the allowable 5 points. Thus the fracture severity was then contrasted with the incidence of complications.

The patients included in this study were all those whose fractures required open reduction and internal fixation. This was carried out using monocortical miniplate osteosynthesis, according to the principles espoused by Champy.[5-7] Any complications that ensued were recorded on the trauma form as an inpatient, and also at the following outpatient visits. These were recommended at 1 week, 3 weeks and 6 weeks postoperatively, and on a needs basis thereafter.

Results

During the period of the study, 324 patients with at least one fracture of the mandible were treated. Of these patients, 247 (76%) were treated by open reduction and internal fixation using non-compression monocortical miniplate osteosynthesis. Overall there were 39 complications, resulting in a complication rate of 15.7% (Table 1).

The complication rate was then compared with the severity of fracture, as determined by the alpha numeric coding score, to see whether or not postoperative complication rate was related to fracture severity. As these two variables were plotted against each other, the correlation coefficient was calculated to measure the strength of the linear association between the two variables. The value of the correlation coefficient (r) lies between -1 and 1, where a positive r indicates positive association[8].

$$r = \frac{1}{n-1} \sum \frac{x-\bar{x}}{S_x} \quad \frac{y-\bar{y}}{S_y}$$

Where x = mean
S_x = standard deviation

It is apparent from Figure 1 that the incidence of complications with miniplate fixation increases as the severity of the fracture (as given by the alpha-numeric coding score) worsens, and that the correlation is strongly positive, correlation = 0.96.

TABLE 1

Complications	No.	%
Plate fracture	2	0.8
Infection resulting in removal of plate	7	2.8
Infection responding to treatment	2	0.8
Malocclusion with corrective op required	13	5.3
Removal of plates	10	4.0
TMJ discomfort	3	1.2
TMJ ankylosis, bilateral reconstruction	1	0.4
Non union	1	0.4
Total	39	15.7

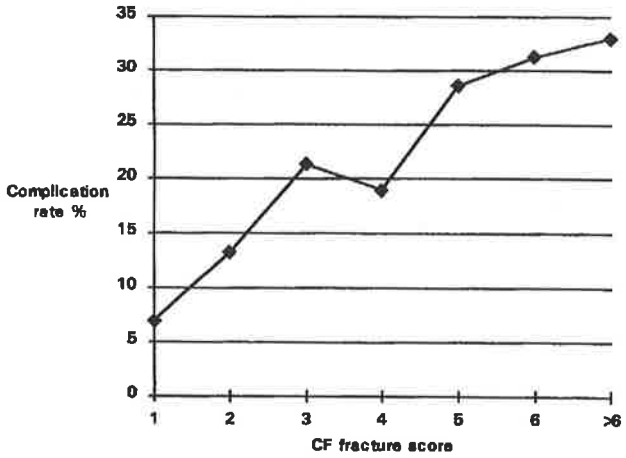

FIG. 1. *Complication rate increases as severity of fracture worsens.*

Conclusion

These figures demonstrate that the incidence of complications associated with the management of mandibular fractures is higher for fractures of greater severity, with a correlation of 0.96 between fracture severity and complication rate. Previously this association, although intuitively recognised, has not been shown statistically due to the absence of an objective and reproducible system of classification of these fractures that includes the location, number, and severity of fractures. The development of the alpha numeric system of coding for craniofacial fractures has allowed an objective and standardised assessment of the degree of severity of the fracture to be made. The recognition of predictor factors such as this enables the clinician to identify patients at greater risk of complications, and may facilitate the development of techniques to reduce the incidence of these complications.

Acknowledgment

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A Comparative Study of Miniplates Used in the Treatment of Mandibular Fractures

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The purposes of this study were to investigate the differences in mechanical properties of major miniplating systems used for noncompression miniplate osteosynthesis of mandibular fractures and to determine whether these properties influence treatment outcome. The study was conducted in two parts. First, six of the major miniplate systems currently used at the Royal Adelaide Hospital were subjected to bending tests at the University of Adelaide Engineering Department to quantify the relative stiffness of each plate. Second, a prospective sample of patients presenting with mandibular fractures was analyzed. These patients were treated with a variety of the miniplating systems. The results of treatment as a whole were compared to identify any direct benefit consequent on the miniplate selected. While significant differences in stiffness were identified between the plating systems, no significant differences in treatment outcome were identified between the noncompression plates employed. Since no observable benefits have been identified by choice of miniplate, selection should be based on surgical preference, biocompatibility, CT compatibility, and unit cost. Because of the variations in materials, design, properties, CT compatibility, and unit cost, it is important not to regard all miniplates as equal and interchangeable. (*Plast. Reconstr. Surg.* 97: 1150, 1996.)

The fixation of mandibular fractures by noncompression monocortical miniplate osteosynthesis according to the tension-band principle was introduced by Michelet et al.[1] and Champy et al.[2] based on the experimental work of Champy et al.,[3] who showed that distraction forces operate at the upper border of the mandible, while compression forces operate at the lower border. This theory has since been contradicted by Rudderman and Mullen,[4] who showed that zones of tension and compression may be reversed when forces are generated along the posterior teeth. Thus the original theory upon which this treatment modality was based has been challenged; however, the method has been retained because the postoperative results and complication rate are comparable with those reported around the world, and the method holds significant advantages over bicortical compression plate osteosynthesis.

The advantages of monocortical miniplate osteosynthesis over bicortical compression plates include the followings:

1. Compression plating often requires an extraoral approach, which is technically more difficult. The necessity for the extraoral approach has been quoted at 60.8 to 78.5 percent of cases.[6,7]
2. Bicortical plates risk damage to the inferior alveolar nerve, whereas the risk of damage to the inferior alveolar and mandibular nerves using the monocortical plates is negligible.
3. Routine use of intraoral incisions with monocortical plates requires minimal dissection and avoids an external scar.
4. The technique is easily taught, and excellent results are achieved by junior registrars.[8]

5. In simple fractures of the mandible, monocortical osteosynthesis provides rigid fixation, and no complications are caused by inadequate stability of fixation. [9,10]

6. It is difficult to make compression plates adapt to the bony curvatures.[9]

The Australian Craniofacial Unit uses a modified Champy approach to the treatment of mandibular fractures, as described by Moore et al.[5] To recognize monocortical miniplate osteosynthesis as the treatment of choice for the open reduction and internal fixation of mandibular fractures is to oversimplify the issue. There is now a myriad of commercially available miniplating systems, and these vary in their materials, design, physical properties, and cost.

With this in mind, the specific aims of this study were, first, to compare scientifically the engineering properties of miniplates commonly used in fracture treatment and, second, in a clinical setting to compare the in vivo performances of the same miniplates to identify which of these properties influence treatment outcome.

Part 1: Comparison of the Biomechanical Properties of Miniplates

Manufacturers have sought to improve miniplates by varying their design, properties, profile, and material composition. This has resulted in a great deal of choice afforded to the clinician. However, despite the large number of obviously different systems, little comparative work has been published to date.

The ideal miniplate will exhibit a number of features. It will be:

1. Cost-effective
2. Easy to mold to the contours of the facial skeleton
3. Sufficiently stiff to maintain rigid fixation and strong enough to resist deformation across the plate during fracture healing
4. Completely biocompatible
5. Of low profile so as not to be palpable
6. Of composition so as not to produce scatter on CT scans
7. Not intrinsically responsible for producing complications

Any comparison of the engineering properties of miniplates must take into consideration their metal composition. This is of particular importance because many of these plates are often left in situ indefinitely, so biologically inert metals are preferred. The three commonly used implant materials are stainless steel, Vitallium, and titanium. The choice of the implant material will influence the strength and stiffness of the implant, the biocompatibility of the implant, and the imaging properties of the implant, particularly with regard to CT investigations. The AO/ASIF group suggests that titanium is the most biologically inert of the three and therefore has the least chance of producing any low-grade immunologic response. No allergic reactions to titanium have been reported.[11] With regard to CT compatibility, titanium is also the preferred implant because it is the most radiolucent.[12]

In choosing a plating system from the product information of the various manufacturers, the clinician may be confounded by the terminology used. For example, the hardness of the component metal may be expressed in a variety of units such as the Vickers hardness number (VHN) and the Rockwell scale (R_B and R_C). The tensile strength and elongation to fracture of the core metal are other parameters often quoted. This information often refers to tests carried out

on the core metal and does not take into account the structural performance of the individual plates. Thus the clinician is not provided with a simple guide to compare different plates directly. In addition, most of the manufacturers make no attempt to link the information they have provided with clinical trials that demonstrate the reasoning behind the miniplate design.

As a result of the lack of experimental data, clinicians are left to select plating systems on the basis of inadequate information. Taking this one step further, the science of selection of the size and strength of plating system for various regions of the craniofacial skeleton also has been neglected, leaving clinicians to estimate the strength of plate that might be required for a specific area, e.g., a "heavy" plate for a mandibular fracture due to the perceived forces applied across the mandible or a "small" plate to stabilise a nasoethmoid fracture due to the absence of large muscular forces applied across this fracture.

Recently, some literature has appeared analyzing the biomechanical properties of miniplates. Damron et al.[13] compared the biomechanical properties of Luhr Vitallium minifragment plates, Synthes titanium minifragment plates, and Synthes stainless steel minifragment plates designed for craniofacial uses but in this study used for dorsal plate fixation of proximal phalangeal fractures. Hegtvedt et al.[14] compared the Luhr minisystem with the Luhr microsystem to provide a comparison of the biomechanical properties of each system. They showed that there is a significant difference in the force required to bend miniplates compared with microplates. They then review some of the expected forces that occur in vivo and make some guarded conclusions about correlating the in vitro biomechanical properties with in vivo forces. For example, if a plate is shown to withstand a certain force in a biomechanical model, does this mean it can withstand a similar occlusal force in vivo? The authors make it clear that clinical studies are needed to confirm such an assumption.

The aim of this study was to produce a clinically relevant comparison of the different mechanical properties of the miniplates. The most important indicators to the clinician are the stiffness of the miniplate and the force required to permanently deform the plate. The clinician will then be able to select a miniplate (taking into account the cost, biocompatibility, and CT compatibility of the plate) able to withstand the expected forces yet still malleable enough to be shaped to the contours of the bone and hence "operator friendly." Since the complex in vivo forces are difficult to calculate, this must be coupled with clinical trials that confirm miniplate effectiveness in individual regions.

Materials and Methods

This study was conducted at the Department of Materials Engineering at the University of Adelaide. Five miniplate systems were selected for investigation, these being the five systems available for use at the Royal Adelaide Hospital, i.e., the Wurzburg, AO/ASIF, Medicon, and Aus Systems and Champy miniplates, along with the Luhr minicompression plates.

Mechanical Properties

When considering the mechanical properties of miniplates, the prime consideration should be their stiffness and strength in bending. Since where the aim of this study was to test the miniplates already in use, not to develop new miniplate design, it was possible to test each miniplate system and its screws as a functional unit rather than testing a standard form of the pure alloy or metal.

Stress versus strain behavior may be represented graphically (Fig. 1). In the elastic section, the strain is reversible, that is to say that the metal returns to its original shape after the stress is removed. Hooke's law suggests that for a

linear elastic material, strain increases in direct proportion to the applied stresses. The slope of the linear elastic section (denoted by *E)* is Young's modulus of elasticity. Young's modulus of elasticity is a measure of the rigidity of the material and is therefore a property of the material.

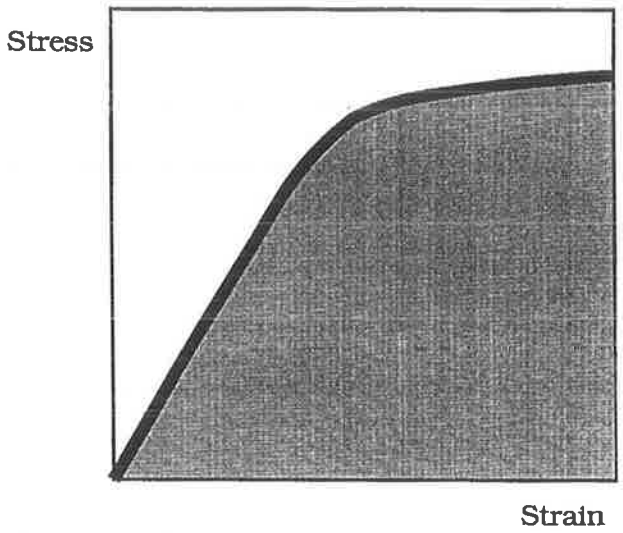

FIG. 1. *Young's modulus of elasticity* E = *stress / strain.*

At a certain point, the deformation of the material ceases to be elastic (reversible) and becomes plastic (permanent). In the plastic region, strain changes are no longer directly proportional to the applied stress. The point at which this occurs is known as the *yield point* and is the most important value for design.

The critical properties of the plate in vivo are those which resist the bending forces across a fracture line, i.e., the stiffness of the plate and its yield load. If *E* is Young's modulus of elasticity and *I* is the moment of inertia of the cross-sectional axis at midspan, then $E \times I$ is the stiffness of the plate (Fig. 2):

$$\text{Stiffness} = EI = \frac{wl^3}{48y}$$

where

w = load

y = displacement at the center of the span

l = length

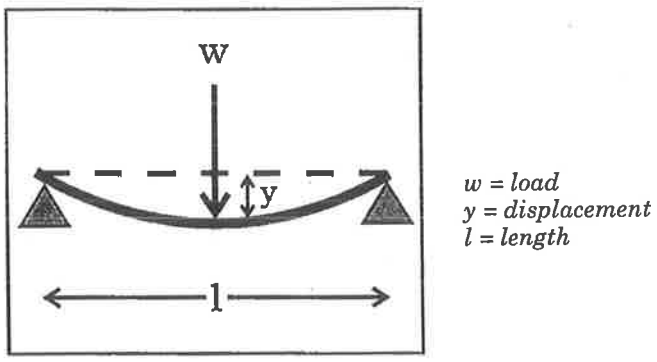

FIG. 2. *Young's modulus of elasticity E times moment of inertia I of the cross-sectional axis equals stiffness (EI=* wl/ 48y).

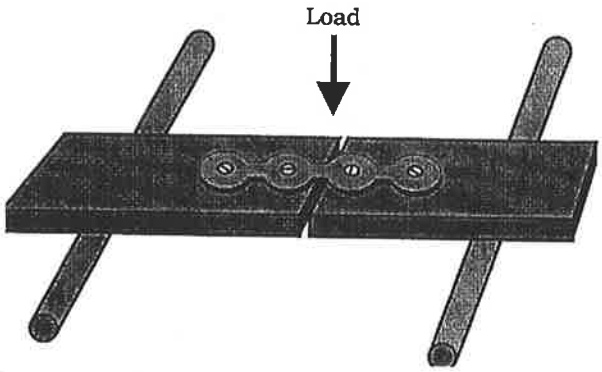

FIG. 3. *Instron 1026 tensile testing machine.*

In conjunction with the Department of Materials Engineering of the University of Adelaide, a testing rig was designed (Fig. 3). A four-hole miniplate was screwed into a brass template with two holes on each side and a 0.25-mm gap to simulate a fracture. The screw holes were pretapped to accept the particular system's screws. This allowed each plating system to be tested as a functional unit. Since the length l is the distance between the grips, the equation gives the empirical value of stiffness for the composite structure (miniplate and brass plates). However, in this model the brass plates were assumed to be infinitely stiff; thus only the deformation of the miniplating system could account for any deformation recorded. Obviously, the distance between the grips is chosen empirically and does not attempt to reflect the real case in vivo. This system was then placed in an Instron 1026 tensile testing machine, which is a threepoint bender exerting a known load on the simulated fracture line. Each plate was tested 10 times, and average stiffness and yield point were established.

Results

The results of the engineering component of the study are shown in Table I. The miniplates were shown to have similar yield points; however, the stiffness of the plates varied significantly.

Thus the clinician is now provided for the first time with a direct comparison of the stiffness and yield point of these plating systems as functional units, i.e., a four-hole plate and screws fixed to an unyielding template.

TABLE I

Stiffness and Yield Point Analysis of the Miniplates

	Yield Point (kg)	Stiffness (*EI*)
Aus Systems miniplates	1.12	295.1
Wurzburg miniplates	1.25	5494.1
Medicon miniplates	2.2	4864.2
AO noncompression miniplates	1.8	2951.1
Champy miniplates	1.8	3699.1
Luhr compression miniplates	25	73981.0

Part 2: Clinical Analysis of Internal Fixation of Mandibular Fractures Using Monocortical Noncompression Miniplates

The in vivo performance of these miniplates was then investigated in a clinical trial designed to identify any difference in treatment outcome related to the selection of miniplate. The clinical sample included all patients with a mandibular fracture requiring surgical fixation under the care of the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital during the 3-year period from 1989 to 1992. During this period, 832 patients with facial fractures were seen. A total of 324 patients had sustained a fracture of the mandible, and of these, 247 were managed by noncompression osteosynthesis.

Surgical Techniques

During the period of this study, the Australian Craniofacial Unit has used Luhr, Medicon, Wurzburg, and Aus Systems miniplates interchangeably. Unfortunately, the selection was not randomised; however, the consultants, registrars, and fellows all used a variety of the systems. No surgeon used one system exclusively, and no protocol was in place for the use of any one system for any particular situation.

Materials and Method

The patients included in this study included all patients with a facial fracture presenting to the Department of Plastic and Reconstructive Surgery at the Royal Adelaide Hospital during the 3-year period from duly 1, 1989 up to and including June 6,1992. The data were collected in a prospective fashion separate from the case notes, thereby eliminating the need for retrospective case note analysis.

Results

A total of 324 patients with mandibular fractures presented during the 3-year period of the study, and of these, 247 (76 percent) were treated by open reduction and internal fixation with miniplates. A total of 77 patients were treated by other methods, the majority having nondisplaced fractures managed conservatively or minimally displaced condylar fractures also managed conservatively or by elastic intermaxillary fixation. Two patients had their fractures treated by lag screws.

The miniplates used were the Aus Systems noncompression monocortical miniplates, the Wurzburg noncompression monocortical miniplates, the Medicon noncompression monocortical miniplates, Luhr minicompression plates (used in a noncompression fashion as described by Munro[15] in 1989), and Luhr compression plates.

Although the selection of miniplate was not randomised, no bias has been identified regarding plate selection. Fractures were coded as to severity according to the alphanumeric system of computer-based coding for craniofacial fractures.[16] The complication rate correlated closely with the craniofacial fracture coding score, the correlation being 0.96.[17] However, there was no significant variation in the distribution of fracture severity among the various miniplates (Table II). In addition, analysis of the distribution of fractures (symphyseal, body, angle, ramus, condylar) showed no significant bias in use between the miniplate groups (Table III). Finally, there was no statistically significant variation in the rate of teeth in the fracture line requiring dental extraction. It was not feasible to look at comparison of individual fracture patterns, since 26 patterns were observed.

The results of open reduction and internal fixation at the Australian Craniofacial Unit will be presented in two parts. First, the results as a whole will be tabled, and second, the results of treatment will be examined to compare the different miniplates in use at the unit to identify any discrepancies in outcome related to the type of plates used. The miniplates used during the period of the study are listed in Table IV. The overall complication rate was 15.8 percent and is listed in Table V.

The complication rates for each of the main systems used on this unit (Aus Systems, Würzburg, Luhr noncompression) were then considered individually in an attempt to identify any difference between the complication rates associated with the use of each plating system (Table VI). Only these three major systems are compared, since the others used in this series were too few to be statistically analyzed. These results were compared with a chi-squared analysis (Table VII); $X^2 = 3.842$ (two degrees of freedom), i.e., $0.15 > p > 0.10$.

TABLE II

Comparison of Fracture Severity versus Miniplate Used

	Craniofacial Fracture Score						
	1	2	3	4	5	6	>6
Würzburg	6	22	26	24	10	8	4
Luhr	5	45	19	19	2	5	5
Aus Systems	8	25	30	23	7	5	4

TABLE III

Comparison of Fracture Location versus Miniplate Used

	Fracture site					
	Angle/Ramus		Condylar		Symphyseal/Body	
Würzburg	29	(33%)	19	(21%)	41	(46%)
Luhr	40	(43%)	12	(13%)	42	(45%)
Aus Systems	65	(41%)	27	(17%)	65	(41%)

TABLE IV

Miniplates Used in the Study at the Royal Adelaide Hospital

Miniplate	Number
Aus Systems	105
Würzburg	50
Medicon	11
Luhr noncompression	62
Luhr compression	19

TABLE V

Overall Complication Rates

Complications	Number	Percent
Plate fracture	2	0.8
Infection resulting in removal of plate	7	2.8
Infection responding to treatment	2	0.8
Malocclusion with corrective operation required	13	5.3
Removal of plates	10	4.0
TMJ discomfort	3	1.2
TMJ ankylosis, bilateral reconstruction	1	0.4
Nonunion	1	0.4
TOTAL	39	15.8

TABLE VI

Complication Rates of Individual Miniplates

Complications	Number of Cases (Percentage)		
	Aus Systems	Würzburg	Luhr Compression
Plate fracture	2 (1.9)		
Infection resulting in removal of plate	1 (1.0)	1 (2.0)	4 (6.5)
Infection responding to treatment	1 (1.0)		1 (1.6)
Malocclusion with corrective operation required	2 (1.9)	4 (8.0)	4 (6.5)
Removal of plates	4 (3.8)		5 (8.1)
TMJ discomfort	2 (1.9)	1 (2.0)	
TMJ ankylosis, bilateral reconstruction		1 (2.0)	
Nonunion			
TOTAL	12(11.4%)	7(14.0%)	14(22.5%)

TABLE VII

Comparison of Complications in the Major Miniplating Systems

	Aus Systems	Würzurg	Luhr	Total
Complication	12	7	14	33
No complication	93	43	48	184
TOTAL	105	50	62	217

Discussion

The information that has been presented on a large series of patients contrasting the use of different makes of noncompression miniplates is the first review of its kind of which we are aware. The complications noted by the Australian Craniofacial Unit have been listed in Table V. Comparing results with those published in the literature is difficult because of the different populations these

studies may represent. A different proportionate representation of certain fracture patterns may strongly influence the incidence of complications. The selection of cases for open reduction and internal fixation also may vary between units. At the Australian Craniofacial Unit, 76 percent of patients with mandibular fractures underwent miniplate fixation of their fractures compared with only 13 percent by Iizuka and Lindqvist.[7] The complication rate quoted is that per patient, not per fracture, as is quoted in many series. Reports in the literature of overall complication rates from compression plate osteosynthesis have ranged from 21 to 37 percent.[7,18-20] Ellis and Walker[21] in 1994 compared the use of double miniplate fixation for angle fractures and found only a slight improvement in complication rate as compared with compression plate osteosynthesis (28 versus 32 percent). They suggest that it is unlikely that fracture instability is the major reason for the development of infections in this area. When pure angle fractures are extracted from our data, the complication rate is 24.1 percent, with an infection rate of 8.6 percent. This would appear to be in line with the low complication rates reported by authors employing miniplate fixation according to the techniques espoused by Champy.[5, 8-10, 22-24] There were two significant classes of complications affecting the patients of this unit. The first was a 5.3 percent incidence of postoperative malocclusion that required corrective surgery. This amounted to 13 cases overall. The second major class of complication was infection, which occurred in 3.6 percent of cases. Of the 9 cases of infection, there were no episodes of osteomyelitis. The policy of the Australian Craniofacial Unit has been to treat all but the mildest cases of infection by removal of the plate, debridement, and irrigation as necessary, followed by replating the fracture with Luhr compression plates. In some cases where the fracture appears rigidly fixed and an abscess has been drained, the existing plate will be left in situ. Resolution of the infection and satisfactory union of the fracture were the ultimate outcome for all cases of postoperative infection.

As stated earlier, plates are not routinely removed on the Australian Craniofacial Unit. Plates will be removed for a variety of reasons, including treatment of infection, exposure of the plate consequent on soft-tissue breakdown, and occasionally by request of the patients when they can feel the plates under the soft tissues. In all, 6.8 percent of patients had their plates removed, 2.8 percent as part of management of infection and 4.0 percent for other reasons. The inclusion of these factors in the overall complication rate figures should be recognised, since those who routinely remove plates postoperatively will not necessarily document these as complications.

Noncompression Plating Comparison

The major plating systems used were compared with each other to identify any influences on complication rate that could be attributed to the noncompression miniplate selected. As can be seen from Table VI, the complication rate was similar in the case of the Aus Systems and Wurzburg plates but higher for the Luhr compression miniplates; however, this observed difference was not statistically significant ($0.15 > p > 0.10$).

Therefore, there is no evidence that the complication rate is influenced by the selection of miniplate in this case. If the Luhr compression miniplate (which showed the highest complication rate) is taken out of the equation, then two similar noncompression miniplates with different bending characteristics can be compared, also by means of the chi-squared analysis. Here, $X^2 = 0.096$ (one degree of freedom), i.e., $p > 0.25$.

Thus, since $p > 0.25$, there is no evidence of a significant difference between the complication rate experienced by either the Aus Systems or Wurzburg plates. Thus, although these plates exhibit different stiffness levels, yield points, design, and materials, no relationship between plate selection and treatment outcome was identified. Aus Systems plates were the most malleable, as found in the engineering component of the study, yet no significant adverse clinical results could be detected in the in vivo study in comparison with other plates; indeed, the Aus Systems plates compared favorably.

Conclusions

It is well known among clinicians that noncompression miniplate Osteosynthesis is the treatment of choice for mandibular fractures but that significant differences in design, materials, mechanical properties, and cost exist among the commercially available miniplates. For this reason, miniplates should not be considered as interchangeable. The absence of true randomisation in this study prevents a clear demonstration of the differences in treatment outcome, but no significant variation in treatment outcome has been identified among the noncompression miniplates examined in this study. If this is the case, then miniplate selection should be based on the unit cost, the biocompatibility of the implant, and the CT compatibility of the implant. Further research is required to establish the most appropriate miniplate for a given discrete region by properly randomised trials. In order to gather sufficient data for such trials, a multicenter approach may be necessary, and in this situation the alphanumeric system of computer-based coding for craniofacial fractures would be useful in comparing results.

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Historical perspectives

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Causes of Injury in the Craniomaxillofacial Region

The Head as a Target in War and Crime

Primate evolution has made the human head very vulnerable to frontal impacts. In other species, the face is more protective: birds have beaks, reptiles have armoured snouts and most mammals have prognathous jaws, evolved to house large olfactory organs or for predatory mastication, but also serving to absorb impact energy. In the evolution of *Homo sapiens,* the sense of smell has become less important and mastication less aggressive. Concurrently, the nose and jaws have receded (Fig. 1.1A), and the cerebral hemispheres — enormous when compared with other primates — bulge forward in the frontal region, an attractive target for blows and missiles (Fig. 1.1 B). The frontal skull is thick and has considerable defensive value, both for the brain and for the eyes recessed in the bony orbits, but this skeletal protection has limits and often fails when challenged by blunt or sharp objects.

The vulnerability of the human head would have fewer consequences if we were less pugnacious and less inventive. But *Homo sapiens* is both aggressive and ingenious: wars and murderous assaults are older than recorded history and in these conflicts the face and head have always been a favoured target. Hand-held clubs, spears and swords have been in military use since remote antiquity and so have missiles. Slingshots and arrows were not new when Troy fell, about 1250 BC; Homer's *Iliad* (c. 800 BC) has many very precise references to missile wounds of the brain and face, penetrating wounds from arrows and bronze-pointed javelins, and blunt impacts by stones that could shatter a soldier's helmet; Adamson (1977) found mention of 59 head or neck wounds in Homer's Iliad, of which 45 were lethal.

Weapons driven by human muscles have limited killing power. Muscular energy can be stored in a stretched bow or a tensed catapult and released to propel an arrow or a bolt; the long history of human violence records many facial and cerebral wounds from these weapons. Perhaps the most famous is the death of Harold, king of the Saxons, supposedly from an orbitocranial arrow wound, sustained in 1066, a year renowned as one of the only two 'memorable' dates in English history (Sellers & Yeatman 1930). Asian history records a similar arrow wound, equally dramatic and better authenticated: the second battle of Panipat (1556) was won, and the future of India was decided, when a Mughal arrow blinded the brilliant Hindu general Hemu at a tactical crisis in the battle. Missiles propelled by chemical energy have far greater power. Gunpowder — a mixture of saltpetre, charcoal and sulphur — came into use c. AD 1300, and by the sixteenth century was employed by all civilized armies. Cannon, mines and the matchlock muskets of Spanish, Turkish and Japanese professional soldiers made this century the age of the gunpowder empires, and inflicted appalling wounds that were so often septic that they seemed to be poisoned.

Advances in the design of offensive weapons were paralleled by improved systems of defence, and the obvious vulnerability of the face and head inspired the helmet (Gurdjian 1973). Awareness of the need for brain protection is evident

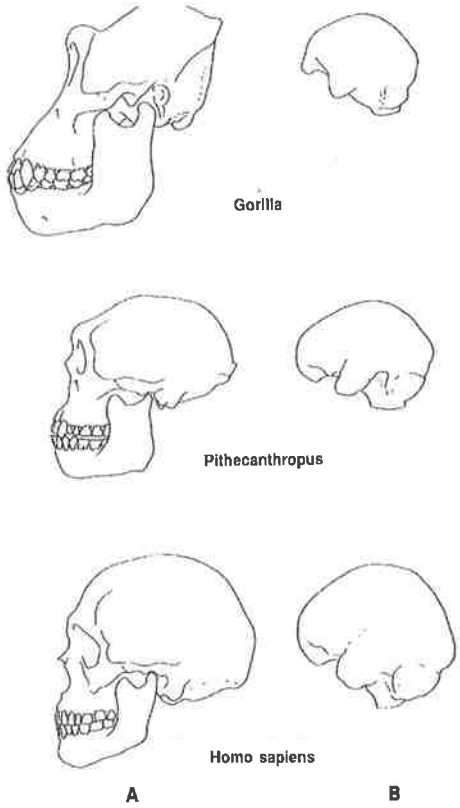


FIG. 1.1. ***The face in primates: a changing target.*** *Craniofacial evolution in the gorilla, a primitive hominid* (Pithecanthropus), *and man* (Homo sapiens). *Reproduced by permission from W. E. LeGros Clark,* The Antecedents of Man. *2nd edn, 1962, Edinburgh University Press.* **A.** *The facial skeleton shows recession of the jaws and increasing prominence of the frontal bone.* **B.** *Endocranial casts show increase in the bulk and prominence of the frontal and temporal lobes of the brain.*

in the leather and metal helmets worn by soldiers in Mediterranean and Middle East armies well before 1200 BC. Greek soldiers also saw a need for facial protection: Greek heavy infantry used well-designed helmets that covered the whole face, with a nasal bar to ward off sword slashes (Fig. 1.2). These full-face helmets restricted vision, and Greek warriors are often shown with their helmets pushed up; the introduction of hinged visors in the European Middle Ages gave the option of an open-faced helmet, and important persons used visored helmets well into the seventeenth century (Fig. 1.3). As late as 1641, a French army commander is said to have inadvertently blown out his brains by pushing up his visor with a loaded pistol. But these visors also reduced the user's field of vision, and some soldiers preferred to take their chances without facial protection. Francois de Guise (1519-1563) got a lance through his face in this way at the attack on Boulogne in 1545: the iron point went in below the eye and out behind the ear, lodging so firmly that it had to be extracted with blacksmith's pincers (Pare 1649, Hamby 1967).

Advances in musketry and military organisation exposed the soldier to increasing numbers of missiles, with increasing accuracy and hitting power (Parker 1988). In the seventeenth century, what would now be called the military-industrial complex, led by arms magnates in Liège, Holland and Sweden, organized mass production of standardised firearms in great quantities. The smoothbored musket increasingly dominated the battlefield (Figs 1.4 and 1.5A); it was soon supplemented by the rifle, more accurate and with greater muzzle velocity. Snipers were armed with rifles as early as the Thirty Years War (1618-1648); rifles were used by guerillas on both sides of the War of American Independence (1775-1781), and by British light infantry in 1800. Breechloaders gave faster rates of fire, but the early designs were unreliable, and in the fierce battles of the American Civil War (1861-1865) both sides chiefly relied on

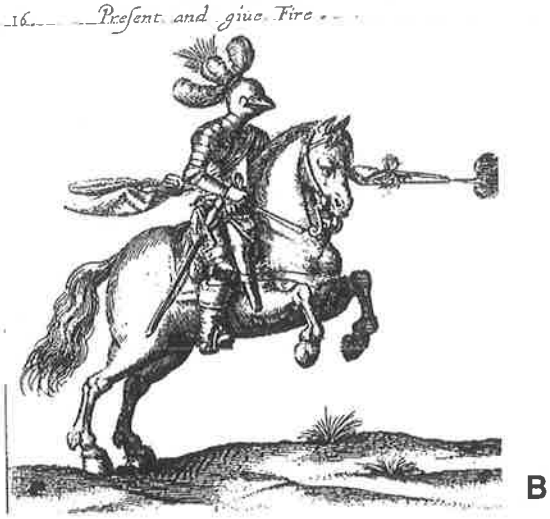
FIG. 1.2. *The Corinthian helmet.* *Bronze helmet, used by Greek heavy infantry (hoplites) in the fifth century* BC. *Reproduced by permission from E. S. Gurdjian,* Head Injury from Antiquity to the Present with Special Reference to Penetrating Head Wounds, *1973, Charles C. Thomas, Springfield, Illinois.*

muzzle-loading 0.58 in. rifled muskets firing 'minny balls', the cylindroconical lead bullets designed by C. E. Minié, a French officer of Chasseurs (Fig. 1.5B). The international armament industry became very competitive: designs were rapidly improved and in the Franco-Prussian War (1870-1871), single-shot breech-loading rifles were in service in both armies. Magazine rifles were introduced in the chief European armies by 1890, and also machine guns. These weapons fired smaller bullets, approximately 0.3 in. in diameter (Fig. 1.5C), driven by new propellants more powerful than gunpowder, giving greater range and impact force. Cordite, a smokeless mixture of nitrocellulose and nitroglycerine, gave the British .303 in. service rifles of 1900 a muzzle velocity in the range 600–800 m/s, whereas smoothbore muskets fired with black powder seldom bettered 300 m/s. The first Hague Convention (1899) outlawed expanding bullets, but to offset this humane aspiration, new bullet designs produced high velocity missiles able to cause still more massive tissue destruction. The effects of higher muzzle velocities on the face and brain are discussed in Chapter 4. Automatic rifles and hand-held submachine guns came into general military use during World War II (1939-1945), at first for close combat, but later as standard equipment; today, the Russian Kalashnikov 7.62 mm assault rifle is the characteristic weapon of the terrorist/freedom fighter throughout the world.

Naval cannon appeared very early, and became the decisive weapon at sea, where the impact of roundshot was multiplied by splinters struck from the ship's timbers. Cannon firing musket balls in bags or in a tin case inflicted appalling wounds at close range. Richard Wiseman (c. 1622-1676), then a young surgeon serving on the king's side in the English Civil War (1642-1646), noted this at the siege of Taunton.

> One of Colonel John Arundell's men, in storming the Works, was shot in the Face by Case-shot. He ... was carried off among the dead and laid in an empty house.
>
> In the morning early, the Colonel marching by that house heard a knocking within against the Door. Some of the Officers desiring to know what it was, lookt in, and saw this man standing by the Door without Eye, Face, Nose, or Mouth ... His Face, with his Eyes, Nose, Mouth, and forepart of The Jaws, with the Chin,

A



B

C

FIG.1.3. Full head protection in the sixteenth and seventeenth centuries. **A.** *Tournament helmet, made for King Henry VIII of England in 1540. The hinged visor gave facial protection but limited vision. By courtesy of the Board of Trustees of the Royal Armouries, UK.* **B.** *Cavalry trooper of the Thirty Years War, charging with his visor down.* **C.** *After he has fired, he reloads with his visor up. From John Cruso,* Militarie Instructions for the Cavallerie, *1632*

FIG. 1.4. ***Frontal missile wounds in the Napoleonic wars.*** *Cerebral fungus or herniation resulting from a musket ball wound, presumably sustained at Waterloo 18th June, 1815. Herniations of this type have been a common sequel of battlefield wounds as late as World War II. From C. Bell,* Illustrations of the Great Operations of Surgery, *1821.*

was shot away, and the remaining parts of them driven in. One part of the Jaw hung down by his Throat, and the other part pasht into it. I saw the Brain working out underneath the lacerated Scalp on both sides between his Ears and Brows. (Wiseman 1977)

The man lived at least 6 days, being able to drink by holding down the root of his tongue.

Concurrently, advances were made in killing by fragmentation missiles. Hand-thrown grenades and bombs shot from mortars were used in the sixteenth and seventeenth centuries; shells fired from howitzers came somewhat later. Difficulties in devising accurate fuses were slowly overcome; in the siege of Gibraltar (1781) Henry Shrapnel, a young English officer, saw the benefits of using shells so that they would burst in the air and by the time of the American Civil War, explosive shells accounted for a sizeable minority of head wounds (Otis, 1870). In the great wars of the twentieth century, more soldiers were killed or wounded by shells than by bullets (Reister, 1975). In World War I (1914-1918), shrapnel shells charged with high explosives such as trinitrotoluene caused so many head wounds that steel helmets came back to give head protection especially against relatively low velocity fragmentation missiles (p. 115). Such helmets remain a military necessity, and are now fitted with polycarbonate visors for eye protection; steel has been replaced by laminated composites of aramid fibres (Kevlar®), giving 35% greater protection without increased weight.

The brothers Wright, and also Graf Zeppelin, made it possible to drop bombs from the air. This was also outlawed by the Hague convention, but ten years after the Wrights' first flight in 1903, the potential of air attacks was being eagerly touted by science-fiction writers like H. G. Wells and by the nascent aerospace industry (Grahame-White & Harper 1914). High explosive bombs were used early in World War I, and also incendiary bombs, containing phosphorus and other combustibles.

Fire is a very ancient weapon and wartime facial burns have a long history. Byzantine warships carried 'Greek fire', an inflammable liquid containing bitumen, naphtha or pitch, which was sprayed by bellows through copper nozzles (Watt 1980); this was used in sea fights with the Arabs as early as the seventh century AD. In 1915, burning oils propelled by compressed nitrogen were used in trench warfare, initially with terrifying effect. Since the first use of gunfire in naval war, burns have been a dreaded feature of sea fights; even modern warships burn furiously, as was seen in the Falklands War. Fire is a horrible concomitant

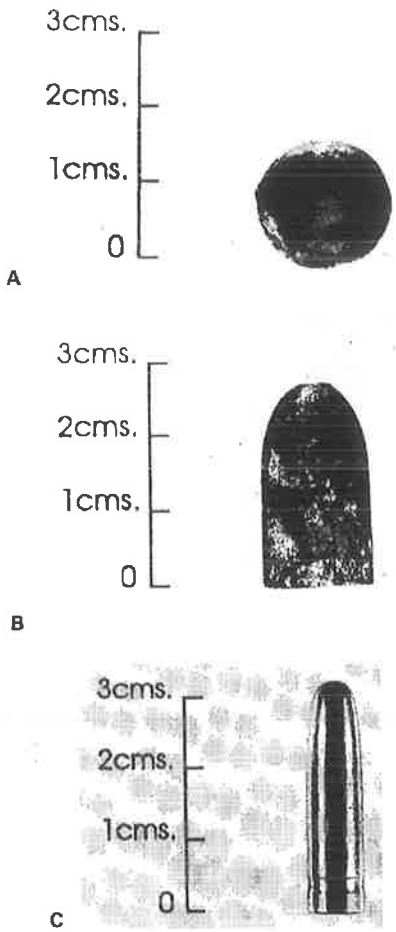


FIG. 1.5. ***A century of military missiles.*** **A.** *British service musket ball, diameter 0.693 in., as used at Waterloo. By courtesy of the Trustees of the Imperial War Museum, London.* **B.** *British Enfield 1853 rifle bullet, diameter 0.550 in., with a hollow base to engage in the rifling of the barrel when fired. Such bullets were used by both sides in the American Civil War, though lacking the grooving of the United States Army pattern Minie bullet. By courtesy of the Trustees of the Imperial War Museum, London.* **C.** *British Army Service rifle bullet, diameter 0.303 in., as used in the First and Second World Wars: probably a Mark VI bullet, approved in 1904.*

of air warfare, both for aircrew and for the victims of bombing; napalm (a mixture of petroleum and palm oil derivatives) has often been dropped from aircraft, with varying degrees of discrimination, and white phosphorus has also been used. These agents are capable of inflicting very severe facial burns (Fig. 17.11).

As might be expected, firearms were early used in crime as well as in war: muskets and pistols were used in several sixteenth-century political assassinations, and private ambushes are mentioned by surgical writers of the period. As military firearms evolved and diversified, the criminal classes were quick to see the utility of the smaller handguns. Pistols, originally used chiefly as a cavalry weapon, were easily hidden in pockets or under cloaks; in violent societies, pistols came to replace the sword for 'self-protection' and as a part of the male image. British duellists took to pistols rather than swords in the eighteenth century, and became even more dangerous. It seems that the usual target was the trunk, but head wounds were not unknown. The Irish surgeon Sylvester O'Halloran (1728-1807), in his racy account (1793) of the high incidence of head injuries in Munster ('our people, invincibly brave, ... and highly irritable, soon catch fire ... to this add the frequent abuse of spirituous liquors, particularly whiskey ...') reported one such duel: the gallant O'C., an excellent shot, was himself struck by a ball in the temple and died of what reads like cerebritis. These duels were usually fought with single-shot pistols. There was felt to be a need for pistols capable of more than one shot and in 1835, Samuel Colt patented

his famous revolver: it was not a new idea, but Colt's reliable weapons became popular throughout the world, and their descendants are still very much with us. While rifles can be used in hunting animals, pistols are primarily intended to kill people, and as soon as they became cheap enough to be purchased by the lower classes, governments began to try to restrict possession of these dangerous weapons to those considered to be socially responsible. In 1602, the viceroy of Catalonia prohibited pistols less than 90 cm long, 'in an attempt to reduce the death rate in the principality' (Elliott, 1963); the spirited Catalans greatly resented this early gun law and it had little effect. Later gun laws have had varied success, but no government has succeeded in the logical measure of total abolition, except the Tokugawa administration in Japan. Ieyasu, the first and greatest Tokugawa shogun, took control of the firearms industry in 1609, and his successors cut back production until the nation was virtually without guns (Parker 1988).

The Head in Traffic Accidents

War is of course not the only historic cause of injuries of the face and head. Traffic accidents are today the leading cause of serious head injuries in most parts of the world, and these also have a long history. The horse has been domesticated for more than four millennia. In Iraq, the Sumerians used horses to pull chariots around 2000 BC and somewhat later horses were ridden, with control by bridles and bits. Nobody who has ridden or driven horses will doubt that this great technical achievement must have led almost immediately to accidents and head injuries. There is an account of a Chinese chariot accident in the oracular archives of the Shang dynasty (c. 1600–1000 BC): one of King Wuding's courtiers was thrown out of his chariot when it hit an obstacle at high speed. (Gao Jian-Guo, personal communication.) A few hundred years later there is a literary reference to a chariot accident causing craniofacial injury in Homer's *Iliad*. Eumelus is thrown in the course of a very dirty chariot race, when his vehicle is sabotaged by the goddess Athene: he suffers facial abrasions and is temporarily speechless, but recovers. No doubt there were many such accidents; a galloping horse can reach a speed of 50 km/h, and at this speed fatal head injuries can easily result from falls, impacts against trees or other overhanging objects, or collisions with pedestrians. In imperial Rome, better roads and a very self-indulgent aristocracy gave rise to complaints about bad driving (Ammanius Marcellinus quoted by Gibbon 1854). Juvenal (c.AD 60-140) also listed heavy road transport as one of the lethal hazards of Roman traffic.

In the Renaissance, there are several case reports of facial and cranial injuries from riding accidents. Giovanni da Vigo (?1460-1520) mentions a gentleman in the service of the duke of Urbino who fell from his horse onto a rock surface: his whole face swelled, and he was mute for 20 days, but recovered with a permanent speech defect that sounds like a dysphasia. Transport accidents were common in the seventeenth century. In the civilian practice of Richard Wiseman, such accidents apparently accounted for a fifth of all the reported injuries; a typical example is the Lady who was wounded 'down the whole length of the Forehead to the Nose, also transverse the left Eye-brow...; her Eye and Face were also much bruised. It happened to her travelling in a Hackney-Coach [i.e. a taxi], upon the jetting [jolting] whereof she was thrown out of the hinder Seat against a Bar of Iron in the forepart of the Coach' (Wiseman 1977). In the eighteenth century, Percivall Pott (1714-1788) described 43 cases of head injury, of which 12 (27.9%) were riding or carriage accidents, some of them alcohol-related: the series gives a picture very familiar to modern accident surgeons (Table 1.1).

In the nineteenth century, the horse was supplemented by steam power. Passenger-carrying steam trains came in England in 1825, and as early as 1839 the Great Western Railway provided a fine museum specimen of a skull base fracture for the Royal College of Surgeons. Steam road carriages followed, but were restrained by a speed limit of 4 miles/h, enforced by an act of parliament that required a man holding a red flag to precede the steam car.

TABLE 1.1

Head injuries in eighteenth century London (from Pott 1779)

		No. of cases	%
Industry	Scaffolding	5	
	Cranes	2	
	Other	4	
	Total	11	25.6
Roads	Carriages and carts	6	
	Riding horses	5	
	Hitting horses	1	
	Total	12	27.9
Assault etc.	By relatives or neighbours	4	
	By rioters	2	
	By school masters	1	
	By the French (naval war)	1	
	Suicides	1	
	Total	9	20.9
Sport	Cricket	2	
	Quoits	1	
	Cudgels	1	
	Total	4	
Miscellaneous	(including falls)	7	16.3
Total		43	100.00

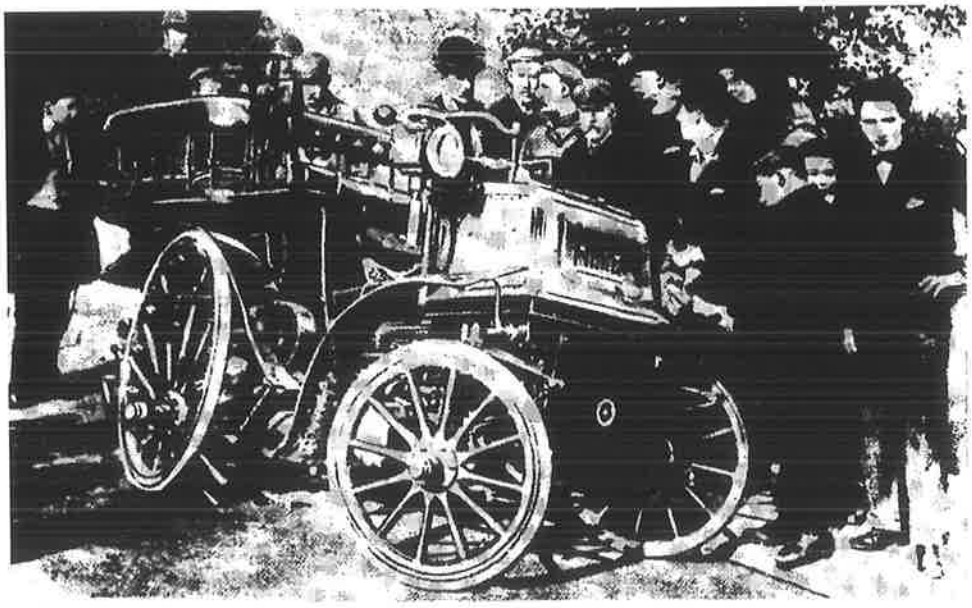


FIG. 1.6. *'Sad fatality at Harrow'.* *Accident to a Daimler waggonette driven by Engineer E. A. Sewell when carrying five gentlemen on 25 February 1899. The right rear wheel collapsed at an estimated speed of 15 miles/h Sewell and his front seat passenger were ejected and killed. By courtesy of Dr G. A. Ryan.*

The modern motor car was born in 1884-1886, when Gottlieb Daimler (1834-1900) invented an effective internal combustion engine powered by petrol, and installed it in a four-wheeled vehicle; the horseless carriage then became a practicable means of road transport (Ovcry 1992, Lay 1993). At first the killing power of the motor car was underestimated, and complaints centred on its effects on the emotional balance of horses. The first automobile crash is said to have

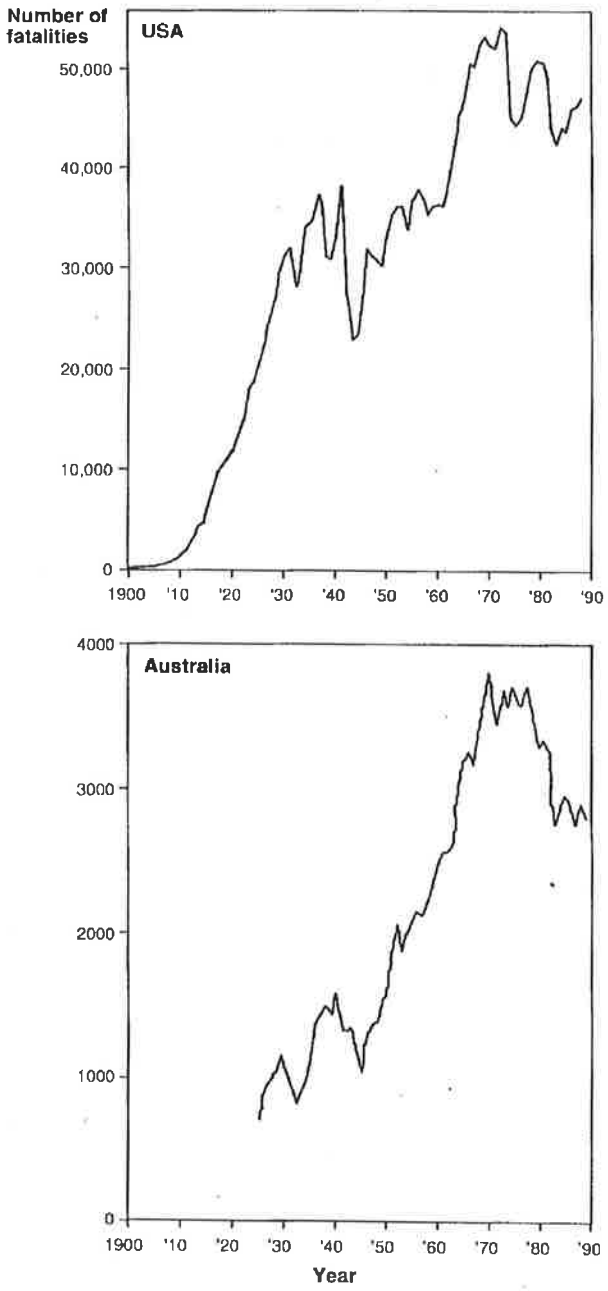


FIG. 1.7. ***Road deaths in the USA and Australia,*** *1900-1990. The graphs show simultaneously a fall after 1970, presumably as a result of safety measures. By courtesy of J. W. Knott (in press) and L. Evans (1991).*

occurred in France, in 1893, when a vehicle driven by Baron de Zuylen struck a motor brake belonging to the Comte de Dion; happily these noblemen escaped serious injury (Ryan 1965). Lay (1993) gives 1896 as the year of the first motor martyrs; a London pedestrian, Ms Bridget Driscoll, was killed despite warning cries from the driver of a car, and in the same year Emile Levassor was killed in a motor race from Paris to Marseilles. In 1899, one of Daimler's fine wagonettes demonstrated its killing power near Harrow (UK). A wheel broke — it was made of inferior (non-British) oak — at perhaps 14 miles/h (Fig. 1.6). The driver (Engineer E. A. Sewell) was killed instantly, and a passenger, Major J. S. Richer, died a few days later in coma (Anonymous 1899). After the World War I the rising mortality rate from car crashes caused great public concern in the USA and other motorized societies, and this has mounted ever since, until at last concern has been implemented in legislation and the road toll has in most countries begun to fall (Fig. 1.7).

The Head as a Target in Sport

Boxing is an ancient sport. Greek and Roman boxers fought wearing the cestus, a leather knuckleduster, and there are literary accounts of well-placed blows damaging the face and brain. In Vergil's *Aeneid* (written 30-19 BC) a champion boxer wears gloves loaded with lead and iron, and stained with blood and brains from previous sporting contests. In nineteenth-century England, boxing was made more humane by padded gloves and by the rules associated with the 8th Marquis of Queensberry, Oscar Wilde's adversary, described by him as the screaming scarlet marquis. Boxing persists today, as the only sport in which brain injury, real or token, is the chief aim.

In the past, there were other sports in which the head and face were targets. Roman gladiators suffered head wounds; the great Galen (c. AD 131-199) spent some years as surgeon to the gladiators of his native town Pergamon, and mentions their depressed skull fractures. Medieval tournaments often ended with head injuries; even the formalized contests of Renaissance elites could have unhappy consequences. Both Henry VIII of England (1491-1547) and Henri II of France (1519-1559) suffered head impacts when jousting in full armour. Henry VIII survived this head injury, and also another sustained in pole vaulting: his apologists have attributed his later behaviour to traumatic or anoxic brain damage. Henri II died of an accidental orbitocranial wound complicated by a chronic subdural haematoma or empyema. For the lower classes there were other martial sports, such as cudgels. Percivall Pott (1779) mentions a young player who received a blow on the forehead: 'it did not seem to himself or to the spectators a severe one, but as it produced blood, it was deemed by the laws of the game a broken head', and he lost the match, as well as developing what sounds like an extradural abscess.

Sports played with balls, or bats and balls, are not intended to cause craniofacial injuries, but have often done so. Again, Pott provides a typical eighteenth-century example: a boy struck in the forehead by a cricket bat, who later developed a frontal extradural abscess, cured by trephining. Cricket ball injuries of the head or face became quite common in the twentieth century, with the rise of fast bowling aimed at the body of the batsman; in the 1977/8 World Cup Cricket Series, a leading batsman first appeared wearing a helmet, and since then serious players of cricket usually wear helmets with visors. Baseball batsmen also use helmets. Football might be thought less likely to lead to head injuries, but the vigour of the game and its opportunities for head contacts make this, in all its forms, a common cause of craniofacial injury, in the past as now. Sir Thomas Elyot (c. 1495-1546) saw the Tudor form of football as 'nothyng but beastely fury and extreme violence, whereof proceedeth hurte and consequently rancour and malice do remayne with thym that be wounded.' However, football is not usually thought to demand protective headgear, except in the USA where fullface helmets have long been considered necessary for serious players. Richard Schneider (1966), an authority on the American form of this robust game, collected 225 cases of serious or fatal injury from football over a 5-year period: of these, 75% were craniocerebral injuries.

Craniofacial Injury in Falls and Industrial Accidents

Falls must always have been responsible for such injuries. Industrial head injuries are doubtless as old as building scaffolds and underground mines; in Pott's (1779) series of head injuries, industrial accidents were almost as numerous as road accidents. Steam engines, introduced into the mining industry in England as early as 1712, presumably increased the risks. In the nineteenth century, concern over industrial accidents was widespread; Hudson (1877) described head injuries among Cornish miners, and mentioned the routine use of protective hard hats. Facial burns and scalds were also a frequent concomitant of the industrial revolution.

Facial Mutilation

In the ancient world, punitive facial mutilation was a favoured way of humiliating and tormenting one's enemies (Adamson,1990). Later, such mutilations were reserved for criminals, especially those of low social rating; in 1637, the King of England's judges removed the ears of a barrister, a clergyman and a doctor of medicine, and this was meant as an especially humiliating punishment for persons of upper middle class status, who would not ordinarily be penalized in this way. In India, nasal mutilation was used on a large scale and for a long time. Thus, Tipu Sultan (1750-1799) the brilliant ruler of Mysore, when at war with the British, offered a pagoda (gold coin weighing about 3.4 g) for each nose removed from a supporter of the English East India Company (McDowell, 1977); modern plastic surgery has been dated from the successful treatment of a victim of this campaign (p. 16). Still more recently, similar mutilations were reported in Africa, during wars of liberation: persons caught smoking enemy cigarettes were likely to lose their lips.

Apart from these punitive disfigurements of the face, many ancient and recent cultural practices involve mutilations of the face and orofacial soft tissues, whether as rituals or for aesthetic reasons. Examples of decorative mutilation include chipping and filing tooth crowns, tooth adornment with inlays or overlays of various materials, evulsion of teeth, facial tattooing, septal perforation, piercing and plugging of the lips, removal of the uvula and facial scarring (Wilson et al 1992).

In most parts of the world today, judicial and political penalties do not include facial mutilation; it is perhaps the only historic cause of facial injury that has been largely eliminated. Decorative mutilations are also less prevalent, though it is possible to find parallels with these in some aspects of modern aesthetic surgery.

The Evolution of Surgical Concepts and Techniques

Craniomaxillofacial Surgery in Antiquity

The record of craniomaxillofacial (CMF) trauma over the last 4000 years is paralleled by the history of endeavours to heal the wounds, mend the fractures and restore an acceptable appearance, undertaken by men and women who can be called surgeons, whether they were seen by their contemporaries as priests, wizards, physicians or skilled craftsmen. The Edwin Smith Papyrus, written about 1600 BC but conveying much older thinking (Breasted 1930), gives in didactic form the practice of an Egyptian surgeon. Cases are described, with treatment policies: nasal fractures and dislocated mandibles are to be treated, but skull fractures with neurological signs such as a squint or gait disorder, or infected jaw fractures, are illnesses not to be treated (Hoffmann-Axthelm, 1982). Whereas much Egyptian medicine is concerned with drugs and incantations, the surgeon behind this papyrus seems rational and objective. Majno (1991) notes that Egyptian wound treatment included skin closure by adhesive tapes and possibly by sutures; he has tested the bactericidal action of the favourite Egyptian wound salve, honey and grease, and found it effective against staphylococci and coliform bacilli.

In ancient Iraq, medical literature surviving in cuneiform inscriptions stresses the therapeutic role of magic. Excavations from Nineveh are said to have discovered scalpels, a saw and a bronze trephine, but there is apparently no literary evidence of surgical treatment, beyond the famous malpractice laws of Hammurabi enacting amputation for unsuccessful operations on persons of noble rank, and lesser penalties for accidents in operations on slaves.

Information on Indian medicine stems from the Sanskrit Vedas; these include the Susruta Samhita, supposedly written around 600 BC, which describes

surgical procedures including restoration of a mutilated nose by swinging down a pedicled flap from the forehead. This procedure is the Indian rhinoplasty, practised by members of a specific caste over the next 2000 years and still in general use (Fig. 17.18).

The surviving memorials of trauma management in antiquity portray the responses of thoughtful surgeons confronted with the challenge of facial wounds and skull fractures. Doubtless there were many such surgeons who left no written memorials, yet transmitted their skills and experience verbally to enrich surgical thinking in India, the Middle East and the eastern Mediterranean.

Graeco-Roman and Arabic Surgery of the Head and Face

In the fifth century BC, Kos, a small Greek island state, produced the towering but shadowy personality of Hippocrates (c. 470-400 BC). The Hippocratic collection, the body of writings attributed to him but now thought to have been written somewhat later, inaugurated a succession of surgical texts that were extended through the Hellenistic and Roman periods, to live on for more than a thousand years in Byzantine compilations. In the field of CMF surgery, the most interesting works in the Hippocratic collection are the treatises 'On Wounds in the Head' and 'On Joints' (Hippocrates, 1927). The first describes trephining for head wounds. It is clear that the operator was careful and thoughtful, with a proper regard for the dangers of penetrating the intact dura mater, and had plenty of experience of wound infection. It is much less obvious why he operated. It appears that depressed fractures of the skull were left to suppurate, in the expectation that the bone fragments would sequestrate and be discharged; fissured fractures were, however, explored aggressively. De Moulin (1988) states that the operator was looking for intracranial bleeding; Majno (1991) sees the operation as intended to let out blood that would otherwise turn to pus. The management of various types of fractures of the mandible is more comprehensible. The fracture was immobilised by interdental wiring, using gold wire or thread, and splinted externally with leather glued to the skin and tied behind the head. Union was expected in only 20 days. Hippocratic surgery also included primary suture of some wounds, especially in the face: the wound was stitched up after being washed out with wine, and dressed with an ointment containing copper oxide and honey. Majno (1991) has tested the antibacterial effect of wine and found it quite potent: it appears that this is due to polyphenols in the wine, not to ethyl alcohol.

The Roman conquest of Greece widened the influence Greek culture; Roman medicine was a partnership based on Italian organisational skills and Greek science. The teachings of Hippocrates and the discoveries of his successors in Alexandria were studied and elaborated by Roman writers such as Cornelius Celsus (c. AD 30). Celsus has been disparaged as a popularizer of current medical knowledge, not an original thinker, yet his very readable work is representative of the best state of the art in imperial Rome. Celsus (1938) gave a good account of plastic closure of areas of facial tissue loss, by two quadrangular advancement flaps sutured together to close the defect; he used thread sutures. Nasal injuries were seen as causes of deformity and airway obstruction: nasal displacements were reduced with the fingers or with a probe or a quill in the nostril(s), and immobilised by internal and external splints. Fractures and dislocations of the mandible were described; interdental horsehair ligatures were used. His account of the surgery of head injury gives some idea of the indications for trephining the skull. He enquired first about neurological symptoms, such as vomiting, loss of speech, and obscured vision. These symptoms might point to a skull fracture in need of treatment. More serious symptoms, such as coma ('torpor'), suggested that the dura mater was violated, and operation might be ineffective. Irritation of the meninges was seen as a cause of harm — a concept that was to have a long life. Celsus recognised the danger of bleeding under the intact skull, presumably extradural, and advised operation for cranial osteomyelitis. His tools included the modiolus, an iron cylindrical trephine, and the terebra, ancestor of the modern perforator: large areas of bone were excised by making a ring of holes with the

terebra and joining the holes with chisel cuts. All in all, it appears that the operative craniofacial surgery of Celsus went to the limits of what was then possible, given the absence of anaesthesia and the consequent need for speed and dexterity: Celsus noted that the surgeon must be compassionate, yet unmoved by his patient's screams.

Galen (c. AD 130-199) is a greater figure in medical biology than in the history of surgery. It is even suggested that he had no practical surgical experience, but this is nonsense. In his work as physician to the gladiators of Pergamon, he certainly had to treat the less fortunate sportsmen, and he wrote confidently of operations on the skull (Galen, 1976). His animal experiments suggest remarkable manual dexterity, and he was a fine anatomist, though he had to study monkeys and other animals, being debarred from using human cadavers. (Human anatomy had been studied in the cadaver in Hellenistic Egypt, but this invaluable aid to good surgery had been abandoned.) His experiments included making brain lesions in conscious animals: he was able to demonstrate the serious effects of damage to the cerebral ventricles, especially the fourth ventricle, and he noted similar effects from accidents in trephining the human head. The awareness of the lethal effects of cerebral compression, both from clinical observation and from animal experiment, foreshadows a central concept in modern neurosurgical thinking. But Galen's main surgical legacy was a very complete system of pathophysiology; his concepts of cardiorespiratory and neurological function were profoundly wrong, but they were very convincing and had enormous influence. Whatever its scientific demerits, Galen's system gave surgeons a set of working dogmas that were doubtless just as helpful as more correct knowledge would have been, given the limitations of what was possible. The Catalan Joseph Trueta (1897-1977), in our times a most influential military surgical writer and a great surgical biologist, blames Galen for many things, and particularly for a fundamental error that prevented progress for fourteen centuries — the belief that suppuration is a benign and indeed essential process in wound healing. In this, he is contrasted with Hippocrates (Trueta 1944). This seems rather unfair. There were writers before Galen who saw suppuration as a favourable event, while Galen himself clearly regarded healing by first intention, i.e. without suppuration, as the most desirable outcome (De Moulin 1988).

Graeco-Roman medicine gave very serviceable rules for the management of facial injuries; for craniocerebral injuries, the Hippocratic legacy was less helpful but at least the basic surgical armamentarium was defined. Galen's concepts of Immoral pathology and his emphasis on venesection were certainly unhappy; he bears much responsibility for the 'therapeutic' bleeding of wounded persons over the next 1700 years. After Galen's death, there were no great speculative or experimental medical biologists in the declining Roman world, but his ideas, and those of his predecessors, were kept alive by surgeons in the Byzantine cities, and by them transmitted to the Arab world.

The great surge of Islam established a brilliant and tolerant cultural unity from Delhi to Cordoba. In the intellectual centres of the Eastern and Western Caliphates, Graeco-Roman medicine was studied and advanced. But surgery received relatively less attention, and most of the great figures of Arab medical science were primarily concerned with internal medicine. The chief exception was Albucasis, in Arabic Abu'l-Qasim (936-1013). His surgical textbook was translated into Latin in Toledo, and widely read in western Europe; he reiterated Hippocratic teaching on the treatment of fractures of the mandible by dental ligatures and external splints. It is also relevant that Albucasis is credited with the belief that atmospheric air will infect wounds. This concept, not an unreasonable deduction from everyday experience, was to have wide currency, and especially in the misunderstanding of brain infections.

Medieval Surgery of the Skull and Face

Some ten centuries separate the political and social disintegration of the Western Roman Empire from the dawn of the Renaissance in Italy. In that long period,

Western Europe produced few surgical writers of distinction or originality, and little of what was written is relevant to the theme of this book. To a considerable extent, learning was confined to the ecclesiastical establishment, and as clerics were prohibited by the Church from shedding blood, there was a cleavage between the learned physician who did not operate and the surgical craftsman who did not study. Nevertheless the picture of medieval scientific sterility and clerical obscurantism has been much exaggerated. In Italy especially, there were priests who did operate, even after this was banned by the Fourth Lateran Council in 1215. In Salerno, a multicultural seaport south of Naples, a lay medical school emerged around 900, and flourished for some 300 years: the surgeons of Salerno studied anatomy, performed autopsies, and reported many cases of cranial and facial wounds, including a fatal extradural haematoma from a slingshot impact (De Moulin 1988). Before operating on the brain, the Salernitan surgeon washed his hands, a practice that had to be rediscovered in the nineteenth century, and also abstained from sexual intercourse and eating garlic. Universities, perhaps the greatest intellectual achievement of the Middle Ages, established medical faculties, first in Bologna (1156) but soon elsewhere, and in Italy these universities taught surgery and anatomy, with cadaver dissections from the early fourteenth century at latest. De Moulin (1988) has reviewed the surgical practice of French writers such as Henri de Mondeville (c. 1260–1320) and Guy de Chauliac (c. 1298-1368): both gave attention to head injury management, and had sensible things to say on wound healing.

The Sixteenth Century

Seen in retrospect, this century appears as a time of exhilarating intellectual adventure. The religious revolution in Europe shattered old beliefs and assumptions. The oceanic voyages of discovery expanded the world enormously, and brought back new diseases and new drugs. Humanist scholars rediscovered or renovated Greek and Latin texts, and the printing press spread their findings in an enlarged intellectual community that extended from Poland to Portugal and beyond to the Americas. Medical texts were studied more widely: Celsus was printed in 1478, and much of Galen's work was published in Latin translation in 1490. An increased interest in human anatomy followed. Surgery, stimulated by the effects of the terrifying new firearms, shared in the expansion of medical science. The northern Italian universities, already old as centres of learning, were in the vanguard of the advance of surgical thinking. Padua, the university of the powerful republic of Venice, was pre-eminent, and its professorial chairs were held by a number of outstanding physician-surgeons, one of whom was Andreas Vesalius (1514-1564) of Brussels.

1543 is often given as the landmark year of Renaissance science, when Vesalius brought out his great textbook of anatomy, *De Humani Corporis Fabrica*. Vesalius was at once a learned Galenist — there are more than 200 citations of Galen in the index of his book — and an iconoclastic revolutionary, correcting Galen's anatomical errors on the basis of his own meticulous dissections. His book gave surgeons a superbly illustrated textbook that emphasized function as well as structure. In particular, it gave a fine description of the skull and facial skeleton, and of the external morphology of the brain and cranial nerves. Vesalius was himself a daring surgeon, not least in the field of CMF trauma, as is evident in a famous case report from his practice after he left Padua. In 1562, Don Carlos, the young and very unsatisfactory crown prince of Spain, fell down a flight of steps 'in hasty following of a wench', and suffered a contused wound of the scalp, which became infected. His conscious level worsened, and Vesalius, who was then one of the royal physicians, evidently diagnosed intracranial suppuration. 'Dr. Vesalius was of the opinion that the lesion was inside and that there was no other remedy but to penetrate the skull to the membranes'. This was not done, but the later course of the illness makes it clear that Vesalius was right in his diagnosis (Simpson 1987).

The sixteenth century saw many notable surgical authors who wrote on the management of CMF trauma. Berengario da Carpi (c. 1460–1530) wrote a

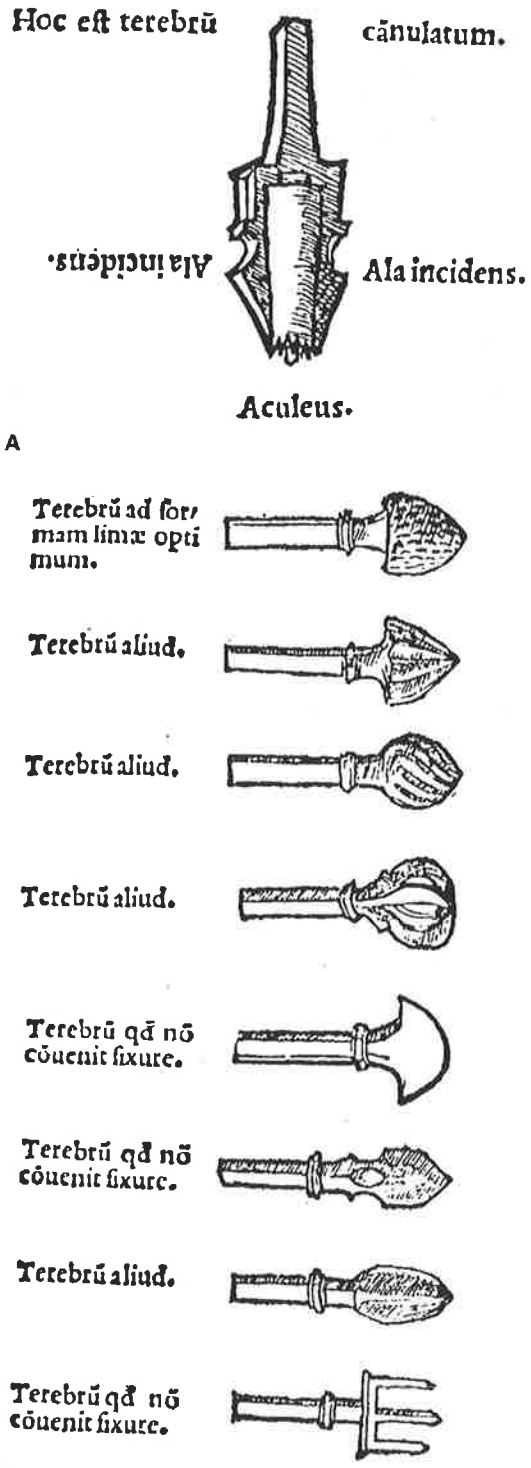


FIG. 1.8. ***Cranial surgery in the Renaissance.*** *Craniotomy instruments used by Berengario da Carpia, 1518.* **A.** *Modiolus or trephine, with adjustable guard against overpenetration.* **B.** *Range of perforators and burrs.*

book, De Fractura Cranei, dealing specifically with head injuries, and remarkable for its emphasis on their symptomatology. This work also figures the instruments of the time; it is interesting to see that the modiolus has guards to prevent overpenetration, and the terebra or perforator is supplemented by a burr (Fig. 1.8) of modern design. Other influential writers were the Italian Giovanni da Vigo (c. 1460–1520), an enthusiast for the cautery, and the German Hieronymus Braunschweig (1450–1533). Also from the Germanic world was the great Paracelsus (Philipp Bumbastus von Hohenheim, c. 1494–1541). Paracelsus exemplifies the Renaissance, in his flamboyant iconoclasm — he ceremoniously burned one of the chief traditional textbooks — and his readiness to explore new

fields; even in his interests in magic and the occult, to us less admirable, there is a gusto that still fascinates. In surgery, his *Grosse Wundarznei* and his *Spitalbuch* have much to say about wound treatment, but they are even more remarkable for the author's total rejection of the traditional separation of medicine and surgery: 'there can be no surgeon who is not also a physician' (Pager 1982). But the best known, best loved and most typical Renaissance surgeon is Ambroise Paré (c. 1510-1590), and his writings have much that is relevant in an evaluation of the evolution of the surgery of CMF trauma.

Paré was born in Bourg-Hersent, a small village in Mayenne, but went early to Paris. It appears that he learned his profession as apprentice to a barber-surgeon, and also in the Hotel-Dieu, the chief public hospital in Paris. He had no university education: in France, unlike Italy, universities did not teach academic surgery. Paré went to Italy in 1537 as personal surgeon to a commander in the French royal army; thereafter, he served in many campaigns in the wars with the German emperor Charles V and his allies. In 1559, he attended his king Henri II after the fatal tourney accident described above, together with Vesalius, who was sent to Paris by the concerned king of Spain, Philip II. Paré also attended many leading figures in the religious wars that broke out after the death of Henri II. He became immensely experienced, he designed many surgical instruments and prostheses and he wrote many books — in vigorous French because he was a poor Latinist. His management of head injuries is described in the tenth book of his 'Complete Works' (Pare, 1649). His chief indication for operation on the head appears to have been a compound depressed fracture, to relieve pressure on the meninges and to allow the escape of 'corrupt and putrid blood'. It is not always clear whether this meant evacuation of pus or blood clot. He used a trephine on a brace, a gimlet perforator, and also a compass saw for larger bone resections. Great care was taken to preserve the dura mater; bone fragments were removed with elevators and forceps. Bone still attached to the pericranium was not removed. Simple scalp wounds were pulled together with a few sutures, or otherwise closed; deeper wounds were often left open to granulate.

Paré described his management of injuries of the face and jaws, and of the eyes, in the same book. Facial wounds were carefully sutured with waxed thread, or closed with strips of sticking plaster sutured together (Fig. 1.9). Fractures of the mandible were treated as advised by Hippocrates, with interdental wiring using gold or silver wire and an external leather restraint sewn to the patient's night-cap. Union of the fracture was expected in 20 days, unless 'inflammation' should supervene. Presumably the figure of 20 days is derived from Hippocrates: Paré qualified it by noting that patients vary in the time needed for union. Paré had little to say about midfacial fractures, though he quoted the gratifying case of a soldier who suffered a disfiguring compound wound of the upper jaw, which became infested with worms because of delay in getting surgical aid, and stank. With appropriate local medication and delayed suture, the wound eventually healed very well. Nasal wounds and fractures were discussed in more detail. Paré noted that an amputated nose could not be replaced, but if there was any adherence to the rest of the adjacent flesh, 'from whence it may receive life and nourishment', then it should be sewn back. His ability to treat eye injuries was limited, but he removed corneal foreign bodies, using a speculum, and was aware that penetrating eye wounds could heal well if the pupil was not injured. Human milk was an excellent irrigating fluid for injured eyes, especially if the donor was suckling a girl. Paré's contemporary Georg Bartisch (1535-1605), court oculist in Dresden, was more adventurous as an eye surgeon: he described the operation of enucleation, and is said to have been the first to note sympathetic ophthalmia as a sequel of eye injury (Albert & Diaz-Rohena, 1989).

Paré described in much detail his various wound dressings; distilled spirits were often used, and may have had some antimicrobial effect, though of course he could not know this. He used frequent venesections for head injuries, in line with traditional Galenic practice, bleeding from the cephalic vein 'according to the strength of the patient.'

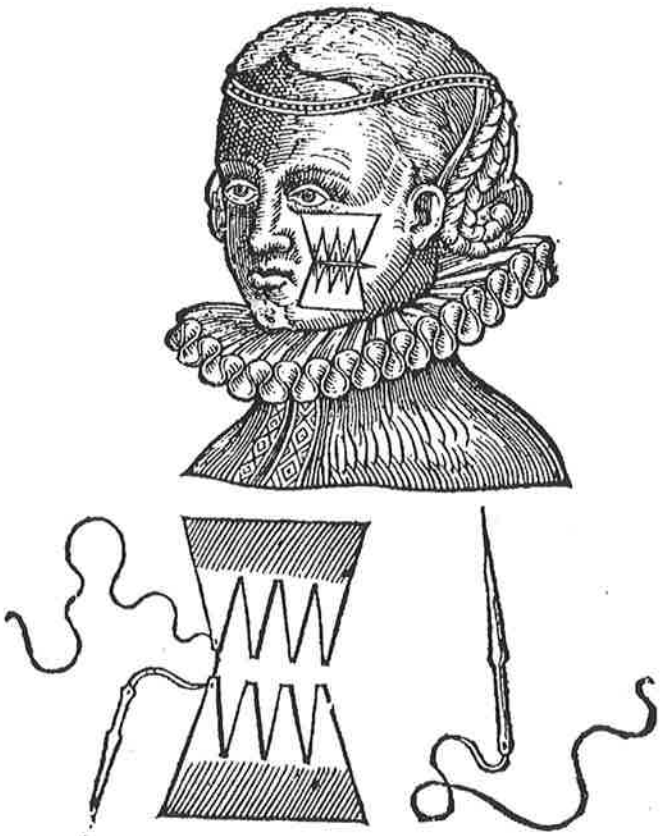


FIG. 1.9. *Skin closure in the Renaissance.* *Paré's 'dry suture' for wounds of the cheek: pieces of cloth are gummed to the skin and sutured together with thread.*

One procedure not in Paré's repertoire was repair of facial tissue loss by a pedicle graft. However, his remarkable contemporary Gaspare Tagliacozzi (1545-1599) was active in this field. Tagliacozzi held a professorial chair in the university of Bologna, and was a very gifted surgeon. To repair noses slashed off in civil or military clashes, he used a pedicle skin graft taken from the inner surface of the arm, sutured to the freshened nasal defect (Fig. 1.10). The arm was bandaged to the head and trunk until union was established. Tagliacozzi's rhinoplasty was a staged procedure, and might take up to five months. He used a similar flap to restore defects in the lips and ears, noting that restoration of a whole ear was not possible. Done without anaesthesia, these operations must have needed great skill in the surgeon and great fortitude in the patient. Tagliacozzi's well-illustrated book is a classic in the history of plastic surgery, but the operation was not widely performed, and the principle of the autogenous pedicle graft was obscured by more or less fantastic tales of homografted noses taken from the rumps of proletarian donors (Gnudi & Webster, 1989).

It is always dangerous to read modern concepts into the minds of the scientists of the past. Renaissance science included much thaumaturgy and belief in astrology and alchemy was widespread; surgeons were not immune to these seductive aberrations. But one can see in the writings of the elite surgeons of the sixteenth century three themes of enduring importance: themes that had indeed been evident in Graeco-Roman surgical practice, but had not previously come so clearly into focus.

First, wound healing and tissue repair were seen as natural if still mysterious processes, and were studied objectively. Early in the century, it had been thought that gunshot wounds were poisoned, and should be purified by the cautery or by irrigation with boiling oil. As is well known, Paré rejected this doctrine on the basis of clinical observation. Paré gave attention to the putrefying effect of the air and the depraved humours of the victim, but suppuration was seen in natural terms as the effect of tissue damage by the force of the missile —

FIG.1.10. ***Tagliacozzi's rhinoplasty****. A pedicled graft from the forearm has been sutured to the mutilated nose, and the arm is immobilised. From G. Tagliacozzi,* De Curtorum Chirugia per Insitionem, *1597.*

The form of a noſe artificially made, both alone by it ſelf, and alſo with the upper-lip covered as it were with the hair of the beard,

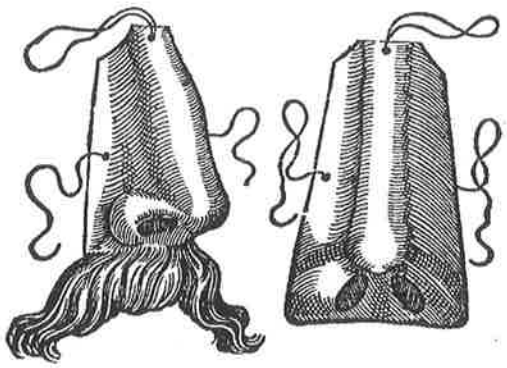


FIG.1.11. ***Paré's prosthesis for a mutilated nose****. The prosthesis was made from gold, silver, paper, or glued cloth, and was coloured appropriately; it was secured to the head or to the hat.*

'the vehemency of the contusion, dilaceration and fracture, caused by the bullets too violent entry' — and to the effects of indriven fragments of cloth, missiles, splinters of bone and bruised flesh (Paré 1649). His contemporary Leonardo Botallo (1530-?) showed that gunpowder is not poisonous, and also attributed the septic complications of gunshot wounds to indriven foreign matter (Trueta, 1944).

Second, there was general concern to protect the brain from the secondary effects of trauma. It is not always clear whether operations on the head were done to evacuate pus or blood clot, or indeed whether they were done prophylactically. But it does appear that there was awareness of the danger of intracranial collections. The distinction between pus and blood may not have seemed so fundamental as it does now, since it was believed that blood was transformed into pus as a natural process.

Lastly, there was full awareness of the aesthetic importance of facial wounds. Here again, Paré speaks for the period, when he warns that bad

management of facial wounds will 'leave deformed scars in the most specious (precious) and beautiful! part of the body' (Paré 1649). This awareness, in that beauty-loving age, doubtless inspired Tagliacozzi's amazing operations and Paré's elaborate designs for facial prostheses (Fig. 1.11). The impact of facial disfigurement is a main theme in this book.

The Seventeenth and Eighteenth Centuries

In these centuries, the philosophy of experimental science became generally accepted, and Galen's physiology was slowly discarded. Wound healing became better understood, especially after the work of John Hunter (I728–1793). He established that healing by primary union, without suppuration, must be the surgical ideal, and should be promoted by wound closure. Like Paré, he favoured closure by adherent plasters because sutures induced ulceration, perhaps from the use of contaminated thread. He studied pus with the primitive microscopes of the time and saw white blood cells; he could not establish their significance, but he came as close to understanding the nature of infection as was possible before Pasteur's work. Most importantly, he denied that air, in itself, could induce suppuration.

Bone healing was also studied: De Moulin (1988) cites the experimental work of the Dutch physician Anton De Heide (1646-169?) on long bone and cranial fractures in dogs. The anatomy of the facial structures, especially the air sinuses, the salivary glands and their ducts, was clarified: the Danish anatomist/bishop Niels Stensen (1638–1686) and the Cromwellian physician Thomas Wharton (c. 1616–1673) are commemorated by the parotid and submandibular ducts respectively. Thomas Willis (1621–1675) published a fine neuroanatomical textbook, illustrated by the young Christopher Wren, which was especially important in defining the anatomy of the cerebral circulation. Understanding of the pathophysiology of head injuries made slower progress. As late as 1752, John Hunter's learned brother William (1718–1783) could say 'as to the uses of the different parts of the brain, we are quite ignorant' (Dowd 1972). However, at the end of the period, one concept of fundamental importance was enunciated: the Monro-Kelly doctrine (Lundberg 1983). The Scottish anatomist Alexander Monro II (1733–1817) stated that the skull is 'a case of bone' whose contents are incompressible and of constant volume; with some important modifications, this observation underlies the modern concept of raised intracranial pressure (p. 36).

The wars of the seventeenth century produced some fine surgeons, notably the German Johann Schultes (1595-1645), better known as Scultetus, a graduate of Padua who practised in Ulm during the Thirty Years War. His management of head injuries has been perceptively studied by Louis Bakay (1971), himself a distinguished neurosurgeon and a competent Latinist. Bakay has shown that Scultetus's procedures were little different from those of Paré and his predecessors, both in technique and in rationale. His surgical armamentarium included the hand trephine, various nibblers and two more complex tools that doubtless express the ingenuity of German craftsmen: a rotary saw and a screw on a tripod to elevate depressed fractures (Fig. 1.12). His medical treatment was traditional: his patients were given enemas and bled vigorously to reduce fever and inflammation. Faith in therapeutic venesection was to persist well into the nineteenth century, despite the intelligent scepticism of the British naval surgeon James Yonge (1646–1721), a pioneer in the study of haemostasis (Watt 1975). In the eighteenth century, Henri-François Le Dran (1685–1770) of Paris and Percivall Pott (see p. 5) of London recognized the clinical importance of the lucid interval before worsening in conscious level, as an indication of extradural haemorrhage, and one must see this as a very notable conceptual advance. Several eighteenth-century writers show awareness of the crucial distinction between the primary effects of head injury and the secondary — often remediable — complications. An article on trephination in the famous French *Encyclopedie* (Anonymous 1765) quoted the royal surgeon F. R. Quesnay (1694–1774) on the indications for operation: these stressed late-onset ('consecutif') symptoms, in

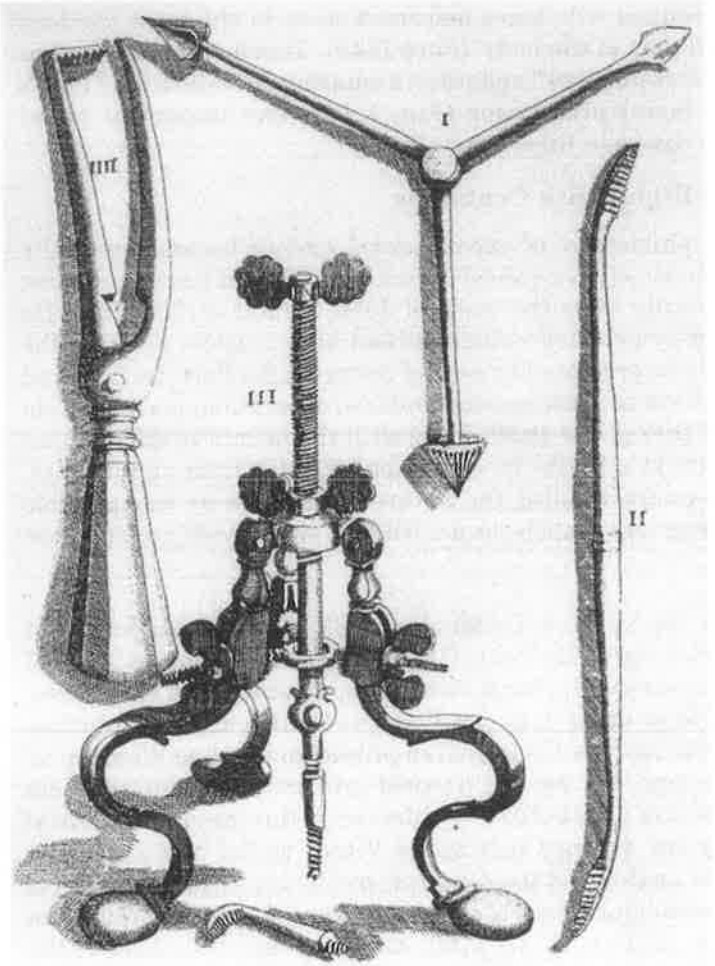


FIG. 1.12. *Cranial surgery in the Thirty Years War.* *A perforator with three points, a simple elevator, an adjustable screw elevator, and a pair of bone forceps. From J. Scultetus,* Armamentarium Chirurgicum, *1655.*

contrast to the primary ('primitif') effects of injury. Both Quesnay and Pott (better known for his puffy tumour of the scalp, associated with cranial osteomyelitis) were also very ready to trephine linear fractures on suspicion of underlying mischief, or even prophylactically (Fig. 1.13). By the end of the eighteenth century, there was general agreement on the detrimental effects of cerebral compression and readiness to relieve it immediately by operation at the site of impact: 'when a Blow upon the Head is attended with considerable Symptoms, you cannot enquire too soon into the State of the Cranium by making a large Incision upon that Part which has received the Blow; and it is far more preferable to make a useless Incision, than to neglect it in a dubious Case' (Le Dran 1740). The surgeons of this period were keen observers, and one comment by Pott deserves record, because of its relevance to CMF trauma: 'I think that I have seen more patients get well, whose injuries have been in or under the frontal bone, than any other bones of the cranium. If this should be found to be generally true, may not the reason be worth enquiring into?' In Chapter 4, this prophetic observation is related to modern research on frontal impacts.

One can see in this period increasing skill in dealing with facial fractures. Richard Wiseman (p. 3) described the reduction of mandibular dislocations along what were then orthodox lines, but also described innovative approaches to fractures of the midfacial skeleton. In one case, a child aged 8 years, he reduced such a fracture with a hook behind the hard palate: the dislocation recurred, and the only recourse available was for members of the family and the child himself to take turns in holding the maxillary segment forward until it became fixed in the proper position. This was done with a good result. Wiseman also described several cases of bullet wounds in the face: he was at pains to extract the slug or ball, especially if it was of iron or brass, as being more likely to 'rust' than lead (p.

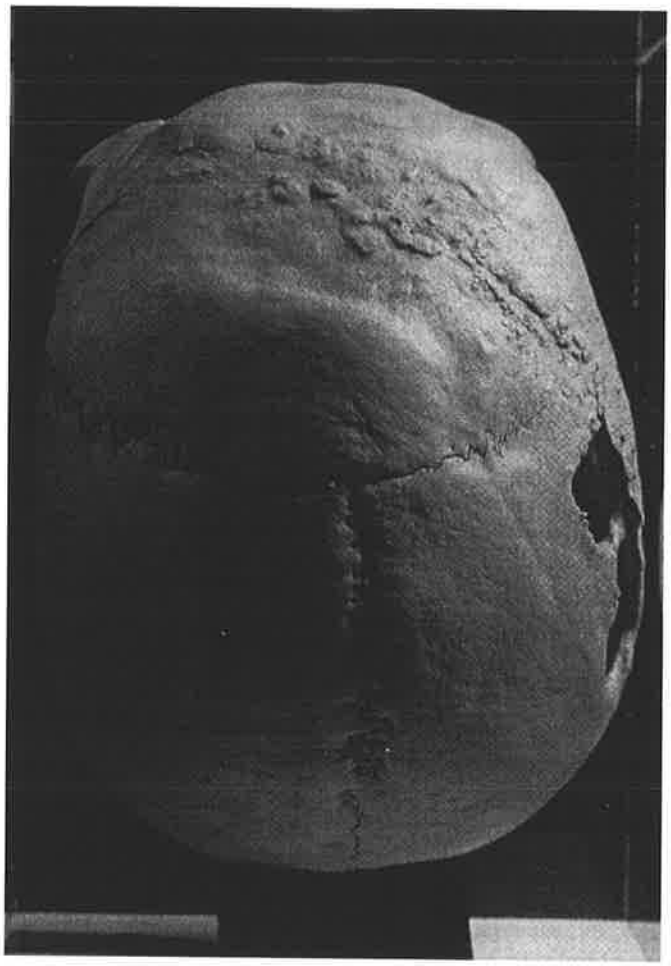


FIG. 1.13. *Cranial surgery in the Age of Reason.* *Skull trephined, possibly by John Hunter. On the right are two healed trephine holes. Extending across the frontal region, is a healed fracture, with many bony nodules, suggesting chronic inflammation.. Reproduced by courtesy of Trustees of the Hunterian Collection, Royal College of Surgeons, England: Catalogue of the pathological series P459.*

153), and he emphasized the danger of indriven rags. In one case, a pistol ball lodged in the nose: it caused chronic discharge — 'a fretting ichor' — and had to be removed through the palate. The palatal defect was closed with a plate.

In dental surgery, there is also evidence of conceptual and technical progress, with use of dental methods in the management of facial fractures. Jean-Francois Capperon (?-1763), Louis XV's dentist, collaborated with the surgeon Henri-Francois Le Dran in a way that anticipated modern craniofacial practice. In 1729 Le Dran treated a man injured in a road traffic accident; he had been run over and both the mandible and the maxilla were fractured. Le Dran immobilized both fractures by dental ligation. 'Being little acquainted with this Method, I thought proper, for the Benefit of the Patient, to desire Mr. Capron, Operator for the Teeth to his Majesty, to go to the Hospital and perform this Operation' — perhaps the first record of interdisciplinary collaboration in the management of CMF trauma. Interdental ligation was not an innovative procedure, but later in the century a new means of immobilisation was designed for mandibular fractures: a kind of G-clamp, with iron plates fitting on the teeth above and under the chin below (Rowe & Killey 1955).

The Nineteenth Century

Napoleonic surgery

The savage battles of the Napoleonic wars produced some great surgeons, but it cannot be said that the theory and practice of the surgery of CMF injuries changed very much in the first half of the nineteenth century. Robert Liston (1794-1847),

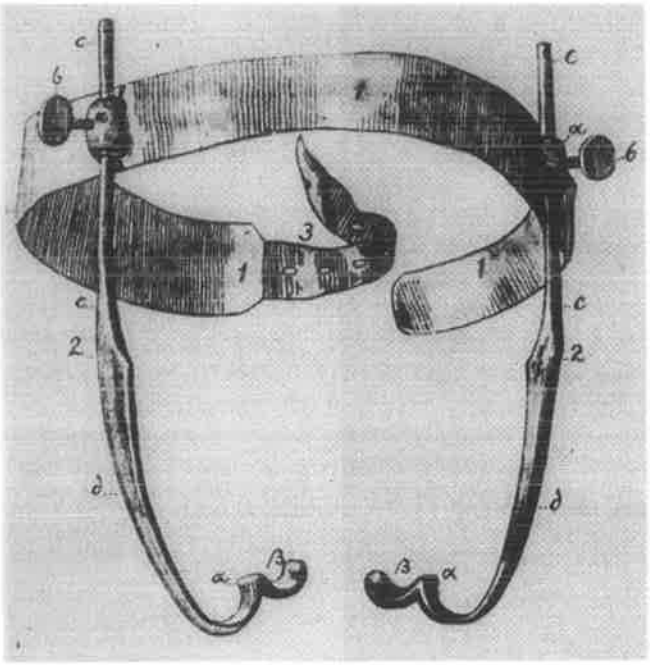


FIG.1.14. ***Prussian maxillofacial surgery in the Age of Biedermeier.*** *Apparatus for splinting fractured maxilla from a headband, devised by C. C. F. Reiche of Berlin, and reported in 1822. By courtesy of Professor W. Hoffmann-Axthelm.*

a Scot who became Professor of Surgery in the new North London (later University College) Hospital, gave a good, succinct account of his treatment of fractures of the nose, maxilla and 'inferior maxilla' (mandible). He used interdental wiring, or 'a machine with blunt hooks and screws, to be had of the instrument-makers', but in general his treatment was little in advance of eighteenth-century practice. However, it is noteworthy that he used metal cap splints fitted to the teeth of the upper and lower jaws and soldered together to immobilise the mandible before excision of a tumour: a device which was to have much application in fracture management (Liston, 1846). He gave credit for this to 'my friend Mr. Nasmyth of Edinburgh', presumably the Scottish dentist Alexander Nasmyth (1789-1848). The cranial surgery of the period is well exemplified by the writings of George Guthrie (1785-1856), who served as an army surgeon from the unusually young age of 16. In 1847, he published a vivid treatise on head injuries, in which the indications for trephining are somewhat more precise than in earlier publications. He operated urgently to elevate compound depressed fractures, to prevent what he called cerebral irritation: his case reports suggest that this was bacterial infection. He also operated for cerebral compression; his cases include five extradural haematomas, trephined on the battlefield, with three recoveries. Guthrie was convinced of the value of bleeding: one of his patients was bled some 4 litres in 3 days, and also purged (Guthrie 1847).

Facial repair was little advanced from Renaissance times. The Indian rhinoplasty became known outside India after 1793, when one of the victims of Tipu Sultan's terror mutilations (p. 8) underwent successful rhinoplasty by an Indian surgeon (McDowell, 1977), and this operation was performed by several European surgeons thereafter. Carl Ferdinand von Gräfe (1787-1840) of Berlin used both the Indian and the Tagliacotian rhinoplasties. Carl Reiche (1796-1860), one of his pupils, reported an apparatus (Fig. 1.14) for fixing a fractured maxilla to a steel head band (Hoffmann-Axthelm 1991). But if Paré and Tagliacozzi had returned to the surgical world of 1845, they would have seen little to surprise them. Over the next 50 years, the theory and practice of surgery changed beyond recognition.

Anaesthesia and the Listerian revolution

In 1846, ether was first given, in Boston, to permit painless surgery; Liston

performed the first operation under ether in Europe in the same year. In 1865, Joseph Lister (1827-1912) first used carbolic acid to prevent microbial wound infection. These landmark events inaugurated modern surgery, yet much had to be done before these revolutionary discoveries became relevant in the routine management of CMF injuries.

John Snow (1813-1858), pioneer of scientific anaesthesiology, noted as early as 1848 that the risk of inhalation of blood made anaesthesia in the surgery of the jaws especially hazardous, and this problem remained unsolved until the advent of endotracheal anaesthesia some 60 years later. Major operations on the facial skeleton were done under chloroform or ether, usually given with a face mask. This entailed very real risks, and for some time, jaw injuries were often managed without anaesthesia. Likewise, the advent of antiseptic surgery was not at first of much significance in the management of jaw injuries: fear of infection continued and it was many decades before internal fixation of mandibular fractures by wiring was considered an acceptable procedure, though the procedure had been described as early as 1846 by M. Fouchard in France and around the same time by Gurdon Buck (1807-1877: see below) in the USA (Rowe & Killey 1955). Indeed, the chief nineteenth-century advances in the management of jaw fractures came from the use of new dental technologies for external fixation. Organized training in dentistry began around 1840, in several countries, but most notably in the USA, producing a generation of capable dentists some of whom made important contributions in the fixation of jaw fractures. In many instances, they utilized new dental techniques and materials, such as vulcanized rubber; metal casting, swaged splints and wire fixation were also used in new ways. In the USA, four dentists were particularly innovative in the treatment of jaw fractures: Thomas Bryan Gunning (1813-1889), James Baxter Bean (1834-1870), Thomas Lewis Gilmer (1849-1931), and Edward Hartley Angle (1850-1930).

In 1861 Gunning, a New York dentist, immobilised mandibular fractures with hard vulcanized rubber splints, made from dental impressions in wax and fixed to the teeth by gold screws; where necessary, the splint was also fixed to the intact maxilla, nutrition being maintained through a hole in the vulcanite splint. Gunning also used his splints in edentulous patients, and for this purpose 'Gunning-type' splints are still in use (p. 522). His papers make fascinating reading (Gunning 1866-7): his cases of mandibular fractures included himself and an unnamed 'distinguished statesman in Washington', who was in fact Lincoln's secretary of state and rival W. H. Seward (1801-1872). Seward had suffered a bilateral compound mandibular fracture in a road accident; nine days later, an inept assassin tried to cut his throat and inflicted an extensive facial wound. Gunning's polite record of his successful treatment of these injuries is a maxillofacial classic.

This attempted assassination took place at the end of the American Civil War, which has been described as the first modern war. It is certainly the first large war to be documented in great detail. G. A. Otis (1870) wrote a magnificent surgical history of the war, giving statistics relating to 3312 hospitalised gunshot wounds of the face (mortality 11.4%), 4350 gunshot wounds of the skull (mortality 59.2%), and 1190 gunshot wounds of the eye, with loss of sight of at least one eye in some 75%. The victims are identified by name or initials, and range in military magnitude from Jefferson Coates of the 7th Wisconsin Volunteers, aged 20, wounded at Gettysburg by a colloidal ball through both orbits, surviving blind and anosmic but 'in good spirits', to 'AL, aged 56 years', who is clearly the commander-in-chief Abraham Lincoln (1809-1865) himself, assassinated by a Derringer pistol shot in the brain (p. 140).

Head injuries of all types were often complicated by intracranial infection; it cannot be said that the war produced real advances in the management of cranial wounds (Table 1.2). The Northern armies were served by a special eye hospital, but the management of eye wounds does not appear to have gone beyond

TABLE 1.2

Mortality from penetrating wounds of the head in wars from 1853 to 1970 (modified from Gurdjian 1973)

	Mortality (%)
Pre-Listerian era	
Crimean War (1853-5)	75
American Civil War (1861-5)	59 – 72
Antiseptic era	
South African War (1899-1902)	40 – 50 (Rawling 1915)
World War I (1914-8)	35 – 50 (29: Cushing 1918)
Antibiotic era	
Second World War (British Army) (1939-45)	14
Korean War (UN Army) (1950-2)	10
Vietnam War (US Army) (1960-75)	10

The range of percentages in earlier wars reflects differences in wound categorisation and sources of data.

excision of irreparably damaged eyes and the provision of glass eyes. Ophthalmoscopy had been introduced in clinical practice in 1851 by Hermann von Helmholtz (1821-1894) and had inaugurated the development of ophthalmology as a clinical science. But ophthalmoscopy was rarely used in the diagnosis of eye injuries in this war and Otis saw it as a superfluous refinement. Among the documented cases of eye injury, there were 40 cases of so-called sympathetic ophthalmitis, though only four went on to blindness. William McKenzie (1791-1868) had given a brilliant description of six cases of this condition in 1830, but it seems likely that its natural history (p. 412) was not yet understood. Secondary haemorrhage from injuries of the face were often fatal, despite heroic operations to ligate major arteries. The treatment of mandibular injuries does appear to have made progress. Gunning's vulcanite splints and prostheses received admiring comment from Otis, who also referred to interdental splints devised for Confederate wounded by Bean of Atlanta, Georgia. Bean's splints were also of vulcanite; like Gunning, he took plaster casts of the fractured mandible, put the pieces into occlusion with the intact maxilla, and then made a vulcanite splint to maintain the apposition (Hoffmann-Axthelm, 1982). Plastic surgical repairs of residual facial deformity were recorded in only 32 cases; some of these were seen as successful, notably three by the pioneer plastic surgeon Gurdon Buck, but Otis was pessimistic about the utility of such procedures in the vast majority of facial injuries.

Oral and maxillofacial surgery

After the Civil War, American dental surgery advanced rapidly. Gilmer, co-founder of the Dental School of North Western University, is credited with the introduction of the practice of immobilising the fractured mandible to the intact maxilla by interdental wiring to preserve a correct occlusal position; he also tried open fixation of a mandibular fracture with platinum wire (McDowell 1977). Edward Angle, the father of orthodontics, pioneered the study of post-traumatic malocclusion and its treatment by devising the comprehensive classification of occlusion that bears his name (p. 347). He also laid the foundations for the use of bands and arch wires to immobilise the teeth in correcting

malocclusion, and advocated a similar system for fixation of mandibular fractures (Fairbank 1936, Hoffmann-Axthelm 1982). The evolution of oral surgery from dentistry was well established by the end of the nineteenth century, and in several American centres, jaw fractures were seen as problems for the oral surgeon rather than for the general surgeon, who was otherwise responsible for trauma management (Johnson 1936). Nevertheless, the most seminal work on maxillofacial fractures at this time came from a French general surgeon, René Le Fort (1869-1951) of Lille. His studies on 35 cadavers subjected to blunt facial impacts (Le Fort 1901) are of enduring importance.

Le Fort's research methodology was robust. Cadaver heads were struck with a block of wood, or thrown against the edge of a marble table, until the skull cracked; the soft tissues were then removed and the fracture or fractures inspected. Le Fort considered nine types of impact:

1. Anteroposterior impact on the upper lip: this tended to produce the transverse (horizontal) lower maxillary fracture described by Alphonse Guérin (1817-1895) in 1866.

2. Lateral impact on the lower part of the maxilla: such impacts were likely to injure the mandible and malar bones, but could also separate the whole upper jaw.

3. Impact from below on the upper alveolar margin: this caused a large symmetrical fracture through the nasal notch, the orbit, below the malar bones and the pterygoid processes, detaching the midface as a large fragment, often with other fractures such as a sagittal crack through the hard palate (p. 294).

4. Impact from in front on the midface: this produced similar fractures.

5. Impact from above downwards on the root of the nose: Le Fort did not himself study this impact, but quoted two cases from the literature.

6. Impact from below upwards on the mandible: fractures of the mandible were often seen but the effect was also to impact the upper alveolar arch from below, causing the fracture pattern resulting from impacts 3 and 4.

7. Impacts on the malar bone — four variants: these impacts forced the malar bone into the maxillary sinus, but could also cause other fractures, running across the midface.

8. Simultaneous facial and cranial impacts: impacts over a large area could separate the whole face from the skull base.

9. Impacts at multiple sites/from multiple directions: Le Fort did not study these himself, but noted that they can occur.

From these findings, correlated with reported clinical experience and with the architecture of the facial skeleton, Le Fort identified his well-known lines of weakness (p. 293). One ran between the skull (i.e. the neurocranium) and the facial skeleton, and fractures in this plane could result from either frontal or lateral impacts on the maxillary complex. This is now called the Le Fort III fracture, and it is interesting that the discoverer saw it as a fracture along the plane that protects the brain box (le boîte crâienne). He identified a second line of weakness in the midface, not wholly independent of the first line, running from the root of the nose above the nasal canal (sic) into the orbital floor and back to the pterygomaxillary fissure. This is the Le Fort II fracture. His third line of weakness ran from the lower margin of the piriform aperture across the canine fossa, below the malar bone and into the pterygomaxillary fissure. He called this the Guérin fracture, but his description is far clearer than that of Guérin, and the term Le Fort I fracture is now usual.

Neurosurgery

While modern concepts of maxillofacial surgery were thus being formulated, neurosurgery was taking shape under the liberating influences of anaesthesia

and antisepsis/asepsis. For long cranial operations, inhalational anaesthesia was an obvious advance, though early techniques often caused brain swelling. The Listerian antiseptic method was of enormous importance in reducing the risk of cerebral infection. Earlier in the nineteenth century, many surgeons had become much more reluctant to operate on the head than their predecessors had been. In the period 1860-1876, four London teaching hospitals recorded mortality rates from trephining in excess of 75%; the mortality for this operation was little lower in the American Civil War. These appalling figures doubtless express not only actual deaths from hospital infection but also deaths after operations delayed because of fear of infection (Hudson 1877). After 1880 surgeons began to operate on the brain with increasing confidence: not only compound skull fractures but also extradural haematomas were treated surgically, and the infective complications of cranial trauma were recognized and treated —sometimes successfully. William Macewen (1848-1924) of Glasgow described cerebral abscesses with great clarity and detail. Of the 64 cases reported in his book (Macewen 1893), 11 had various kinds of post-traumatic intracranial suppuration, and six of these made good recoveries after surgical drainage. Macewen also made important contributions in the prevention of brain infection by proper primary wound cleansing and suture. Greater readiness to operate on the brain also led to more interest in cerebral physiology and especially in the physiology of raised intracranial pressure: this work was summarized by the Swiss surgeon Theodor Kocher (1841-1917) in a remarkable monograph on brain injuries (Kocher 1901), which includes experimental work done in Berne by the brilliant young American surgeon who was to be the founder of modern neurosurgery — Harvey Cushing (1869-1939).

Radiology

Lastly, the nineteenth century saw the advent of in vivo imaging of bone and organs. Conrad Röntgen (1845–1923) described his 'new kind of rays' late in 1895, and radiology entered the field of CMF trauma as early as 1898, as a means of localizing intraocular and intracranial missiles. Thereafter, clinical radiology grew with a speed that seems amazing today, when innovative procedures are delayed by so many safeguards, necessary and unnecessary.

World War I, 1914-1918

Carnage

Modern concepts of the specialized management of CMF trauma took shape during this long and dreadful war. In 1914, these concepts were still embryonic. It was still an age of omnicompetent general surgeons. Admittedly, ophthalmology, ENT surgery and oral surgery were recognised and although there were very few committed neurosurgeons in the world, it was accepted that the surgery of the brain required special knowledge and skills. Plastic surgical techniques had evolved from rhinoplasty to employ skin flaps in many situations, and split-skin grafts had been known for some 40 years, but plastic procedures were usually performed by general surgeons. The chief surgical textbooks of the period contained chapters on the surgery of the brain and the jaws by men with a special interest in these fields, and from these one can deduce that the common CMF injuries of peacetime were well understood. Nor were missile wounds ignored: indeed, the surgical literature from Wilhelm II's Germany showed an ominous preoccupation with the effects of the new weapons. Yet when war broke out in August 1914, the army medical services of the chief combatants — Germany, Austria-Hungary, France, Russia and Great Britain — were soon overwhelmed by the sheer numbers of wounds of the head. It is said (Zilz, 1917) that in France and Belgium alone, there were 90 000 head injuries in the first 10 months of war. Facial wounds aroused special concern, because of their appalling appearance and because the victims survived in large numbers, often bitterly aware of disfigurement. Brain wounds also received attention, especially when the high incidence of delayed death from brain abscess became tragically apparent. Eye

wounds, very numerous from the new fragmentation missiles, made the blinded soldier a familiar figure. It is said that eye wounds required removal of the eye in two cases out of three. Perhaps this high rate of enucleation in part reflected a dread of sympathetic ophthalmitis, which in fact appears to have been a rare complication in this and later wars (Albert & Diaz-Rohena 1989); nevertheless, only 6 months after the outbreak of war, British ophthalmologists were aware that the risks of the demon of ophthalmitis did not justify sacrificing a damaged eye if there was some useful vision (Jessop 1915).

Specialist management: Morestin, Gillies and Cushing

Management plans took different courses in the various combatant countries, yet converged at the end of the war. For France, the war was chiefly fought on French soil, not far from civilian hospitals of great distinction, well equipped to give multidisciplinary care for maxillofacial injuries. In Hippolyte Morestin (1869–1919), Paris had already a plastic surgeon of acknowledged genius, and the dental management of jaw injuries had been well taught by Claude Martin (1843–1911) of the Lyon school of military medicine (Hoffmann-Axthelm 1982). French wartime literature contains many references to the benefits of collaboration between surgeons, stomatologists and dental technicians in the management of jaw injuries; a short-lived bilingual periodical, *La Restauration Maxillofaciale,* was published to foster this collaboration. German medical authorities also saw the importance of organized multidisciplinary centres for the care of facial injuries: a 225–bed hospital was allotted for this purpose in Düsseldorf as early as August 1914. Brain wounds were given special attention both in France and in Germany: the famous Parisian hospital La Salpêtrière cared for many thousands of brain-injured soldiers, and the University Clinic in Frankfurt am Main played a similar role for Germans wounded on the Western Front: it was here that the neurologist Kurt Goldstein (1878–1965) inaugurated modern neurorehabilitation. For Austria–Hungary, the war was fought on many fronts, often under great logistic handicaps: perhaps for this reason, the pros and cons of early versus late closure of head wounds came into clear focus in Austrian surgical thinking. The Viennese Nobel Laureate Robert Baranyi (1876–1936), during the siege of Przemysl, argued for primary closure. Austrian surgeons on more remote fronts, where wounds were already stinking when first seen, saw this as Utopian and accepted the formation of a brain hernia as the best hope of avoiding a lethal brain infection (Albrecht & Feuchtinger 1916). It seems that the Russian armies, fighting under notorious administrative handicaps, failed to evolve special services for CMF injuries, although fine work was done by individuals, notably V. P. Filatov of Odessa. He is credited with the invention of the tubular pedicle skin graft (McDowell 1977).

For the British army, the position was somewhat different. The chief effort was in France and Belgium: the trench line changed very little during the war, and medical policy dictated that primary surgical treatment should be done within a few miles of the line. British surgeons believed that brain wounds travelled badly after operation, and for this reason definitive treatment was done as soon as possible, often in advanced hospitals. In this there seems to have been a divergence from French policy: the leading French neurosurgeon of the day, Thierry de Martel (1875–1940), believed that brain wounds did not require urgent operation (Chatelin & de Martel 1918). British surgeons also agreed that eye wounds should be treated as soon as possible. But maxillofacial wounds did not seem so urgent, and were sent for definitive treatment in England; there were obvious psychological advantages in this. In 1917, the Queen's Hospital was established at Sidcup in Kent for these injuries, eventually disposing of 1000 beds and treating about 5000 cases. It became the focus of innovative CMF surgery of the highest quality, under the inspiration of the New Zealand surgeon Harold Gillies (1882–1960). Gillies had originally been an ENT surgeon; he had been impressed by French work on facial injuries and inspired British military medical authorities to form a special hospital for such cases. Australian, Canadian, New Zealand and US sections were added; the Australian section was commanded by

Henry Newland (1873–1969), a South Australian general surgeon and one of the founders of Australian plastic surgery and neurosurgery (Hughes 1972). Gillies' team included the dental surgeon W. Kelsey Fry (1889–1963), Ivan Magill (1888–1986) the pioneer of endotracheal anaesthesia, and Henry Tonks (1862–1937), a distinguished academic artist whose delicate pastel drawings of facial wounds are haunting reminders of the brutality of war (Bennett 1986). Tonks was joined by the young Australian artist Daryl Lindsay (1889–1976): his water-colours of similar wounds in Australian soldiers are less well known but are equally precise and moving (Fig. 1.15).

US troops did not come to the Western Front in substantial numbers until 1918, but individual US surgeons were associated with French and British units much earlier, and two of these made very important contributions in CMF surgery. V. H. Kazanjian (1879–1974), a Harvard dental specialist, worked at first in prosthetic reconstructions: he later became a leading plastic surgeon (Converse 1977). Harvey Cushing, already the acknowledged leader of American neurosurgery, served with a British frontline medical unit in 1917, and demonstrated the merits of early wound exploration and closure in two classic papers, which are still well worth reading (Cushing 1918a,b). He reported a rigorous analysis of 133 head wounds with dural penetration treated over 3 months during the terrible battles around Passchendaele: in this period, his early mortality fell from 54.5% to 28.8%, an improvement attributed by him to better technique rather than to case selection. Cushing is not remembered as a modest man, but this report is remarkable for its generosity to British colleagues and for a total absence of comment on the fact that the work was done in a casualty clearing station dangerously close to battles which the author vividly described in the diary he kept during his exhausting work (Cushing 1936).

The end of World War I saw the management of CMF trauma well advanced. Gillies and his team had firmly established the value of the multidisciplinary team in the total care of maxillofacial injuries. His remarkable book (Gillies 1920) described the contribution of the dental prosthetist in fixation of jaw fractures (Fig. 1.16); he detailed the various skin flaps evolved to repair regional facial defects, including the tubed pedicle grafts for which he is famous. This procedure was first used by him in a case of burns, a field in which he also made fundamental contributions, in management strategy as well as in techniques; his principles of burn management, still very relevant, are discussed in Chapter 17. The battle of Jutland (31 May/1 June 1916) brought naval casualties to Sidcup, among them Able Seaman Vicarage, one of the few pioneering patients whose name has not been forgotten (Fig. 1.17). Vicarage had suffered appalling facial burns in a cordite fire in the battleship HMS Malaya; Gillies gave him a new face 18 months later by swinging two tubed pedicle grafts from the chest (Gillies 1920, case 338; Pound 1964).

Gillies also used and extended French techniques (Imbert & Real 1917) of free bone grafting, using iliac or costochondral grafts. Bone grafts had been used with success in long bone injuries; in the later years of the war, several small series of mandibular reconstructions with free grafts were published, but the results were not brilliant (Cole 1918), sepsis being a frequent cause of failure (Fig. 1.18). Gillies has a central place in the history of the management of CMF injuries, and he was a great teacher. Many Sidcup techniques have become standard procedures, but perhaps more fundamental has been the Sidcup emphasis on aesthetic reconstruction as the prerequisite of psychological recovery.

Concurrently, Cushing had shown the merits of early operations, by neurosurgeons, for craniocerebral wounds; X-ray control, gentle debridement by suction and irrigation became routine procedures, and Baranyi's vision of primary wound closure was accepted as standard policy.

Advances in anaesthesia made these surgical achievements possible. Cushing, and some German plastic surgeons (Lexer 1931), favoured local or

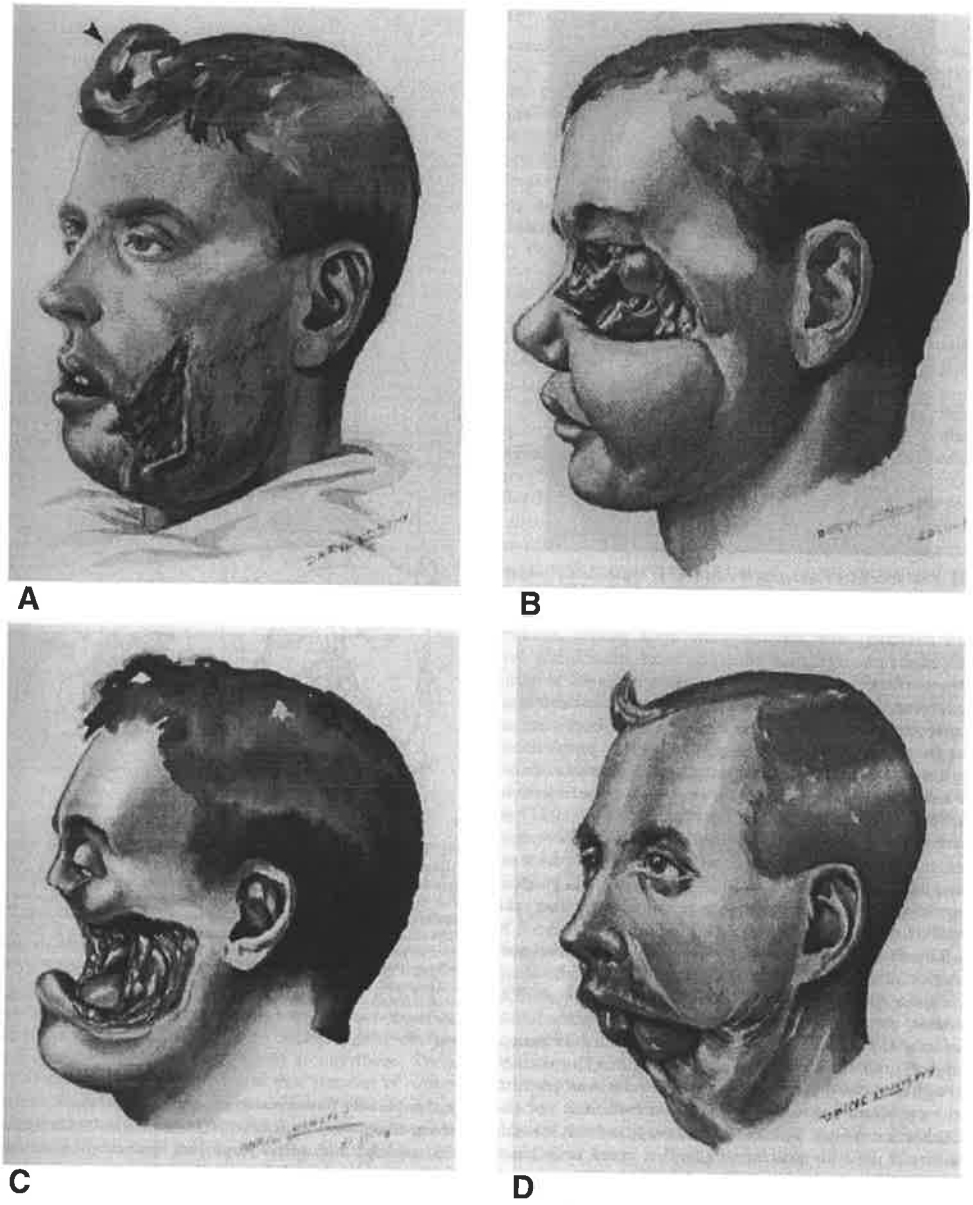


FIG 1.15. ***The disasters of war.*** *Four young wounded soldiers, treated in Queen Mary's Hospital, Sidcup, 1918-19. Watercolours by Sir Daryl Lindsay. By courtesy of the Royal Australiasian College of Surgeons.* **A.** *Facial and frontal wounds; the frontal swelling appears to be a cerebral hernia.* **B.** *Massive orbital destruction.* **C.** *Massive ablation of upper jaw and nose.* **D.** *Ablation of lower jaw.*

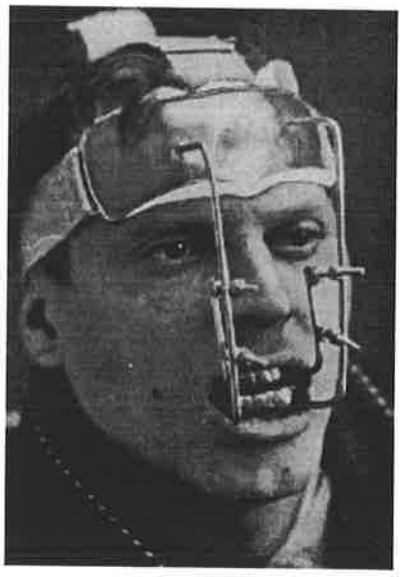


FIG. 1.16. ***Maxillary fixation in World War I.*** *Apparatus for forward replacement of the maxilla, devised by Major Rischworth, a New Zealand dental surgeon. From H. Gillies (1920),* Plastic Surgery of the Face, *Oxford University Press.*

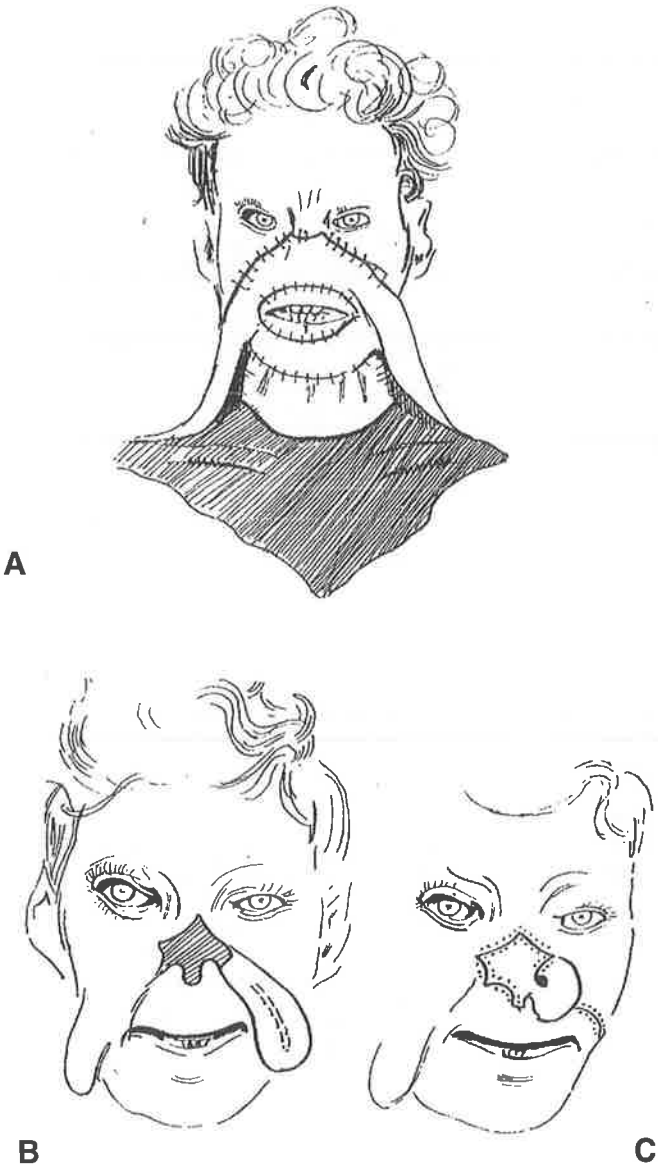


FIG. 1.17. ***Tubular pedicle grafts for facial deformity after burns: Gillies' first case.*** *The patient had been severely burned some 18 months earlier. Gillies planned to excise the scar tissue and replace it with two flaps of vascularized skin from the thorax. He was inspired to convert these flaps into tubes.* **A.** *Diagram of the plan of operation. The flaps took well, but the skin over the nose became necrotic* **B.** *When the pedicles were vascularized, the left pedicle was detached and used to cover the nose. Later operations corrected ectropion on both sides and replaced the eyebrows with scalp grafts. The nose was reconstructed with an allograft of cartilage. The final appearance was considered to be aesthetically successful, and the eyelid functions were much improved.*

regional blocks, but Magill's routine use of endotracheal anaesthesia for CMF trauma showed the way to the future (Rowbotham & Magill 1921).

In retrospect, one can see some gaps in this record of achievement. The need for long-term neurorehabilitation was ignored or soon forgotten. Good as interdisciplinary collaboration between dentists and plastic surgeons had been in war, there was in many countries no systematic professional formulation for continuing this collaboration in peacetime. Nor did the need for routine collaboration between plastic surgeons, oral surgeons and neurosurgeons emerge during the war; indeed, increasing specialisation may have made this collaboration less likely. Earlier in the war, a military writer on the surgery of the head could discuss brain and jaw wounds together, in the knowledge that the same surgeon might well treat both forms of CMF trauma (Rawling 1915). Later in the war, a definite conceptual separation is evident. Thus, Cushing made reference to the problems of craniocerebrofacial wounds, but did not enlarge on the contribution

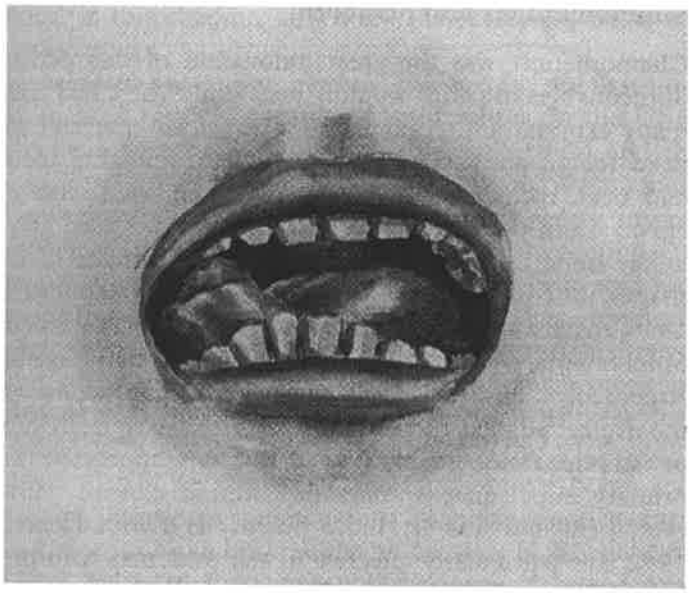


FIG. 1.18. ***Bone grafting In World War I.*** *Tibial bone graft (arrow) extruding from the mandible: wounded soldier treated September 1917 to February 1919. Watercolour by Sir Daryl Lindsay. By courtesy of Royal Australasian College of Surgeons.*

of the plastic surgeon; his preferred use of triradiate scalp incisions (Fig. 1.19) suggests that he would have benefited from collaboration of this type. These issues remained unresolved and perhaps unrecognised in the post-war years.

World War II, 1939-1945

Logistics: Tönnis and Cairns

This war saw important advances in the logistic and organisational aspects of CMF trauma management, both in the field of maxillofacial surgery and in neurosurgical treatment. These wartime developments have had enduring consequences in peacetime surgery. At the beginning of the war, two great neurosurgeons were in positions of power in their respective countries: Wilhelm Tönnis (1898-1978) as consultant surgeon to the German airforce(Luftwaffe), and Hugh Cairns (1896-1952) as neurosurgical consultant in the British army. Both had served in the World War I, and both saw the need for an integrated service that would give expert neurosurgical care as soon as possible, and would maintain that care until rehabilitation was completed.

Tönnis created, from the outset of the war, a comprehensive service of this type. Great use was made of air transport. When Poland was attacked in 1939, Luftwaffe aircraft, chiefly the famous three-engined Junkers JU52, brought some 2500 casualties back to established neurosurgical units in Berlin, Breslau and Vienna: the average flying time was under three hours. Priority was given to brain, eye and jaw wounds. The Luftwaffe was not the first service to use aircraft in this way; indeed, aircraft had been used for peacetime accident cases in outback Australia for many years. But the Luftwaffe's massive achievement impressed even its enemies, and other combatants soon organized air ambulance services. The small Australian air force pioneered with such a service in the Middle East theatre of war. In the Pacific theatre, where there was often no alternative means of transport, US medical authorities deployed special air evacuation units, providing trained medical and nursing staff for care of patients in military transport aircraft; these anticipated the modern air medical and paramedical retrieval teams.

Hugh Cairns, a South Australian and the much-loved founder of the Oxford neurosurgical school (Fraenkel 1991), approached the logistic challenge of head injuries in a somewhat different way. He established a 300-bed base hospital for head injuries in Oxford, but his most original concept was the 'mobile

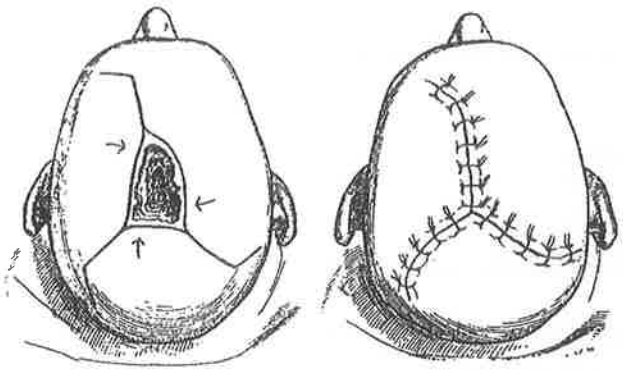


FIG. 1.19. ***Scalp closure In World War I.*** *Cushing's well known tripod or three-legged incision for wounds of the cranial vault. Personal experience has shown that closure by a rotating flap is usually preferable. From Cushing (1918).*

neurosurgical unit', a small self-contained team that could go anywhere. These very flexible units, staffed by a dozen persons of whom only one was a fully trained neurosurgeon, were equipped to work either close to the battlefield, or in a base hospital; Cairns (1947) has told their story well and for the present purpose it is only necessary to say that these teams both advanced the technical management of brain wounds and pioneered in the multidisciplinary management of complex craniofacial injuries.

The Trinity

Maxillofacial wound management had a parallel evolution. Harold Gillies and Kelsey Fry, with the prestige and experience of their Sidcup achievements, were well qualified to advise on the organization of services in this field. Base units were established in Great Britain, the best known being in Basingstoke under Gillies himself. Rooksdown House in Basingstoke became a great teaching centre during and after the war: from it came the classic textbook *Fractures of the Facial Skeleton,* written by two great oral surgeons, Norman Rowe (1915–1991) and Homer Charles (Paddy) Killey (1915–1976). In addition to the base maxillofacial and plastic units, mobile units analogous to Cairn's mobile neurosurgical units were formed and deployed in various theatres; eventually there were six of these in the British army and two more in India.

There were also in the British army special mobile opthalmological units, deployed as far forward as possible (Goulden 1953). It was common practice to group special neurosurgical, ophthalmological and maxillofacial units closely together: as early as 1943, this grouping was being called 'The Trinity'. This interdisciplinary grouping can be related to the increasing interest in complex craniofacial and cranio-orbital wounds, which constituted up to a quarter of all brain injuries in one large series.

Concern over the treatment of injuries involving the anterior cranial fossa was relatively new. Between the two world wars, neurosurgeons had begun to repair the anterior fossa in cases of post-traumatic cerebrospinal fluid leak: Walter Dandy (1886–1946) had reported a successful case in 1926. Wartime experience of a high incidence of infection after wounds opening the paranasal air sinuses made it logical to carry out such repairs as part of the primary wound closure. However, many wounds of this type showed extensive tissue loss in the fronto-orbital region, and elaborate plastic procedures were needed to close the defects. Thus, interdisciplinary collaboration in CMF trauma management was carried a step further. This is evident in the remarkable *War Surgery Supplement* of the *British Journal of Surgery* which appeared in 1947: it was edited by Cairns, and includes superb illustrations, many of which show craniofacial wounds (Fig. 1.20).

The war saw advances in management of faciomaxillary trauma. Bone grafting of mandibular fractures became increasingly successful. In England, Gillies' pupil Rainsford Mowlem (1902–1988), also a New Zealander, demonstrated

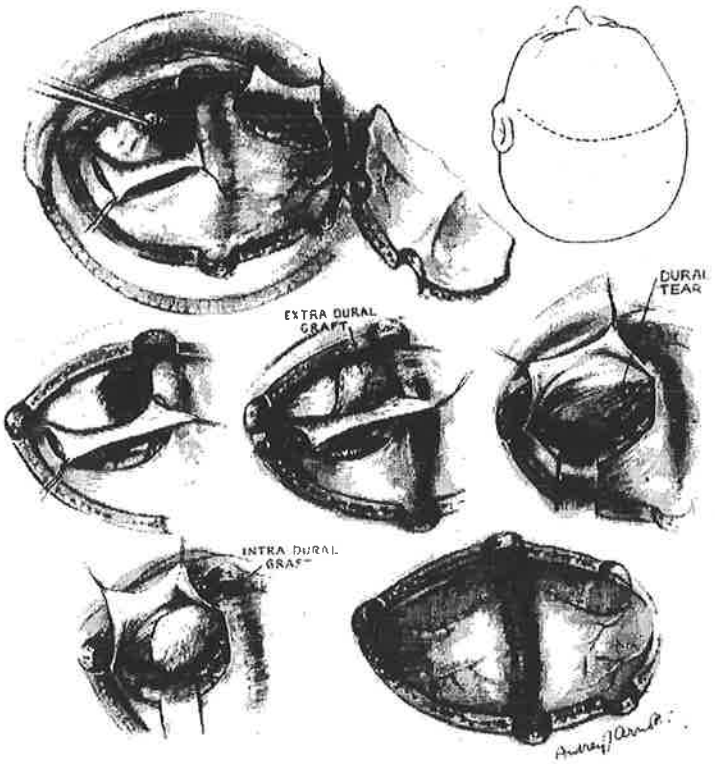


FIG. 1.20. *Craniofacial wound in World War II.* *Repair of an orbitonasofrontal wound, caused by a shell fragment, which had lodged in the ethmoid air cells. Drawn by Audrey Amot, whose illustrations of operations done by Cairns and his pupils are rightly famous. From Calvert (1947), by courtesy of Messrs Heinemann.*

the value of cancellous bone (Mowlem 1944). Blocker & Stout (1949) performed what would now be called a meta-analysis of US wartime experience of bone grafts, and found a final success rate of 97%. However, the authors noted that poor follow-up made this imposing achievement somewhat suspect. Fixation of fractures of the mandible by external pins (Fig. 1.21) was also used where other means failed: fear of infection had deterred surgeons from much use of open fixation of mandibular fractures, but the advent of chemotherapy opened up this possibility also (Fry 1953). Maxillary fractures received more attention than in the past; fixation to a plaster headcap became a routine procedure (Wallace 1985).

Sulphonamides and penicillin

Chemotherapy was the great innovation of this period. Surgeons dealing with wounds in World War I had tried many external antiseptic agents without success and the need for an antibacterial agent in the circulating blood had been recognised. Antisera had been tried with no effect; urotropine, now a forgotten drug, had been given in the hope that it would generate formaldehyde in the tissues. But until the discovery of the sulphonamides in 1935, there was no chemotherapeutic agent of real worth. Sulphonamides were indeed effective, especially against streptococci, and they were given liberally, sometimes to the neglect of good surgery; Tönnis (1943) wrote critically of 'sulphonamide fanatics' who did not appreciate the primary importance of operative wound closure. However, penicillin was far more potent, and its advent in 1942 was particularly effective in the treatment and prevention of brain infections. Penicillin was brought into surgical use by Cairns, in collaboration with his fellow South Australian Howard Florey (1898-1968); it proved its value in CMF trauma management in the 1943 Italian campaign.

World War II brought the surgery of brain wounds in general, and especially craniofacial wounds, to a stage that is recognisably close to modern practice (Table 1.2): later wars have emphasised the lessons learned then, and have only slightly modified them (p. 373). Head injury rehabilitation was also stimulated by the

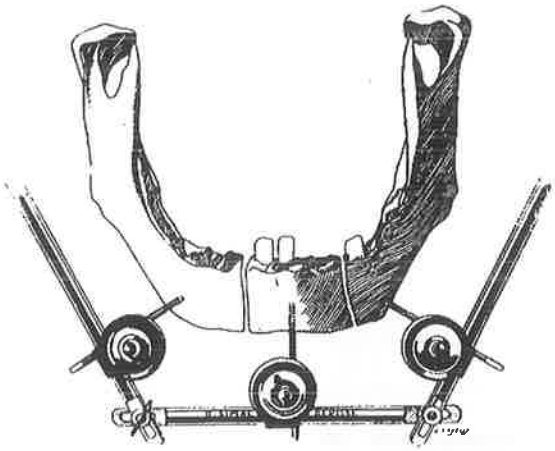


FIG. 1.21. ***External fixation in World War II.*** *Pin fixation of mandibular fracture, tile method employed by Roger Anderson for other Fractures, and by G. Maurel in 1940. From Rowe & Killey (1955).*

challenge of so many young men with neurological disabilities. In Russia, Aleksandr Luria (1902–1977) advanced views on the nature of recovery from brain injury that have since inspired many workers in this field; in the USA, the importance of systematic rehabilitation was emphasized by Goldstein, who had emigrated to New York from Nazi Germany.

Multidisciplinary Management

Road crashes and trauma management

After 1945, the USA and Western Europe experienced a mounting pandemic of injuries due to road crashes, and within a few years, increasing motorization brought this pandemic to most other parts of the world. Injuries in the CMF region attracted particular attention, and not only as causes of death: with better airway management and nutritional support, prolonged survival in what was later called the vegetative state became a common sequel of severe brain injury.

Surgeons responded to the challenge of these CMF injuries, often with multiple injuries elsewhere, in many ways. These responses are now largely embodied in modern thought and practice. Much of what was innovative in the decade after the World War II is still very relevant, and will be discussed in appropriate parts of this book: in this chapter, only a few major developments are considered.

Accident prevention became, perhaps for the first time in surgical history, a leading consideration. In the USA, the neurosurgeon Elisha Gurdjian (1900–1985) and the engineer Herbert Lissner (1907–1965) made Wayne State University in Detroit a multidisciplinary centre for research in the bioengineering aspects of head impacts, and this has had enormous importance in head and face protection. Colonel John P. Stapp and other volunteers explored the effects of high velocity accidents on themselves and on anaesthetised animals, using a rocket-propelled sled devised by German wartime research workers concerned with aircraft crashes. Stapp saw the relevance of these studies to road accidents and annual Stapp Car Crash Conferences commemorate his initiative and courage. In Oxford, Cairns inspired the physicist A. H. S. Holbourn (1907–1962) to investigate the dynamics of closed head injuries, and this led directly to Sabina Strich's historic discovery of what is now called diffuse axonal injury (p. 139). In Australia, the concern of a number of surgeons did much to give that country priority in the mandatory use of crash helmets and seat belts and in alcohol control.

Trauma management benefited from advances in diagnostic radiology. Brain imaging began in 1918 with air contrast ventriculography and

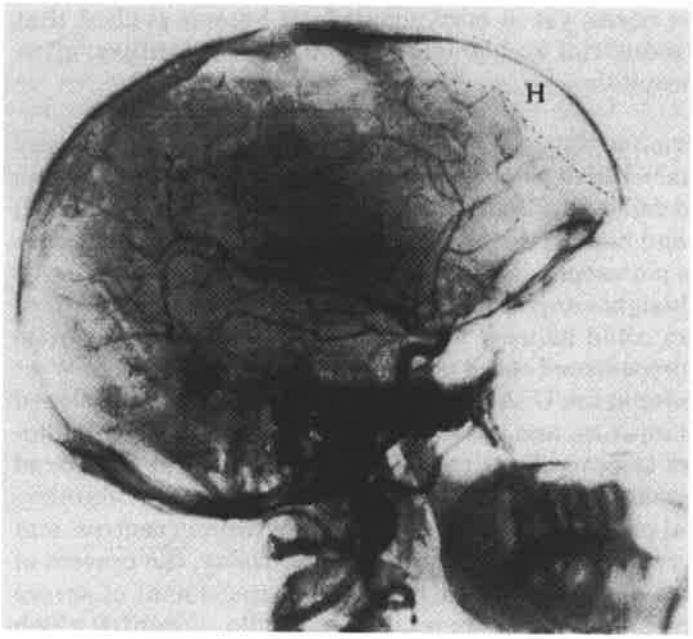


FIG. 1.22. ***Portuguese contribution: cerebral angiography.*** *Cerebral angiography, introduced by Egas Moniz, has been an important advance in the management of CMF injuries. The Lisbon school pioneered intracarotid injection of thorotrast, which visualised surface clots very well. Unfortunately, the long-term complications of this radioactive contrast agent were often devastating. From Lima (1950), by courtesy of Oxford University Press.*

encephalography, but these procedures had little place in the management of acute brain injuries. Cerebral angiography, first reported by the Portuguese neurologist Egas Moniz (1875–1955) in 1927, was used to exclude complications of head injury as early as 1936 (Lohr quoted by Lima 1950). After 1950 percutaneous angiography was increasingly used even in acute cases (Fig. 1.22). However, these invasive procedures were largely superseded by computed tomography (CT), which came into clinical use in 1973. Tomography with a moving X-ray source and conventional silver halide film was used to visualize facial fractures as early as 1940 (Curler 1940); plain tomography was found invaluable in delineating the temporomandibular joint and the cribriform plate. In 1973, the British physicist G. N. Hounsfield's inspired combination of computer analysis with scintillation detection made computed axial tomography the safest and most effective means of imaging the brain, and it still holds this place in trauma management despite the later advent of magnetic resonance imaging (p. 186). The first generation of CT scanners did not provide sufficient definition to visualize facial fractures, but later models could do this, and with software programs allowing three-dimensional reconstruction, the visualisation of facial trauma was further advanced.

These increasingly complex methods of imaging the damaged brain and the shattered face brought logistic problems: there was a large capital cost and trained radiological staff had to be available at short notice. The care of the comatose patient was also very demanding. Modern coma care began around 1950 with the general use of tracheotomy, both for the 'stoved-in face' (Nelson 1958), and for the comatose craniocerebral injury where death from respiratory complications would have been inevitable 20 years earlier (Echols et al 1950). The success of tracheotomy encouraged other methods of maintaining normal cerebral physiology, especially oxygenation and intracranial pressure (ICP). Continuous ICP monitoring was introduced in France in 1951 (Guillaume & Janny 1951) and in Sweden a few years later (Lundberg 1983); monitoring has since become a routine part of neurosurgical intensive care, in conjunction with mechanical ventilation. Intensive care was even more demanding in medical and nursing staff and in laboratory services. Only large, well-funded metropolitan

hospitals could meet these needs, yet in most countries it became evident that road crashes, unlike the industrial accidents of the nineteenth century, often occurred far from major hospitals.

The concept of the 'trauma centre' was evolved to provide expensive, highly specialized multidisciplinary management for the victims of severe peacetime trauma. This concept had indeed its roots in the experience of World War II, after which Hugh Cairns and his successors had established in Oxford a trauma service that embodied this philosophy. But experience in the long and tragic war in Vietnam brought new insights and techniques. There, one side enjoyed total air superiority: helicopters could be used to transport the wounded to mobile surgical hospitals with unprecedented speed and greatly superior results. In West Germany and somewhat later in the USA, similar rescue services were developed for road and other civilian injuries, and based on metropolitan hospitals. In San Francisco, rapid transport to centralized trauma management was compared favourably with less systematic care in another area, Orange County. Trunkey (1983), in a very influential paper, made a case for regional trauma centres, and these have been widely accepted in the USA and Canada. Today, the concept of the Trauma Centre is implicit in the multidisciplinary management of severe CMF trauma in many parts of the world, not least in Australia, a country which suffers more than most from what has been called the tyranny of distance — the logistic constraints of geography and demography.

Advent of craniofacial surgery

After 1970, a new surgical philosophy began to modify and refine the management of facial injuries. In the preceding decade, Paul Tessier and his colleagues at the Hôpital Foch in Paris had devised innovative surgical procedures to correct congenital facial dysmorphisms, especially those involving severe orbital deformity. In doing this, Tessier created the subspecialty of craniofacial surgery, which in his hands embodied not only a variety of carefully designed anatomical corrections in three dimensions, but also a multidisciplinary, team–based system of assessment and holistic treatment. Tessier's achievements in the surgery of congenital malformations lie outside the scope of this book. But the insights and the methods of craniofacial surgery have greatly enriched the management of CMF trauma.

Tessier was trained in the grand mainstream of French surgical teaching, which has stressed the importance of trauma since the time of Paré, and his formative experiences began during the World War II. He has told how in 1944, only 2 years after his internship, he became an assistant in the Centre of Maxillo-Facial Surgery in the Paris Military Region, which treated many facial wounds in the tough fighting that liberated Paris. In 1946, this army service was transferred to the Hôpital Foch, which for a while housed separate military and civilian units. It will surprise nobody who loves France that these units 'did not appreciate each other'; as an expression of this mood, the senior army surgeon refused to give the young Tessier access to the hospital's prosthodontic department. This forced Tessier to develop that reliance on internal fixation which is so fundamental in modern craniofacial technique (Wolfe & Berkowitz 1989). Paris was not the only place where it proved hard to maintain wartime cooperation, both interdisciplinary and personal, in peacetime surgery, where the isolationism of each specialty becomes stronger as time passes; it is not the smallest of Tessier's achievements that he institutionalised the multidisciplinary craniofacial team. Such teams need constant practice; Tessier (1971) emphasized that engagement in a busy trauma service keeps the team's skills sharp and ready to deal with the more complex problems of dysmorphia.

Tessier inspired many young plastic surgeons to form craniofacial units. What is now the Australian Craniofacial Unit began in 1974, when one of us (D.J.D) saw a need for a service of this type in South Australia, and entered into close collaboration with a small group of neurosurgeons already much engaged

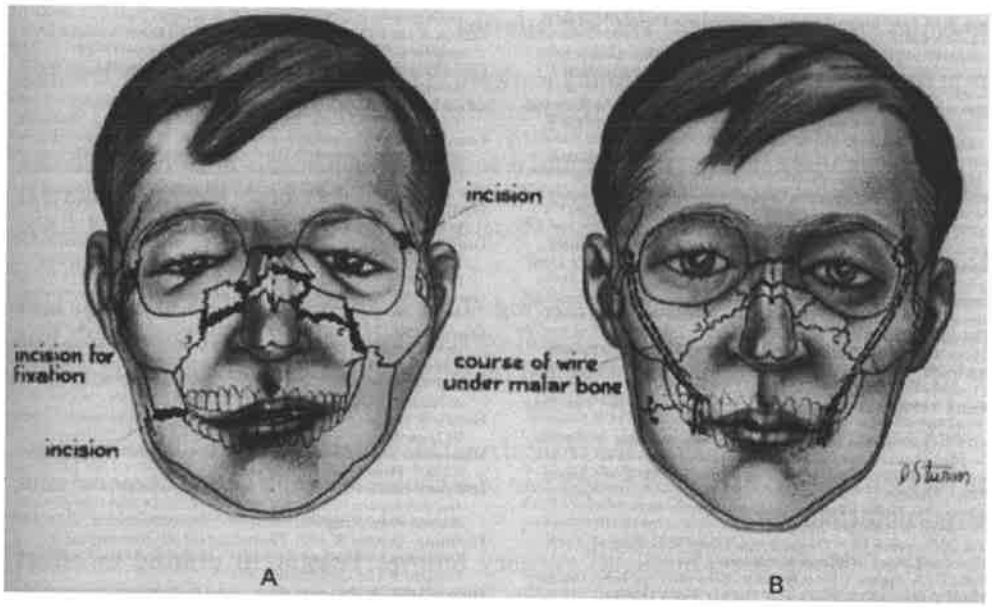


FIG. 1.23. ***Internal fixation of midfacial fractures.*** *The use of internal fixation of fractures by wiring of the middle third of the face was reported by Adams (1942). His paper is a landmark in the management of CMF injuries.* **A.** *Comminuted fracture of maxilla, presumably Le Fort III type, with fractures of both zygomas; fracture of right mandible.* **B.** *The fractures have been reduced by open operation; the maxillary component was fixed with wires secured to upper teeth and passed to the frontal bone on each side. The left zygoma and the mandible were also fixed by wiring. From Adams (1942).*

in trauma management; several of them also had some acquaintance with missile trauma in less peaceful countries. Similar groups, each with its own mix of specialties and its own personal dynamics, were established at the same time in a number of centres in North America, Europe and elsewhere. The treatment of CMF trauma received great impetus from multidisciplinary groups of this type.

Internal fixation of facial fractures

In 1942, Adams of Memphis (Tennessee) advocated internal suspension and fixation of midface fractures by wiring (Fig. 1.23); he reported a low incidence of infection, and the advent of chemotherapy made surgeons braver in accepting techniques involving open exposure of fractures. Tessier's reconstructive techniques entailed internal fixation of mobilized components of the facial skeleton by wires passed through small drill holes, and the success of these procedures encouraged many surgeons to use similar methods to fix fractures of the mandible and maxilla. Metal plates offered greater stability than wires. Metal plates had been used for fixing long bone fractures since the nineteenth century, but the use of inappropriate types of steel had led to inflammatory complications resulting from metallic corrosion. In the decade preceding World War II, experimental studies had shown the importance of biocompatibility in implanted materials. Venable et al (1937) championed the alloy Vitallium, but chromium — nickel-molybdenum (18/8/Mo) steel was also found to perform well in the tissues, and was easier to forge. With the availability of these biocompatible metals, the Swiss Arbeitsgemeinschaft für Osteosynthesefragen (AO) reopened in 1958 the question of internal fixation of fractures, and soon European maxillofacial surgeons were successfully plating fractures of the facial skeleton (p. 237). Titanium, originally used in orthopaedic procedures, was found to be better tolerated than steel or vitallium, and has been used widely for facial fracture fixation and cranioplasty (pp. 270 and 549).

Contemporary management of CMF injuries

From the shared experience of many workers in the management of CMF trauma has emerged what can be called the craniofacial perspective:

1. Complex injuries demand a team. Tessier began his work with the orbit as the centrepiece: he deployed there the skills of both the maxillofacial surgeon and the neurosurgeon. Other structures in the CMF region demand other skills, especially in the oral area.

2. Modern methods of imaging show the nature of the damage, and allow decision on the timing of surgical correction: delay is often beneficial, but the neurosurgical and ophthalmological complications of CMF trauma sometimes need urgent action.

3. Wide exposure of the craniofacial skeleton is needed, either through the bicoronal scalp flap, or through one of a choice of periorbital, intraoral and extraoral incisions.

4. Modern craniofacial surgery follows Tessier in aiming to effect internal fixation by primary bone grafts, together with wires, plates and screws. Small plates constructed of biologically acceptable materials have greatly facilitated fixation.

5. Microvascular repair, introduced some 30 years ago (Jacobson & Suarez 1960), has enlarged the scope of grafting with soft tissues and with bone; aspects of the remarkable history of microvascularized bone grafts are given on p. 623.

6. Advances in prosthetic design and materials can supplement surgical correction of traumatic defects by a range of intraoral and extraoral prostheses, including osseo-integrated implants.

7. Advances in conservative dentistry have made it possible to salvage injured teeth that would have been condemned in the past. The science of endodontics has come of age, and the rationale and techniques of endodontic therapy are now integrated in the modern management of maxillofacial trauma.

The surgery of CMF injuries has come a long way in the last four millennia and without doubt it will go further. This book is an endeavour to assess current techniques, and to identify the chief principles of treatment. In the perspective of history, these principles are not new, but their interpretation is constantly changing, and finality remains an unattainable goal.

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Functional anatomy

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Definition of the Craniomaxillofacial Region

An understanding of trauma in this region presupposes an appreciation of the bony and soft tissue anatomy of the entire head, including the skull and the cranial viscera. The skull comprises three principal structures: the calvaria or vault, the cranial base and the facial skeleton. The calvaria provides mechanical protection for the brain against external violence. The cranial base provides a platform for the brain, with exit foramina for the cranial vessels and nerves. It is the template of the skull: from it is suspended the facial skeleton and above it rises the domeshaped vault. Base and vault constitute the neurocranium. The facial bones (sometimes called the viscerocranium) enclose the eyes, the upper parts of the airway, and the upper digestive tract; they are coated with muscles and ligaments which give the face much of its form and function. The frontal component of the head, designated the craniomaxillofacial (CMF) region by clinicians, comprises the facial skeleton and the associated viscera and integuments, together with the anterior cranial fossa, the calvaria anterior to the coronal sutures, and the frontal lobes of the brain (Fig. 2.1).

The Bones and Their Articulations

Calvaria

This part of the skull is formed chiefly by the frontal, parietal, temporal and occipital bones. The thick frontal bone forms the forehead and is connected anteriorly with the facial skeleton by the frontonasal, frontomaxillary and frontozygomatic sutures. The thin paired orbital plates curve posteriorly to articulate with other components of the cranial base by the sphenofrontal and fronto-ethmoid sutures. Posteriorly the frontal bone articulates with the parietal bones by the coronal suture. The parietal and occipital bones, which are outside the CMF region as here defined, articulate with each other, with the temporal bones and with the greater wings of the sphenoid laterally. The temporal bone forms part of the cranial base and its squamous part articulates with the mandible by the temporomandibular joint. The calvarial bones are in principle composed of inner and outer plates of cortical bone, separated by cancellous bone which in childhood contains haemopoietic marrow (Fig. 2 2). The layer of cancellous bone constitutes a plane of cleavage, often burst open in comminuted calvarial fractures, and exploited surgically in taking calvarial bone grafts (p 241).

Cranial base

The cranial base is divided into the anterior, middle and posterior fossae: while only the anterior fossa enters into the CMF region, the anterior part of the middle fossa and the central part of the posterior fossa are important relations (Fig. 2.3).

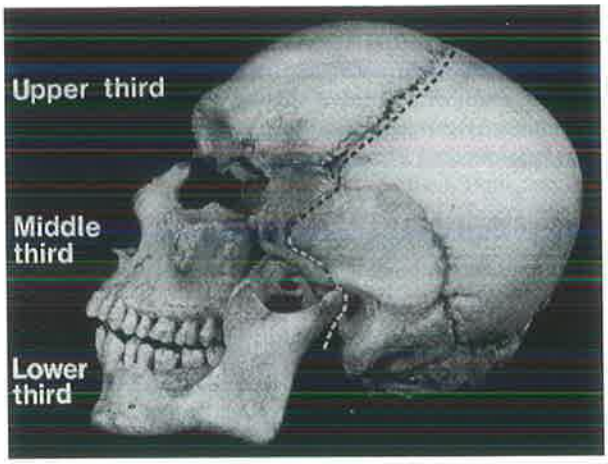


FIG. 2.1. ***Demarcation of the craniomaxillofacial region.*** *A disjoined view of a normal skull: the dotted line shows the demarcation of the CMF region by the coronal suture, the sphenoid bone and the external auditory meatus. Within this region, it is usual to divide the face into thirds: upper, middle and lower*

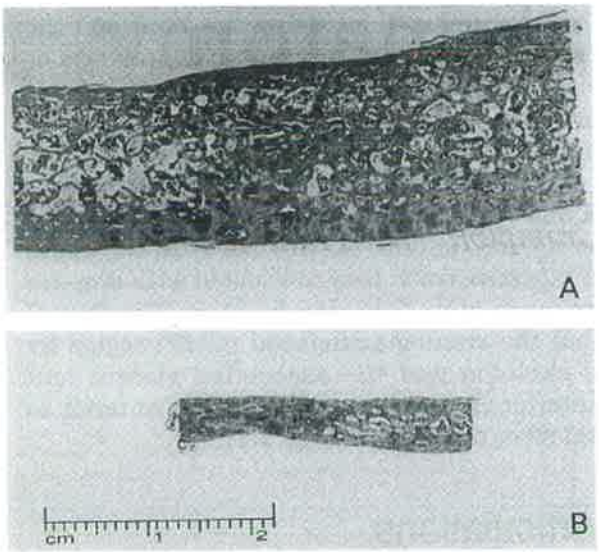


FIG. 2.2. ***Adult and infant calvarial structure.*** *Sections of parietal bone of adult* **A.** *and of 1-year-old infant* **B.** *(H & E stain).*

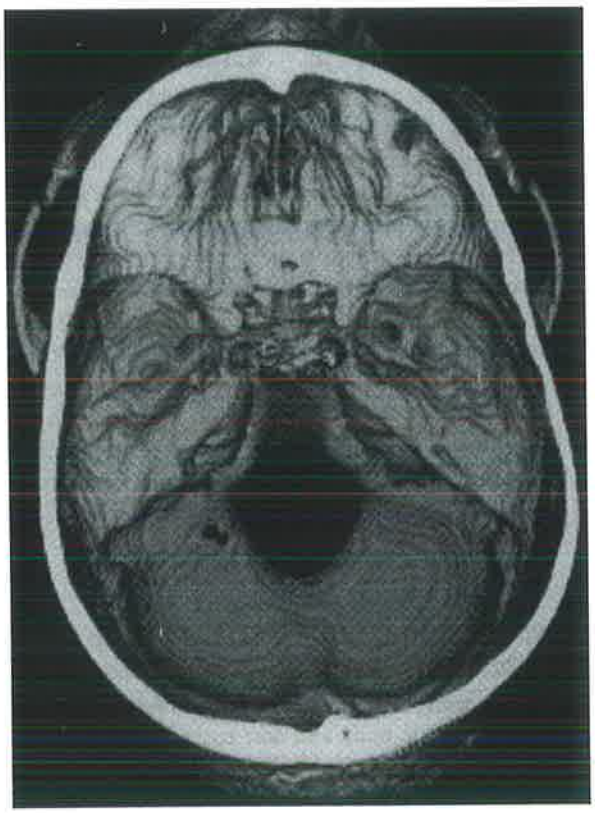


FIG. 2.3. ***The anterior cranial fossa.*** *Interior of skull, reconstructed from fine cut (1.5 mm) CT scan. The slice lines give a contour map of the anterior and middle cranial fossae, showing bony prominences which correspond with impressions in the frontal and temporal lobes; in closed head injury, contusions are often seen in relation to these. In contrast, the posterior fossa contours are smooth.*

The anterior cranial fossa

The anterior cranial fossa is bounded anteriorly and laterally by the frontal bone which contains the frontal air sinuses. The largest part of the floor of the anterior cranial fossa is formed by the curved orbital roofs. Medially the floor on each side dips downwards to the cribiform plate, which, with the crista galli, is a part of the ethmoid bone. The cribriform plate is directly related to the roof of the nose; on either side lie the anterior and middle ethmoid air cells. The lesser wing of the sphenoid laterally forms the crescentic posterior borders of the fossa. Centrally the body of the sphenoid roofs over the sphenoid sinuses; the optic canals run lateral to the body, being formed by the two roots of the lesser wing of the sphenoid (p. 57). Fractures of the anterior fossa floor may therefore involve the frontal, ethmoid or sphenoid sinuses or the nasal cavity itself through the cribriform plate; the optic canals may also be involved when the fracture line runs posteriorly (p. 419). The extent of the frontal sinuses is extremely variable, ranging from almost no pneumatization to extensive pneumatization involving the roof of the orbits. The dura of the cribriform plate is penetrated by the fine olfactory nerves; they are accompanied by arachnoidal sheaths (Lang 1983). Stripping dura from the cribriform plate will lead to anosmia, and possibly to CSF leakage.

In making extradural approaches to the supraorbital region, certain anatomical features need to be borne in mind. The orbital roof may dip steeply down towards the cribriform plate which itself may be quite narrow. On average the cribriform plate is 8 mm below the nasion, the most deeply depressed point on the bridge of the nose (Lang 1983). In frontal craniotomies, access is greatly improved by temporary removal of a bar of bone including the upper margins of the orbits: this entails opening the frontal sinuses.

The middle cranial fossa

This is composed of portions of two bones — the sphenoid and the temporal bone. The sphenoid is surely the most fascinating bone in the craniofacial complex. It sits centrally as the keystone of the skull and has its manifestations in the middle fossa, anterior fossa, infratemporal fossa, pterygomaxillary fossa and orbital cavity. The body of the sphenoid forms the centre of the middle fossa and is in close proximity to the vital neurovascular structures in the cavernous sinuses. Anteriorly, the middle fossa is formed by the greater and lesser wings off the sphenoid, and between these the superior orbital fissure; the wings form part of the lateral wall of the orbit. Laterally the middle fossa is formed by the squamous temporal bones, articulating with the mandibular condyles on their inferior surfaces. Its posterior aspect is buttressed by the strong petrous temporal bones. At the anterior margin of the apex of the petrous temporal bone is a cluster of foramina (lacerum, ovale, spinosum); the bone is penetrated by the ear clefts and traversed by the carotid canal. Although the petrous temporal bones are inherently strong their anterior borders are relatively weakened and the foramina act as areas of stress concentration. The common hinge fracture of the skull base runs along this line of weakness, crossing the midline through the body of the sphenoid. Medially, the middle fossa ascends to join the pituitary fossa (sella turcica).

The floor of the middle fossa is penetrated by branches of the trigeminal nerve — the ophthalmic nerve through the superior orbital fissure, the maxillary nerve through the foramen rotundum and the mandibular nerve through the foramen ovale. The internal carotid artery lies immediately beneath the floor in the carotid canal, which may have no bony roof; this artery ascends into the cavernous sinus. The artery then curves upwards and forwards to pierce the dura medial to the anterior clinoid process. The middle meningeal artery also pierces the floor of the middle fossa, through the foramen spinosum posterolateral to the foramen ovale.

Facial skeleton

Nasal bones
These fragile bones are connected above with the frontal bone by the frontonasal suture and together they separate the frontal processes of the maxillae with which they also articulate. Posteriorly the joined nasal bones are attached to the perpendicular plate of the ethmoid and to the nasal spine of the frontal bone.

Ethmoid bone
The ethmoid bone is delicate and complex. The cribriform plate is perforated by the exit foramina of the olfactory filaments, running from the olfactory bulb to the nasal epithelium. Lang (1983) found on average about 40 osseous perforations on each side; however, there are fewer dural perforations, as the olfactory filaments are grouped into larger bundles as they pass into the dura. The central perpendicular plate of the ethmoid contributes dorsally to the crista galli, and ventrally to the nasal septum; it articulates with the vomer and the septal cartilage. The medial and lateral plates enclose the ethmoid air cells on each side; the lateral plates, suspended from the cribriform plate above, form the medial orbital wall. The lateral wall is paper-thin, hence its alias lamina papyracea, and in some individuals there are bony dehiscences. The ethmoid bone provides the skeletal elements of the superior and middle nasal conchae, also known as turbinate bones. The inferior concha is a separate bone articulating with the maxilla and palatine bone. The ethmoid bone is connected to the frontal bone, the sphenoid bone, the nasal bones and the maxillae.

Zygomatic bones
These form the prominence of each cheek and contribute to the lateral wall and inferior margin of each orbit. The zygomatic bone is interposed between the frontal bone, the sphenoid bone and the maxilla and connects with the zygomatic process of the temporal bone forming the zygomatic arch. It is the length of this arch which determines the forward projection of the cheek bone, and in reconstructing the arch after injury, it is important to maintain its anteroposterior dimension (p. 316).

Maxillae
These paired bones form the keystone of the midface (Fig. 2.4). Anteriorly the maxillae join in the midline, below and behind the anterior nasal spine. They contain the teeth of the upper jaw and together with the palatine bones form the hard palate; they enclose the largest air spaces in the skull, the maxillary air sinuses, which occupy most of their bodies. The terminal branches of the infraorbital nerve and artery exit through the infraorbital foramina below the inferior orbital margins. The paired frontal processes of the maxilla connect with the frontal bone above and the nasal bones medially. Laterally the maxillae flare out to join with the zygomatic bones at the zygomaticomaxillary sutures. The very thin orbital floor components of the maxillae are connected to the ethmoid bone medially. Posteriorly the maxillae articulate with the palatine bones and the pterygoid processes of the sphenoid bone. The keystone position of the maxillae enable them to absorb the forces of mastication. These physiological forces are transmitted from the occluding teeth to the cranial base through the maxillae, which articulate with the frontal and zygomatic bones by the frontomaxillary and zygomaticomaxillary sutures. Thus the maxilla is an essential component of the system of vertical pillars or buttresses discussed below, together with the nasal septum, the ethmoid bone, and the strong buttresses provided by the junction of the pyramidal process of the palatine bone and the pterygoid laminae of the sphenoid bone. The maxilla is composed of thin plates of compact bone, which contain little cancellous bone, except in the alveolar region, where the upper teeth and their roots are embedded in bone of this type; nevertheless these thin plates are able to carry heavy loads when stressed in the vertical plane. They are less able to sustain horizontal stresses. The thinness of the maxilla must be remembered when screws are inserted in reconstructing the vertical buttresses of the facial skeleton with miniplates (p. 304); the walls of the maxillary

air sinus are often only 0.5 mm thick or even less, but thicker bone is found lateral to the piriform aperture.

Mandible

This robust bone occupies a prominent and exposed position in the facial skeleton. It articulates with the cranial base through its condyles which lie in the glenoid fossae: condyle and glenoid fossa form the temporomandibular joint (TMJ), a synovial joint divided into two cavities by a disc of fibrocartilage (Fig. 2.5). This important joint acts as a hinge, but also allows sliding movement in the anteroposterior plane.

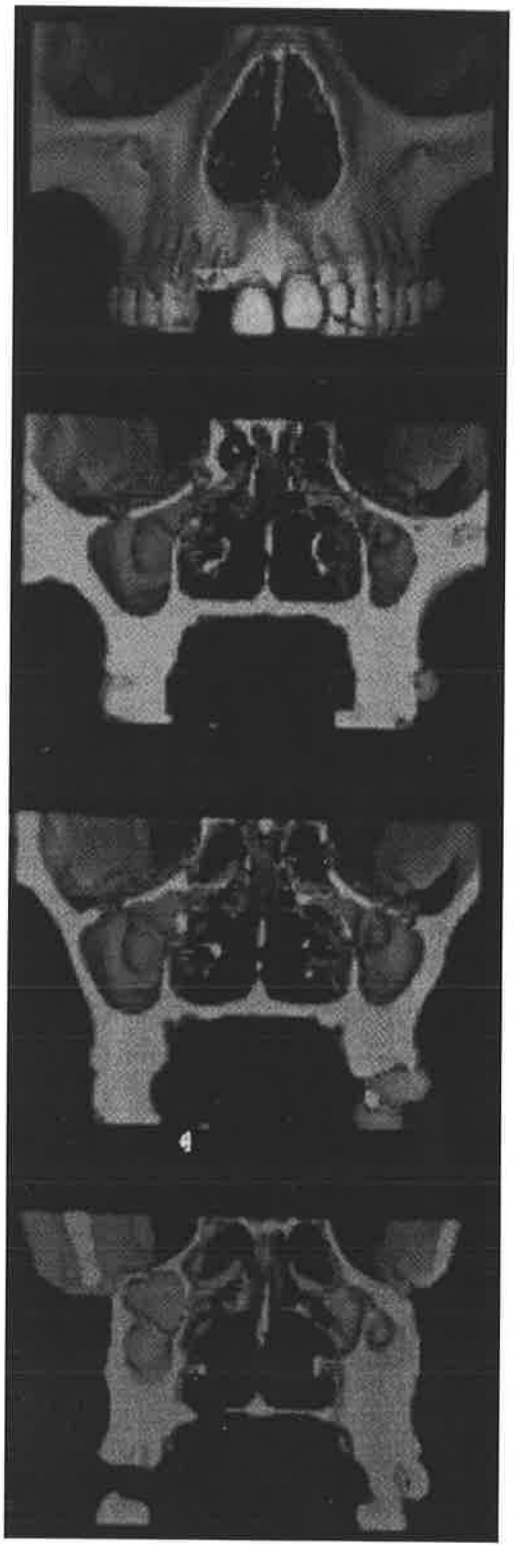
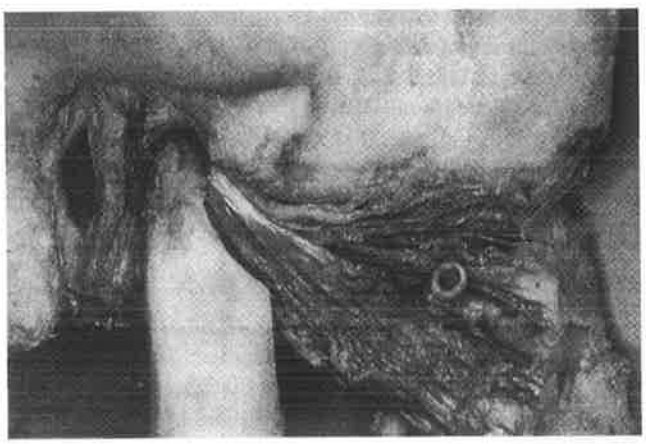


FIG. 2.4. *The maxillae.* *Seen in coronal section, the maxillae comprise dense alveolar bone supporting the teeth, connected transversely by the strong hard palate. The vertical elements of the midface are stronger laterally and behind; centrally the walls of the nasal cavity and the septum are very thin.*

The mandible comprises a number of named components: the condyles, the coronoid processes, the rami, the angles and the two halves of the body, joined anteriorly at the symphysis. The body of the mandible is shaped like a horseshoe, though more open posteriorly; the rami, vertical platelike pillars, project upwards from the free ends of the horseshoe. Viewed from the lateral aspect, the ramus forms an angle of about 120° with the body of the bone in the adult, slightly more in the very young and the elderly. Viewed from the superior aspect there is a slight divergence of the rami. The alveolar portion of the bone supporting the molar teeth does not follow the same line as that formed by the lower border of the mandible.

From a functional viewpoint, the mandible can be regarded as two conjoined L-shaped cantilevers, acting under the influence of the masticatory muscles. The temporalis muscle inserts predominantly into the medial surface of the coronoid process, the masseter to the lateral surface of the ramus and angle. The medial pterygoid muscle inserts on the medial surface of the angle and lower ramus; together with the masseter, it forms a powerful sling acting on the ramus of the mandible. The two heads of the lateral pterygoid muscle unite to insert into the anterior part of the capsule of the TMJ and the pterygoid fovea of the neck of the condyle, with a few fibres passing to the articular disc (Fig. 2.5). The action of this muscle is important in functional recovery after intracapsular fracture of the condyle, and its insertion must be preserved (p. 286).

In cross-section, the body of the mandible is seen as a flattened tube or U. being composed of strong inner and outer cortical plates of bone, between which are inserted the teeth and their roots, embedded in cancellous bone (Fig. 2.6). The plates of compact bone have the typical structure for such a tissue, being built up of haversian systems (osteons) and composed in the outer surface by circumferential lamellae. Between the two plates of cortical bone runs the mandibular or inferior dental canal, containing the inferior dental nerve on its way from the mandibular foramen to the mental foramen. Knowledge of the location of this nerve is of great importance in siting screws for plate fixation.



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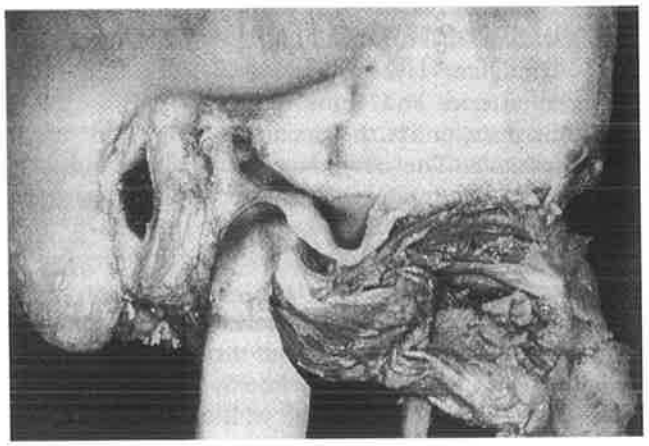


A

FIG. 2.5. ***Dissection of the temporomandibular joint and lateral pterygoid muscle.*** **A.** *The condyle of the mandible is in its resting position, located within the articular fossa, the temporomandibular disk is hardly visible.* **B.** *The mandibular condyle is pulled forwards and downwards to simulate the action of the lateral pterygoid muscle during a jaw-opening movement. The fusion of the superior and inferior pterygoid heads and their insertion into the neck of the mandible are shown. The temporomandibular disk with the insertion of a few fibres of the superior head of the lateral pterygoid into its anterior region is clearly visible. Photographs by courtesy of Professor G.C. Townsend, The University of Adelaide.*

The mandible is well adapted to withstand the vertical forces generated in mastication; it has areas of weakness, notably at the condylar process, the angle (especially if there is an unerupted third molar), and in the vicinity of the canine tooth (Abbott et al 1992; p. 264).

The morphology of the mandible and the maxilla reflect the state of the dentition. In infancy and childhood, the jaws become progressively more prominent as the tooth buds successively enlarge and erupt. Loss of the teeth, especially in old age, results in regression of the alveoli and muscular processes, with a significant reduction in the strength of the jaws. Alveolar regression is associated with a relative upward migration of the inferior dental nerve—another factor to be remembered when screws are inserted.

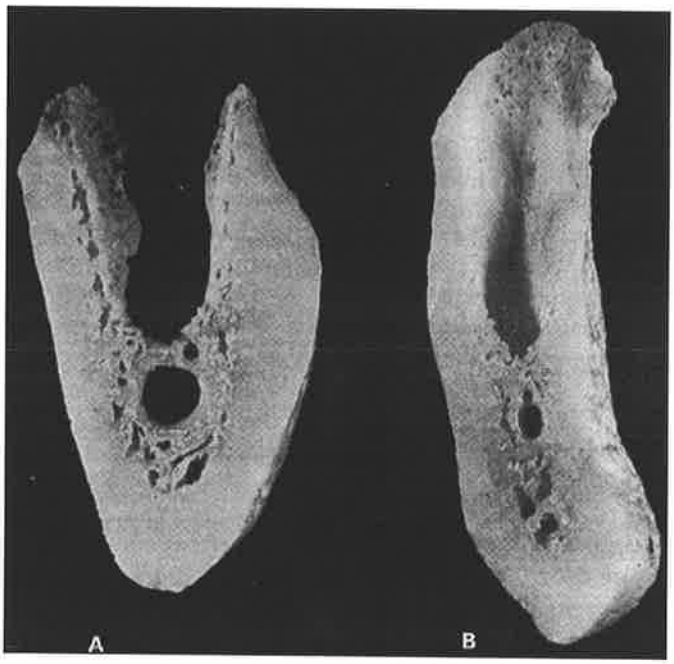


FIG. 2.6.*Adult mandible.* **A.** *The mandible is sectioned through the second molar socket: the mandibular canal is about 3 mm in greatest diameter. On the buccal surface, the cortical plate is 2.7 mm thick at its thinnest point.* **B.** *The mandible is sectioned through the socket of the canine tooth: the canal, now termed the incisive canal, is ~1 mm in diameter. The cortical plates are 2.5–3 mm thick.*

TABLE 2.1

Major and minor zones in the calvaria and skull base, identified alphabetically (Cooter & David 1989)

Major zone	Major code	Minor zone	Minor code
Frontal	F	calvarial	FC
		frontal sinus anterior	FSA
		frontal sinus posterior	FSP
		anterior fossa	FA
		cribriform plate	FCP
		coronal suture	F:P
Parietal	P	Calvarial	PC
		Sagittal suture	P:P
		squamosal suture	P:T
		lambdoid suture	P:OC
Sphenoidal	S	lesser wing	SL
		greater wing	SG
		sph.-frontal suture	S:F
		basal	SB
		sph.-occ. Synchond	S:OC
Temporal	T	calvarial	TC
		basal	TB
		petrous	TP
Occipital	OC	cavarial	OCC
		basal	OCB

TABLE 2.2

Major and minor zones of the facial skeleton, identified alphabetically(Cooter & David 1989)

Major zone	Major code	Minor zone	Minor code
Naso-ethmoidal	NE	nasal bone	N
		naso-frontal suture	N:F
		maxill. frontal process	NMX
		ant. Ethmoid	EA
		post. ethmoid	EP
Zygomatic	Z	arch	ZA
		body	ZB
		zyg. -frontal suture	Z:F
		zyg. maxill. suture	Z:MX
Orbital	O	roof	OR
		medial wall	OM
		lateral wall	OL
		floor	OF
		inferior rim	OI
		superior rim	OS
Maxillary	MX	ant. Wall	MXA
		buttress	MXB
		palate	MXP
		dento-alveolar	MXD
		pterygoid	MXT
Mandibular	MD	condyle	MDC
		coronoid process	MDP
		ramus	MDR
		angle	MDA
		body	MDB
		symphyseal	MDS
		dento-alveolar	MDD

TABLE 2.3

Numerical coding of fracture severity (Cooter & David 1989)

0	=	no fracture
1	=	undisplaced fracture
2	=	obviously displaced fracture
3	=	comminuted and/or compound fracture

Computer coding of CMF bones

Cooter & David (1989) have devised an alphanumeric system of coding fractures of the skull according to their anatomical sites, coded alphabetically, and to their severity, coded by a numerical scale (0-3). The bones of the skull are classed as major zones; the sutures and various bone parts or regions are classed as minor zones. The code letters for the major and minor zones are set out in Tables 2.1 and 2.2; the severity scale is set out in Table 2.3. The clinical use of this alphanumeric system is considered in Chapter 11.

The Craniomaxillofacial Cavities

Cranial cavity

The cranial cavity is lined by the dura mater, a strong membrane of great surgical importance. It is composed of interlacing collagen fibres, and some elastic fibres, interspersed with fibrocytes and fibroblasts. The dura mater is both the inner periosteum of the skull bones and the outer meningeal investment of the brain,

and in keeping with its dual roles, it is composed of two separable layers. The outer layer is osteogenic; it contains the meningeal arteries and veins, and also the meningeal nerves; the dura, unlike the brain and the leptomeninges, is pain-sensitive. The inner layer is separated from the arachnoid by the subdural space, which is lined by flat cells of fibroblastic type. Nabeshima et al (1975) considered that the subdural space is really intradural since electron microscopy shows that the outer layer of the arachnoid is composed of mesothelial cells with tight junctions: in their interpretation, this layer is the true barrier between dura and arachnoid, and the subdural space is a cleft in a fascial plane within the dura. Haines et al (1993) have endorsed this concept. However this may be, the subdural space is very real and very important in the surgical pathology of head injuries, and it is very easily opened up, even by the entry of air into the cranial cavity, if intracranial pressure is low (Fig. 19.14).

Once the cranial sutures have fused and the fontanelles have closed, the walls of the cranial cavity are rigid and the total intracranial volume is fixed. The three major components (brain, spinal fluid and blood) are incompressible: an increase in any one of these three, or the addition of another volume such as a clot or abscess, will cause a rise in intracranial pressure (ICP) unless an equal volume is displaced extracranially. Cerebrospinal fluid (CSF) can be displaced from the cranium, through the foramen magnum to the more distensible spinal subarachnoid space. Venous blood can be displaced via the dural venous sinuses to the extracranial veins. This compensatory capacity is limited both in volume and in rate of displacement. Once the compensatory reserve is exceeded, ICP rises exponentially. In patients with head injuries compensatory reserve may be reduced by brain swelling, which may take several days to subside. Intracranial surgery is then rendered difficult by the inability to retract the brain without excessive force (p. 377) and nursing care and anaesthesia must be conducted in such a way as to prevent any additional rises in ICP. This may be increased inadvertently by overhydration, hypercarbia, hyperpyrexia and the use of volatile anaesthetic agents (p. 252). The clinical measurement of ICP is discussed in Chapter 13: in adults it is normally 5-15 mmHg, being somewhat lower in children and lower still in infants.

Orbital cavity

The orbit is pyramidal in shape (Fig. 2.7). It contains the globe of the eye, the optic nerve, and the external ocular muscles, embedded and sheathed in the orbital fat; the lacrimal gland lies in the upper outer corner of the orbit, in a bony shallow fossa. The orbital cavity is not as high as it is wide and the globe of the eye is therefore nearer to the roof and floor than to the sides (Deuschle 1969). The maxillary component of the orbital floor is very thin as is the medial orbital wall formed by the lateral plate of the ethmoid; these parts of the orbit are often fractured, either as part of a more extensive midfacial fracture, or in isolation as an effect of hydraulic pressure transmitted to the orbital floor through the orbital fat after blunt impact on the globe of the eye (p. 104). The orbital roof is also thin and may be fractured easily; such fractures are often part of a more extensive craniofacial injury, especially an injury resulting from oblique frontal impact. In contrast, the orbital margins are relatively thick, and constitute strong vertical and transverse components of the facial skeleton. Lang (1983), quoting G. Oehmann, gives the following mean lengths of the adult orbit:

roof: 50.5 mm

floor: 48.4 mm

lateral wall: 47.2 mm

medial wall: 40.5 mm

The eye is protected from blunt trauma by the orbital rim, this protection being least effective in the inferolateral area. The optic canal enters the orbit at the apex of the pyramid: Lang (1983) found that the canal is on average 9.8 mm long and 4.6 mm wide at its narrowest point. The optic nerve is often injured

within the canal, either by impact forces transmitted through the intact bone of the canal (Jend & Jend-Rossmann 1984) or orbital contents, or when a fracture enters the canal. The nerve is protected from impact by the orbital fat and from stretching by its tortuosity.

Nasal cavity

The irregularly shaped nasal cavity extends from the roof of the mouth to the skull base and communicates with the other pneumatized cavities in the craniofacial region (Fig. 2.8). It is divided in the midline by the nasal septum comprised in part by the crest of the maxilla, the vomer, and the vertical plate of the ethmoid, together with its cartilaginous component. It is further subdivided by the three paired conchae (turbinate bones). The inferior concha demarcates the inferior meatus, into which debauches the nasolacrimal duct. The middle concha demarcates the middle meatus, which is extended anteriorly as the ethmoid infundibulum; into this drain the ostia of the anterior ethmoid air cells, the maxillary air sinus, and in some individuals the frontal sinus. In other individuals, the frontal sinus drains directly into a recess of the middle meatus, the anatomy being dependent on the position of the uncinate process (Stammberger 1991). Many anatomists have described the frontal sinus drainage as an actual duct (frontonasal or nasofrontal duct). Stammberger has shown that this is a rare finding; in most individuals, the ostium of the frontal sinus lies at the junction of two funnel-shaped cavities — the upper leading down from the floor of the sinus and the lower extending up as the frontal recess, which opens into the middle meatus or infundibulum. In this book, the terms frontonasal duct and frontal recess are used interchangeably and with no implication that the drainage system is either a cylindrical duct or a funnel-shaped recess. The superior concha, which is often split into two components, demarcates the superior meatus, into which drain the posterior ethmoid air cells. Above and posterior to the superior concha is the spheno-ethmoid recess, in which the sphenoid air sinuses have their ostia.

The nasal cavity is enclosed by the bony and cartilaginous skeleton of the nose. This comprises an upper immobile bony portion and a lower, mobile cartilaginous portion. Pollock (1992) subdivides the cartilaginous element further, into a middle subregion which equates with the upper lateral nasal cartilages, and a lower area corresponding to the lower lateral or alar cartilages.

The upper osseous portion comprises the paired nasal bones capping a pyramid whose base is formed by the frontal process of the maxilla on each side. Articulating above with the frontal bone, the nasal bone is thick; inferiorly it becomes thin as it meets the upper lateral cartilages. Lying deep to the nasal bones is the perpendicular plate of the ethmoid, and the septal cartilage, and with the vomer at the bone-cartilaginous junction of the septum. In the middle section the upper lateral cartilages overlie the septal cartilage alone, whilst more inferiorly the alar cartilages are tethered to the anterior nasal spine via their medial crura.

The septal cartilage varies in thickness, being strongest and providing most support superiorly at its junction with the vomer. The caudal end and dorsal border are also thickened with a central, relatively thin portion. The cartilage connects to the perpendicular plate of the ethmoid posterosuperiorly, and to the vomerine groove postero-inferiorly. Septal injury is of great importance in more severe nasal fractures, and if not treated may cause nasal obstruction (p. 333).

The nasal cavity is lined with pseudostratified columnar ciliated epithelium (Moran et al 1991), except in the nasal vestibule and nostrils, where the epithelium is stratified squamous, becoming keratinized as it approaches the skin. The epithelium contains goblet cells, secreting mucus, and ciliated cells. The mucus forms on the surface of the nasal cavity in two layers: a fluid inner layer, termed the sol phase, and a viscous outer layer, the gel phase (Stammberger 1991). The

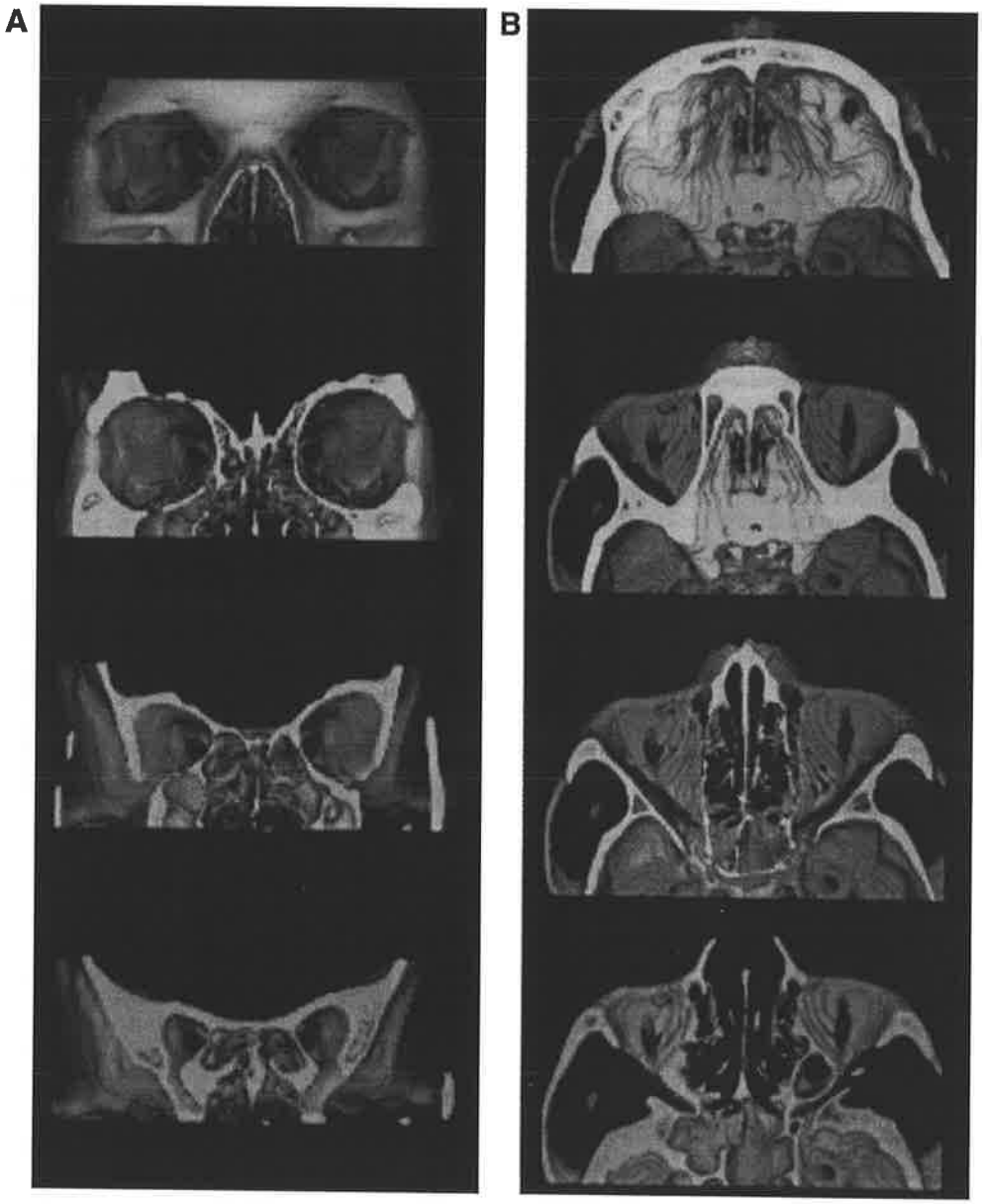


FIG 2.7. ***The orbital cavities.*** **A.** *Coronal CT sections show that the lateral wall is relatively thick, the floor and medial wall very thin, the roof variable but sometimes very thin.* **B.** *Horizontal sections show the pyramidal shape and the close relations with the ethmoid air sinuses.*

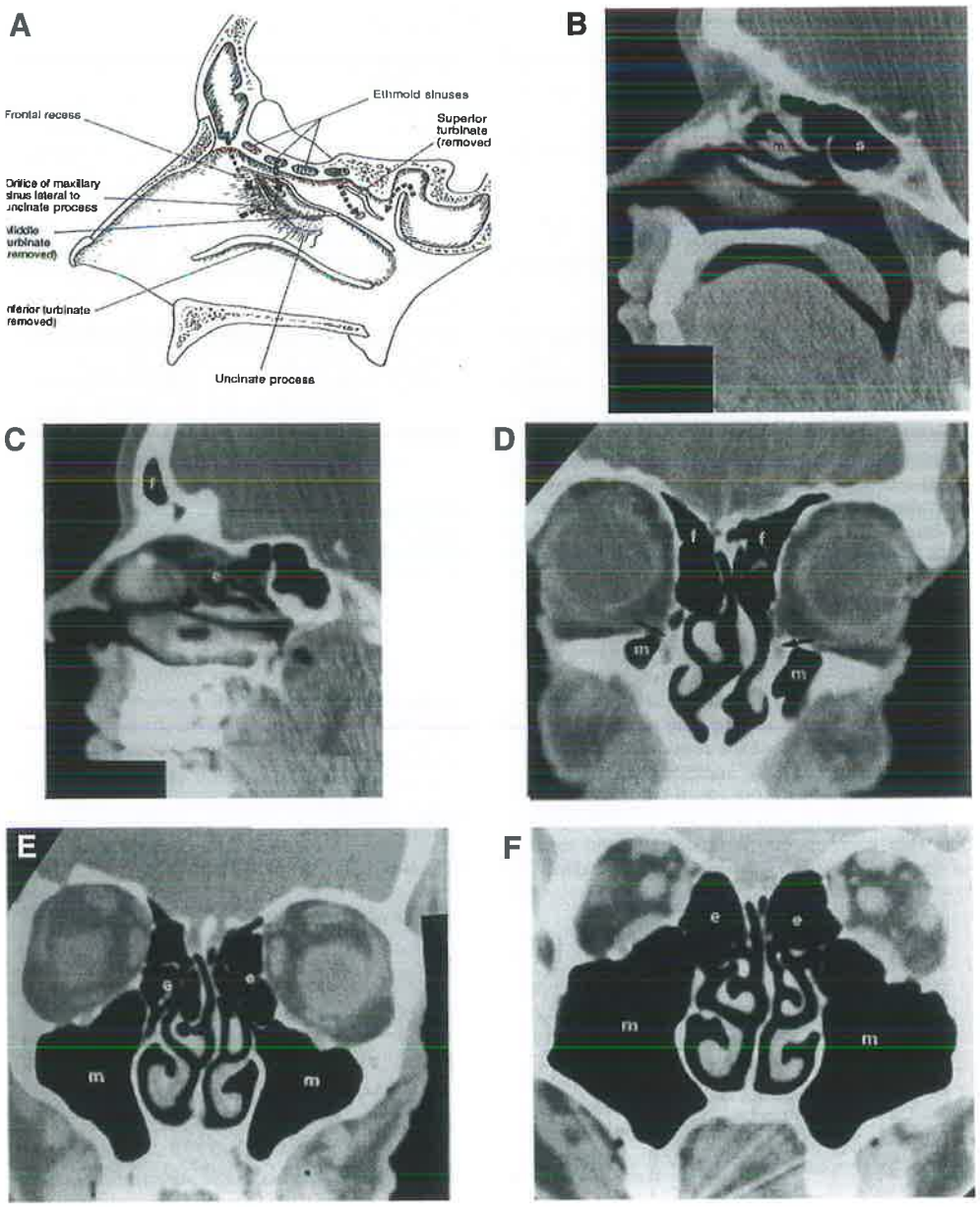


FIG. 2.8. ***The nasal cavity and paranasal air sinuses.*** **A.** *Diagram shows the lateral wall of the nasal cavity after removal of the turbinate bones. The drainage routes of the paranasal air sinuses are shown (broken arrows).* **B.** *CT scan: a parasagittal cut shows the inferior (i) and middle (m) nasal conchae; the upper part of the bulla ethmoidalis is seen above the attachment of the middle concha. The sphenoid (s) sinus is well seen.* **C.** *CT scan: more laterally, the ethmoid (e) and frontal (f) air sinuses are seen.* **D.** *CT scan: an anterior frontal cut shows the frontal (f) and anterior ethmoid (f) air cells; the maxillary air sinuses (m) are also seen. The lacrimal ducts (arrows) are seen on each side* **E.** *CT scan: more posteriorly, the maxillary air sinuses (m) are well developed. On the right side, an arrow indicates the site of the drainage of the maxillary air sinuses into the nasal cavity under cover of the uncinate process. The ethmoid air cells (e) are separated from the orbital contents only by the thin lamina papyracea.* **F.** *CT scan: skull more posteriorly, the maxillary (m) and posterior ethmoid (e) air cells are well developed; the ethmoid air cells have a large surface relation to the dura of the anterior cranial fossa.*

cilia beat continually in the sol layer, slowly moving the overlying gel layer back to the pharynx; foreign material caught on the viscid surface layer is thus moved out of the nasal cavity and swallowed. The nasal epithelium is adherent to the periosteum or perichondrium of the underlying skeletal structures, and contains mucous and serous glands, as well as many blood vessels; the epithelium regenerates well after injury. The nasal conchae have a bony skeleton, covered with a thick mucosa containing venous sinuses with erectile properties. The erectile tissue intermittently dilates and contracts in the normal nasal cycle which regulates air flow through one nostril or the other. In the roof of the nose above the superior concha, the epithelium over an area of about 2 cm^2 is specialized for olfaction: here are found the ciliated olfactory receptor cells. These are bipolar neurons which send their axons through the olfactory filaments to the olfactory bulb; they have considerable regenerative capacity, being replaced by undifferentiated basal cells, but this capacity is lost if the olfactory bulb is destroyed (Fig. 2.9).

The skin cover of the nasal skeleton and cartilages is of variable thickness. Fascial interconnections exist between the lower cartilaginous structures aiding in the maintenance of shape and structure. These tend to be strong in the region between the alar domes but more lax in the junction between alar and upper lateral cartilages.

Paranasal air sinuses

These sinuses are lined with columnar ciliated epithelium, containing mucus-secreting goblet cells; both cilia and goblet cells are less numerous than in the nasal mucosa. The mucus is transported by ciliary action to the ostia of the sinuses, and then into the nasal cavity; endoscopic studies (Stammberger 1991) have shown that each sinus has its characteristic pattern of transport of mucus secretions.

The anatomy of the paranasal air sinuses is very variable, and these variations are best studied in high resolution thin-section CT scans (Fig. 2.8B-F). This is especially important when endonasal or transnasal surgery is planned.

Maxillary sinuses

At birth, all the paranasal sinuses are rudimentary. The maxillary sinuses are present as narrow slits or pouches in the wall of the nasal cavity; they expand slowly at first, and more rapidly after the eruption of the permanent dentition.

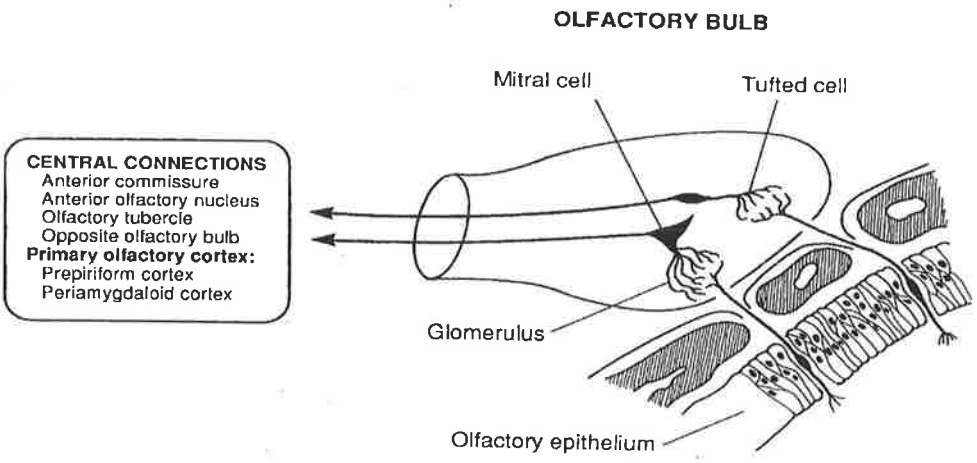


FIG. 2.9. ***Olfactory pathways.*** *The diagram shows the olfactory bulb in the cribriform fossa, and its connections with the olfactory epithelium; some of the centripetal pathways are shown, with their central connections*

In adult life, the maxillary sinus usually fills the entire body of the maxilla; it may even extend into the zygomatic arch and below the floor of the nose. The maxilla has fragile walls and is easily fractured.

Ethmoid sinuses
The labyrinthine ethmoid air cells are variable in number, shape and size. They are well formed but small at birth, when they have no direct relation with the dura of the anterior cranial fossa; however, they expand rapidly and by the third year of life they are quite capacious, being separated from the dura by only a thin plate of bone, and in relation to the anterior fossa over an area of about 5 cm^2 (Caldicott et al 1973). In adult life, ethmoid pneumatization may be extensive; the crista galli), and even structures outside the ethmoid bone, may be pneumatized.

Frontal sinuses
The frontal sinus develops between the inner and outer tables of the frontal bone; pneumatization is said to become evident during the third year, but in our experience these air sinuses do not usually develop a relationship with the anterior cranial fossa until the age of 4 years or even later. The anterior and posterior walls of the frontal sinus may be quite thin and easily fractured. The sinuses may be large and then contribute to the fullness and shape of the frontonasal region.

Sphenoid sinuses
These sinuses are evident at birth but enlarge slowly and are the last of the paranasal sinuses to establish a relation to the anterior cranial fossa (Fig. 2.10); much of their development takes place after puberty.

Ostia
The orifices of the paranasal sinuses (Fig. 2.8A) have surgical importance. If they are obstructed, the secretions of the sinus are retained and a tension mucocele or chronic sinusitis may result. The frontal sinus ostium and frontal recess are often involved in depressed frontal fractures, and post-traumatic frontal mucoceles are not uncommon; measures to prevent infection or mucocele formation are discussed on page 372. The ethmoid ostia may also be blocked, resulting in local infection or orbital cellulitis. Farmand & Gottsauner (1991) reported on the findings of endoscopy of the maxillary air sinuses after injury; they could visualize the ostium of the sinus in only half their cases, but concluded that it is rarely obstructed except when there is a severe deviation of the nasal septum.

Oral cavity

The oral cavity is incompletely enclosed by the inner surface of the alveolar processes and the lingual surfaces of the teeth: these form lateral anterior walls, the roof being formed by the hard palate. Posteriorly, the mouth is continuous with the oropharynx, the posterior wall of which is formed by the upper two cervical vertebrae. It is lined by stratified non-keratinizing squamous epithelium, which is heaped up in papillae of several types over the surface of the tongue: the filiform and circumvallate papillae contain chemoceptive cells organized in taste buds and responsible for perception of the basic tastes — salt, sweet, sour and bitter. The oral cavity is irrigated by saliva from the sublingual, submandibular and parotid salivary glands and from numerous smaller buccal and labial glands (see below).

Alveoli and teeth

The teeth are supported by the alveolar processes of the maxillae and mandible. The alveolar bone is formed as the teeth develop from tooth germs, emerge and eventually take up their positions within the dental arches. When teeth are lost through disease or trauma, the alveolar processes or parts of them are resorbed; the edentulous jaw may indeed be devoid of all alveolar processes (Fig. 19.11)

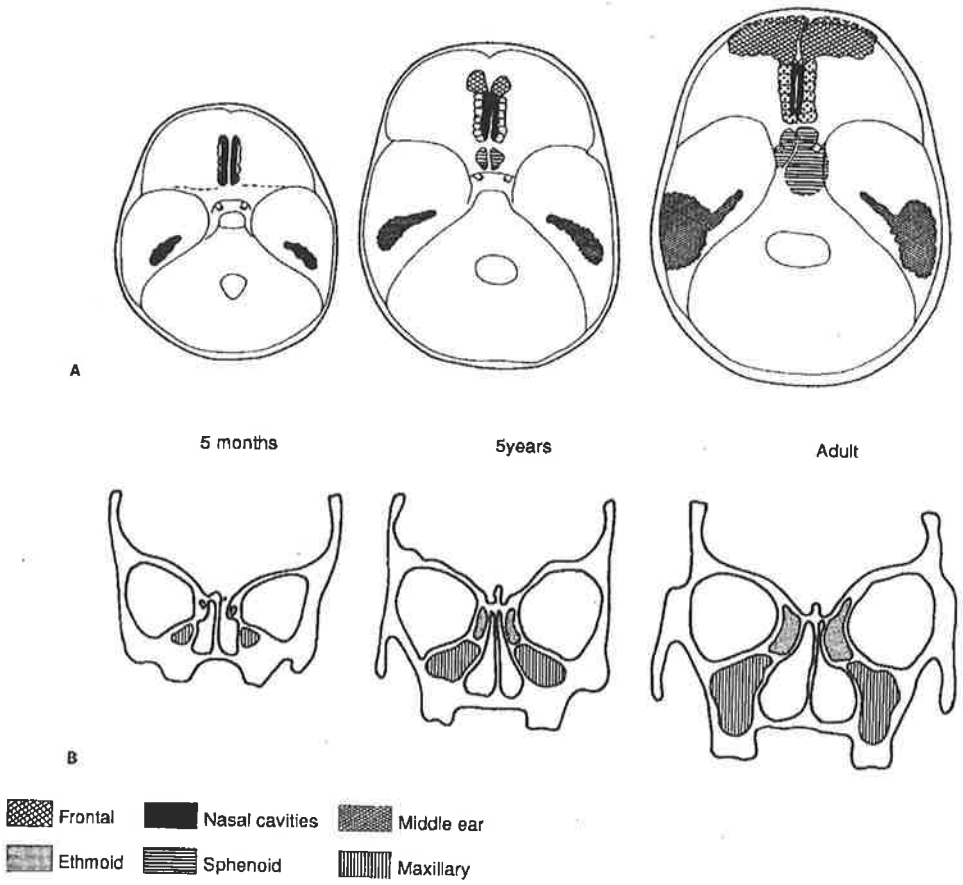


FIG. 2.10. ***Growth of the paranasal air sinuses.*** *Diagrams, based on dissections or CT scans, show the approximate size of the air sinuses at 5 months, 5 years and in adult life. There is much individual variation.* **A.** *The air sinuses as seen from above, showing areas separated by thin bone from the dura mater. After Caldicott et al (1973), by courtesy of the Journal of Neurosurgery.* **B.** *The air sinuses as seen from in front.*

and is then extremely fragile.

Between about 6 months and 2 years, the primary dentition of eight incisors, four canines and eight molars erupts through the alveolar bone and covering gum tissue (Table 2.4). The permanent teeth, eight incisors, four canines, eight premolars and twelve molars erupt later, between about 6-20 years (Tables 2.5 and 2.6). For a period of time, around 6-14 years, the mixed dentition is present with some teeth from both deciduous and permanent dentitions (Fig. 2.11).

Each tooth is held firmly in its socket within the alveolus by the tough periodontal ligament passing between cementum on the tooth root and the cortical lamina dura lining the tooth socket. The periodontal ligament has an important sensory function also as it contains proprioceptive nerve endings that provide information to guide mandibular movements during function. The morphology of a tooth and its supporting structures is shown in Fig. 2.12. Relations of teeth and alveoli can be visualized in a panoramic radiograph of the jaws (Fig. 2.13).

Normally each tooth contacts a neighbouring tooth at the regions of maximum crown curvature (Fig. 2.12). The adjacent tooth roots, each within their individual sockets, are separated by wedges of alveolar bone which form the interdental septa. These septa are composed of cancellous bone with a thin covering of compact cortical bone termed the lamina dura. A healthy lamina dura appears as a thin radio-opaque line on radiographs. The septa are covered by gingival tissue which extends some distance into the interdental space. This tissue is known as the interdental papilla.

TABLE 2.4

Emergence times of deciduous teeth (months) in two Caucasian populations studied longitudinally

A Maxilla **Population***	**Authors**	***n***	i^1	i^2	**c**	m^1	m^2
Umea, Sweden	Lysell et al (1962)	171	10.2	11.4	19.2	16.0	29.1
London, England	Leighton (1968)	84	9.2	10.6	18.2	14.7	26.3
B Mandible **Population***	**Authors**	***n***	i^1	i^2	**c**	m^1	m^2
Umea, Sweden	Lysell et al (1962)	171	8.0	13.2	19.7	16.3	27.1
London, England	Leighton (1968)	84	7.3	11.5	18.3	14.8	25.7

* Male and female data combined in absence of significant differences between sexes.

TABLE 2.5

*Emergence times of permanent teeth (years) in South Australian school children**

A Maxilla		I^1	I^2	**C**	Pm^1	Pm^2	M^1	M^2
Males	5th Percentile	5.8	6.4	9.5	8.9	9.7	5.1	10.3
	Median	7.4	8.6	11.8	11.3	12.1	6.7	12.7
	95th Percentile	9.1	10.9	14.2	13.6	14.4	8.4	15.1
	Range	3.3	4.5	4.7	4.7	4.8	3.3	4.8
Females	5th Percentile	5.6	6.0	8.8	8.6	9.2	4.8	9.9
	Median	7.2	8.2	11.2	10.8	11.7	6.6	12.3
	95th Percentile	8.7	10.5	13.7	13.0	14.2	8.3	14.7
	Range	3.1	4.5	4.9	4.4	5.0	3.6	4.8
B Mandible		I^1	I^2	**C**	Pm^1	Pm^2	M^1	M^2
Males	5th Percentile	5.0	6.0	8.9	9.0	9.7	5.0	9.8
	Median	6.6	7.8	11.0	11.2	12.1	6.6	12.2
	95th Percentile	8.3	9.6	13.1	13.3	14.5	8.3	14.5
	Range	3.3	3.6	4.2	4.3	4.9	3.5	4.7
Females	5th Percentile	4.8	5.7	8.0	8.5	9.1	4.9	9.4
	Median	6.4	7.5	10.1	10.6	11.7	6.4	11.8
	95th Percentile	8.0	9.3	12.2	12.7	14.2	8.0	14.1
	Range	3.2	3.5	4.2	4.3	5.1	3.1	4.7

* Percentiles derived by logistic regression of data from over 37 000 children aged 4–16 years who were examined in 1988 (Diamanti 1991).

The gingival tissues, which act as cuffs around all the teeth, are composed of tough stratified squamous epithelium and fibrous connective tissue. The gingival crest is the free part of tissue separated from the neck of the tooth by a capillary space, the gingival crevice. Gingivae are attached to the necks of teeth at their gingival attachments and to the periodontal ligaments and septal bone. This region, where the gingival tissues surround the teeth, is light pink in colour and tightly bound to the underlying fibrous tissue, giving the characteristic 'orange-peel' appearance of healthy gums. Over the apical part of the alveolus, however, the gingival tissues merge, at the vermilion border, with the alveolar mucosa which is reddish and highly vascular like the epithelium lining the oral cavity.

In the upper jaw, the maxillary sinus is an important superior relation to the premolars and molars, often extending from the region of the first premolar posteriorly as far as the maxillary tuberosity which it often invades, particularly in later life. In radiographs, the maxillary posterior tooth roots sometimes appear to perforate the floor of the sinus but there is a layer of bone, albeit very thin, separating the roots from the lining of the sinus.

Immediately behind the maxillary third molar lies the tuberosity, the hamular notch and the hamular process of the medial pterygoid plate around which the tendon of the tensor palati muscle passes. The hard palate can be seen on orthopantomograms lying above the level of the tooth roots.

In the mandible, the most important relation of the tooth roots and alveoli is the mandibular or inferior alveolar canal (Fig. 2.6) with its enclosed neurovascular bundle. This canal passes very close to the third molar roots and, rarely, it may be encircled by them. The canal passes below the posterior teeth to the mental foremen located in the region of the apex of the second premolar tooth. Here, the canal divides into mental and incisive terminations.

Another important relation of the lower alveolus is the lingual nerve which arises in the infratemporal fossa and gains access to the oral cavity by passing below a small ridge of bone lingual to the last mandibular tooth, which in the adult is the third molar. In this position the nerve is protected only by this ridge of bone, which has been termed the endalveolar crest, and by the covering mucosa (Volchansky & Makings 1984; Penhall 1992). Figure 2.14 illustrates the relations of the maxillary and mandibular alveoli in the region of the second molar. The mylohyoid muscle, suspended from the mylohyoid lines on each side of the mandible and attached in front to the inside of the symphysis and below to the hyoid bone, forms the interface between the intraoral structures and those lying in the submandibular region. Important structures lie superior to the mylohyoid muscle in the floor of the mouth in close relation to the mandibular alveolus: the sublingual gland, submandibular duct, lingual nerve and at a deeper level the hypoglossal nerve and lingual artery.

TABLE 2.6

Emergence times of permanent teeth (years) in four populations

A Maxilla **Population**	**Authors**		I_1	I^1	I^2	C	Pm^1	Pm^2	M^1	M^2
USA (Blacks)	Garn et al (1972)	Males	6.8	8.1	10.9	10.2	10.8	6.5	12.3
		Females	6.8	7.9	10.2	9.7	10.5	6.2	12.0
USA (Whites)	Garn et al (1972)	Males	7.3	8.4	11.5	10.9	11.5	6.2	12.5
		Females	7.1	7.8	10.7	10.4	10.8	6.4	11.7
Hong Kong (Chinese)	Lee et al (1965)	Males	7.4	8.7	11.3	9.8	10.9	6.4	12.6
		Females	7.2	8.3	10.4	9.5	10.4	6.2	12.0
New Guinea (Kaiapit)	Malcolm & Bue (1970)	Males	6.5	7.4	10.7	10.2	11.3	5.7	11.2
		Females	6.7	7.3	9.3	9.5	10.4	5.7	10.3
B Mandible **Population**	**Authors**		I_1	I^1	I^2	C	Pm^1	Pm^2	M^1	M^2
USA (Blacks)	Garn et al (1972)	Males	6.1	7.2	10.2	10.3	11.2	6.2	11.9
		Females	5.8	6.7	9.4	9.7	10.7	5.9	11.3
USA (Whites)	Garn et al (1972)	Males	6.2	7.5	10.9	11.0	11.8	6.2	10.2
		Females	6.4	7.1	9.9	10.5	11.2	6.2	11.5
Hong Kong (Chinese)	Lee et al (1965)	Males	6.2	7.5	10.5	10.4	11.3	6.0	11.9
		Females	6.1	7.2	9.6	9.8	10.7	5.9	11.3
New Guinea (Kaiapit)	Malcolm & Bue (1970)	Males	6.2	6.8	10.0	10.4	11.4	5.2	11.3
		Females	6.5	6.9	8.9	10.1	10.4	5.5	10.3

* From data tabulated by Eveleth & Tanner (1976).

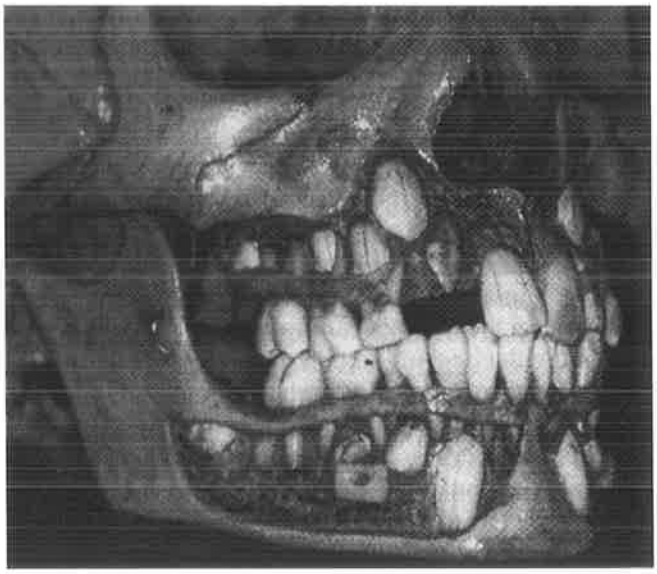


FIG. 2.11. ***The mixed dentition.*** *Dissection of a young (8-9-year-old) skull showing some permanent incisors and the first permanent molars fully erupted. Also visible are the primary molars and a number of succedaneous permanent teeth developing within their crypts.*

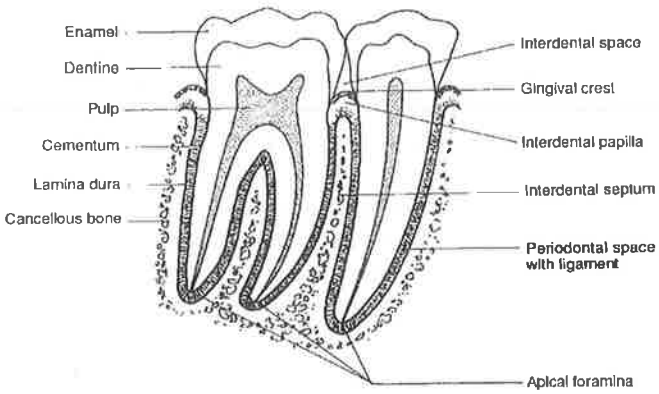


FIG. 2.12. ***Morphology of teeth and surrounding tissues.*** *Section through a mandibular second premolar and first molar showing internal dental structures and the features of the alveolus and interdental region.*

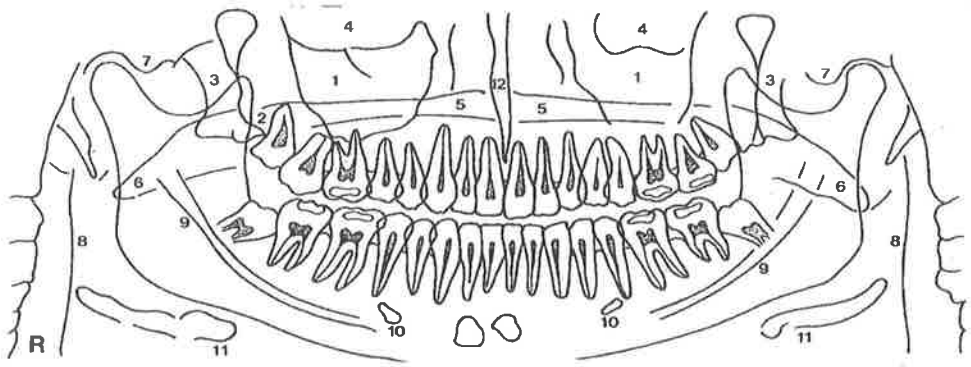


FIG. 2.13. ***Anatomical relations of the teeth and alveoli.*** *Drawing of an orthopantomogram showing the principal anatomical relations of the teeth and alveoli: (1) maxillary sinus; (2) maxillary tuberosity; (3) lateral pterygoid plate; (4) orbital cavity; (5) hard palate; (6) soft palate; (7) articular eminence; (8) posterior pharyngeal wall; (9) inferior alveolar (mandibular) canal; (10) mental foramen; (11) hyoid bone; (12) nasal septum.*

Geometric Concepts of the Craniofacial Skeleton

There have been several attempts to produce geometric models of the craniofacial skeleton. These models present simplified interpretations of the complex anatomy of the bony structural pillars that transmit the forces of mastication, devised to further the understanding of fracture patterns (Fig. 2.15).

The dispersion of masticatory forces through vertical bony columns to the broad areas of the skull base was appreciated by Le Fort himself (p. 17). Rowe & Killey (1955) emphasized the mechanical strength of the alveoli and the transverse palatal arch, on which rest the paired buttresses of the maxillae, the palatine-pterygoid buttresses posteriorly and the zygomatic complexes laterally; they saw additional support in the central buttress of the vomer and ethmoid vertical plate (Fig. 2.15A). In a functional analysis of the facial skeleton, Sicher & Du Brul (1975) considered that the facial structures were anchored to the skull base by three pairs of curved vertical pillars: the canine pillar, the zygomatic pillar, and the pterygoid pillar (Fig. 2.15B). Sturla et al (1980) performed cadaver impaction studies, on the basis of which they proposed a lattice-shaped structure whose central and lateral vertical pillars spanned between a superior cranial shelf and an inferior palatal platform (Fig. 2.15C). Their lattice shaped model had a layout resembling the architectural concept of Rowe & Killey (1955): both were essentially anatomical schematizations of the face with the thin laminae of bone removed. The projection of these vertical facial pillars to the main structural zones of the cranial base has been reviewed by Fain (1980) who examined the effects of facial bone trauma on the basal structures The concept of structural pillars of the facial skeleton has also formed a basis for fracture management whereby the anterior vertical buttresses are reconstituted to preserve the facial height (Manson et al 1980; Gruss & Mackinnon 1986).

In a less anatomical fashion, Gentry et al (1983) conceived the face geometrically as a series of triplanar osseous struts (Fig. 2.15D; Tables 7.1-7.3; p. 183). They identified three major struts in the horizontal plane, five in the sagittal plane, and two in the coronal plane; also considered were the relationships of the soft tissues to each strut. This analysis was based on high resolution computer tomography of the traumatized face, and was not designed to illustrate mechanical concepts of the facial skeleton. Luce (1984) also proposed a less anatomical format when he illustrated the relationship between the anterior cranial fossa, frontal sinus, orbits and the ethmoid air cells with a diagram in which each region is depicted as a box. This cellular concept of CMF anatomy is of interest in relation to the mechanical role of the paranasal air sinuses. The sinuses form a large aerated honeycomb: from a teleological view, this structure seems designed to absorb the energy of facial impact and so to protect the brain and the sensory organs (Blanton & Biggs 1969). When force is transmitted from below, the pyramidal orientation of these sinuses, with the maxillary sinuses forming the base and the sphenoid sinus as the apex, constitutes an architectural structure that is particularly well suited to a protective, energy-absorbing role (Riu et al 1960).

The forces required to fracture the craniofacial bones are impressively high; in Chapter 4, the data derived from cadaver studies are reviewed. It is necessary to relate the skeletal anatomy of a fracture to the direction of the impacting force as well as to its magnitude. The facial skeleton is well adapted to withstand the vertical forces of mastication, but it is less tolerant of forces in the horizontal plane, which may detach the alveolar complex from its supports, or cause complete craniofacial disjunction. The bones of the craniofacial region are intricately linked and the majority of their junctions are immovable sutures. As a result of this configuration there is a high propensity for a stereotyped pattern of fracturing to result from a single blow and the angle of impact may be an important determinant of the final fracture pattern. From a knowledge of the normal spatial relationships of the bones, their major structural components and their impact tolerances, a

clear insight can be gained into the dynamic responses to severe impact, the final result of which is the fracture pattern.

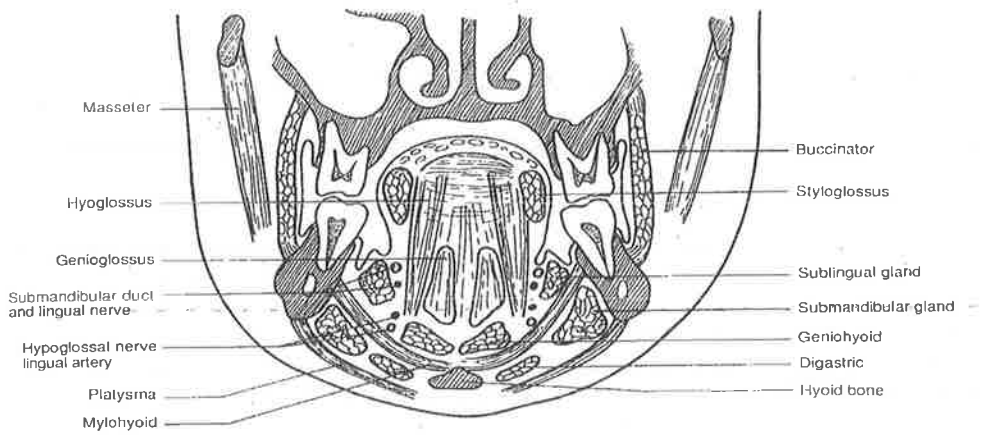


FIG. 2.14. *Sublingual and submandibular regions.* *Coronal section in the region of the second molars showing the sublingual and submandibular relations of the teeth and alveoli.*

Blood Vessels

The blood supply of a region is studied to assess the viability of structures selected for skin flaps and bone flaps, to design surgical approaches and to deal with primary and secondary haemorrhage. Modern concepts of craniofacial surgery have added the need to understand the effects of wide periosteal stripping of the craniofacial skeleton on fracture healing, and to know how to preserve blood vessels and nerves contained in the soft tissues (p. 293). Furthermore, microvascular surgical reconstruction now has a great and increasing role in all aspects of CMF surgery, and this demands knowledge of the vascularity of a region and the potential scope for microvascular anastomosis.

Arterial supply

The arterial blood supply of the CMF structures is derived from branches of the external and internal carotid arteries. There is free anastomosis across the midline (Har-Shai et al 1992) and anastomosis between the internal and external systems in the region of the orbital and nasal cavities (Fig. 2.16).

The common carotid artery divides, usually at the level of the disc between the third and fourth cervical vertebrae, into the internal and external carotid arteries. The internal carotid artery does not branch in the neck and proceeds to enter the base of skull through the carotid canal in the petrous temporal bone. The artery leaves the carotid canal at the foramen lacerum and pierces the dura mater; it then runs forward within the cavernous sinus for about 2 cm before turning upwards and piercing the dura again to enter the subarachnoid space. Within the cavernous sinus, the internal carotid artery gives rise to several small but important arteries. Following Parkinson (1984), most surgical anatomist authors identify the meningohypophyseal trunk, the artery of the inferior cavernous sinus, and McConnell's artery supplying the capsule of the pituitary gland (Stephens & Stilwell, 1969). lithe meningohypophyseal artery typically gives rise to three branches: the tentorial artery, the dorsal meningeal artery and the inferior hypophyseal artery, which supplies the posterior lobe of the pituitary gland (see below). Lang (1983) describes two arterial trunks, a superior

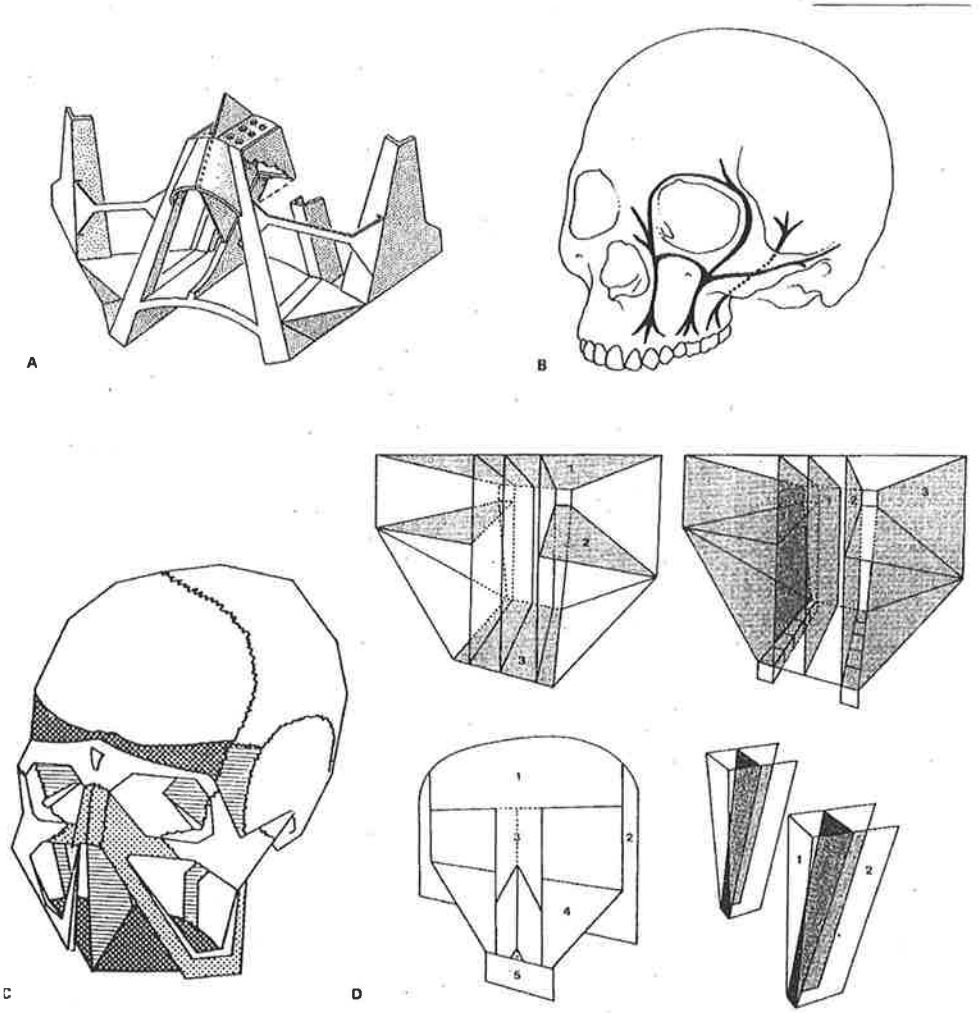


FIG. 2.15. ***Geometric concepts of the facial skeleton.*** *The complex structure of the facial skeleton has been considered in terms of its capacity to absorb the forces of mastication, and diagrammatic interpretations have been published.* **A.** *Rowe & Killey (1955) saw three paired girders transmitting force evenly to the skull base, with an additional midline strut; in their architectural diagram, the arched palate and the inferior orbital rims join the anterior and lateral girders.* **B.** *Sicher & Du Brul (1975) also recognised the importance of the three paired pillars in transmitting force to the skull base. These authors gave greater prominence to the horizontal pillars connecting the curved vertical pillars, especially the supraorbital bar. Redrawn after Sicher & Du Brul by courtesy of Messrs Mosby, St Louis.* **C.** *Sturla et al (1980) emphasised the importance of the vertical pillars, but also stressed the importance of the transverse platforms—the hard palate below and the frontal and sphenoid bones above. Reproduced by courtesy of* Plastic & Reconstructive Surgery *and the authors.* **D.** *Gentry et al (1983) studied thin-section CT scans of cadavers and identified: (i) three horizontal struts (shaded): superior (1), middle or orbital (2), and inferior or palatal (3); (ii) three sagittal struts (shaded): median or septal (1), parasagittal (2) and lateral (3); (iii) five anterior coronal struts: frontal (1), zygomaticofrontal (2), nasofrontal (3), anterior maxillary (4), and anterior alveolar (5); (iv) two paired posterior coronal struts: posterior maxillary (1) and pterygoid (abutting on 1). This complex schematization can be clearly related to anatomical structures seen in coronal and axial scans. Reproduced by courtesy of* American Journal of Roentgenology *and the authors.*

caroticocavernous trunk supplying the pituitary and the clival region, and a lateral caroticocavernous trunk supplying the trigeminal ganglion and the ocular motor nerves. Avulsion of one of these branches can be a cause of carotid-cavernous fistula (pp. 146 and 392). The internal carotid artery then gives off its important branch the ophthalmic artery. From this arises the anterior ethmoid artery which arises in the orbit, enters the ethmoid in its own canal, traverses the cribriform plate, and enters the nose through the cribro-ethmoid canal to anastomose with branches of the maxillary artery. The ophthalmic artery also gives rise to the central artery of the retina and the supraorbital and supratrochlear arteries, which anastomose with the facial artery (Fig. 2.16). The internal carotid artery then gives off the anterior choroidal, anterior cerebral and middle cerebral arteries and anastomoses with the vertebrobasilar system through the circle of Willis. The details of the vascular supply of the brain lie outside the scope of this book.

The external carotid artery gives off named branches to the pharynx, occiput, thyroid, tongue and face; it then divides into the superficial temporal artery and the maxillary artery.

Together with the ophthalmic and occipital arteries, the superficial temporal artery supplies the scalp, and is therefore the nutrient artery of many useful flaps. It divides into two main branches, anterior (frontal) and posterior (parietal), and there are several variations on this pattern (Marano et al 1985). These main branches (which are usually palpable) divide repeatedly, and as they near their terminations they become more superficial; this can prejudice the survival of galeal flaps dissected away from the scalp (Har-Shai et al 1992). The superficial temporal artery contributes to the temporalis muscle and fascia, though the main supply of the temporalis muscle is the deep temporal artery.

The maxillary artery's relationship to the condylar neck and posterior maxilla is important surgically (p. 293), and its anastomosis with the internal system, in and about the nose, has significance in primary and secondary haemorrhage from this area. The maxillary artery gives rise to the middle meningeal artery, which enters the middle cranial fossa, sending an anterior branch to the region of the pterion, and the inferior dental artery, which enters the mandibular canal.

The middle meningeal artery supplies a wide area of the dura mater; it contributes to the supply of the anterior cranial fossa, anastomosing with branches of the ophthalmic artery, the posterior and anterior ethmoid arteries, and with the small accessory meningeal artery, which may arise from it or from the maxillary artery itself. The extradural haemorrhages complicating impacts in the CMF region (p. 385) are usually the result of bleeding from the small meningeal arteries in the anterior fossa rather than from the main trunk of the middle meningeal artery, which is typically lacerated by vertical fractures of the temporal bone. The flat bones of the calvaria receive their blood supply from the pericranium externally, and internally from the dura mater, whose outer layer is functionally equivalent to the pericranium.

Further in its course, the maxillary artery supplies the maxilla and the palate, through the infraorbital artery, the greater palatine artery and the superior dental arteries, and terminates as the sphenopalatine artery. The teeth and their supporting structures are supplied by the inferior and superior dental branches of the maxillary artery but with contributions from the lingual and facial arteries. As the maxillary artery crosses the pterygomaxillary fissure it gives rise to the posterior superior dental artery from which branches enter the maxilla through small foramina above the maxillary tuberosity. These then divide into smaller branches supplying the upper molar and premolar teeth, the lining of the maxillary sinus and the surrounding alveolus. Other branches run outside the alveolus to supply the alveolar mucosa and other soft tissues in the posterior maxillary region. The maxillary artery passes anteriorly along the infraorbital canal, becoming the infraorbital artery and giving off the anterior, and sometimes

middle, superior dental branches to the remaining teeth and supporting tissues. The infraorbital artery provides terminal branches to the upper lip and to the nasal vestibular tissues.

The maxillary artery also gives palatine branches as it crosses the pterygomaxillary fissure. The greater palatine artery enters a canal, passes downwards and emerges through the greater palatine foramen in the posterior region of the palate. From here it passes forwards supplying the palatal mucosa and the gingival tissues on the lingual surfaces of the maxillary teeth. In the region of the incisors it anastomoses with the terminal nasal branches of the maxillary artery emerging through the incisive canal. Lesser palatine arteries also emerge near the posterior border of the hard palate to supply the soft palate where they communicate with branches of the tonsillar and ascending pharyngeal arteries.

The mandibular teeth are supplied by the inferior dental artery. Passing forwards in the mandibular canal the inferior dental artery supplies the posterior mandibular teeth, tooth sockets and cancellous bone of the mandible before dividing into mental and incisive branches in the region of the premolar teeth. The mental branch emerges through the mental foramen to supply lower lip, cheeks and gum tissue as far as the midline where it anastomoses with branches of the facial artery. The incisive branch continues within the mandible to supply anterior teeth and supporting bone.

The buccal mucosa over the mandible is supplied by the buccal branch of the maxillary artery which pierces the buccinator muscle to gain access to the intraoral region. Anastomoses with branches of the facial artery occur in this region also. The lingual mucosa in the mandible is supplied by branches of the lingual artery.

The lingual artery supplies principally the tongue and the floor of the mouth. It arises from the external carotid artery near the tip of the greater cornu of the hyoid bone where it lies on the middle constrictor of the pharynx. The artery then loops above the tip of the hyoid and is crossed by the hypoglossal nerve before descending to enter the floor of the mouth by passing beneath the hyoglossus muscle. Near the anterior border of hyoglossus the lingual artery gives its submental branch to the sublingual gland and floor of the mouth and continues as the deep artery of the tongue which gives many branches to the muscles and mucosa of the tongue.

The facial artery is the main nutrient of the facial musculature and soft tissues. It arises in the neck from the external carotid just superior to the lingual artery. It then passes upwards, grooving the submandibular gland, before passing over the gland and descending between the gland and the ramus of the mandible. While in the neck it gives the ascending palatine and tonsillar branches to the region of the oral fauces. The facial artery crosses the lower border of the mandible at the anterior border of the masseter muscle where the arterial pulse can be felt quite readily. The facial artery takes a tortuous course across the face giving branches to lower and upper lips; it terminates at the side of the nose where it becomes the angular artery, usually passing in its course within the muscles of the region. During its course across the face, the facial artery anastomoses with other arteries in the region, particularly branches of the maxillary, superficial temporal and ophthalmic arteries. The chief superficial facial branches are shown in Fig. 2.16. It is of surgical importance to note that the mandible is unique in the CMF region in the nature of its blood supply. The other facial bones, like the calvarial bones, derive their blood supply from superficial periosteal arteries. The mandible has in addition a central artery, the inferior dental artery, and in this it is comparable with the limb bones. The inferior dental artery is vulnerable in fractures of the body of the mandible. In elderly people, this artery is often obliterated, presumably from atherosclerosis (Bradley 1975; Pogrel et al 1987).

The facial artery is the artery of choice in providing blood supply by microsurgical anastomosis for a free bone graft in restoring a mandibular defect (p. 621). The superficial temporal artery has also been used, or even the occipital artery; the superior thyroid artery is sometimes too small. All the branches of the external carotid artery have relatively thin walls and are apt to go into spasm when microvascular anastomosis is done.

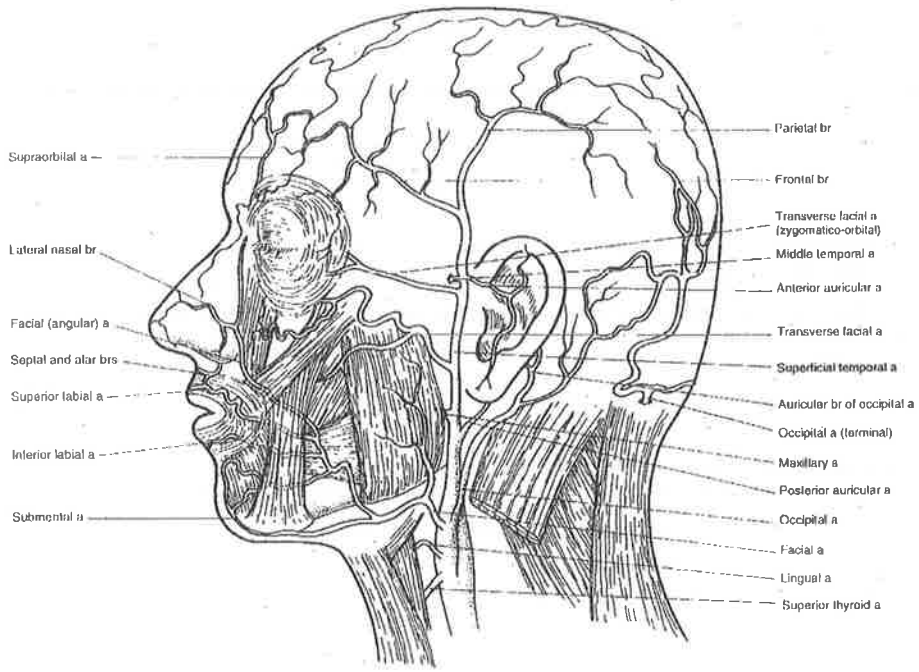


FIG. 2.16. ***Arteries of the face and scalp.*** *With the exception of the supraorbital artery (a branch of the ophthalmic artery), all the named arteries in the diagram are derived from the external carotid artery. There are many variations in origin, calibre and anastomoses; these variations often determine the feasibility of microvascular replantations. Redrawn after Gray's Anatomy and other sources.*

Venous drainage

The posterior part of the superficial facial venous system (Fig. 2.17) drains into the external jugular vein through the retromandibular vein from the upper part of the face and temporal region; the facial vein, arising in the medial canthal region, drains into the internal jugular vein, but has anastomotic connections both with the external jugular vein and the cavernous sinus. There are no valves in the superficial facial veins. There are deep venous systems of surgical significance, the lingual veins, the pharyngeal plexus and the pterygoid plexus, all of which drain into the internal jugular vein. The large pterygoid plexus, lying around the pterygoid muscles, anastomoses with the facial vein and also with the cavernous sinus; it receives venous blood from the meninges and the orbit as well as from the subtemporal region. The venous drainage of the orbit is in communication with both the pterygoid plexus and with the cavernous sinus; these communications become very obvious in the presence of a carotid-cavernous fistula (Fig. 13.14) and are relevant in determining the spread of infection.

The venous drainage of the maxillary and mandibular teeth is through veins that accompany the arteries and pass forward into the anterior facial vein or backward into the pterygoid plexus and so into the cervical jugular system.

The venous blood from the cranial cavity finds its way out through the sagittal transverse and sigmoid sinuses to the jugular veins, and to a much lesser extent through the cavernous sinus system to the facial venous plexuses. The cavernous sinus is an intradural blood space situated adjacent to the pituitary

fossa and body of the sphenoid sinus (Fig. 2.18). It is pyramidal, with the apex anteriorly, and lies between two layers of dura. The medial and inferior walls are formed by the periosteal dura of the body of the sphenoid and the dural fold which forms the lateral wall of the pituitary fossa. The superior surface is triangular and formed by the free and the attached edges of the tentorium and by the interclinoid ligament.

Medially the cavernous sinus is related to the pituitary fossa and the sphenoid air sinus; laterally to the temporal lobe. The posterior wall lies within the posterior fossa and is related to the brain stem. The apex of the cavernous sinus is related to the apex of the orbit and the superior orbital fissure. Inferiorly lies the sphenoid air sinus.

The major venous tributaries of the sinus are: the superior ophthalmic vein which leaves the orbit through the superior orbital fissure to enter the cavernous sinus anteriorly; the sphenoparietal sinus which lies along the edge of the lesser wing of the sphenoid; the superior and inferior petrosal sinuses which connect the cavernous sinus with the sigmoid sinus and jugular bulb respectively; the superficial and deep middle cerebral veins.

The cavernous sinuses on each side are united by a variable pattern of intercavernous sinuses which lie within the dural wall of the pituitary fossa and by a basilar plexus of dural veins which lie between the dura overlying the clivus. Other venous tributaries connect the cavernous sinuses to extracranial venous plexuses such as the pterygoid plexus. The sinus contains the internal carotid artery, and the third, fourth, fifth and sixth cranial nerves. Within the cavernous sinus the internal carotid artery gives rise to the small but important branches described above (p. 47).

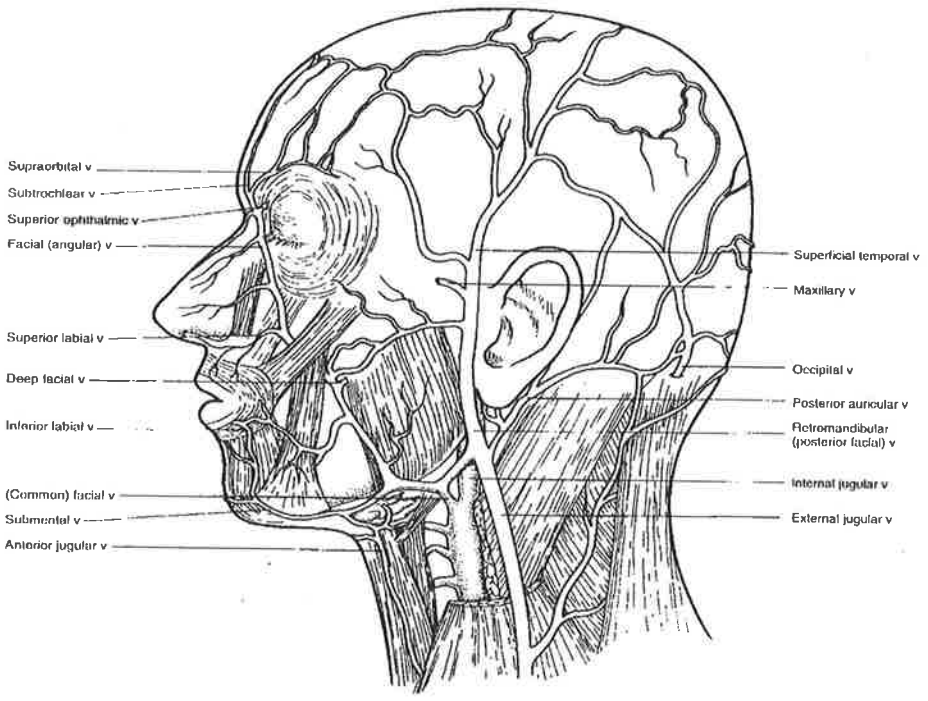


FIG. 2.17. *Superficial veins of the face and scalp.* *These show even more variations in origin, calibre and anastomoses than the arteries, and are also of importance in microvascular replantations. The superficial veins anastomose with the deep venous system at many points: (i) at its origin, the facial (angular) vein connects with the superior ophthalmic vein, and so with the cavernous sinus; (ii) the maxillary vein connects with the pterygoid venous plexus; (iii) the occipital, superficial temporal and supraorbital veins connect through emissary veins (not shown) with dural veins and venous sinuses. These connections may be routes for spread of infection, resulting in cavernous sinus thrombophlebitis or subdural abscess (p. 147). Redrawn after* Gray's Anatomy *and other sources.*

Lymphatic drainage

Lymphatic vessels from the scalp and the superficial facial tissues drain into named and unnamed lymph nodes: the chief of these are the submental, submandibular, parotid, retroauricular and occipital nodes. These in turn drain into deep cervical nodes. Lymphatics from the deeper oral viscera and from the nasal, nasopharyngeal and tympanic cavities also drain into deep cervical nodes. lymph drainage of the teeth and supporting tissues reaches regional nodes in the sublingual and submandibular region before entering the deep cervical vessels. The dura mater has a rich lymphatic system (Miller & Woollam 1962), but the brain, like the eye, has no lymphatic drainage.

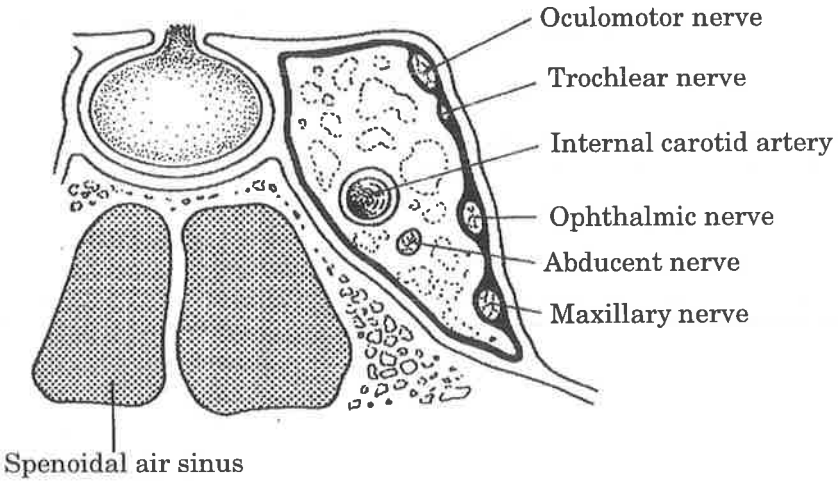


FIG. 2.18. ***The cavernous sinus.*** *The diagram shows the sinus and its contents in coronal section. The sixth (abducent) cranial nerve is within the lumen of the sinus. The maxillary and ophthalmic branches of the fifth cranial nerve are embedded in the deep dural plate of the lateral wall Of the sinus, as are the third (oculomotor) and fourth (trochlear) nerves: Lang (1983) has shown that these nerves are sheathed in arachnoid for short distances after they enter the wall of the sinus.*

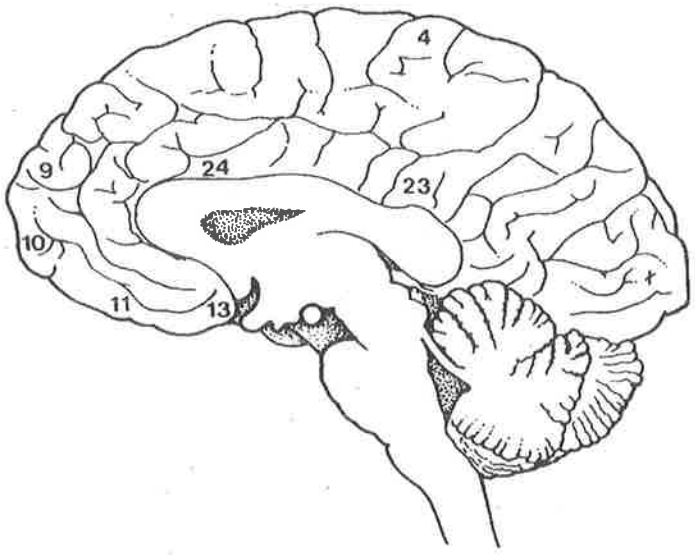


FIG. 2.19. ***Medial aspect of cerebral hemisphere.*** *Diagram showing the chief cortical areas of the medial frontal lobe; the numerals indicate cortical areas identified by Brodmann.*

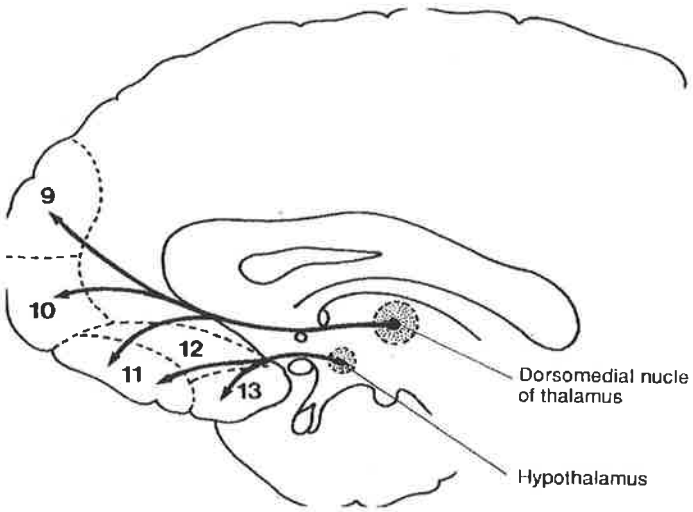


FIG. 2.20. ***Fibre connections of prefrontal and orbital cortex.*** *The granular prefrontal areas have strong two-way connections with the dorsomedial nucleus of the thalamus through the thalamofrontal bundle. The posterior orbital cortex has connections with the hypothalamus through the medial forebrain bundle.*

Brain

Within the cranial cavity is the brain, lying in the protective bath of CSF provided by the subarachnoid cisterns. Magnetic resonance imaging (MRI) makes it possible to visualize cerebral lesions and anatomical structures as small as 5 mm in diameter, or even smaller. Knowledge of the gross anatomy of the brain has therefore become clinically more relevant than in the past, while functional neuroanatomy remains indispensable in understanding the neurological effects of head injury. However, gross anatomy is best studied in correlative atlases (Lang 1983; Duvernoy 1991), and clinical neuroanatomy is too vast a subject to be reviewed here; for those interested in the anatomy of nuclei and fibre systems, Brodal (1981) and Carpenter & Sutin (1983) provide very detailed textbooks, while Noback et al (1991) give a well-illustrated introductory account. However, it is necessary to consider the clinical and surgical anatomy of those parts of the brain which are in direct relation to the frontal bone and the floor of the anterior fossa, and to consider their functions in the context of CMF trauma.

Frontal lobes

Any part of the brain may be injured by impacts striking the CMF region (p. 138), but the frontal lobes are especially vulnerable. The neuroanatomy of the frontal lobes is therefore clinically very important. in understanding post-traumatic cerebral disabilities (p. 653) and their neuropsychological evaluation (Appendix III).

The inferior surface of each frontal lobe conforms closely with the contours of the anterior cranial fossa. Medially, the narrow gyrus rectus extends back from the region of the crista galli to the level of the tuberculum sellae. It is bounded laterally by the olfactory sulcus, in which rests the olfactory tract, and medially by the interhemispheric fissure; it extends back to the medial olfactory strict, one of the terminal components of the olfactory tract. Lateral to the olfactory sulcus are the orbital gyri, overlying the orbital plates and often showing sulci conforming with ridges in the bony surface. Histologically, these gyri are composed of typical six-layered neocortex, and except in the posterior part, the cortex shows numerous granule cells, especially in layer IV. It is usual to identify this granular

part of the frontal cortex by the Brodmann numbers 9, 10 and 11, though one may be sceptical of the precise demarcations in the mosaic maps of cortical areas devised by Korbinian Brodmann (1868-1918) and later students of the cytoarchitectonics of the cortex (Fig. 2.19). Posteriorly, the cortex contains fewer granule cells, and indeed the most posterior part is agranular (area 13 in current terminology); there is considerable individual variation in the demarcations of these areas (Beck et al 1950).

The orbital cortex receives a strong projection from the dorsomedial nucleus of the thalamus through the compact thalamofrontal bundle (Fig. 2.20). There are also numerous fibre connections with the hypothalamus, suggesting autonomic functions. The orbital frontal cortex is often damaged in anterior fossa fractures, especially when the orbital plates are shattered. The clinical effects are striking only when the damage is massive and bilateral (Fig. 2.21). Surgical isolation of the orbital cortex, by cutting through the white matter, was a standard operation in the era of limited psychosurgery. It was found that bilateral lesions of this selective type caused flattening of mood, or sometimes mild euphoria, but without intellectual deficits detectable by the tests then available (Tow S: Lewin 1953). More extensive lesions extending back into the posterior orbital cortex, the anterior striatum and the nasofrontal nuclei, were sometimes complicated by severe apathy or even stupor, nutritional disturbances and persisting urinary incontinence (Beck et al 1950).

The medial surface of the frontal lobe is related to the medial segment of the falx and to the opposite frontal lobe (Fig. 2.19). Anteriorly and inferiorly, the cortex is granular and is an extension of areas 9-11. Posteriorly is the large cingulate gyrus (areas 23 and 24), curving around the corpus callosum. The cingulate gyrus receives fibres from the anterior nucleus of the thalamus, which is connected with the mammillary body in the hypothalamus (Fig. 2.22) through the mammillothalamic tract. The medial mammillary nucleus in turn receives a tightly organised, point-to-point projection from the hippocampal complex (Simpson 1952) through the fornix. The cingulate gyros, the mammillary nuclei and the hippocampal complex have been conceptualised as part of the limbic system, and functionally related both to mood and to memory. The cingulate gyrus is rarely injured in isolation, but psychosurgical cingulate ablation was once much used for obsessive states; the effects of this operation included transient memory impairment (Whitty & Lewin 1960), but this did not constitute a permanent disability. Lesions in the mammillary bodies (Dusoir et al 1990), or bilateral destruction of the hippocampal complex, cause gross impairments of memory.

On its convex lateral surface, the frontal lobe is related to the frontal bone, and more posteriorly to the parietal bone (Fig. 2.23), since the precentral gyrus — the posterior limit of the frontal lobe — lies well behind the coronal suture. Anteriorly, the cortex is rich in granule cells and is a part of areas 9-11: thus, these cortical areas are located on all the surfaces of the frontal pole and are often called prefrontal cortex. More posteriorly, the cortex (areas 8, 6, 45 and 46) shows fewer granule cells, and large pyramidal cells become more conspicuous; the precentral gyrus (area 4) is agranular, containing large pyramidal neurons, some of which send axons to synapse directly on spinal motor neurons. The posterior frontal convexity cortex is concerned especially with motor function, including limb movements (areas 4, 6 and 8) and conjugate eye movement (area 8); on the dominant side, the frontal cortex is concerned with expressive speech (supposedly areas 44 and 45). Traumatic lesions posterior to the coronal suture are often associated with contralateral limb or facial weakness, or with non-fluent dysphasia when the dominant hemisphere is injured. But lesions in the prefrontal region, even those causing substantial cortical and subcortical damage, may show no limb signs or speech impairment, and in the past this has given the mistaken impression that this is a 'silent area'. In fact, bilateral prefrontal lesions often cause very disabling changes in intellect and personality.

At present, the evidence does not allow exact correlations between the neuropsychological effects of frontal lobe injury and localized damage to specific cortical areas. But there is general agreement that destruction of the dorsolateral quadrants of the frontal lobes is more disastrous than damage to the inferior and medial quadrants, which may cause little or no obvious clinical impairment (Fig. 2.21). From his large experience of the effects of psychosurgery and of head injury, Walsh (1978, 1985) has made some general correlations between lesion site and neuropsychological sequelae. He associates the dorsolateral cortex with the ideational preparation and execution of actions; patients with lesions in this region may suffer severe intellectual disabilities in planning and learning, despite normal performance in standard IQ tests. There is a characteristic difficulty in integrating data to solve a problem; thinking tends to be rigid or inflexible (Walsh 1978), and this is reflected in the content of speech, though rigid thinking is seen after injuries elsewhere in the brain. The medial frontal cortex, presumably including the anterior cingulate gyrus, is concerned with self-initiated action and sustaining behaviour at an appropriate level. Severe damage in this region may explain the adynamia—lack of drive—so often seen after frontal lobe damage. Walsh (1985) relates the basal (orbitofrontal) and basomedial cortex with 'the flexible control of excitation and inhibition and the emotional control of behaviour': large bilateral lesions in this region may be associated with socially disastrous disinhibition, loss of insight and egotistic behaviour. In practice, the frontal lobe lesions seen after CMF injury are rarely confined exactly to a particular region, and when they are, the effects are not always what one would forecast. But the changes in personality and behaviour described by Walsh and others are very familiar as disastrous sequelae of frontal lobe damage: they are seen in various combinations, which no doubt express differences in anatomical damage as well as different pretraumatic personalities.

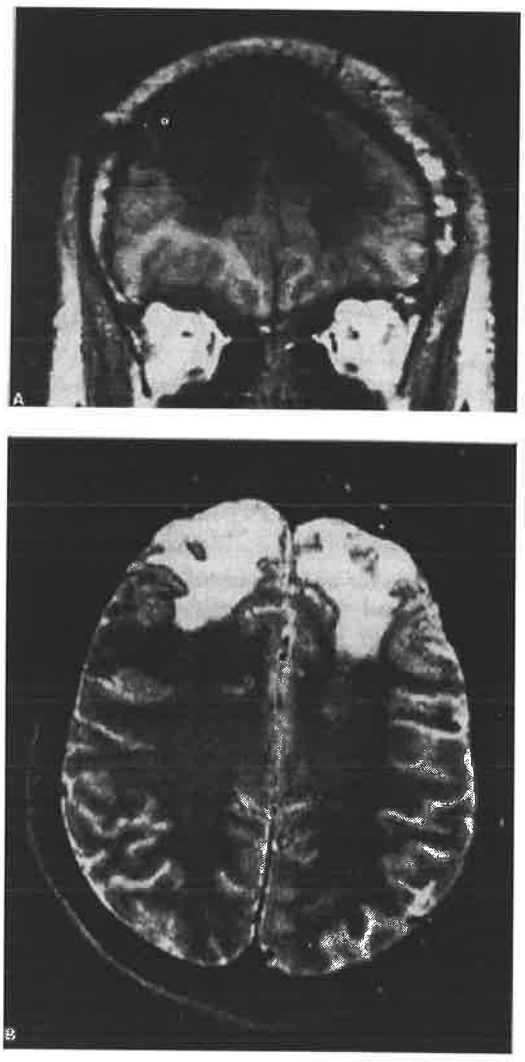


FIG. 2.21. *Bifrontal polar damage.* *MRI scan of a young man who had suffered a central frontal impact in a car crash at age 5 years. The victim has a stable personality and intelligence within the normal range.* **A.** *Low signal abnormalities in the T1-weighted coronal image: the white matter is largely destroyed, especially in the dorsomedial quadrants.* **B.** *Bifrontal high signal abnormalities in T2-weighted images.*

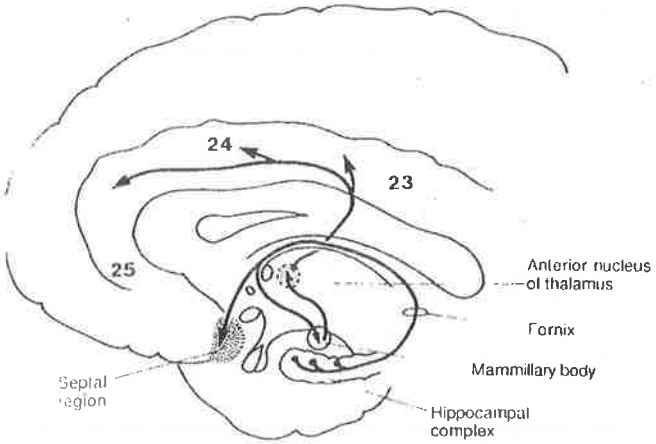


FIG. 2.22. ***Pathways connecting the mammillary nuclei with the anterior thalamic nucleus and the cingulate gyros.*** *Neurons in the subiculum (part of the hippocampal complex) project through the fornix to the medial mammillary nucleus and also to the septal nuclei and other basal forebrain structures.*

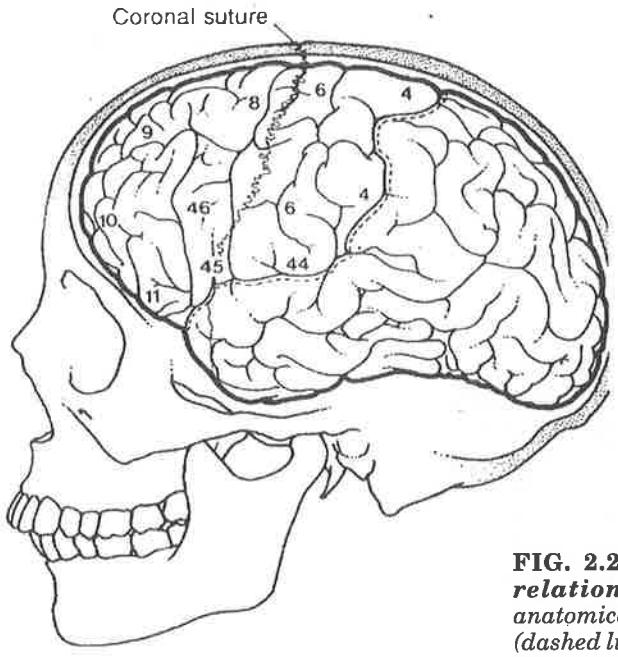


FIG. 2.23. ***Frontal convexity, in relation to coronal suture.*** *The anatomical frontal lobe is demarcated (dashed lines) by the central sulcus and the sylvian fissure. The chief cortical areas are identified by Brodmann's numbers.*

Corpus callosum

The genu and the rostrum of the corpus callosum constitute a massive commissural system joining the two frontal lobes. In our experience, surgical lesions in the middle and posterior sections of the corpus callosum may cause mild but detectable impairments of interhemispheric transfer (Jeeves et al 1979), but later writers have not confirmed this, and certainly small surgical transections in the body of the corpus callosum are well tolerated. Surgical transection of the genu does not cause any obvious clinical effects.

The corpus callosum is often injured in closed head injuries. MRI scans visualize callosal lesions well (Fig. 5.16), though it should be remembered that there are numerous variations in the normal structure of the body of the commissure.

Basal forebrain nuclei

The posterior margins of the gyrus rectus and the medial orbital gyri are demarcated by the bifurcation of the olfactory tract. The tract forms a lateral olfactory stria,which goes to the primary olfactory cortex in the medial temporal

region (see below), and a medial stria which terminates in the basal forebrain area. Posterior to the termination of the olfactory tract is the anterior perforated substance, so called because it is perforated by the medial striate arteries and more laterally by the recurrent artery of Heubner, a branch of the anterior cerebral artery. Dorsal and posterior to the anterior perforated substance are a number of important basal frontal nuclei, which are linked with the frontal cortex by the medial forebrain bundle (Fig. 2.20) and with the hippocampal complex by the anterior component of the fornix (Fig. 2.22). Of these, the basal nucleus of Meynert, the medial septal nucleus and certain other cell masses are composed of large neurons which send cholinergic axons to the neocortex and also to the limbic system. These magnocellular basal nuclei are thought to be a cholinergic arousal or modulatory system and dysfunction in this system has been seen as part of Alzheimer's dementia; to our knowledge, this has not been postulated in post-traumatic dementia, but as the area is sometimes damaged in closed head injuries, the possibility deserves consideration.

Temporal lobes

These lie postero-inferior to the frontal lobes to which they are linked by several fibre systems. The anatomy of the temporal lobes is too complex to be reviewed here: it is however important to note that the medial parts of the temporal lobes contain the hippocampus and the amygdaloid nucleus, and these are both connected with the basal forebrain nuclei and diencephalon (Fig. 2.22). Of all the structures concerned in the functions of memory, Amaral (1987) concluded 'that the hippocampal formation and the amygdaloid complex are principal candidates for roles in memory processing', and clinical experience has shown that bilateral ablation of these structures can cause very severe and prolonged impairment of memory. Graff-Radford et al (1990) have argued that it is necessary to destroy components of both the hippocampal and amygdaloid systems to produce permanent amnesias, but this is still debatable.

Brief post-traumatic memory impairment is the chief clinical characteristic of minor head injury, and the duration of post-traumatic amnesia is a much used measure of the severity of the injury (p. 384). At present, it cannot be said with confidence what are the anatomical correlates of post-traumatic amnesia, or of the prolonged dysmnesias that sometimes follow head injuries from which in other respects the patient appears to have made a good recovery. But it is noteworthy that bilateral contusions or ischaemic infarctions of the medial and polar temporal lobes are very common results of blunt impact to the head.

Cerebral blood supply

The frontal lobes receive their arterial supply from branches of the anterior cerebral artery, while the middle cerebral artery makes an important contribution to the supply of the frontal convexity. The proximal parts of both arteries send striate branches to the basal frontal nuclei, and, together with the anterior choroidal and posterior cerebral arteries, supply the basal ganglia and the diencephalon.

Two superficial venous systems drain the frontal cortex. Several large veins from the frontal convexity bridge the subarachnoid space to enter the sagittal sinus; laterally, veins run into the superficial middle cerebral vein to drain ultimately into the transverse sinus, with an anastomotic connection to the sagittal sinus. These anastomoses usually prevent venous infarction when cortical veins or even the anterior third of the sagittal sinus are injured; however, it is unwise to presume on this, and even the origin of the sagittal sinus should not be divided unless there is a real need to transect the falx (p. 380). Rupture of the medial bridging veins is one of the causes of subdural bleeding and haematoma formation after head impact.

Hypothalamus

This small but vital area lies posterior to the basal frontal nuclei and the optic chiasm; it forms the ventral walls and floor of the third ventricle. From it the vascular infundibulum or pituitary stalk runs forward and downward to join the posterior lobe of the pituitary gland (neurohypophysis) (Fig. 2.24). The mammillary nuclei constitute the posterior part of the hypothalamus. The hypothalamus has an array of nuclei, which are concerned with the regulation of body temperature, various vegetative and metabolic functions, and certain aspects of behaviour, including aggression. Hypothalamic nuclei also regulate the endocrine activity of the pituitary gland.

The well-defined paraventricular and supraoptic nuclei (Fig. 2.25A) are composed of large neurons which synthesize the peptide hormones vasopressin and oxytocin. These hormones pass by axoplasmic flow to the pituitary stalk and the neurohypophysis, from which they are liberated into the blood stream. Vasopressin is liberated in response to changes in serum osmolality, to which osmoceptive cells in the nuclei respond; vasopressin preserves homeostasis in water balance by regulating diuresis. Damage to the stalk results in diabetes insipidus. The hormonal output of the adenohypophysis (anterior lobe and pars intermedia) is regulated by a different system (Fig. 2.25B). Neurons in the ventral hypophysis, especially the small-celled tuberal nuclei, liberate hypophysiotropic peptides (hormonal releasing or inhibiting factors) into the system of portal vessels running down the infundibulum into the gland, and these induce the release of stored hormones formed by the cells of the adenohypophysis. The hypothalamus is often damaged in severe head injuries (Crompton 1971), and partial or complete lesions of the pituitary stalk are also often seen, especially in cases of anterior fossa fracture (p. 382).

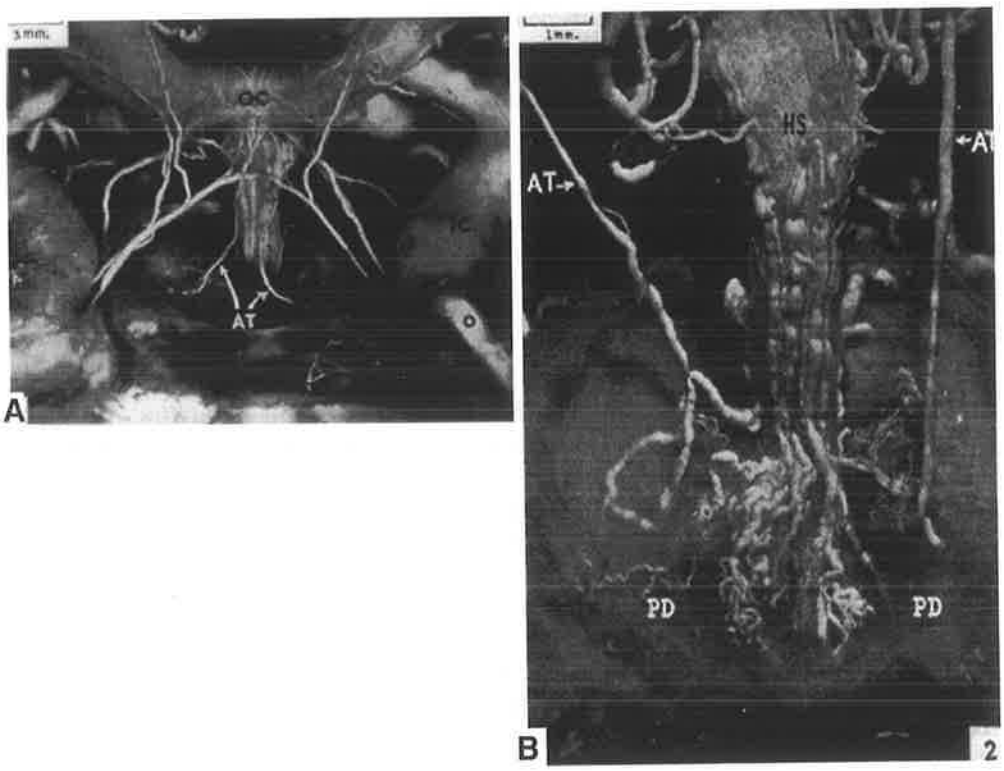


FIG. 2.24. ***Arterial supply and portal drainage of the hypothalamus and pituitary stalk.*** **A.** *The superior hypophyseal arteries run from the internal carotid arteries (IC) to supply the optic chiasma (OC) and then give descending arteries to the stalk. One of these, the artery of the trabecula (AT), goes directly to the posterior lobe of the pituitary gland. The ophthalmic artery (O) is not normally involved in the blood supply of the pituitary gland.* **B.** *Large portal veins run down along the hypophyseal stalk (HS) to enter the vascular sinusoids of the anterior lobe of the pituitary (pars distalis — PD). Reproduced by permission from Xuereb et al (1954a,b).*

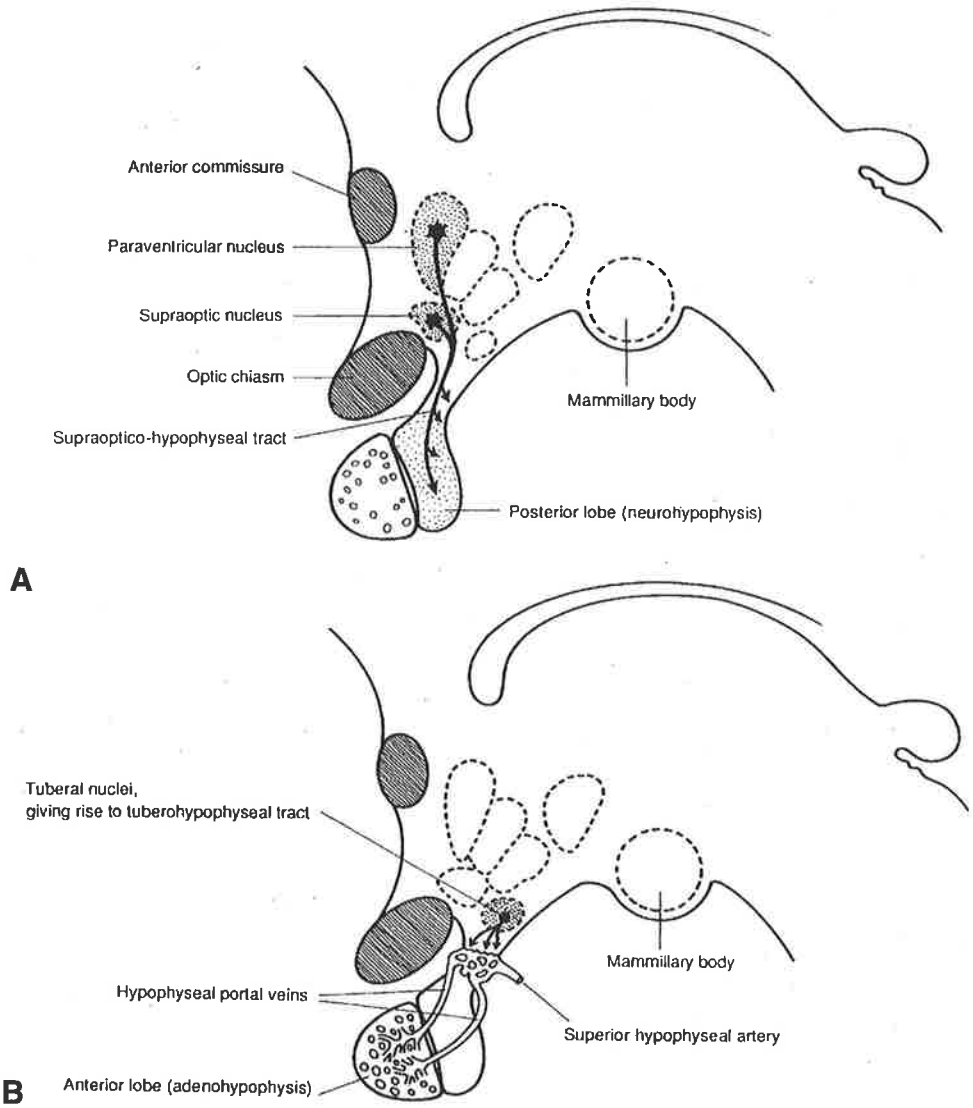


FIG. 2.25. ***Hypothalamic control of pituitary secretion.*** **A.** *Vasopressin and oxytocin are secreted in the paraventricular and supraoptic nuclei of the hypothalamus and transported in the axons of the supraoptico-hypophyseal tract to the posterior lobe of the pituitary gland: these hormones are then released into the circulation.* **B.** *Hypothalamic releasing hormones are formed in the hypothalamus and transported to a capillary plexus formed by the superior hypophyseal arteries around the median eminence and infundibular stem of the hypothalamus; these hormones there enter into the vascular system and are taken by the portal veins to the adenohypophysis. Here they stimulate, or inhibit, the release of the hormones of the adenohypophysis.*

Pituitary gland (hypophysis)

The gland is well protected in the sella turcica. It has its own capsule, separate from the dura-periosteum of the sella. On either side, it is flanked by the cavernous sinuses; ventrally it is separated from the sphenoid air sinus by a variable thickness of bone. It is covered by the diaphragma sellae, which is an extension of the dura of the skull base. The diaphragma has a central foramen through which the infundibulum passes to join the posterior lobe of the pituitary gland, of which it is functionally and histologically a part. There is an arachnoid cistern around the infundibulum, which extends into the sella to spread out over the surface of the anterior lobe (Lang 1983).

Direct injury to the pituitary gland is unusual, at least in surviving cases. Fractures through the body of the sphenoid may injure the gland, and in theory could open a fistulas communication with the subarachnoid space, but in our experience CSF leakage through the sella turcica is rare. However, the pituitary may be infected by trauma to its blood supply.

The gland has a dual blood supply. It receives a direct supply from the inferior hypophyseal artery (p. 47). It is also supplied by the superior hypophyseal artery, arising from the internal carotid artery after it leaves the cavernous sinus (Fig. 2.24A). The superior hypophyseal arteries send branches to capillaries in the hypothalamus and the infundibulum (Xuereb et al 1954a,b); from these capillaries arise portal veins which run down the infundibulum to the sinusoids in the adenohypophysis (Fig. 2.24B). These portal veins convey the hypophysiotropic factors elaborated in the hypothalamus; transection of the pituitary stalk interrupts this pathway, and usually causes infarction of much of the gland. In our experience, the subcapsular glandular tissue may be spared, perhaps because it is supplied by the inferior hypophyseal arteries. Hypopituitarism is occasionally seen after head injury (Winternitz & Dzur 1976), and presumably the basis is an infarction of the adenohypophysis. Daniel et al (1959) showed that autopsy evidence of infarction is quite often seen after severe head injuries of different types; transection of the superior portal vessels seems the likeliest cause.

Disturbed function of the neurohypophysis is a commoner finding in CMF injuries. Damage to the hypothalamus, or traumatic transection of the infundibulum, interrupts the hypothalamohypophyseal tract, causing failure of vasopressin secretion and temporary or permanent diabetes insipidus (p. 382). The converse state of overproduction of vasopressin is common after brain injury as a stress response, and often reaches a level inappropriate to the physiological requirements of the injured person (Fox et al 1971).

Cranial Nerves

First (olfactory) nerve

The sensory receptors are in the olfactory epithelium in the roof of the nose. The fine olfactory filaments traverse the cribriform plate in bundles, which are enclosed in an arachnoid sheath, and terminate in the olfactory bulb (Fig. 2.9). The complex central connections of the olfactory tract are described by Carpenter & Sutin (1983) and in more detail by Shipley & Reyes (1991); for present purposes, it is relevant that secondary sensory axons from the olfactory bulb run back in the olfactory tract and the lateral olfactory stria to the primary olfactory cortical areas (prepiriform cortex and periamygdaloid cortex) in the medial temporal lobe. While it is likely that posttraumatic anosmia is usually due to damage to the fragile olfactory filaments, bulb or tract, the possibility that damage to the central connections may be responsible has not been excluded; Sumner (1976) suggested that post-traumatic loss of both taste and smell could be due to central

damage, though we have not seen a case that supports this interpretation. Lesions in the primary olfactory cortex may result in epileptic seizures with an olfactory aura, but this type of epilepsy is rare after head injury. The tract and bulb are exposed in intradural subfrontal craniotomies, and the bulb is easily avulsed from the olfactory fossa. However, the bulb can be preserved by freeing it from the overlying gyrus rectus.

Second (optic) nerve

In the orbit, the optic nerve runs back in the centre of the cone of muscles; this segment of the nerve is about 25 mm long. The complex anatomical relations are described and illustrated by Lang (1983) and by Blinkov et al (1986). The nerve then enters the optic canal, between the roots of the lesser wing of the sphenoid bone. Ventral to the nerve runs the ophthalmic artery. This artery has a surgically important relation to the nerve: it arises from the internal carotid artery (see above), usually ventromedial to the nerve, but the point of origin is variable. The final section of the optic nerve is intra cranial: the nerve runs back to the chiasm, a distance ranging from 7 to 16mm (Lang 1983). The nerve can be injured in any part of its course (p. 418), but clinical interest has centred on the intracanalicular section (Fig. 2.26), since here the possibility of remediable compression arises. Maniscalco & Habal (1978) dissected 83 cadavers, of unspecified ages, and found that the canals were on average 9.22 mm long, ranging from 5.5 to 11.5 mm. The canal is elliptical in cross-section, the widest diameter being horizontal at the intracranial end and vertical at the narrower orbital end. The wall of the canal is thickest at the orbital end of the canal, sometimes called the optic ring, and it is here that the canal is narrowest. On its medial side, the canal has a close relation with the sphenoid air sinus, and indeed there is usually a bulge in the wall of the sinus, representing the canal (p. 420). In some individuals, there is a bony deficiency in the canal, when the optic nerve is covered only by dura mater and the mucosa of the sinus (Renn & Rhoton 1975).

The optic nerve is formed from the axons of the retinal ganglion cells. Fibres from the nasal half of each retina, serving the temporal half of each visual field, run back in the nerve, those from the macula being in the centre; they decussate in the optic chiasm, where they are joined by uncrossed nasal fibres from the opposite eye to form the optic tract (Fig. 2.27). The fibre arrangement explains the field defects seen in cases of CMF trauma. Thus, injuries of the optic nerve, if not complete, may cause central visual loss of scotomatous type when the macular bundle is damaged. When the superior surface of the nerve is damaged — not a rare event — there is an inferior horizontal field defect, sometimes bilateral. Injury of the decussating chiasmal fibres gives a bitemporal hemianopia. Injury at the part of the optic nerve joining the chiasm — the anterior chiasmal angle — may affect the upper temporal field of the opposite eye, because the decussating lower nasal fibres from the opposite retina loop a variable distance into the ipsilateral optic nerve; the field defect sometimes takes the form of a junctional scotoma. Posterior to the chiasm, field defects are homonymous.

The optic nerve is surrounded by a sheath of dura mater and an extension of the subarachnoid space; at the inner orifice of the optic foramen, the dural sheath leaves the nerve to merge with the dura of the anterior and middle cranial fossae, while the arachnoid sheath forms part of a cistern around the nerve.

Third (oculomotor) fourth (trochlear) and sixth (abducent) nerves

These nerves arise in the midbrain (third and fourth nerves) and lower pans (sixth nerve). They course through the subarachnoid space to enter the cavernous sinus through separate portals. The third nerve pierces the dura midway between the anterior and posterior clinoid processes and runs in the lateral wall of the sinus. The portal of the fourth nerve is more posterior and lateral; it also runs in the wall of the sinus. The sixth nerve enters the dura over the clivus and runs through the inferior petrosal sinus to enter the cavernous sinus, where it lies

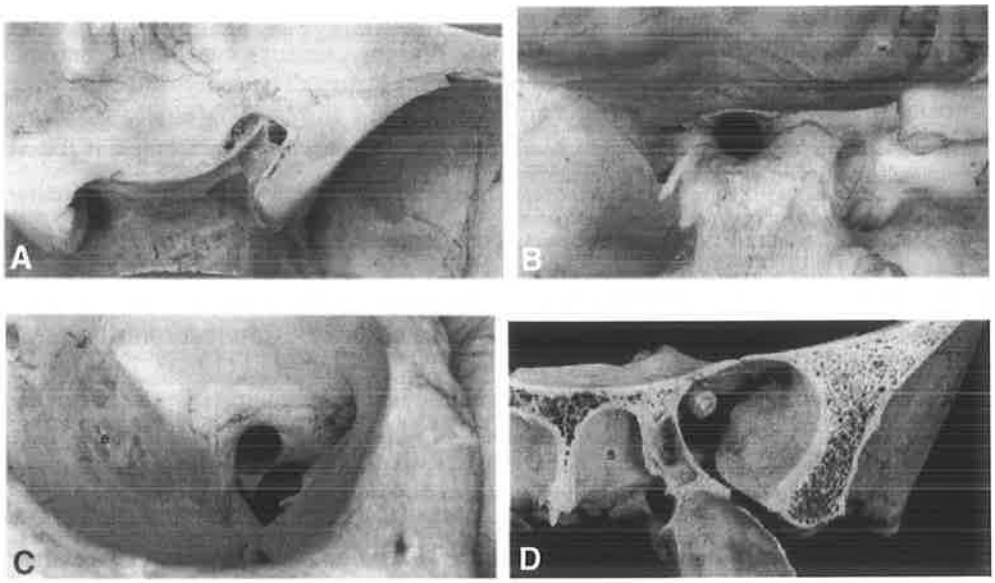


FIG. 2.26. *Optic canal.* *The canal lies between the two roots of the lesser wing of the sphenoid bone. It transmits the optic nerve and the ophthalmic artery. It is in close but variable relation with the sphenoid and ethmoidal air sinuses.* **A.** *The right optic canal is exposed by removal of its thick roof It is a funnel-shaped canal, narrowing as it enters the orbit.* **B.** *Seen from behind over the dorsum sellae, the optic canal is oval and narrowed in the vertical diameter. In life, the sharp upper margin is prolonged back by a thin fold of dura, the falciform process The anterior clinoid processes (arrow) lie lateral to the optic nerves.* **C.** *Seen from in front through the orbit, the optic canal is formed by a thick bony ring; it is narrowed in the horizontal diameter. The lamina papyracea of the ethmoid bone (e) lies medially.* **D.** *The canal, containing the optic nerve (simulated: white arrow head), has a medial relationship with the sphenoid (s) and posterior ethmoid (e) air sinuses. In this specimen, there is poor pneumatization and the canal is protected by a relatively thick layer of bone. In many cases, the canal bulges into the sphenoid sinus and the optic nerve may be covered by a very thin layer of bone, or even by mucosa only.*

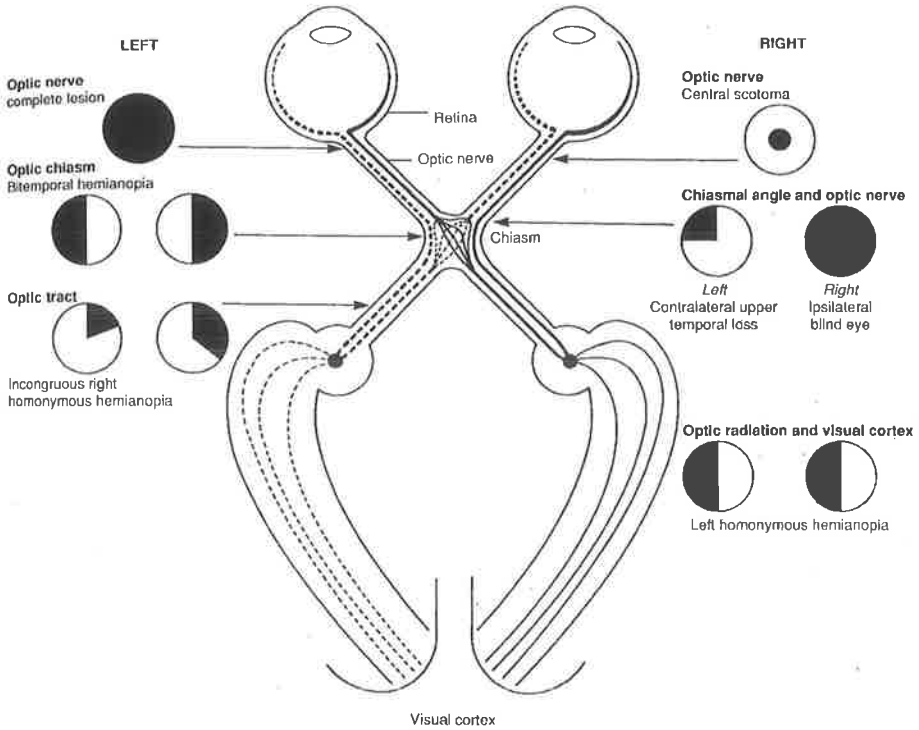


FIG. 2.27. *Visual pathways.* *The diagram shows the nasal and temporal retinal fibres passing through the optic nerve, with the nasal fibres (subserving the temporal hemifield) decussating in the chiasm, synapsing in the lateral geniculate body and then passing in the optic radiation to the visual cortex. Lesions of an optic nerve produce a unilateral visual field defect, except at the anterior chiasmal angle where a junctional scotoma with a contralateral upper temporal field loss is produced, as shown in the diagram Chiasmal lesions produce bitemporal field defects. Retrochiasmal lesions produce homonymous field defects which become more congruous as the fibres pass back towards the visual cortex.*

between the internal carotid artery and the dural envelope of the trigeminal ganglion (Fig. 2.18). These nerves are sheathed by sleeves of arachnoid as they enter the dura (Lang 1983). They leave the sinus to enter the orbit through the superior orbital fissure, together with the ophthalmic division of the trigeminal nerve. Before it leaves, the third nerve divides into two divisions. The superior division supplies the superior rectus muscle and the levator palpebrae superioris. The larger inferior division supplies the medial and inferior rectus muscles and the inferior oblique muscle. It also sends parasympathetic fibres to innervate the smooth muscles of the iris and the ciliary body, through the ciliary ganglion and the short ciliary nerves. It is thus the motor component of the light, accommodation and convergence reflexes. The fourth nerve passes above the upper tendinous head of the superior rectus muscle to supply the superior oblique muscle.

The sixth nerve passes below the upper tendinous head, together with both divisions of the third nerve, to supply the lateral rectus muscle.

The sympathetic nerves supplying the pupillary dilator muscle usually join and travel with the sixth nerve in the cavernous sinus, though in some individuals they may travel with the third or fourth nerve.

These nerves are often injured. Nuclear or supranuclear damage is seen as a result of severe brainstem damage. The nerves may be contused or avulsed either at their origins from the brainstem (Heinze 1969), or where they pierce the dura; this usually results from a severe impact, though third and fourth nerve paralyses occasionally follow less severe violence. The third nerve may be compressed in its subarachnoid course by an anterior transtentorial herniation: the parasympathetic fibres are especially sensitive, and pupillary paralysis is therefore the classical sign of transtentorial herniation (Fig. 2.28). Posterior transtentorial herniation may be evident as signs of compression of the midbrain nuclear and supranuclear control of third nerve function — bilateral ptosis and inability to look up. The sixth nerve is often injured in transverse petrous fractures, presumably in its intradural course. All three nerves may be involved in the cavernous sinus, either by the primary injury or from the effects of a carotid-cavernous sinus fistula. Because of the often close relationship of the sixth nerve and the sympathetic pupillodilator fibres, the combination of a Homer's syndrome and a sixth nerve paralysis suggests a lesion in the cavernous sinus. Injury in the superior orbital fissure from skull base fracture may result in simultaneous oculomotor and ophthalmic nerve paralysis. The clinical signs of paralyses of these nerves are set out in Table 2.7, and are further discussed in Chapter 6.

Fifth (trigeminal) nerve

This large nerve takes origin from the upper pons; its brainstem nuclei extend from the midbrain to the second cervical cord segment. Its motor and sensory roots run through the subarachnoid space to the trigeminal sensory ganglion, lying snugly in its aural cave lateral to the cavernous sinus. The motor root passes below the ganglion to enter the mandibular division. Injuries of the roots or the ganglion are rare. Through its three divisions, the trigeminal nerve supplies sensation to almost all the structures in the CMF region (Fig. 2.29): the ophthalmic division provides sensation to the forehead, the maxillary division to the middle third of the face and the mandibular division sensation to the lower face, as well as innervating the muscles of mastication. The supraorbital and supratrochlear nerves emerge from the orbit separately over the supraorbital margin; the supraorbital nerve passes through a notch or foramen, from which it has to be freed by chisel cuts in the standard frontal exposure of the orbits (p. 238). The infraorbital nerve passes through the orbital floor and the infraorbital foramen, supplying sensation to the lateral side of the nose and upper lip and the maxillary teeth via the superior alveolar nerves. Zygomaticofacial and zygomaticotemporal branches emerge through foramina in the bones, being often damaged during

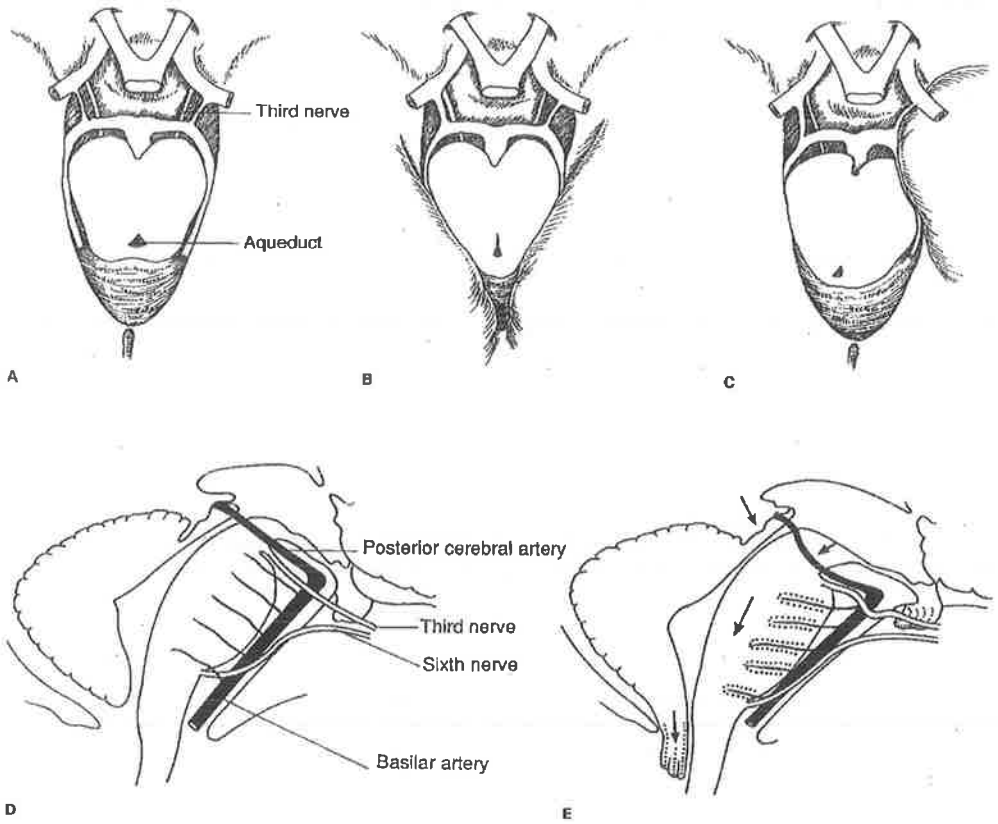


FIG. 2.28. ***Clinical anatomy of intracranial displacements.*** *An intracranial mass, such as a haematoma, may expand and displace cerebral tissue medially under the falx and downwards through the tentorial hiatus.* **A.** *The normal tentorial hiatus: the midbrain, seen from above in horizontal section, is surrounded by the quadrigeminal and ambiens cisterns (not shown). Anteriorly are the basilar artery and the third nerves running forward to the cavernous sinuses; the posterior cerebral arteries curve around the upper brainstem on their way to supply the occipital visual cortex.* **B.** *Posterior transtentorial herniation: the medial temporal lobes on both sides are forced into the hiatus and compress the dorsal midbrain* (clinical signs: bilateral ptosis, inability to look up and drowsiness). *One or both posterior cerebral arteries may be blocked* (clinical signs: hemianopia or cortical blindness). *The quadrigeminal cisterns are compressed* (CT signs: obliteration of cisterns). **C.** *Anterior transtentorial herniation: one medial temporal lobe is displaced by an ipsilateral mass lesion, e.g. a middle meningeal extradural haematoma, into the hiatus, compressing the lateral side of the midbrain and Inking the third acre* (clinical signs: ipsilateral papillary dilatation and ptosis, drowsiness). **D.** *The normal brainstem in sagittal section: the basilar artery supplies the ports and ventral midbrain.* **E.** *Brainstem displacement and cerebellar herniation: accompanying transtentorial herniation is a downward displacement of the whole brainstem. The blood supply of the pans and midbrain is imperilled; there may be haemorrhages (Duret type) into the ventral brainstem* (clinical signs: deepening coma, decerebration, bradycardia, fixed pupils, respiratory failure). *The cerebellar tonsils are forced into the foramen magnum.*

periosteal stripping as well as by trauma. The inferior dental nerve enters the mandibular canal (Fig. 2.6) at the lingula and traverses the body of the mandible to give sensation to the posterior teeth; in this part of its course, the nerve is easily injured (p. 265). The nerve terminates as the mental and incisive nerves to provide sensation to the anterior teeth, chin and lower lip.

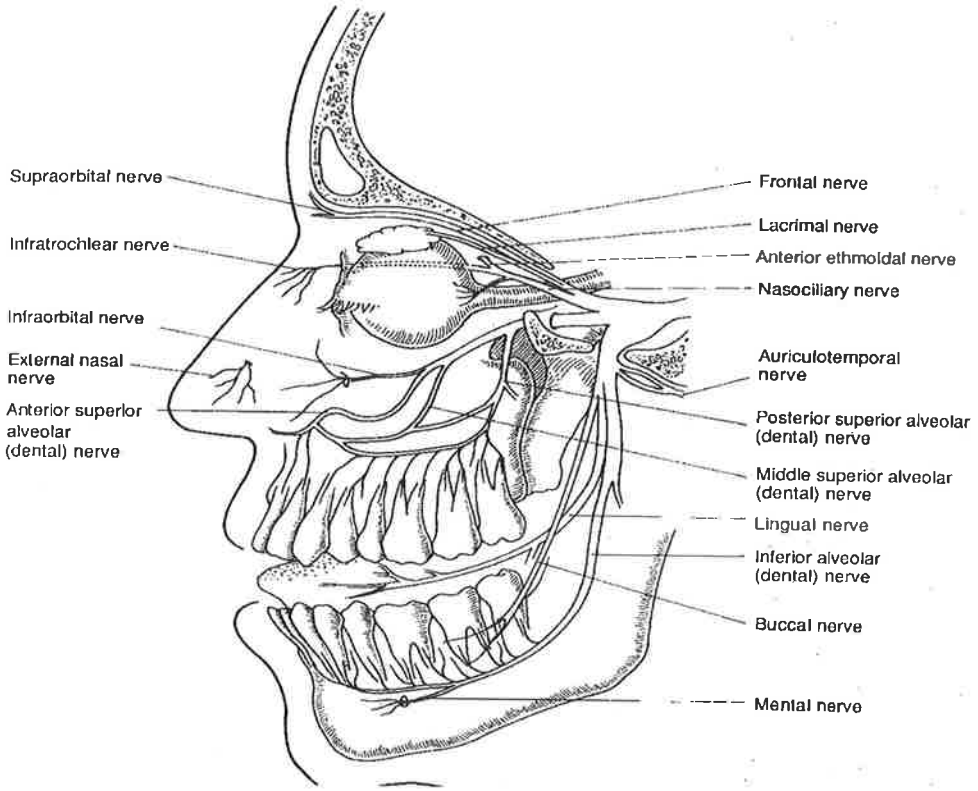


FIG. 2.29. ***The trigeminal nerve.*** *Diagram of the sensory distribution of the trigeminal nerve. The intracranial course of the anterior ethmoidal nerve is not shown: after entering the ethmoid bone, the nerve passes across the cribriform fossa anterior to the filaments of the olfactory nerve and enters the nasal cavity, which it supplies, to terminate as the external nasal nerve.*

Seventh (facial) nerve

This largely motor nerve has its nucleus of origin in the pons. It runs through the subarachnoid space into the internal auditory meatus and enters the petrous temporal bone, to emerge at the stylomastoid foramen. It then runs into the substance of the parotid gland from which it emerges to supply the muscles of facial expression (Fig. 2.30). Kempe (1980) has shown by stimulation under local anaesthesia that there is functional segregation in the facial nerve even within the stylomastoid canal. He has identified three components, each constituting a fascicle. One innervates the perioral muscular sphincter, the orbicularis oris and a second supplies the ocular sphincter, the orbicularis oculi. The third fascicle, termed by Kempe the mimic fascicle, supplies a crescent of facial muscles including the frontalis muscle, the zygomaticus major, the levator labii superioris, the depressor anguli oris, the mentalis and the platysma (Fig. 2.30A).

Through its chorda tympani branch, the facial nerve transmits sensation from taste buds in the anterior two thirds of the tongue (Fig. 2.31). The facial nerve also contains parasympathetic secretomotor fibres to the lacrimal and the submandilular and sublingual salivary glands, via the chorda tympani and the greater superficial petrosal nerve (Fig. 2.32). These secretomotor fibres have their cells of origin in the superior salivatory nucleus, in the pons.

Injuries of the facial nerve are common, both in its petrous course and in the region of the parotid gland. These injuries are discussed in Chapter 15.

Eighth (vestibular and cochlear) nerves

These nerves are outside the scope of this book; their neuroanatomy is well described by Brodal (1981) and by Carpenter & Sutin (1983). The eighth cranial nerve may be injured in fractures of the skull base involving the petrous bone (p. 439).

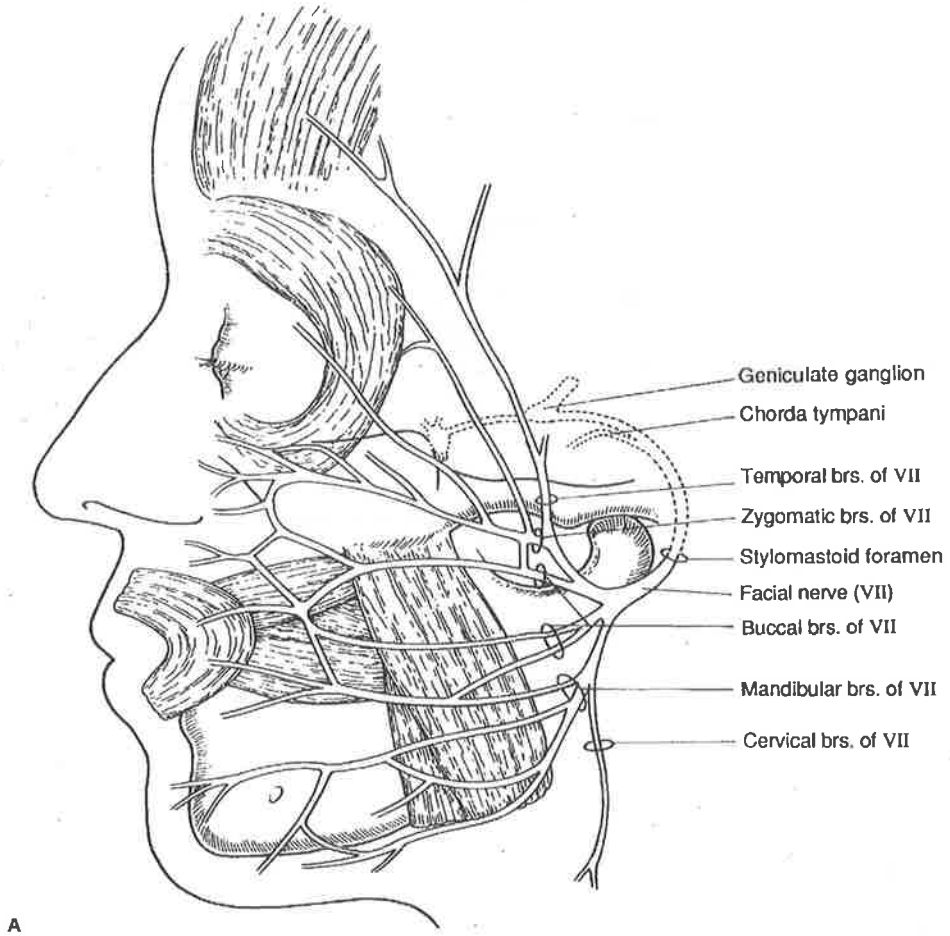


FIG. 2.30. ***The facial nerve.*** **A.** *Within the petrous temporal bone the nerve is already divided into fascicles destined to innervate individual muscles of facial expression. Kempe (1980) has identified three groups innervating: (1) the orbicularis oris sphincter, (2) the orbicularis oculi sphincter, (3) the muscles of facial expression. Reproduced by courtesy of the* Journal of Neurosurgery.

Ninth (glossopharyngeal), tenth (vagal) and eleventh (accessory) nerves

These supply the striated muscles of the pharynx and larynx; the spinal root of the accessory nerve supplies the trapezius and sternomastoid muscles. The nerve trunks are rarely injured, except in cases of penetrating wounds of the neck. The glossopharyngeal nerve conveys sensation, including taste, from the posterior third of the tongue (Fig. 2.31), and supplies parasympathetic secretomotor fibres (Fig. 2.32) to the parotid salivary gland through the small tympanic nerve and the lesser superficial petrosal nerve (Brodal 1981). These secretomotor fibres have their cells of origin in the inferior salivatory nucleus of the medulla. The visceral distributions of these nerves are chiefly outside the CMF region.

Twelfth (hypoglossal) nerve

This motor nerve is often injured by missile wounds in the region of the mandible. The nerve leaves the skull base through its own deeply placed canal, and runs deep to the internal carotid artery and the jugular vein to the level of the angle

of the mandible. Here it passes forward superficial to the internal carotid artery, usually crossing the lingual artery, and then runs deep to the mylohyoid muscle to supply the muscles of the tongue.

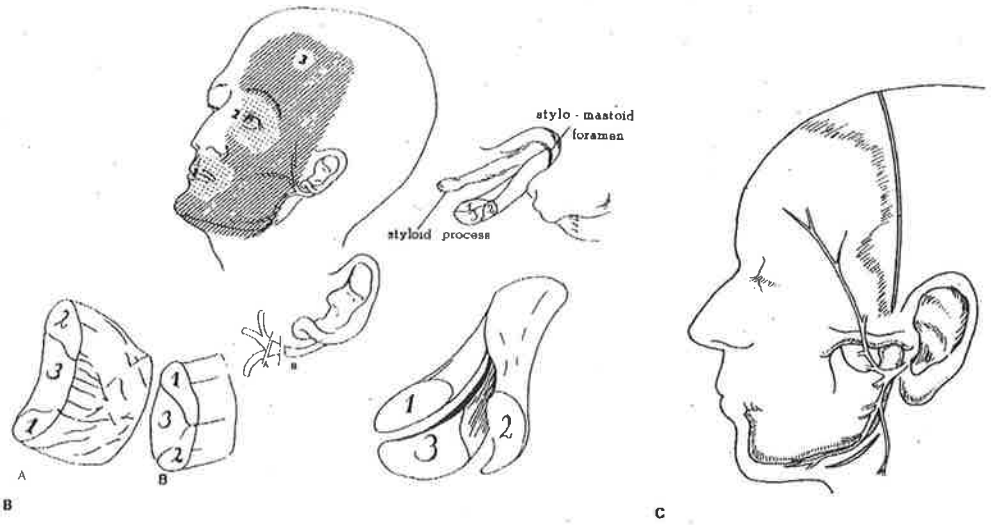


FIG. 2.30. ***The facial nerve.*** **B.** *After leaving the stylomastoid foramen, the facial nerve enters the parotid gland and breaks up into branches supplying the muscles of the face.* **C.** *The standard coronal scalp incision should be sited so as to spare the frontal branch of the facial nerve; submandibular incisions should spare the mandibular branch.*

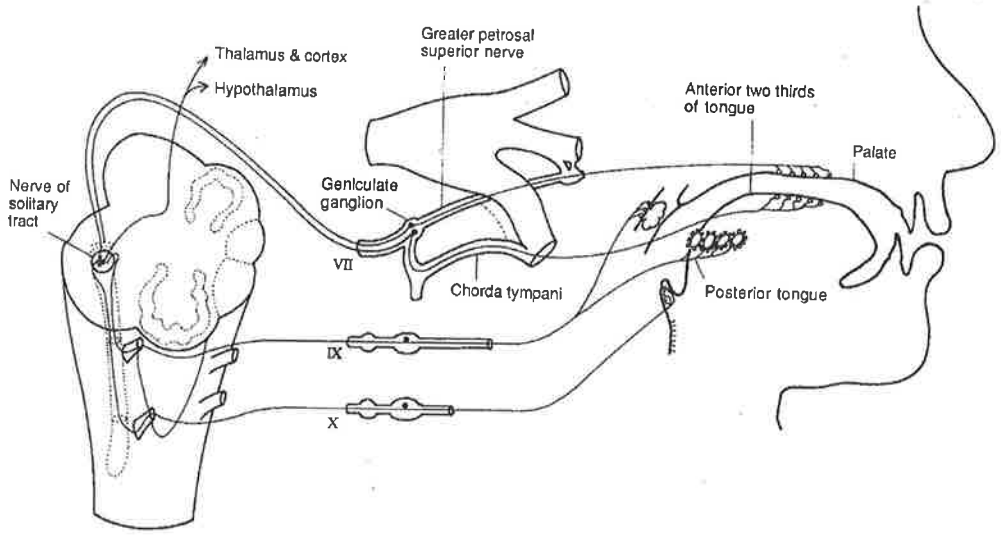


FIG. 2.31. ***Taste pathways.*** *Chemoceptive cells in the anterior two-thirds of the tongue are supplied by sensory fibres which travel in the chorda tympani nerve; their cells of origin are in the geniculate ganglion, and they establish central connections in the nucleus of the solitary tract. Similar nerve fibres innervate the posterior third of the tongue; they travel centrally in the glossopharyngeal nerve and have their cell bodies in the superior glossopharyngeal ganglion. They also terminate in the nucleus of the solitary tract together with fibres transmitted by the vagus nerve from the epiglottis.*

Sympathetic innervation

The whole CMF region receives postganglionic, and some preganglionic, fibres from the cervical sympathetic chain, which are distributed through branches of the cranial and upper cervical nerves, and along perivascular plexuses. These fibres innervate blood vessels, sweat glands, salivary glands, and the smooth muscle of the orbital muscles, iris and ciliary body. Ablation of the superior cervical sympathetic ganglion causes immediate pupillary constriction, mild ptosis, and

abolition of sweating; there is also vasodilatation, often causing a stuffy nose on the side of the ablation, from vascular dilatation in the nasal mucosa. But injury to this ganglion is unusual in CMF injuries, and damage to the peripheral branches of the sympathetic system is not usually of clinical significance, unless as a sign of a cavernous sinus lesion.

The Salivary Glands

Saliva

Saliva is a viscous, colourless liquid produced in volumes of around 1.5 l per day by the major salivary glands and the many smaller glands lying under the oral mucosa.

It has many functions. Saliva assists mastication and swallowing by moistening food and by enzymatic predigestion of starch, and it aids taste perception by acting as a solvent for chemical substances. Saliva moistens the oral lining surfaces, facilitating movements of the mouth in speech and mastication, and helping to maintain oral hygiene in the self-cleansing of the teeth. Moreover, salivary lactoperoxidase has an important antimicrobial role.

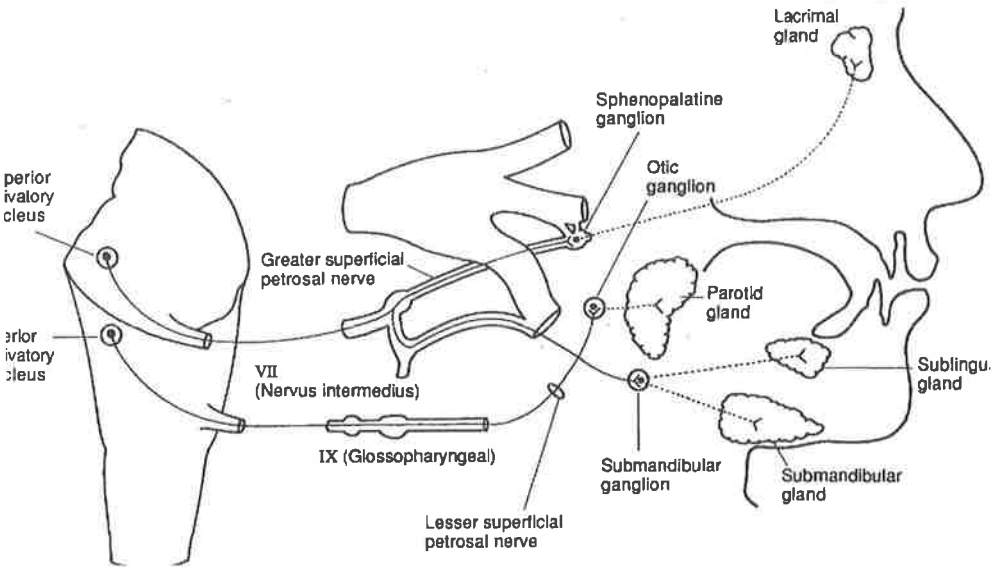


FIG. 2.32. ***Parasympathetic innervation of the chief salivary glands and the lacrimal gland.*** *Preganglionic fibres arise in the brainstem, travel in the 7th, 9th and 10th cranial nerves and terminate in small peripheral ganglia, from which postganglionic fibres are distributed to innervate secretory cells in the named and unnamed salivary glands and the lacrimal gland.*

Parotid gland

This is a pure serous-secreting gland, which is enclosed in a dense fibrous capsule bound to adjacent structures by the deep investing fascia of the neck. It is lobulated, irregular in shape and wedged into the space bounded anteriorly by the posterior margin of the mandible and posteriorly by the styloid and mastoid processes and their attached muscles. It extends up to the zygomatic arch and the temporomandibular joint, and an extension of the gland lies on the masseter muscle. Within the gland are the external carotid artery, dividing into its terminal branches, and the superficial temporal and maxillary veins, anastomosing to form the posterior facial or retromandibular vein, which usually divides to form the common facial and external jugular veins. The facial nerve enters the gland from its posterior aspect, and divides into upper and lower divisions, which ramify to supply the facial muscles; the nerve lies superficial to these veins, in what is

termed the faciovenous cleavage plane. The gland is divided into deep and superficial components by this plane, a cleavage of considerable surgical importance when the facial nerve has to be exposed. The parotid duct forms within the gland, emerges from its anterior aspect, and runs subcutaneously over the masseter to pierce the check muscles and enter the oral cavity at the level of the crown of the second molar tooth. The gland and the duct are easily wounded; branches of the facial nerve are often cut at the same time (p. 439).

The parasympathetic secretomotor supply (Fig. 2.32) is derived from the glossopharyngeal nerve: the preganglionic fibres synapse in the otic ganglion and the postganglionic fibres are distributed by the auriculotemporal branch of the mandibular nerve. Injury to these parasympathetic fibres, followed by aberrant regeneration along sympathetic nerves, is thought to explain the finding of facial sweating and flushing on gustatory stimulation, sometimes seen after surgical or traumatic damage to the parotid gland.

Submandibular gland

This has both mucous and serous secretory cells. It lies below and deep to the body of the mandible and comprises superficial and deep components, divided by the posterior border of the mylohyoid muscle. The submandibular duct runs from the deep surface of the gland, under the mylohyoid, to enter the mouth through the sublingual papilla on the side of the frenulum. The gland receives preganglionic parasympathetic innervation from the chorda tympani nerve (see below); the postganglionic relay fibres arise in the submandibular ganglion.

The sublingual gland

This has predominantly mucous secretory cells. It lies beneath the mucosa of the floor of the mouth, medial to the sublingual fossa in the inner surface of the anterior part of the mandible, dorsal to the mylohyoid line. The ducts of the gland drain directly or via the submandibular duct into the mouth. The nerve supply is, like that of the submandibular gland, from the chorda tympani.

Muscles

Muscles of facial expression

R. J. Last in his excellent textbook of anatomy emphasizes that the muscles of facial expression are developed from the mesoderm of the second pharyngeal arch (McMinn 1990). During their migration, they drag with them the nerve of that arch, the seventh (facial) cranial nerve. These muscles are part of the panniculus carnosus, the sheet of subcutaneous striated muscles which arises from and inserts into the dermis. In lower mammals this sheet is very extensive; in man, it is largely confined to the muscles influencing facial expression. From a functional viewpoint, the facial muscles are grouped around the orifices of the orbit, the nostrils and the mouth; they provide sphincters to close these orifices and dilators to open them.

Muscles of the eyelids

The orbicularis oculi surrounds the orbital entrance and spreads over the eyelids. It is opposed by two dilator muscles, the levator palpebrae superioris and the occipitofrontalis. The orbicularis oculi comprises two main parts: palpebral and orbital. Some anatomists identify a third lacrimal component (Davies & Coupland 1967).

The palpebral fibres arise from the medial canthal ligament, and arch across both lids in front of the tarsal plates, to form the lateral canthal ligament. These important ligaments are variously designated as palpebral and canthal; the medial ligament is so well defined that it is often termed a tendon, while the lateral

ligament is considered by some anatomists to be no more than a rapine of muscle fibres. In this book, the terms medial and lateral canthal ligaments are preferred.

The orbital portion of the sphincter has a wider extent, and its fibres form loops around the orbit, without interruption on the lateral side; they arise and insert in the nasal part of the frontal bone and the frontal process of the maxilla. The sphincter is opposed by the levator palpebrae superioris, arising from the under surface of the lesser wing of the sphenoid bone just above the optic canal and by the occipitofrontalis, the frontal belly of which runs from the galea aponeurotica of the scalp to insert in the muscles of the orbit and nose. Contraction of the occipitofrontalis elevates the eyebrows and creases the frontal skin; its nerve supply may be damaged by frontal trauma, or iatrogenically in raising a bicoronal scalp flap. The dilator elevator capacity of this muscle is utilized in brow suspension operations for ptosis (p. 423). The levator palpebrae superioris is supplied by the third cranial nerve, and may be paralysed by injury to that nerve, resulting in a ptosis much more severe than that caused by paralysis of the occipitofrontalis.

The lacrimal or pretarsal fibres of orbicularis oculi are attached to the fascia over the lacrimal sac and insert in the tarsi or interlace in the lateral canthal ligament or rapine; their important role in the blink reflex is described below (p. 76).

Medial canthus

An understanding of the anatomical relationships at the medial canthus is necessary for successful post-traumatic reconstruction: the surgeon must take into account the medial canthal ligament, its relationship to the lacrimal apparatus, its attachments to bone, and its relationships to the orbicularis oculi muscle, skin and conjunctive. Displacement or detachment of the medial canthal ligament may produce a change in the shape of the palpebral fissure.

The medial canthal ligament (also termed a tendon) is a strong, complex structure (Fig. 2.33). It attaches to the bone of the medial orbit by an element that passes deep to the lacrimal sac, and another more massive component that passes superficial to the sac and inserts into the frontal process of the maxilla. Zide and McCarthy (1983) have described a vertical component to the superficial part of the ligament which inserts into the medial orbital rim adjacent to the nasofrontal suture. The deep portion of the ligament arches, above and below the ampullae of the canaliculi, and inserts into the lacrimal sac behind the posterior lacrimal crest. The strong, thick, superficial part of the medial canthal ligament is attached firmly to the anterior lacrimal crest and extends onto the nasal bone beyond (Rodriguez & Zide 1988). The anterior lacrimal crest protects the lacrimal sac as it nestles in its fossa and the palpable part of the ligament is medial and lies over the bone.

The telecanthus and canthal dystopia which result from damage in this area can be related to severance of all or part of the ligamentous attachment, or to displacement of the bones to which it is attached. The relationship of ligament to lacrimal drainage apparatus is vital when considering diagnosis and treatment of trauma in this area (p. 309 and 582).

Lateral canthus

The anatomy of the lateral canthal region is not well defined. There is some controversy as to whether or not there is a true ligamentous attachment of the tarsi to the orbital bone or whether there is just a thickening of the periosteum. Couly et al (1976) came down on the side of a definite structure, describing a 'true' ligament, divided into two divergent bundles. The base of this ligament which has the form of a Y, is attached to the external angle of the two tarsi. The bundles diverge, a deep part attaching to the frontal process of the malar bone

2 mm behind the orbital rim, the superficial part continuing over the frontal process and attached to its periosteum.

Whitaker (1984) gave some credibility to the existence of the lateral canthal ligament, and others workers in different disciplines, seeing the technical applications of adjustments to this region, make reference to it. In contrast to its medial counterpart its structure is not impressive but in surgical manoeuvres for establishing the correct positioning of the tarsi in relationship to the bone, the lateral canthal ligaments are important none the less.

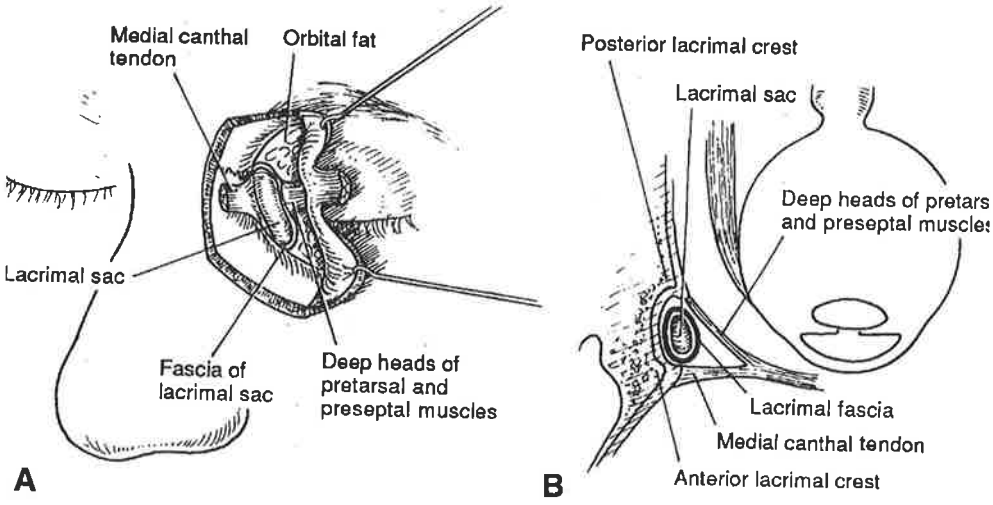


FIG. 2.33. ***The medial canthal ligament.*** **A.** *The medial canthal ligament (tendon) shown with its superficial part peeled back revealing the lacrimal sac.* **B.** *This strong fibrous band is shown attaching to the anterior lacrimal crest and roofing the lacrimal sac; the ligament (tendon) has been divided and its Y-shaped attachment to the tarsal plates is not shown. The preseptal and pretarsal fibres of orbicularis oculi are shown in simplified form lying posterior to the lacrimal sac and attaching to the posterior lacrimal crest. Redrawn from Rodriguez & Zide (1988).*

Muscles of the nostrils

The sphincter of the nostril is the compressor naris, which surrounds the alar cartilages and arises from the maxilla; it is opposed by the dilatator naris, which also arises from the maxilla and is inserted into the ala. The tip of the nose is elevated by the procerus muscle, an extension of the frontal belly of the occipitofrontalis muscle; it is assisted by the levator labii superioris alaeque nasi, and opposed by the depressor septi.

Muscles of the lips and cheeks

The orbicularis oris muscle forms the sphincter of the mouth; the dilators are facial muscles radiating from the lips. The orbicularis oris muscle has fibres which attach near the midline to the upper and lower jaws; these intermingle with fibres of the dilator muscles at the angles of the mouth on each side at a point of convergence called the modiolus, where some fibres actually cross in the form of a chiasma. This modiolus is often difficult to reconstruct after injury.

The buccinator muscle arises from both jaws opposite the molar teeth and from the pterygomandibular rapine (Fig. 2.14). It arises from the whole length of this rapine, interdigitating with fibres from the superior constrictor of the pharynx. The muscle converges on the modiolus, where fibres arising from the rapine decussate. It is pierced by the parotid duct and by the buccal branch of the mandibular nerve, whose fibres supply the muscle. The buccinator is the muscle of the cheek pouch and is lined by adherent mucous membrane; it forms the basis of a very useful musculocutaneous flap for intraoral reconstruction. It is basically a muscle of mastication, but has other functions.

The dilator muscles of the lips radiate from the orbicularis oris like the spokes of a wheel and are inserted into the lips and the modiolus. When these muscles all contract together, they open the lips to the widest possible extent. Of these radial dilators, the levator labii superioris alaeque nasi arises from the frontal process of the maxilla to insert into the alar cartilage (see above) and the lateral part of the upper lip, which it elevates. The levator labii superioris arises from the inferior orbital margin and is inserted into the remainder of the upper lip, also as an elevator. The levator anguli oris arises from the canine fossa below the inferior orbital margin; it inserts into the angle of the mouth, where it merges with the depressor anguli oris. The zygomaticus minor arises from the zygomaticomaxillary suture and the zygomaticus major further out on the zygomatic bone; both muscles converge on the modiolus. Risorius, a variable muscle derived from the platysma, inserts into the angle of the mouth. The depressor anguli oris arises from the oblique line of the mandible; it is a superficial muscle and passes through the modiolus to insert into the angle of the mouth. It is in part continuous with the levator anguli oris above, and in part with the platysma below. The depressor labii inferioris arises deep to the depressor anguli oris and inserts into the lower lip. The mentalis is a muscle arising from the mandible on each side of the symphysis mentis; its fibres pass downwards through the depressor labii inferioris to insert into the skin of the chin. It is an elevator of the centre of the lower lip and has significance in facial aesthetics and in denture control (McMinn 1990).

All these muscles are supplied by the seventh cranial nerve (see above p. 62). There are many variations, some of them important to the surgeon (Fig. 9.4).

Muscles of mastication

The temporalis muscle originates from the temporal bone and inserts on the apex and medial side of the coronoid process of the mandible (Fig. 2.34). Its functional importance in closing the jaws is considerable. The temporalis muscle is stripped away in the routine bicoronal approach to the craniofacial skeleton (p. 238), and we urge meticulous reattachment to the temporal lines and margins of the temporal fossa. Segments of the muscle can be stripped from the bone and used for reanimation of the face; the blood and nerve supply are preserved. A segment of muscle can be mobilized with a piece of underlying attached calvarium for bony reconstruction of the face (p. 622).

The powerful masseter muscle arises chiefly from the zygomatic arch and inserts on the ramus of the mandible, and assists in closing the jaws. The extensive upper attachment to the zygoma is of the utmost importance in fractures of this bone and their tendency to displace after correction (p. 315). The pull of the masseter and the pterygoid muscles also influences fractures of the angle of the mandible.

The lateral pterygoid muscle takes origin by two heads, from the infratemporal surface of the greater wing of the sphenoid bone and from the lateral surface of the lateral pterygoid plate. It has a firm round attachment high up to the condylar neck (Fig. 2.5) and to the meniscus of the temporomandibular joint, important in the pattern of displacement of fractures and fracture dislocations of this region. It is the chief agent in opening the mouth.

The medial pterygoid muscle arises from the medial surface of the lateral pterygoid plate and the adjoining palatine bone; it inserts on the ramus and body of the mandible. The medial pterygoid muscle closes the mouth and also moves the mandible in the horizontal plane in side-to-side chewing. The masseter laterally and the medial pterygoid muscle medially enclose the ramus and angle of the mandible.

The digastric muscle with its interesting central tendon bound down to the hyloid bone and its dual innervation from the fifth and seventh nerve assists

in the opening of the lower jaw, and also depresses and retracts the chin. It may be used for reconstructive purposes.

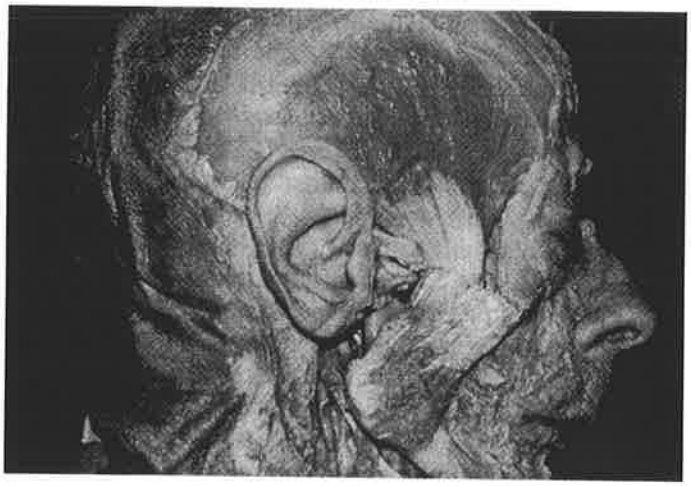


FIG. 2.34. *Muscles of mastication.* *Deep dissection of the face showing the temporalis and masseter muscles. The temporalis fibres converge inferiorly to form a tough ligamentous insertion into the coronoid process of the mandible. Photograph by courtesy of Professor G.C. Townsend, The University of Adelaide.*

Muscles of speech and swallowing

The tongue with its intrinsic and extrinsic musculature, lying in the floor of the mouth and attached to the mandible anteriorly and laterally, helps to form the shape and size of this bone as it develops. McMinn (1990) notes that the intrinsic muscles act in changing the shape of the tongue, whereas the three paired extrinsic muscles stabilize the organ and change its position. Of the extrinsic muscles, the genioglossus arises from the superior mental spine of the mandible, the hyoglossus from the body and greater horn of the hyoid bone; these muscles are likely to be detached or destroyed in avulsive injuries of the anterior mandible. The smaller styloglossus muscles are situated more posteriorly and have less individual importance.

The motor supply is by the twelfth or hypoglossal cranial nerve. Loss of sensation and motor supply to the tongue are both clinically significant, as is loss of the mandibular muscular attachment support which may produce a retrodisplacement of the tongue and airway obstruction.

The soft palate consists of the tensor veli palatini muscle, which arises from the scaphoid fossa at the base of the medial pterygoid plate, the spine angularis of the sphenoid and the anterolateral aspect of the cartilage of the eustachian tube. It runs antero-inferiorly and narrows towards the hamulus, where some of its bundles are attached, and widens like a fan towards the centre of the palate. It terminates along the oral side of the aponeurosis which occupies the whole of the anterior third of the velum or directly into it. The levator veli palatini is a cylindrical muscle which arises from the undersurface of the apex of the petrous temporal bone and from the edge of the canal for the passage of the internal carotid artery. Its anterior bundles arise from the posteromedial side and base of the cartilaginous auditory tube. This portion of the Eustachian tube elevates and shifts the soft palate backwards, the muscles of both sides form a sling suspended from the base of the skull. The palatopharyngeus muscle passes from the thyroid cartilage and the adjacent part of the pharyngeal wall through the palatopharyngeal arch to a fan shaped origin from the posterior border of the hard palate. It comprises two parts. The pterygopharyngeal part of the muscle runs from the posterolateral part of the pharynx and attaches to the hamulus and the palatine aponeurosis. The salpingopharyngeal part is the weakest portion: its bundles are attached to the inferior edge of the cartilage of the eustachian tube orifice and blend with the stylopharyngeus muscle (McMinn 1990) The

palatoglossus is a slender muscle arising from the transverse bundles of the tongue; it passes up into the palatoglossal arch and inserts fan-wise into the muscles of the soft palate. Together with its opposite muscle it forms the anterior pretonsillar sphincter. The superior pharyngeal constrictor is a quadrangular muscle surrounding from behind and laterally the upper third of the pharyngeal wall. It is the deepest of the pharyngeal constrictors.

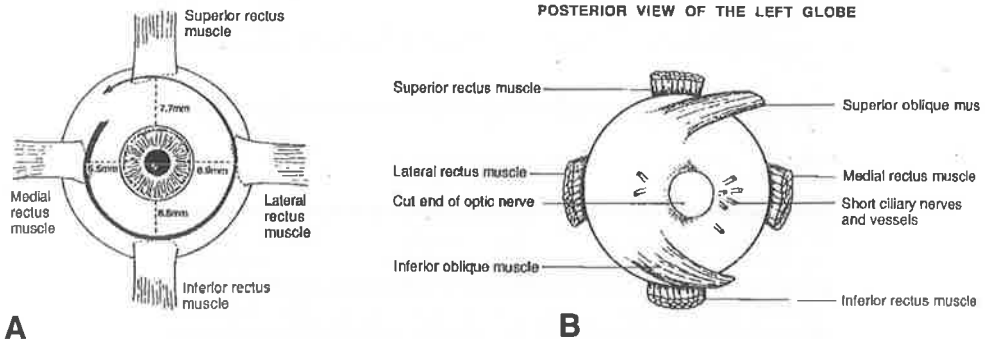


FIG. 2.35. ***The extra-ocular muscles.*** *The diagram shows the insertions of these muscles.* **A.** *The left eye from in front, shoving the four rectus muscles inserting into the globe at varying distances from the limbus, as shown by the spiralling arrow.* **B.** *The left eye from behind, showing the insertions of the two oblique muscles: these are attached lateral to the axis of the globe and posterior to its equator. Also shown are the entry of the short ciliary nerves and veins into the posterior segment of the eye.*

Extraocular muscles

Eye movement is controlled by the six striated extraocular muscles which include the four rectus muscles and the two oblique muscles (Fig. 2.35). At the apex of the orbit is the annulus of Zinn which is continuous with the dural sheath of the optic nerve and with periorbital and apical connective tissue. From this annulus arise the rectus muscles. The annulus has an upper tendon (of Lockwood) and a lower tendon (of Zinn). Pathological processes such as trauma may affect all these structures simultaneously, because of their intimate relationship.

Anteriorly, the rectus muscles insert on the globe Some 5.5-7.9 mm posterior to the limbus. The medial rectus inserts closest to the limbus; the inferior, lateral and superior muscles insert at increasing distances from the limbus. The superior oblique muscle originates just superior to the annulus and runs forward and becomes tendinous to run through the trochlea which is some 4 mm posterior to the orbital margin and just medial to the supraorbital notch. After passing through the trochlea, the tendon extends in a slightly posterior and lateral direction to fan out inserting on the superior aspect of the globe. The inferior oblique muscle arises from the bone just posterolateral to the nasolacrimal fossa, extending in a similar posterior lateral direction, coursing beneath the inferior rectus and inserting on the inferolateral aspect of the eye. The superior oblique muscle is innervated by the trochlear nerve, and when it contracts the muscle depresses the globe and causes incyclo-version (intorsion or 'wheel-in') of the vertical axis of the eye. The superior oblique has maximum depressing ability when the eye is adducted and maximum rotational ability when the eye is abducted. The muscle also acts as an abductor. The lateral rectus is innervated by the abducent nerve and this muscle abducts the globe. The remaining four extraocular muscles are innervated by branches of the oculomotor nerve. The inferior rectus depresses the globe whilst the medial rectus adducts it and the superior rectus elevates the eye particularly when the eye is abducted. The superior and inferior rectus muscles also act together as adductors. The inferior oblique muscle acts both as an elevator (this is maximal when the eye is adducted) and outward rotator of the vertical axis of the eye, with maximal excyclo-rotation (extorsion or 'wheel-out') occurring in abduction; with the superior oblique it acts as an abductor. Table 2.7 gives the clinical signs of acute paralyses of individual muscles.

The approximate anteroposterior lengths of the rectus muscles and the belly of the superior oblique muscle are 4 cm. Their nerve supply enters at the junction of the posterior and middle thirds of the muscle bellies. The inferior oblique is some 3.5 cm in length; its nerve supply arises from the inferior division of the oculomotor nerve and enters the muscle belly posteriorly after running lateral to the inferior rectus and behind the equator of the globe.

The Eye and its Adnexae

The structure of the eye is very complex (Fig. 2.36): those requiring a full review are referred to such texts as Last (1977). Some aspects of ocular anatomy and physiology require discussion because of their relevance to the pathology and management of eye injuries.

The globe of the eye can be seen as three concentric layers of tissue, each serving a different function. Externally, the fibrous sclerocorneal layer provides structural integrity. Within this layer, the vascular uveal layer (iris, ciliary body and choroid) provides nutrition to the rest of the globe, and especially to the innermost neurosensory layer, the retina.

TABLE 2.7

Clinical characteristics of paralyses of third, fourth and sixth cranial nerves and of the muscles innervated by them. The signs vary according to the degree of paralysis

Nerve	Muscle	Diplopia	Other signs
Third (oculomotor)	Medial rectus	Horizontal: on gaze to opposite side	Ptosis, fixed dilated pupil, divergent squint; eye may be turned slightly down; paralysed accommodation
	Superior rectus	Vertical: on gaze up	
	Inferior rectus	Vertical: gaze down	
	Inferior oblique	Vertical: on gaze up, with torsional element	
Fourth (trochlear)	Superior oblique	Vertical: on gaze down, with torsional element	Often subtle signs: compensatory head tilt to opposite side, difficulty in looking down
Sixth (abducent)	Lateral rectus	Horizontal: on gaze to same side	Compensatory face turn to same side, weak abduction, convergent squint, worse for distant fixation

Sclera and cornea

The sclera is composed of collagen and elastic fibres arranged in criss-crossing bundles, with sparse fibroblasts between the fibres. It is about 1 mm thick at the posterior pole of the eye, where the optic nerve enters, and only 0.3-0.4 mm where the rectus muscles insert. The sclera is continuous anteriorly with the cornea, the limbus being the junctional line.

The translucent cornea (Fig. 2.37) is composed chiefly of a stroma of collagen fibrils arranged in regular fashion; between the fibres are stromal cells with fibroblastic capacity. Anteriorly, the cornea is bounded by a layer of non-keratinizing stratified squamous epithelium, resting on Bowman's membrane, which is a homogeneous condensation of the corneal stroma. On electron microscopy, the epithelial cells are seen to have microvilli projecting into the tear film (see below). Posteriorly, the cornea is bounded by a single endothelial layer of hexagonal cells, resting on Descemet's membrane, a basement

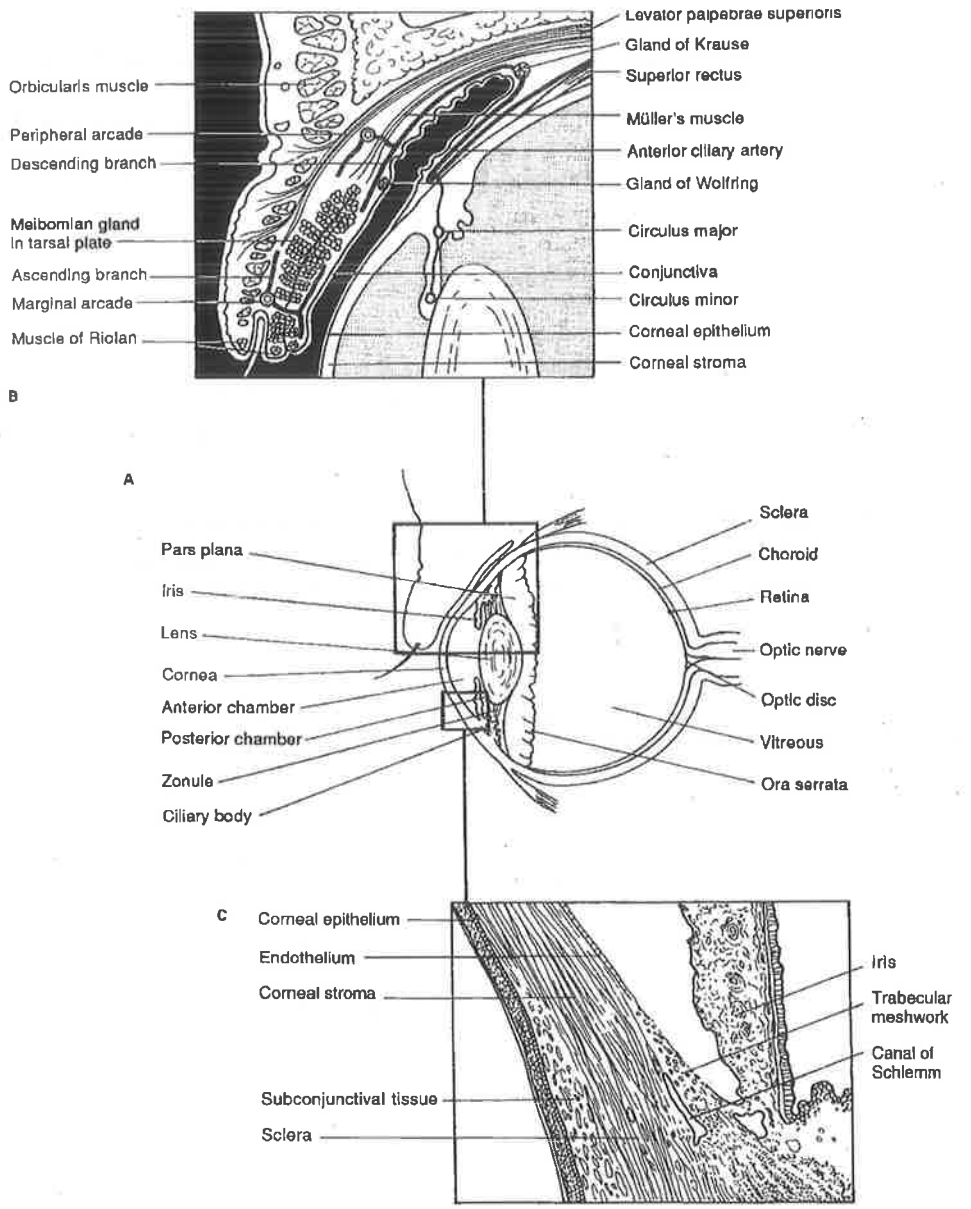
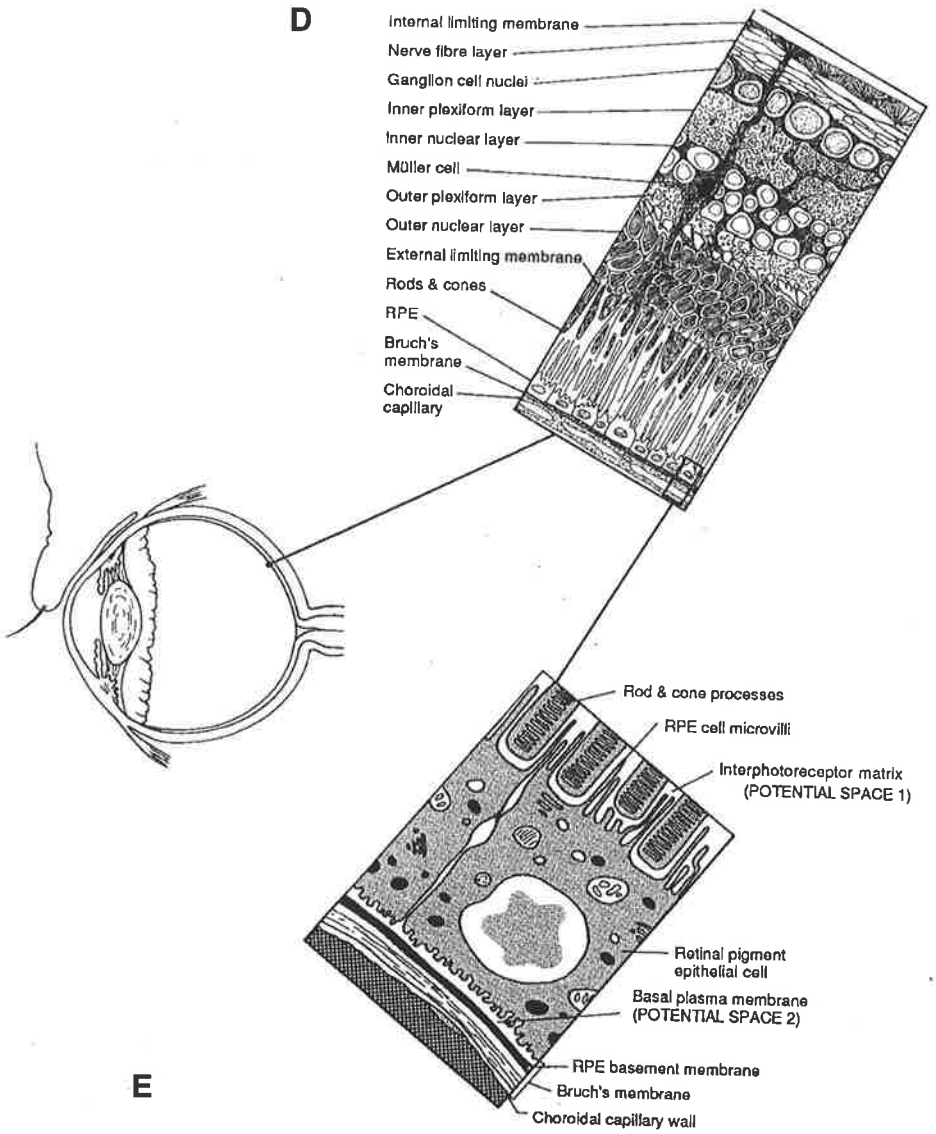


FIG. 2.36. ***The structure of the eye.*** **A.** *Diagram showing the eyelid and the anterior segment of the eye; the various muscles and glands are shown together with the blood supply of the lid.* **B.** *Schematic diagram of the major anatomical structures of the eye.* **C.** *Diagram showing the transition from sclera to cornea, with the structures in the drainage angle of the eye.* **D.** *Diagram showing the nine layers of the retina, and its membranes.* **E.** *Diagram showing the ultrastructure of a retinal pigment cell in relation to the rod and cone layer (right side) and the choroid (left side).*



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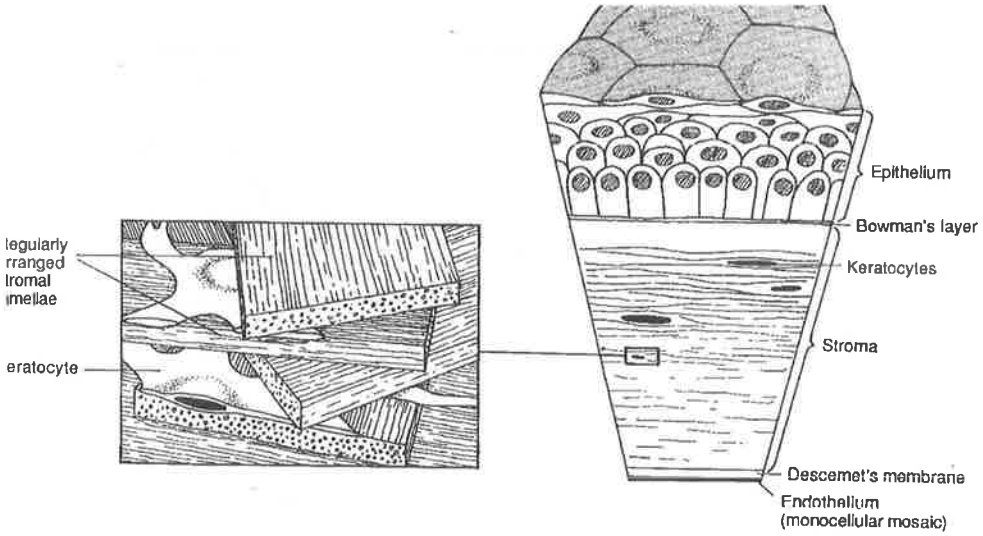


FIG. 2.37. *The layers of the cornea.* *Diagram showing a section of the corneal stroma: the stromal lamellae are arranged regularly in layers. On the inner surface (anterior chamber) there is a monocellular lining layer of endothelial cells, and on the outer surface there is a layer of stratified nonkeratinized epithelial cells.*

membrane containing elastic fibrils. The cornea is thinnest (about 0.5 mm) centrally and thickest where it joins the sclera.

It is the major refracting structure in the eye, providing three-quarters of the optical power of the eye as a lens system.

The cornea is wholly avascular, deriving nutrition chiefly from the aqueous humor; the corneal stroma is hydrophilic, and attracts water, glucose and other requirements to maintain the active metabolic requirements of the cellular elements, especially the epithelial cells. Oxygen is also obtained by diffusion, externally through the tear film, internally from the aqueous. The thickness of the cornea, and also its transparency, relate to the water content; dehydration of the cornea causes thinning and loss of normal lucency. The intraocular pressure, in adults normally 16–20mmHg, is also important in maintaining corneal clarity, and excessive pressure causes corneal oedema.

The cornea is sensitive to pain, touch and cold (Walsh & Hoyt 1985); it is innervated by the ophthalmic division of the trigeminal nerve, through the long ciliary nerves. It has been argued that in some individuals, the lower part of the cornea is innervated by the infraorbital nerve; Rowbotham (1939) found no evidence for this, but our clinical observations suggest that this may still be still an open question.

Uveal layer

The iris is pigmented, reducing light scatter, and contains a diaphragm of constrictor and dilator smooth muscle fibres which represents the effector component of the pupillary light reflex. The vascular ciliary body produces the aqueous humor, which fills the anterior segment of the eye and is absorbed through the trabecular meshwork into the canal (or canals) of Schlemm, which drains through collecting channels into the conjunctival veins (Fig. 2.36C). The ciliary body also contains smooth muscle, acting on the lens through the suspensory zonule in reflex accommodation.

The choroid, also pigmented, is highly vascular and provides nutrition to the outer two thirds of the retina; it also rids the retina of metabolic waste products, and acts as a heat sink.

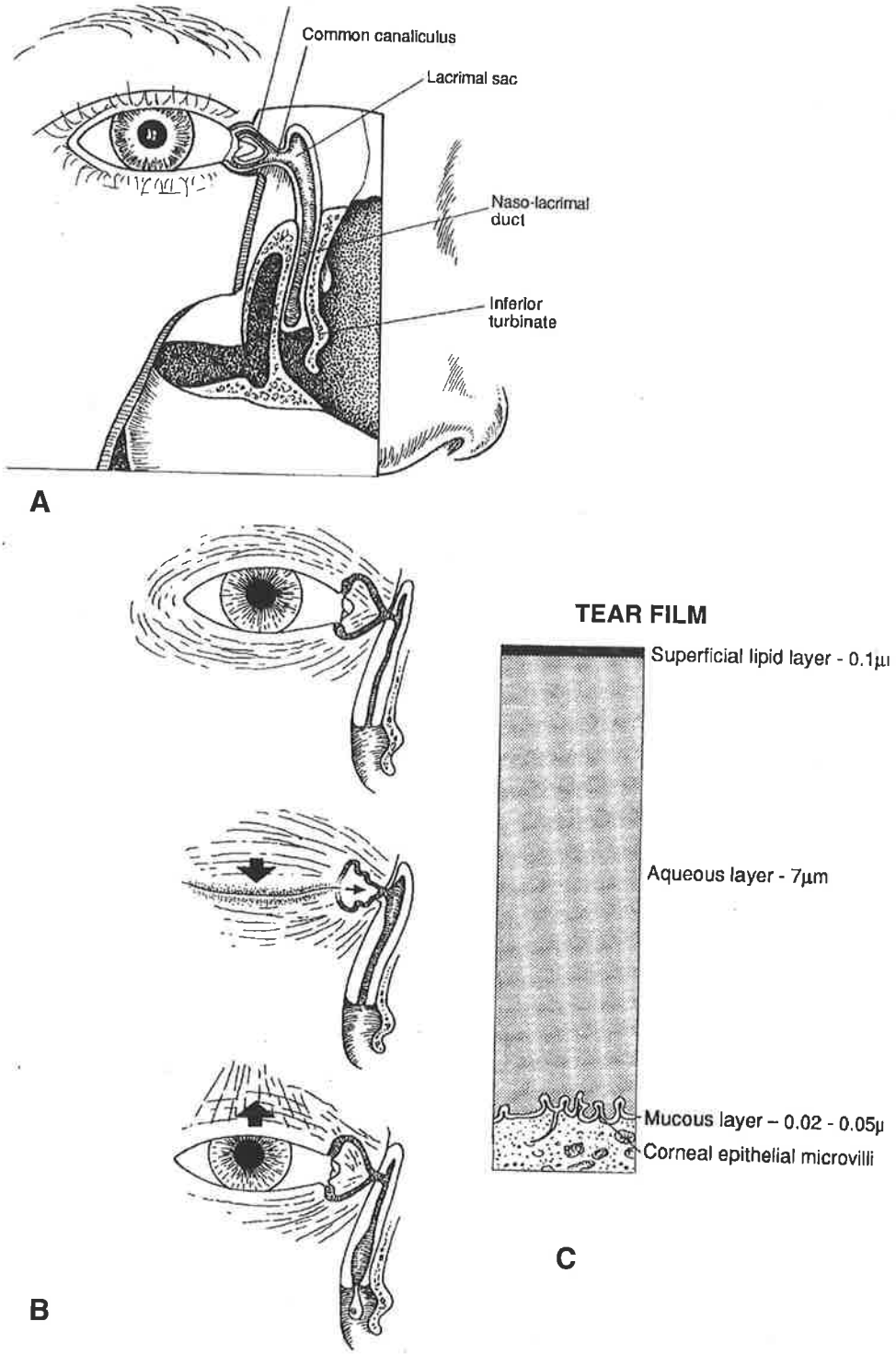


FIG. 2.38. ***The lacrimal apparatus.*** **A.** *Cut-away diagram showing the tear drainage system from the conjunctival sac to the nasolacrimal duct entering the inferior meatus of the nose.* **B.** *Diagram showing the active lacrimal pump mechanism, motored by the orbicularis oculi muscle and involving the lacrimal diaphragm. Upper diagram: with the lids open, the puncta are in contact with the lacrimal pool — the tears tend to collect medially. The canaliculi remain patent as the lacrimal sac collapses. Middle diagram: with eyelid closure, tears are milked to the medial side. The deep heads of the pretarsal muscle contract, shortening the canaliculi and closing their ampullae. At the same time the deep heads of the preseptal muscles pull the lacrimal diaphragm laterally, thereby creating negative pressure within the sac. Lower diagram: as the lids re-open, the lacrimal diaphragm returns to its relaxed position, thereby collapsing the lacrimal sac and propelling the tears into the nasolacrimal duct. The canaliculi reopen to admit more tears and the cycle is repeated.* **C.** *The tear film on the cornea: microvilli of corneal epithelial cells protrude into the tear film, which is a complex sandwich of mucous, aqueous and lipid layers, less than 8 mm thick.*

The lens lies behind the iris and is suspended from the ciliary body by the fibres of the zonule. It is the variable part of the dioptric power of the eye, and has the capacity to absorb ultraviolet light. The lens is composed of tightly packed epithelial cells which elongate to produce long prismatic lens fibres, contained within a capsule 2-20μm thick and made of an elastic, collagen-like substance. Like the cornea, the lens is nourished by diffusion from the aqueous humor, and also from the vitreous body; also like the cornea, the lucency of the lens depends on its hydration. Throughout life the epithelial cells of the lens are forming new lens fibres by mitotic division, and like all mitotically active cells they are sensitive to radiation: this is the basis of radiation cataract (p. 186).

The vitreous body occupies the posterior segment of the globe, a volume of about 4 ml. It is a hydro-gel containing 99% water: nevertheless it is in a dynamic metabolic state, providing nutrition to the lens and to the retina, and also mechanical support. The junctional surface between the vitreous and the retina is the internal limiting lamina of the retina, a basement membrane delimiting the internal layer of the retinal glial cells and a layer of dense collagen fibrils.

Retina

This comprises two layers, the outer pigmented epithelial layer, and the inner neuroepithelial layer (Fig. 2.36D,E). Between these layers is a potential space, and it is here that separation occurs when the retina is detached (p. 403). The retina is a most complex structure, containing not only light-sensitive rods and cones, but also neurons engaged in visual processing. The most sensitive part of the retina is the macula, whose centre — the foveola — is situated some 3 mm temporal to the optic disc; the retina varies in thickness, being thinnest at the foveola, and thickest where the retinal nerve fibres enter the optic nerve.

Blood supply

The eye is supplied by branches of the ophthalmic artery, the central artery of the retina, the long and short posterior ciliary arteries and the anterior ciliary arteries. These anastomose, but effectively the retina is dependent solely on its central artery. The retinal neuroepithelial cells are vulnerable to anoxia, but less so than might be supposed. Bock et al (1963) found that ischaemia for 6 min obliterated the electroretinogram, but even after 60 min. recovery of vision was possible; total irreversible loss of retinal function was established after 120 min. This finding of recovery after prolonged retinal ischaemia is supported by recent experimental studies in a monkey model (Young et al 1992).

The eyelids and lacrimal apparatus

The firm yet flexible nature of the eyelids (Fig. 2.36B) is given by the tarsal plate, the skeleton of the lid. The tarsal plates are composed of fibrous tissue; the upper plate is more prominent, and receives fibres of the aponeurosis of the levator palpebrae superioris, and also smooth muscle fibres which arise from that muscle and are under sympathetic innervation. The tarsal plates are connected to the circumference of the orbit by the orbital septum, and at their ends, they are attached to the lateral and medial palpebral ligaments. Each tarsal plate contains some 30 Meibomian glands, which open on the lid margin and secrete the oily element in tears. The lids are covered anteriorly by thin skin with lax, easily distensible subcutaneous tissue; posteriorly, they are covered by the conjunctiva, a stratified columnar epithelium containing goblet cells which contribute to the tear film. The lid margin, the free edge of the lid, has anteriorly the row of eyelashes, and posteriorly the row of orifices of the Meibomian glands: the 'grey line' between these rows is an important landmark in the surgery of the eyelid. There are some 150 lashes on the upper eyelid and about half that number on the lower lid; each lash is a typical short stout hair growing from a follicle that contains a sebaceous gland adding sebum to the tears. At the medial end of each eyelid is the lacrimal punctum, draining tears through the lacrimal canaliculi

into the lacrimal sac, and so through the nasolacrimal duct into the inferior meatus of the nasal cavity (Fig. 2.38). The bulk of the tears goes by this route; the rest (some 25%) is lost by evaporation. Tear flow is promoted by gravity, capillary attraction and the massaging action of the orbicularis oculi (Zide & Jelks 1985).

The blink and the tears

The tear film is said to be made up of three layers, a superficial oily layer, an aqueous layer, and a layer of mucus opposed to the microvilli of the corneal epithelial cells (Fig. 2.39C). Each has a specific function. The oily layer helps to prevent tear spillage. The mucus promotes adherence to the corneal epithelium. The aqueous layer, in bulk the largest, has nutritive and excretory functions, as described above, and is essential in maintaining corneal hydration; it is secreted by the lacrimal gland and other accessory glands which open into the conjunctival fornices. It is thought that the accessory lacrimal glands maintain tear secretion at a basal level, while the lacrimal gland proper responds to corneal irritation or emotional stimuli. The tear film is spread by the blink, and this is essential to maintain comfort and optical clarity. The normal blink is produced by contraction of the pretarsal fibres of orbicularis oculi, combined with relaxation of the levator muscle and contraction of the superior rectus muscle (Bell's phenomenon); forced lid closure is effected by the orbital component of orbicularis oculi. During normal blinking, the lower lid remains almost stationary, while the upper lid closes like a blind, with a final zipper-like narrowing of the palpebral fissure from lateral to medial canthus: the action helps to move tears toward the lacrimal puncta. In man, the normal blink rate is about 25/min, each blink taking about 0.3 s.

Growth of the Face

Early development

The face of a young child is not simply the miniature of the adult face; the face does not have the same proportions between the various regions or parts at all stages of growth (Fig. 2.39). In one of the earliest X-ray cephalometric studies, Broadbent (1937) showed that whereas the cranial vault and the orbits are approximately of adult size as early as the age of 10 years, the facial skeleton continues to enlarge until growth ceases after about 21 years only minor growth continuing thereafter. During the first year of life, both the calvarial and facial components of the skull increase in size by about 30%; in the adult, however, the calvarial skeleton is about 60% greater than its neonatal size, whereas the facial skeleton has enlarged by some 93%. The transformation from neonatal to adult proportions involves a general displacement of the facial skeleton away from the cranial vault in a downward and forward direction; furthermore, mandibular growth tends to outpace maxillary growth, so that the facial profile becomes straighter with age. Thus, the forehead of the child is relatively more prominent — and thus more vulnerable — than the lower facial structures, whereas the mandible is less prominent. This can be related to the greater incidence of childhood injuries in the frontal region, and the relative rarity of mandibular fractures (p. 502).

At birth, the fetal chondrocranium has been largely ossified, and membrane bones have formed in the fibrous capsules of the brain and the facial viscera. New bone is formed by osteoblasts adjacent to proliferating cartilage in the synchondroses which separate the components of the cranial base: the spheno-occipital synchondrosis is the chief of these. This process of endochondral ossification likewise takes place in the septo-ethmoid region and in the mandibular condyle. Bone is also produced by osteoblasts in the periosteum and endosteum covering the developing bones and in the sutures that separate them: the calvarial and facial sutures constitute an important mechanism for continuing ossification of membrane bones in response to the tissue-separating effects of growing viscera such as the brain, the tongue and the nasal cavity. As growth progresses, and in

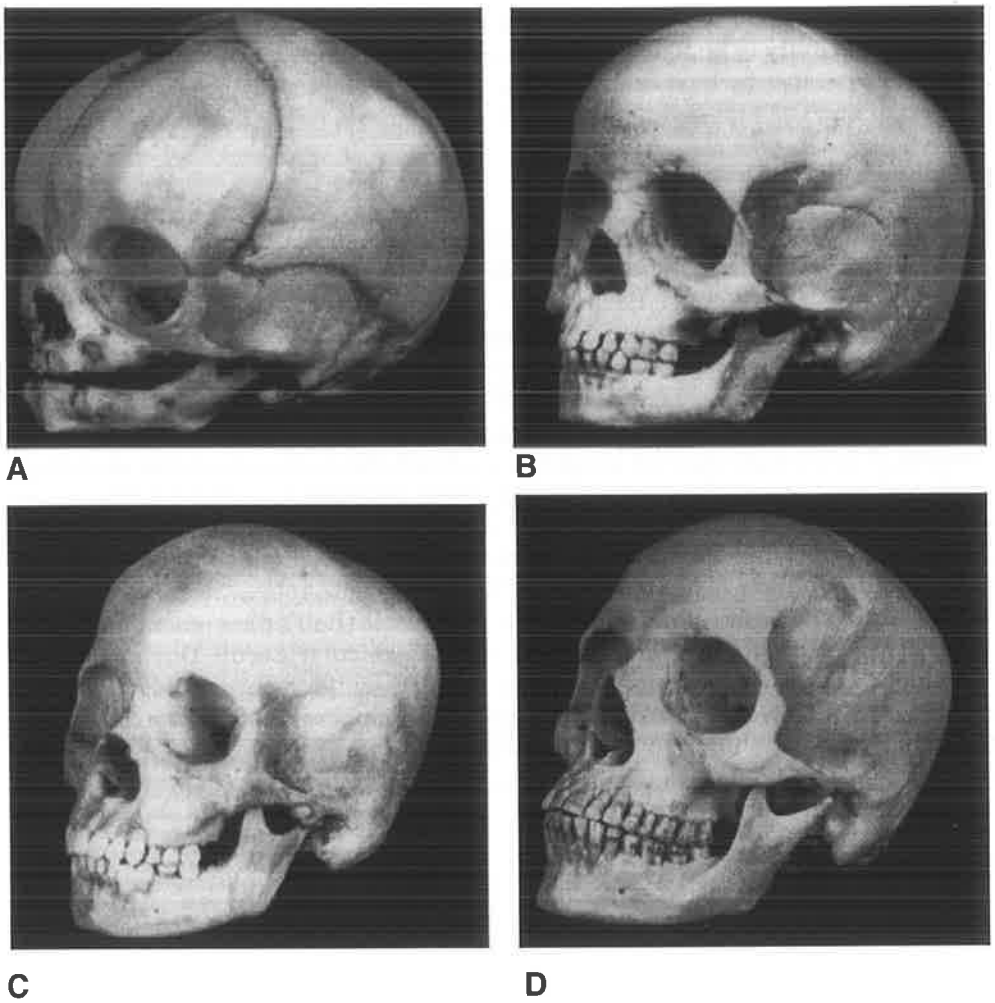


FIG. 2.39. *Growth changes in the skull.* *Skulls in lateral oblique view, demonstrating the changing proportions between cranial vault and facial skeleton with growth. The older skulls have been reduced to the approximate size of the foetal skull* **A.** *Late foetal or early postnatal skull.* **B.** *Child's skull* **C.** *Adolescent skull.* **D.** *Adult skull.*

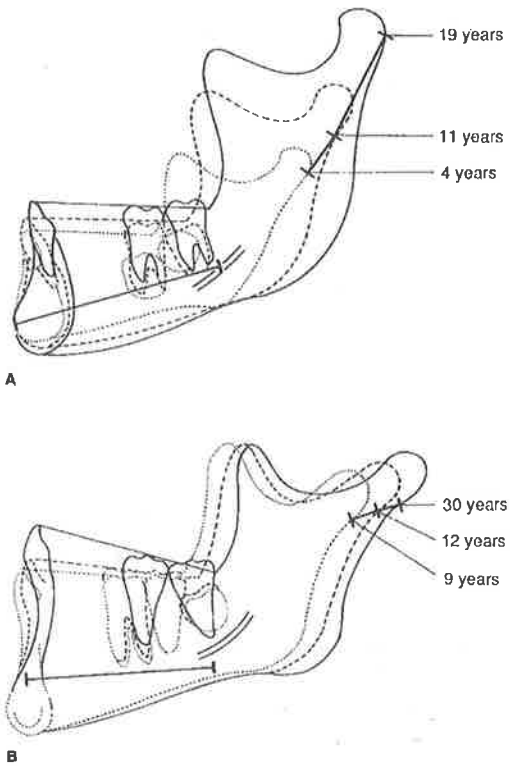


FIG. 2.40. *Mandibular growth patterns.* *Mandibular tracings from cephalometric radiographs superimposed on a line joining two stable metallic implants to show remodelling during growth. Anterior rotation in relation to the implant line is shown in* **A.** *and posterior rotation in* **B.** *In each case there has been resorption along the anterior surface of the ramus and deposition along the posterior. The mandibular border also shows remodelling in conjunction with the anterior rotation. Upward growth of the condyle is accompanied by downward and forward displacement of the mandible relative to the cranial base. Redrawn after Bjork & Skieller (1983).*

response to growth requirements, the bones are remodelled. Surface remodelling is effected by a combination of bone deposition by osteoblasts and bone resorption by osteoclasts. This results in changes in the size and sometimes also the shape of the cranial bones.

Bone remodelling and translation

Although these histological mechanisms are well understood, there is controversy over the nature of the dynamic forces that govern the complex process of bone remodelling and translocation of elements of the skull. Enlow (1982) clarified understanding of these changes by emphasising the concepts of *drift* and *displacement*. Drift represents remodelling of the borders of growing bones; displacement is seen when a bone moves in response to growth elsewhere, as in the descent of the body of the mandible in response to the upward growth of the condyles. Enlow was also interested in the relationship between bone remodelling and the direction of bone growth. His working hypothesis was that the surface of new bone formation always faces the direction of growth.

Bjork, in Copenhagen, used metallic implants placed under the periosteum at various key sites in the maxilla and mandible to study craniofacial growth in children. These implants constituted a stable system of markers of growth. Superimposed serial cephalometric radiographs allowed visualisation and quantification of the processes of drift and displacement over time (Bjork 1969, Bjork & Skieller 1972, 1974, 1976, 1983). Bjork was able to show that the maxilla and mandible rotate away from the cranial base during growth, causing changes in the angulation between the anterior cranial base and a line joining two markers. With the face viewed in left profile, anterior rotations occurred when the implant line rotated in a clockwise direction, and posterior rotations when the line turned counterclockwise (Fig. 2.40). Each type of rotation produced characteristic morphological results. Thus, pronounced anterior rotation of the mandible tended to give a closed bite, an anterior facial height short in relation to posterior facial height, and a squarish face; the reverse morphology resulted from pronounced posterior rotation. Despite substantial rotations and remodelling, the basic shape of the mandible remained fairly constant. Bjork & Skieller (1977) also showed that the maxilla likewise rotates during growth, and this rotation correlates with that of the mandible; the paths of dental eruption are to some extent determined by these rotations. The implant studies also allowed quantification of the anterior migration of the whole dentition and the associated alveolar remodelling that provides space for the later erupting molar teeth.

Postnatal growth: sites, centres and forces

Earlier workers stressed intrinsic bone growth as a driving force in craniofacial growth, especially endochondral ossification in the spheno-occipital synchondrosis, the septal cartilage, and the mandibular condyles (Scott 1967) Weinmann & Sicher (1947) noted that many sutures are parallel to each other and at right angles to the direction of facial growth downwards and forwards. This was correlated with the older view that sutures also produce a growth force — a view not widely held today: sutural growth is now seen as largely a passive response to expansion of the craniofacial viscera.

Moss (1971, 1972) built on his own and earlier work to formulate his still controversial *functional matrix* theory of craniofacial growth. He saw the head as a structure designed to carry out many functions, notably neural integration, respiration, speech, ingestion and mastication, and the special sensory functions. Each function is exercised by a functional matrix of soft tissues and spaces, and each functional matrix is supported and protected by a micro- or macroskeletal unit. Moss postulated that the growth of skeletal units is subordinate to the growth of the related functional matrices. In this view, the skull bones have no inherent genetic information to direct their growth, which is entirely provided by the functional matrices — the brain, the eyes, and the muscles, teeth, fat and glands.

Growth in the CMF region is still a controversial subject: those interested are referred to the authors cited here and to the series of monographs published by the Center for Human Growth in the University of Michigan, Ann Arbor. Clinicians who manage paediatric injuries must be aware of these controversies. Unquestionably, visceral growth plays a crucial role in determining the morphological appearance of the face, but it is still uncertain whether independent skeletal growth is also a factor.

In the growth of the cranial base, the spheno-occipital synchondrosis is regarded as a major postnatal growth centre: endochondral ossification takes place on both sides of the cartilaginous plate, causing an increase in the length of the skull base posterior to the sella turcica. The synchondrosis closes in females between 11 and 14 years of age, and in males between 13 and 16 years.

The cranial base forms a junctional zone between the calvaria and the facial skeleton, and the growth of these structures is interdependent. The shape of the calvarial vault expresses both the growth of the brain and the growth of the cranial base. The membrane bones of the calvaria are separated by sutures which are sites of bone deposition during the period of rapid brain growth (Fig. 2.41). Sutural growth ceases when the brain ceases to grow, though surface deposition of bone enlarges the calvarial skull for some time into adult life. The sutures fuse at various ages, the metopic suture usually in infancy, the others much later (30–40 years) or never.

There is also little doubt that the form of the skull base influences facial morphology (Bjork 1960). Marked flexion of the skull base, measured by the angle between the plane of the clivus and the floor of the anterior cranial fossa (planum sphenoidale), is associated with a low middle fossa, prognathism and a short anterior facial height; conversely, a flat cranial base is associated with the reverse appearance of the face.

The morphology of the upper third of the face and the orbits also relates to the growth of the skull base as well as to the growth of the brain and the eyes (Fig. 2.42). However, the middle third displays a more general growth pattern, with greater postnatal growth relative to the growth of the calvaria (see above), and in particular an adolescent spurt. During postnatal growth, the entire facial skeleton is displaced downwards and forwards away from the skull base; the implant studies of Bjork & Skieller (1976, 1977) have shown that there is much individual variation in this movement.

The nature of this displacement is in dispute. Most authorities no longer regard growth in the complex system of sutures separating the facial bones as a dynamic force carrying the face down and forwards, though like the calvarial sutures these sutures show continuing deposition of bone during the period of facial growth, and there is concurrent bone remodelling by apposition and resorption on the external and internal surfaces of the maxilla and other facial bones. Bone remodelling is responsible for much of the growth in facial breadth and depth, and in the formation and modification of the alveolar processes to accommodate the primary and later the secondary dentition. Bone remodelling is also involved in the downward relocation of the hard palate—an example of bone drift.

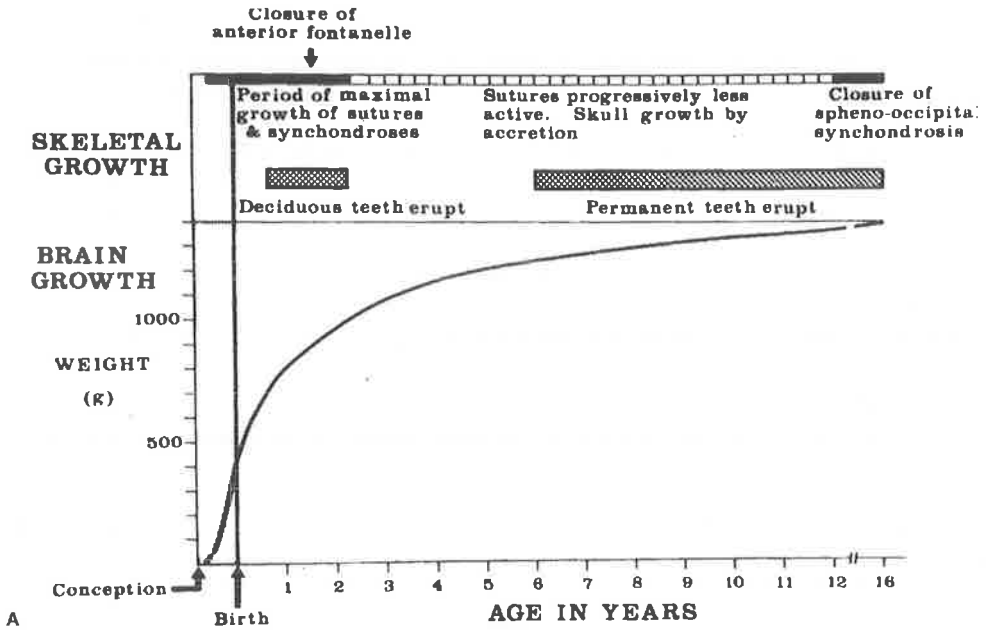
The nasal septum is also involved in this downward and forward displacement. The septum comprises the vertical plate of the ethmoid, the vomer and the septal cartilage, which is a persisting non-ossified component of the embryonic nasal capsule. There is a site of endochondral ossification at the septo-ethmoid junction, and a site of cartilage proliferation at the septo-vomeral junction. The cartilage retains its osteogenic potential for some time after birth, and bone is deposited along its posterior edge. The time at which septal growth ceases is debatable; Scott (1967) stated that the process ends by the tenth year. The role of the nasal septum is contentious. Is it a primary growth centre, driving

the downward movement of the face, or does it respond passively to other growth forces, such as the expansion of oronasal soft tissues and organ spaces? This unsolved question is central in the debate over Moss's functional matrix theory.

Because the teeth must meet in occlusion, growth of the middle third of the face is coordinated with growth of the mandible. Growth changes in the hard palate and maxillary dental arch are also important. During the transitional phase of dental development there are changes in the positions of individual teeth as occlusal relationships are established (Bjork & Skieller 1972). As the primary teeth are gradually replaced, there is a decrease in the depth of the dental arch measured from the contact between central incisors to a line tangential to the mesial surfaces of the first permanent molars. This reduction is associated partly with the size differences between the larger primary molars and the succeeding but smaller permanent premolars (Brown et al 1990). Arch breadth increases slightly, particularly in the posterior region, as a result of growth at the midpalatal suture combined with alveolar remodelling, and the incisors become more upright. The mandible is largely formed by membrane bone laid down lateral to Meckel's cartilage, the embryonic skeleton of the first visceral arch; remnants of Meckel's cartilage persist near the symphysis (Fig. 2.43). Secondary growth cartilages appear in the condylar process about 12 weeks after conception; after birth these condylar cartilages persist as a zone of proliferating cartilage cells under the fibrous covering of the articular surface of the temporomandibular joint. Under this zone of proliferation, new bone is formed by endochondral ossification. Thus by generation of new cartilage above, and new bone below, the condylar cartilages gradually add to the height of the mandibular rami, and displace the mandible downwards.

Growth at the condyles and bone remodelling are the main growth processes responsible for the postnatal changes in the size and shape of the mandible. Nevertheless, despite many experimental studies, there is still uncertainty about the role of the condylar cartilage as a primary growth centre. When the condyle is resected experimentally in the monkey, the resulting deformity is limited to the operated side, the other components of the mandible being relatively unaffected; this is also the case when condylar growth is disturbed by injury or disease in early human life, unless bony ankylosis occurs, when gross deformity results (p. 598). Moreover, transplanted components of the rat mandible do not show the independent growth capacity exhibited by transplanted epiphyseal cartilages, unless the transplanted component includes some of the ossified ramus (Koski 1971). These observations suggest that the mandibular condyle is not a primary growth centre but a compensatory structure maintaining correct relationships between the functional components of the temporomandibular joint as the face enlarges.

Bone remodelling is also crucial in postnatal growth of the mandible. Deposition and resorption of bone preserve the shape of the mandible and are also responsible for the development of the alveolar processes under the stimulus of the emerging teeth. Although the symphyseal region remains relatively unchanged with age, there is extensive remodelling elsewhere. Resorption along the anterior border of the coronoid process provides room for the eruption of the third molar teeth; on the other hand, deposition along the superior border increases its height. Remodelling also occurs along the lower border of the body and the posterior border of the ramus, preserving the basic shape of the mandible and its relations with the growing sub- and retromandibular tissues. Though increase in the breadth of the skull base may be a contributory factor, growth of the width of the mandible is predominantly due to remodelling — bone drift again!



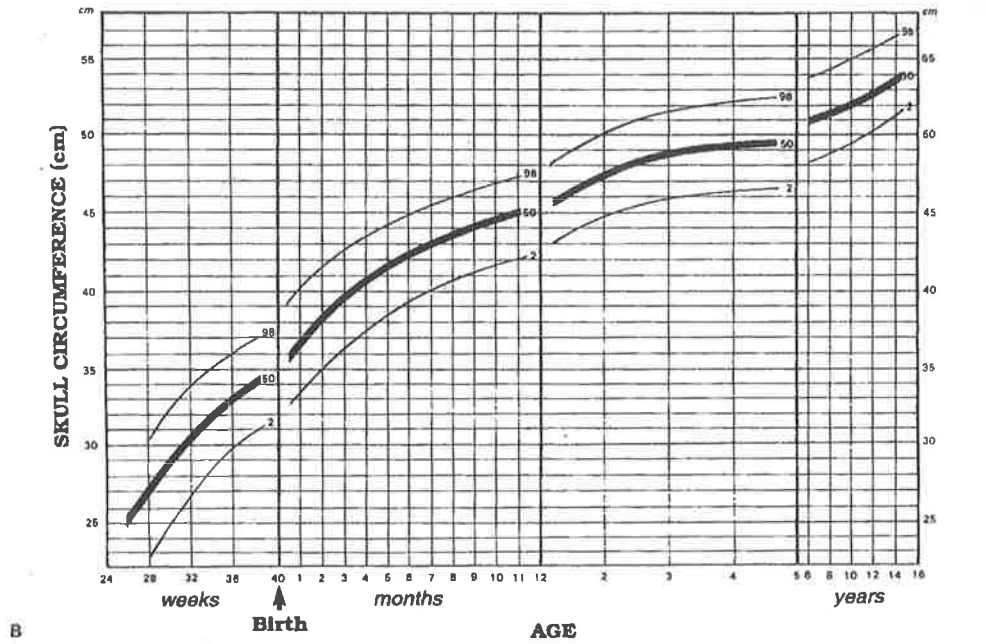


FIG. 2.41. ***Brain growth in relation to the growth of the skull the teeth.*** **A.** *Brain mass increases rapidly in utero and during the first 2 years of life; in the same period, calvarial growth is correspondingly fast and takes place especially across the calvarial sutures. By courtesy of Blackwell Scientific Publications.* **B.** *Head circumference of females, in utero, to 12 months, to 5 years, and to 16 years.*

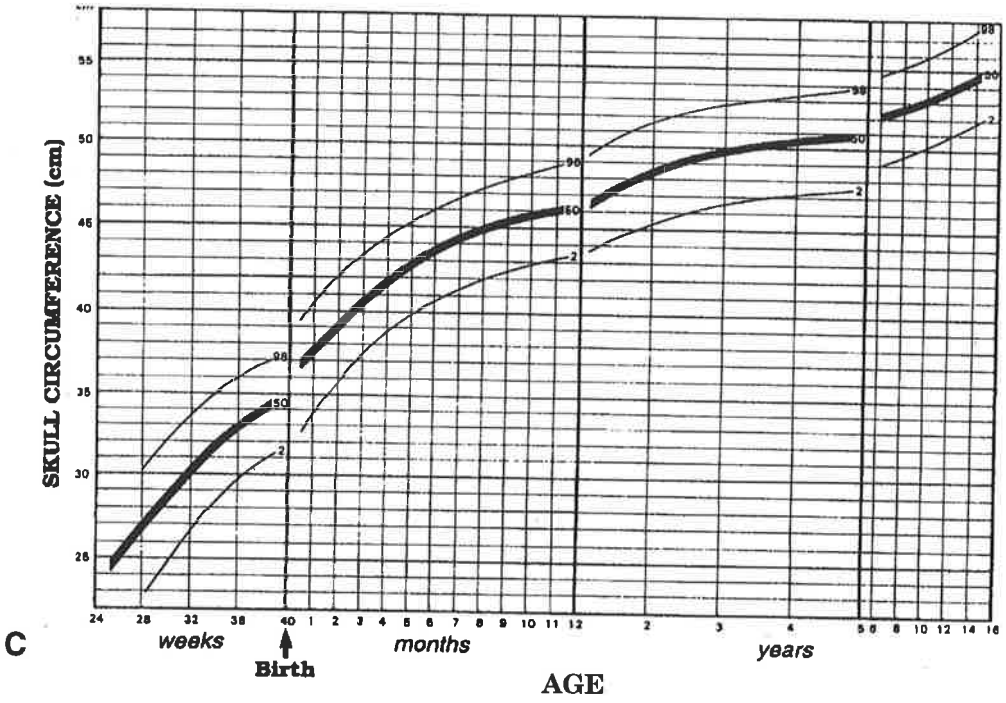


FIG. 2.41. ***Brain growth in relation to the growth of the skull and the teeth.*** *C. Head circumference of males, in utero, to 12 months, to 5 years, and to 16 years. For clinical convenience, percentile charts of age-appropriate sections of the skull circumference curve are used. By courtesy of National Health and Medical Research Council of Australia.*

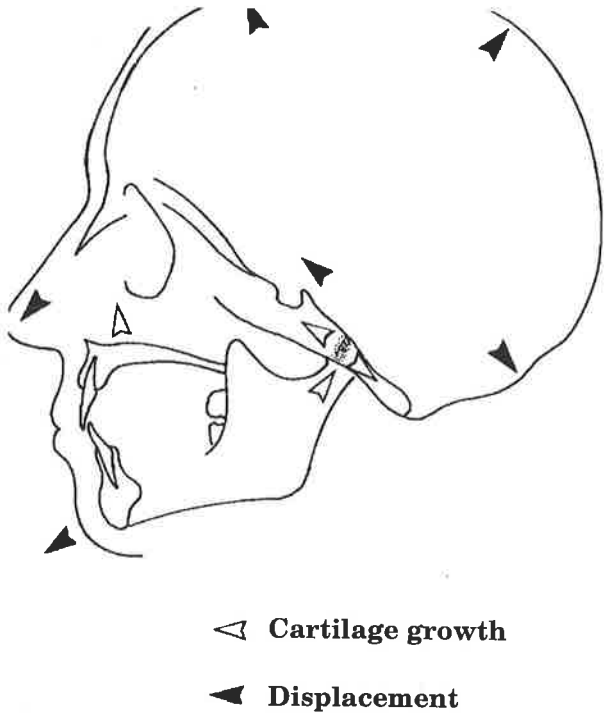


FIG. 2.42. ***Craniofacial growth.*** *Displacement of the craniofacial structures with growth and sites of cartilaginous ossification. Redrawn from David et al (1982) by courtesy of Springer-Verlag.*

Aesthetics of the Face

Ideal proportions

Aesthetics is the science and art of sensual perception. Greek philosphers defined it as harmony, balance and proportion, expressed most subtly in the 'golden section': the concept of subdividing an object so that the smaller part is to the greater as the greater is to the whole. This formula for beauty implies that all beautiful things, including the face, are divisible into parts expressing the golden relation 1: 1.618 (Fig. 2.44). As a measure of beauty, this proportion still has its champion: Ricketts (1981, 1982) has designed a golden divider to ensure that facial landmarks are related in this way. Some medieval artists used the magical number 7; others used the number 5. In the Renaissance, Leonardo da Vinci expressed facial proportions in geometric terms; Converse (1977) has shown that these remain relevant in art and in aesthetic surgery, and modern artists still take from Leonardo the rule that the distance between each eye is the width of one eye. This rule has been formulated in the vertical division of the frontal face into fifths, each the width of the eye from canthus to canthus (Powell & Humphreys 1984); in the deformity of hypertelorism, the intercanthal distance is greater (telecanthus) than one-fifth of the face, and conversely smaller in hypotelorism. The search for the mathematical basis of beauty has persisted so that we still analyse faces using cephalometric X-ray pictures and tracings from these, and relate normal populations to the abnormal, both beautiful and ugly. Nevertheless, aesthetic surgeons who make adjustments of the facial skeleton to beautify normal faces do so on intuitive appreciations and on the wishes of their patients as well as on cephalometric norms (Rosen 1992; Munro 1992).

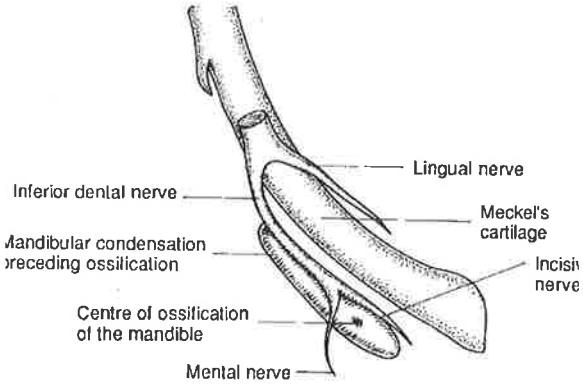
Measurement

Anthropometry is concerned with the measurements of points on the face itself. Measurement has grown to be very sophisticated, and may be made on 3-D images obtained from CT scans or 3-D wire diagrams from biplanar cephlometrics; the production of CAD/CAM (see p. 549) models of faces is a natural progression of the search for the mathematical secrets of beauty. Hard tissue proportions are embodied in the many cephalometric analyses available. Steiner's S-curve and -line correlate soft-tissue points of the nose, lips and chin; Holdaway's H-angle endeavours to relate hard tissue to soft tissue. Fig. 2.45 shows these, and two other profile relationships, which we have found helpful in surgical planning. Powell & Humphreys (1984) have analysed the beautiful profile in terms of the following soft-tissue geometric relationships: the nasofrontal, nasofacial, nasomental, and mentofacial angles, and the nasomental line. For each, norms have been derived from supposedly beautiful models, celebrities and patients.

Value judgements

There are many different ideas about the essentials of beauty, ranging from Hogarth's 'smooth serpentine line' to Francis Bacon's need for some element of strangeness, perfect symmetry being boring; the Miss Universe contests give much support to Bacon's view. Kant believed in simplicity, and this view periodically surfaces in aesthetic fashions. Some modern psychologists believe that aesthetic judgments are the result of a whole pattern of experiences at a particular time — the total experience. The total experience necessarily involves the brain behind the eye of the beholder if beauty is to be 'in the eye of the beholder'. The beholders, with few exceptions, include the persons themselves.

Freudian psychologists have related beauty to potential sexual function. Vigorous young men and healthy young women present the strength of one and the child-bearing potential of the other. These views have been roundly attacked



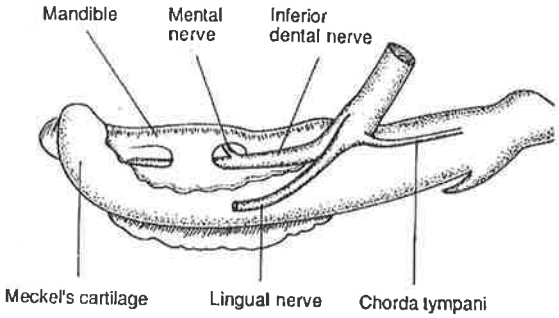


FIG. 2.43. *Early development of the mandible.* *Ossification Of the mandible commencing lateral to Meckel's cartilage at the junction of the mental and incisive branches of the inferior alveolar nerve as viewed from above and medially. Redrawn after Scott & Dixon (1978)*

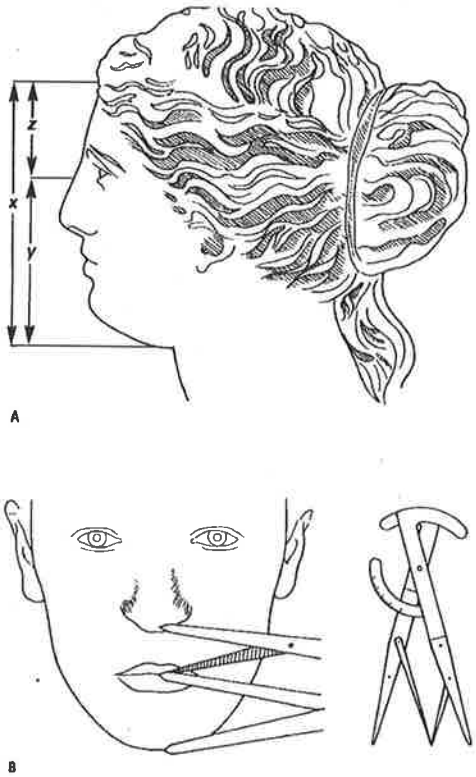


FIG. 2.44. *Classical aesthetics and the 'golden section'.* *The Greek ideal of facial beauty was expressed in geometrical terms; the ratio 1.0:1.618 was seen by some as the ideal (golden) proportion.* **A.** *In this profile drawn from a Hellenistic statue, this proportion is almost achieved in the relations of the hair line — the pupil — the junction of chin and neck. In establishing the soft-tissue landmarks, there is much imagination and even fantasy. In the classical Greek profile, there is an almost unbroken line from the forehead to the nose, with very little indentation at the nasion. Greek sculptors stressed gender differences in the rounder female chin and jaw line.* **B.** *The golden section and the golden dividers: Ricketts has designed dividers to establish the ideal proportion of 1.0:1.618 in assessing the dimensions of the face from hairline — tip of nose — mouth line — chin point. Redrawn from Powell & Humphreys (1984) by courtesy of Thieme Medical Publishers, Inc.*

by Schopenhauer and more recently by Simone de Beauvoir and her followers; but they seem to have relentless persistence. What also persists is the connection between goodness and beauty — an everyday assumption in modern film and television.

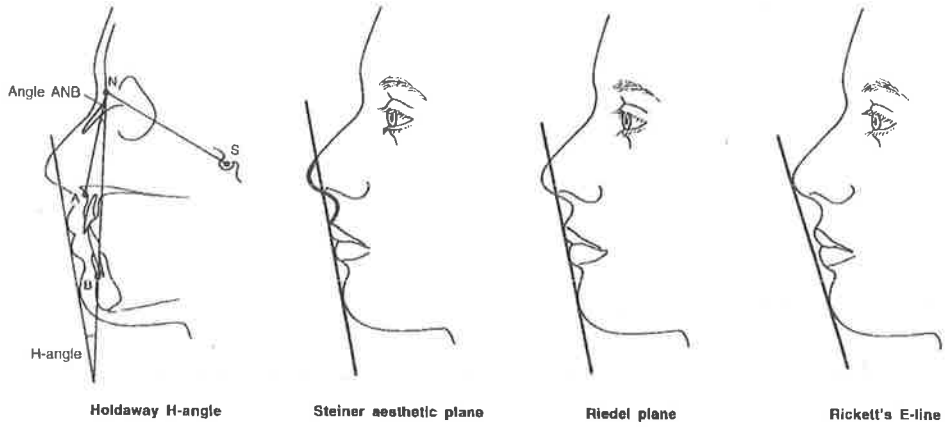


FIG. 2.45. ***Aesthetic geometry in the profile.*** *In planning surgical reconstructions that will affect the profile, geometric concepts of the ideal adult profile are used. A. Holdaway's H-angle: bone - soft tissue. Lines are drawn from the nasion N to the supramentale point B (deepest point in outer mandibular concavity) and the subspinale point A (deepest point in outer premaxillary concavity). The H line is drawn tangential to the soft tissue of the chin and upper lip; it makes an angle H with the line B. When the angle between NA and NB is 1-3°, the angle H ought to be 7-9°. B. Steiner's aesthetic soft-tissue plane. The lower border of the columella and the upper lip form a lazy S curve (heavy line in profile): a line is drawn from the middle of the S to the chin. The lips should lie on this line. C. Riedel's soft-tissue plane. The upper lip, lower lip and chin should lie on a straight line. Redrawn from Powell & Humphreys (1984) by courtesy of Thieme Medical Publishers, Inc. D. Rickett's soft-tissue E-line. A line from the nasal tip to the chin should be about 4 mm anterior to the upper lip and about 2 mm anterior to the lower lip.*

When the Scottish philosopher David Hume (1711-1776) threw his weight behind the concept that beauty is in the eye of the beholder, there was an imperative need to examine the eye of the beholder both in individuals and in the societies that mould them, in which there is infinite variety. If to some extent the face that we see (other than our own in the mirror) is a vehicle for personal projections, then there is an argument for examining the mind behind the eye of the beholder. Impressionist painters gave us every opportunity to use our eyes and our imaginations, and the surrealists such as Magritte show us some of the disturbances that may emerge.

Race provides a great variety of aesthetic ideals. There are significant variations of facial form and colour between the major human races, and within each of these are superimposed local cultural and personal preferences (Vistnes & Eskenazi 1991). Concepts of beauty vary radically on a national and cultural basis, and underlying these ethnic stereotypes are the desires and prejudices of the individual.

Trauma and aesthetics

In the setting of recent trauma, the aesthetics of the face may be disrupted in many ways; integument, bone, contour and muscle function may all be deformed or destroyed. With the exception of injury occuring in infancy and early childhood, it is the change that has been wrought that is important in planning treatment. The results of the treatment to correct the deformity produce a wide range of feelings in the affected individual, the family and the community. It is always helpful to have some idea of the appearance of the individual before the trauma, especially in a severely injured patient.

Aesthetic reconstruction is very complex, dealing as it does with bone, muscle, fat, skin and all the specialized structures of the face including the eyes, the nose, and the very special functions involved in movement, such as facial muscular action and eyelid closure, down to the beautiful lines of the lips with its vermillion and white roll borders. In trauma the problem for the treating team is not how to agree on the aesthetic ideal as visualised by the patient and then to conceptualise and deliver it. It is the problem of restoration where possible, as close as possible, to the pre-existing appearance as known to the patient. The aim is restoration not only of self-image but of body-image, so the problems of aesthetics that come into so many other aspects of cosmetic contouring of the craniofacial skeleton are set against a need to return the patient to the former self. This puts the use of measurements against objective standards raised from particular populations into perspective. The demanding requirement to restore the patient to the former self does indeed require some knowledge of the mensuration of ideal facial form but it is not necessary to have more than a reference to this as the emphasis is on restoring the former self. An excellent up-to-date perspective of aesthetics in facial skeleton surgery is given by Rosen (1992) with expert commentary by Munro. Ricketts (1991) and Barkovic (1991) provide similar up-to-date references from the orthodontic point of view.

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Pathology of injury and repair

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Introduction

The pathology of craniomaxillofacial (CMF) injuries expresses the general pathology of trauma, modified by the structural and functional peculiarities of the CMF region and its component tissues and organs. Of these, the brain, the eyes and the teeth are so specialised that their responses to trauma are in many respects unique: each has its own special pathology. Even for less specialised tissues such as skin and bone, the primary effects of injuries in the CMF region show features that demand individual consideration.

The secondary effects of trauma are very diverse. Some result from acute disorders of normal physiology: the secondary pathophysiology of brain injuries is a subject in itself. Other secondary complications represent microbial colonisation, either by organisms normally colonising epithelial surfaces or by exogenous bacteria; the face, the mouth and the nasopharynx have rich and variable microbial populations.

The reparative processes of the CMF tissues also require individual consideration. As elsewhere in the body, epithelial surfaces and bone heal well; the peripheral nerves of the face regenerate exceptionally well. Cartilage cells and striated muscle fibres have only limited regenerative capacity, but wounds of the facial cartilages or muscles do not as a rule have serious consequences, whereas the complete inability of the neurons of the central nervous system to regenerate is the chief cause of permanent disability from injuries in the CMF region. The processes of repair are mediated by a large number of biologically active chemical substances, of which the proteins identified as specific growth factors are especially important from the surgical viewpoint. Some growth factors are now being used experimentally as therapeutic agents.

In the management of any injury, the surgeon's fundamental aim is to utilize and foster natural healing. When there is massive tissue loss, it may be necessary to supplement local reparative processes by a transplant from elsewhere in the body, or from another individual, or even from another species: reactions to autografts, allografts and xenografts are part of the surgical pathology of trauma. Finally, some injuries are best managed with the help of a surgical implant: the choice and use of implants require understanding of the tissue reactions to foreign materials.

Microbial Flora

Linton & Hinton (1990) have described the normal flora, or more correctly the microbiota, of surfaces and cavities in the CMF region, notably the skin, the upper respiratory tract and the mouth. They emphasize that the 'normal' resident microbes are constantly supplemented by transient intruders from the environment or from other parts of the body. Ecosystems vary between individuals and in the same individual, according to age and health; severe injury may reduce local or systemic resistance and more virulent organisms may establish themselves. In abnormal environments, such as intensive care units, cross

infection may alter the individual's microbiotic ecosystem; invasive procedures may allow intrusion by organisms into cavities which are not ordinarily colonized. Diseases, such as aquired immune deficiency syndrome (AIDS; p. 535), and therapeutic interventions, such as antibiotic therapy, create situations where organisms not usually pathogenic can cause opportunistic infections. Yet, within limits, the composition of the chief regional ecosystems in the CMF region is remarkably constant.

The incidence of infections after wounds of the face and scalp is low compared with wounds in other sites, such as the feet, presumably because the number of endogenous potential pathogens is lower, and also because of the rich blood supply. The facial skin harbours large numbers of coagulase-negative staphylococci, especially *Staphylococcus epidermidis,* which has a predilection for sebaceous glands. Coryneform bacteria are also numerous, especially where the skin sebum content is high. These organisms are weak pathogens, but the skin may also be colonised by coagulase-positive *Staph. aureus.* These often aggressive organisms are especially prevalent in hairy areas, such as the beard and the scalp. The scalp microbiota also includes organisms of little clinical importance, such as the *Pityrosporum* yeasts; these seem incapable of causing infection, even in the favourable environment of a penetrating wound. *Staph. aureus,* on the other hand, is the organism usually responsible for the cerebral infections complicating scalp wounds (Carey et al 1971, de Louvois et al 1977), except when the use of prophylactic antibiotics has fostered gram-negative organisms.

Respiratory tract

The upper respiratory tract harbours a number of separate ecosystems. The anterior nares are often colonised by potentially pathogenic coagulase-positive staphylococci; Wheat et al (1981) quote carrier incidences of 10-50% in healthy populations. The nasopharynx commonly has a normal microbiota of streptococci, especially of the viridans type, and gram-negative cocci. These include many organisms that are not pathogenic, or pathogenic only under favourable conditions. But it is of great clinical importance that *Streptococcus pneumoniae, Haemophilus influencae* and the meningococcus may normally or transiently colonise the nasopharynx. The pneumococcus is the chief cause of meningitis after CMF trauma; repeated attacks of pneumococcal meningitis occurring long after injury (p. 388) may relate to intermittent transient colonization by virulent pneumococci in association with a poorly healed fracture of the anterior cranial fossa. The accessory nasal air sinuses are normally sterile.

Conjunctiva

The conjunctiva is sometimes colonised by *Staph. epidermidis* or by diphtheroids; however, serious pathogens are rarely found except when there is clinical evidence of infection. This paucity of significant conjunctival organisms is attributed to the efficiency of lacrimation and lacrimal drainage: the tears contain lysozyme and other antibacterial agents, and the eyelids, acting like a wind screen wiper, maintain movement of tears to the nasolacrimal duct (p. 74).

Oral cavity

The mouth has an extremely complex microbiota, including yeasts, viruses and protozoa as well as bacteria. The dental age of the individual, the state of dental hygiene and the presence of foreign materials such as dentures are important in determining the composition of the oral microbiota. Notably absent from the normal oral microbiota are coagulase-positive staphylococci and the intestinal gram-negative bacteria. Organisms of potential clinical importance include numerous types of streptococci, including *Strep. milleri,* and anaerobes such as bacteroides; also of clinical interest is the gram-negative facultative anaerobe *Eikenella corrodens,* a coccobacillus found on mucous membranes but capable of causing infection. Yeasts of the candida genus and actinomycetes may assume pathogenic roles in abnormal circumstances. It might be supposed that oral

wounds would show a high incidence of infection, but this is not so (Lieblich & Topazian 1991): the oral mucosa has antibacterial defences, including secretory immunoglobulin A, and infection is rare unless there is associated bone injury. Fractures of the mandible are notoriously prone to cause osteomyelitis if not treated effectively; the organisms responsible often include anaerobes which are not easily cultured. Actinomyces species are often found in the normal mouth and chronic actinomycotic infections may complicate orofacial trauma. The human oral microbial population assumes a different significance when man bites man (or woman): some organisms not often seen as wound pathogens may then cause infections in the victim (p. 496). *E. corrodens* is not uncommonly found in infected human bites.

Intestinal tract

Under abnormal conditions, the microbiota of the gut often complicate the management of CMF injuries. After extensive burns and other forms of very severe trauma, the gut mucosa may atrophy, causing breakdown of the mucosal barrier. It is believed that this contributes to the passage (translocation) of gram-negative bacteria and enterococci into the lymphatic system and bloodstream, resulting in septicaemia, endotoxaemia and multiple organ failure (Rush et al 1988). The experimental evidence for this concept is strong, and there is circumstantial support from clinical studies. It is suggested that an insulin-like growth factor (IGF- 1) may have a protective effect in maintaining mucosal integrity (Huang et al 1993); epidermal growth factor (EGF) has also been shown to reduce bacterial translocation after severe burn injury in an experimental model (Ahdoot et al 1992, Zapata-Sirvent et al 1993).

Soft Tissues

Skin wounds

The general processes of skin wound healing have been much studied and several excellent reviews of the recent literature hate been published (Feinberg & Larsen 1991, Colon et al 1992). Healing follows a sequence of overlapping phases: haemostasis is followed by an inflammatory phase, commonly lasting up to 5 days; this merges into a phase of proliferation and after perhaps 3 weeks to a phase of maturation (Fig. 5.1).

Haemostasis and inflammation

Incised wounds of the face usually heal by primary union (healing by first intention). If the skin edges are not widely separated, they are first coapted by a fibrinous coagulum, formed during the immediate haemostatic response to the injury: blood platelets, tissue thromboplastin and the plasma coagulation factors together mediate the formation of this weak union. Within a few hours, a leukocyte response initiates an antibacterial defence process. Neutrophil polymorphs appear in large numbers in the wound margins, and kill bacteria. It has been supposed that their chief role is to phagocytose organisms, but it appears that their more important function is to liberate proteolytic enzymes and oxygen-derived free radicles, which have microbicidal effects (Wahl & Wahl 1992). Blood monocytes appear, at first in small numbers; they transform to become macrophages, which remove dead tissue and in other ways promote wound healing. After the first few days, the macrophages are more numerous than the polymorphs, which have a much shorter cell half-life; the macrophage is seen as the most important cell in the first stage of wound healing. These leukocyte responses are stimulated by numerous mediator agents released from damaged tissues and from the blood platelets, as well as by activated complement from the plasma (Fig. 5.1). In addition to chemotactic agents derived from tissue breakdown and bacterial action, current opinion emphasizes the roles of cytokines, hormone-like regulators of cellular activity (McKay & Leigh 1991). Of these cytokines, the peptide growth factors appear especially important; some of their sites of action and the

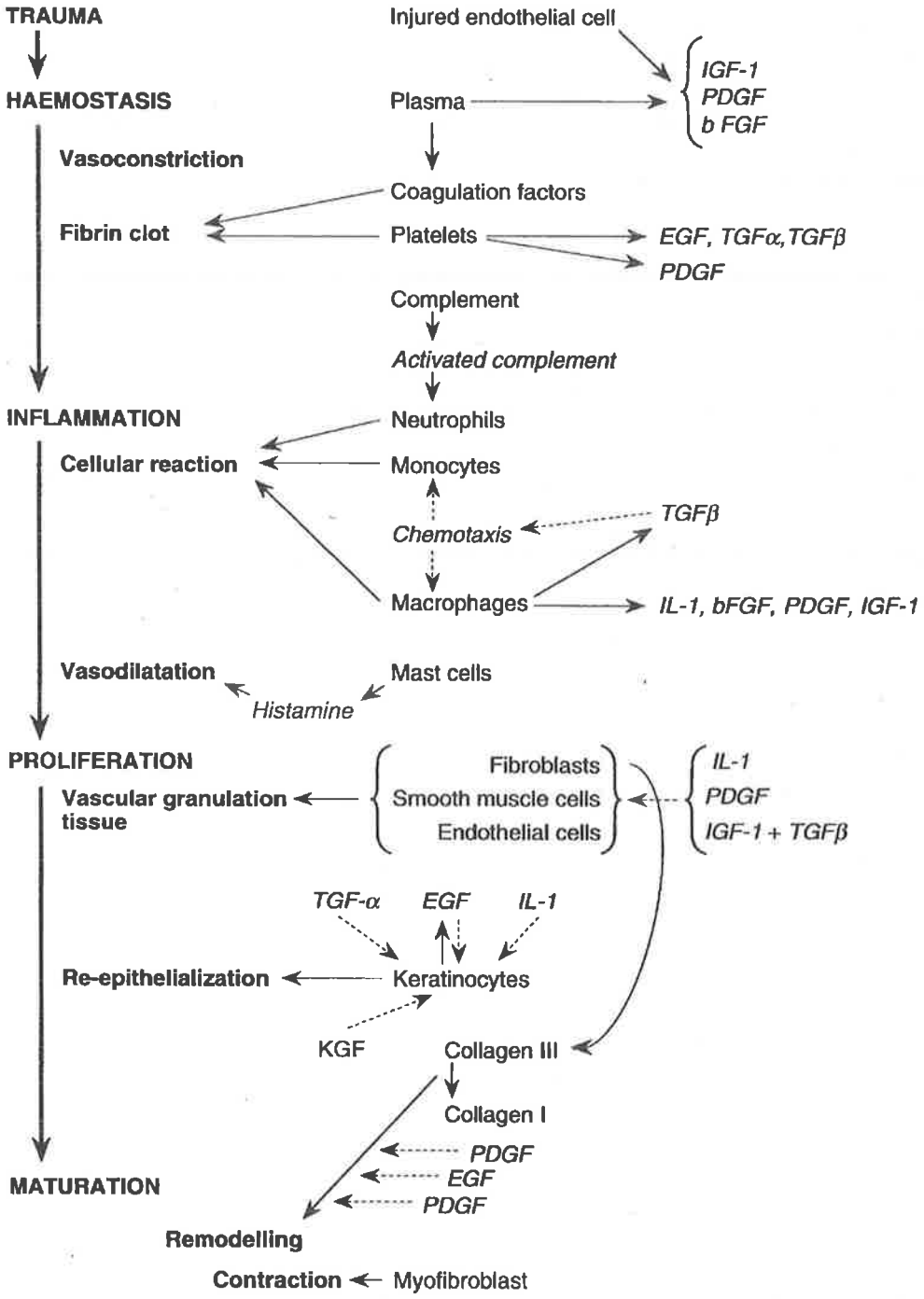


FIG. 5.1. ***Skin wound healing: the roles of chemical mediators.*** *Schematic summary of skin wound healing; the phases of healing overlap, and the linear sequence given in the diagram is an oversimplification. Some of the many chemical mediators are shown in italics: EGF, epidermal growth factor; b-FGF, basal fibroblast growth factor; ICF-1, insulin-like growth factor; IL-1, interleukin, KGF, keratinocyte growth factor; PDGR, platelet derived growth Actor; TGFα, TGFβ: transforming growth factors.*

abbreviations used to identify them are set out in Fig. 5.1. The platelet-derived growth factor (PDGF) is vital in promoting the cellular response to injury (Ross et al 1986); however, it is only one of an array of growth factors contained in the platelets and believed to be important in wound healing. The mast cells, very numerous in the subcutaneous connective tissues, liberate histamine and chemotactic substances (leukotrienes). Blood vessels dilate under the influence of histamine and other agents; endothelial cells alter in morphology and begin to proliferate under the stimulation of mitogenic agents, including the fibroblast growth factors (FGF: Schweigerer et al 1987), of which seven have been identified (Werner et al 1992). Lymphocytes and macrophages become active in defence mechanisms; they produce lymphokines and monokines, active agents among which the interleukins have been shown to stimulate the local cellular inflammatory responses as well as the systemic metabolic response to injury. Interleukin-1 (IL-1), which is produced by activated macrophages, is especially important in the inflammatory response, and also has a role in epidermal regeneration.

Concurrently, and also in response to mediator agents, epithelial cells (keratinocytes) become active. Keratinocytes in the basal layer of the epidermis at the wound edges show amoeboid movement: they proliferate by mitosis and grow into the wound gap. The agents responsible for this include epidermal growth factor (EGF), keratinocyte growth factor (KGF: Werner et al 1992) and other cytokines; Lynch et al (1989a) have shown that two of these, one of the transforming growth factors (TGFß) and an insulin-like growth factor (IGF-1), act only in synergy. However, epithelial growth does not provide wound cover until there is a substrate of granulation tissue.

The formation of granulation tissue begins only a little after the leukocyte reaction. Capillary buds grow into the wound, together with a migration of fibroblasts and lymphocytes. This inflammatory response is chemically mediated, being promoted by a cascade of mediator agents including PDGF which stimulates fibroblast growth as well as leukocyte migration, and the transforming growth factors (TGFα and β) which have many actions, including the stimulation of collagen formation Jeffrey 1992). Lymphocytes have many roles in wound healing, not all of which are yet understood; the T-lymphocytes produce agents promoting fibroblast proliferation.

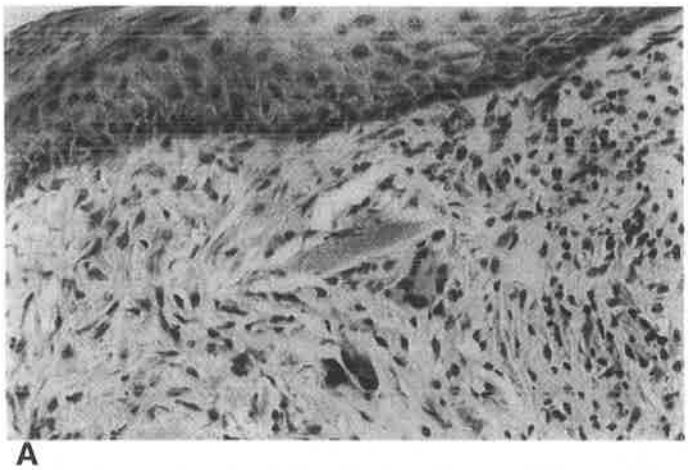
Proliferation

The inflammatory phase is followed by the second or proliferative phase of wound healing. Epithelialization, fibroblastic activity and deposition of collagen dominate this stage. Epidermal cells migrating into the wound meet other epidermal cells and establish continuous epithelial cover; they then cease to migrate and by continued mitosis they establish a mature stratified epidermal layer. When migration ceases, the keratinocytes lay down a new basement membrane composed of type IV collagen.

The fibroblasts secrete extracellular matrix; they lay down collagen in the form of an unorganized gel as early as the second day after injury. Initially, collagen of the embryonic type III is deposited in the extracellular matrix Collagen formation is promoted by the growth factor TGFβ but retarded by glucocorticoids.

Maturation

This is the third stage of healing and continues for some months. In the deeper layers of the wound, cross-linked type I collagen replaces type III collagen, and gives a stronger union. Estimates of wound tensile strength depend to some extent on the technique of measurement chosen; after the third or fourth day, strength increases, but it is stated that as late as 3 weeks, skin wounds commonly have only a third of their normal tensile strength. Collagen fibres are remodelled and tensile strength increased though experimental wounds in aponeurotic tissue are said to be less strong than normal controls even a year later (Douglas 1963).



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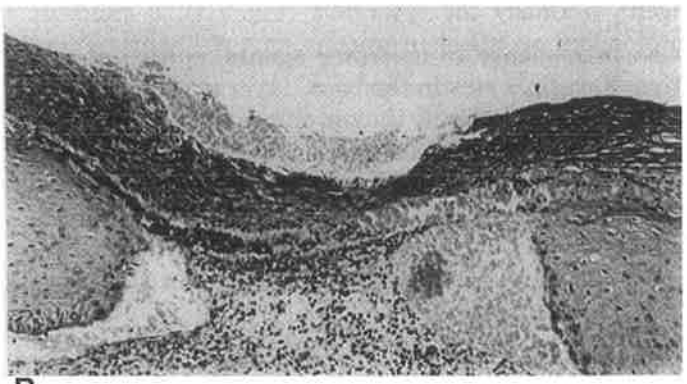

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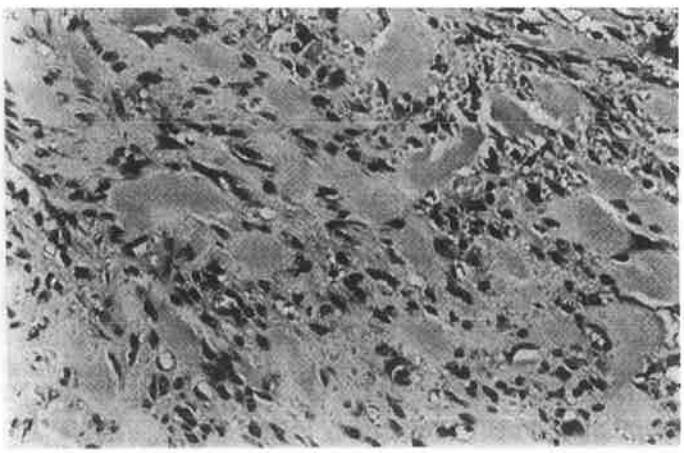


C

FIG. 5.2. ***Abnormal healing in skin wounds.*** *These biopsies shout various complications in wound healing, all of which can result in delayed healing and/or ugly scars.* **A.** *Inflammatory reaction, with giant cells in relation to a foreign body, the overlying epithelium is thickened. Haematoxylin and eosin (H&E); × 169* **B.** *Dehiscence: a layer of dead epidermis covers the separated edges of healthy epidermis and a cellular exudate has formed in the centre of the wound. HOE, × 73.* **C.** *Subcutaneous keloid: bundles and sheaves of coarse collagen, with many fibroblasts. H&E, × 169.*

The wound becomes less vascular, by regression of newly formed capillaries; however, larger vessels are formed, giving better flow of blood across the line of the healed wounds The wound becomes a scar and maturation of the scar may continue for some years. Maturation and scar shrinkage are unpredictable processes, variable both in time and in extent; this unpredictability explains the occasionally disappointing aesthetic result of some reconstructive surgical procedures, especially in the orbital region (p. 573).

Complications

The idealized process of primary wound healing is seen after surgical incisions in the face and scalp. and many clean accidental wounds follow a similar course. However, facial injuries due to blunt impact may be bruised or abraded; there may be implanted foreign matter (Fig. 5.2A). Abrasion of the epidermis and even of the dermis is often seen, and this may lead to delayed wound healing (healing by second intention). The abraded surface is covered by a fibrinous coagulum, which hardens as a scab. Epithelialization may then result from cellular migration from the margins of the abraded area, or from residual skin appendages such as swear glands and hair follicles under the scab. This process is facilitated by contraction of the injured area: fibroblasts (Derby et al 1990) in the granulating wound develop the contractile properties of smooth muscle cells and the action of these so-called myofibroblasts (Guber & Rudolph 1978) reduces the area to be covered by epithelial cells. However, the process of contraction may have adverse aesthetic and functional effects (p. 475). Wound contraction is more effective in young tissues (Catty 1965).

The general processes of wound healing can take different forms in various parts of the CMF region. Scalp wounds usually heal well, unless there is impairment of blood supply, when areas of marginal necrosis may be seen: this is especially likely if the wound is closed under tension, for the scalp has less elasticity than the facial skin (Fig. 5.2B). Incised wounds of the face also heal well; however, facial abrasion and tissue loss can lead to an unsightly scar, as can indriven foreign matter. Explosions can drive grains of earth or debris into the skin causing permanent tattooing. Keloid formation (Fig. 5.2C) represents an abnormal and excessive overgrowth of fibrous tissue in a wound; keloid tissue is characterised by nodules of vascular and fibroblastic hyperplasia, with poorly organised bundles of collagen (Murray & Pinnell 1992), often including large hyalinized fibres. Keloid formation is not especially common in the CMF region, but is by no means rare.

Facial burns

Thermal or chemical injury causes denaturation of cellular proteins and consequent cellular necrosis (p. 115). Depending on the intensity and duration of the injurious event, the cells may suffer immediate coagulative destruction, or may appear intact but undergo delayed death, or may show reversible swelling (Sevitt 1957). Necrosis is first evident in the tissue layer closest to the injurious agent, normally the epidermis (Fig. 5.3); if exposure is prolonged, as when an unconscious accident victim lies against a hot structure, the considerable protective capacity of the epidermis is destroyed and deeper tissues undergo necrosis — first the dermis and then muscle, bone and even brain. In a superficial skin burn, keratinocytes in the outer layers of the epidermis die, but the basal cell layer remains intact (Fig. 5.4A). The subcutaneous capillary plexus shows vasodilatation, evident as erythema, but there is no blister formation. The epidermis regenerates in a few days and the erythema subsides. In deeper burns, both epidermis (Fig. 5.4B) and dermis (Fig. 5.4C, D) are involved. Tissue damage evokes an outpouring of inflammatory mediators which increase capillary permeability, allowing escape of serum to form oedema fluid. This may separate viable from non-viable tissue; in partial thickness (second degree) burns, oedema fluid raises a blister by separating necrotic or partly necrotic epidermis from living dermis. When the blister ruptures, the protein-aceous fluid leaks out and the dermis is exposed, typically as a moist surface with intact pinprick sensation and intact capillary circulation. Such burns will usually heal spontaneously. Modern biologically compatible dressings allow more rapid re-epithelialization and give better cosmesis and function. More severe burning destroys an increasing depth of dermis. In a full-thickness burn all epithelial structures are lost and the dermal collagen shows coagulation necrosis, losing its normal eosinophilia and fusing into coarse strap-like bands (Fig. 5.4D). There may be complete capillary stasis, and secondary thrombosis may be seen. Between these extremes, there may be burns of intermediate depth, in which a few epidermal structures are

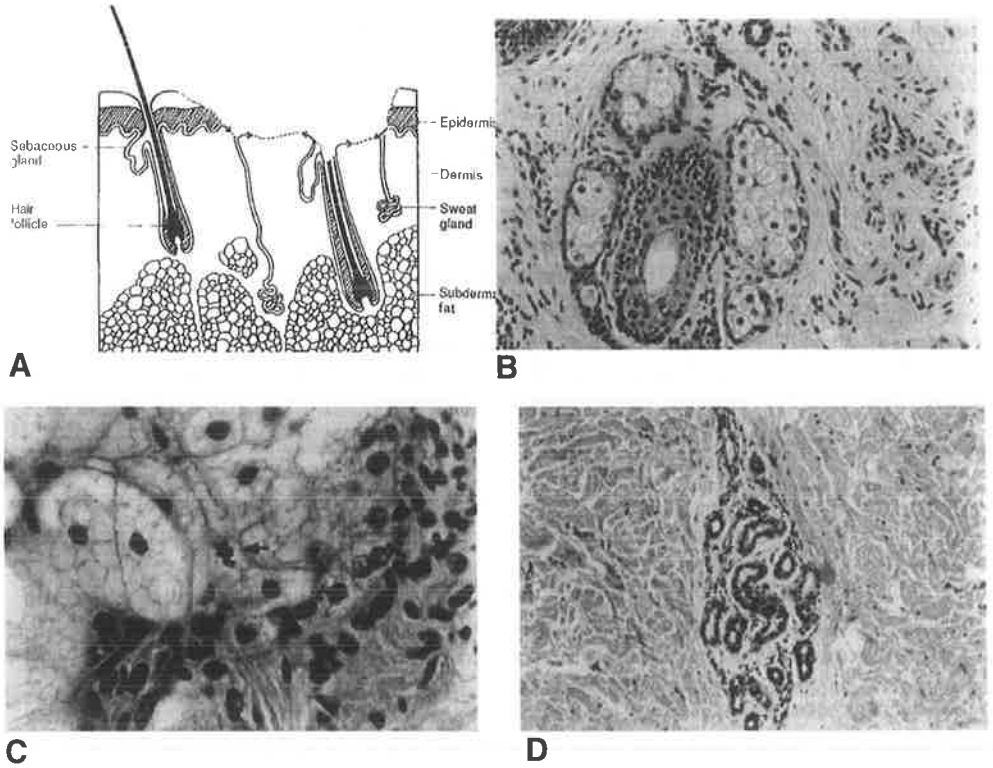


FIG. 5.3. ***The skin: thermal defence in depth.*** *The epidermis constitutes a significant barrier against thermal and chemical injury, the epidermal glands and hair follicles provide mitotically active cells for regeneration of the epidermis if this has been destroyed.* **A.** *Diagram of a skin burn showing the layers and the chief sources of epithelial regeneration.* **B.** *Hair follicle, surrounded by sebaceous glands; a single coil of a sweat gland is shown at the top of the held (arrow). H&E, ×169.* **C.** *One of the cells in a sebaceous gland is in mitosis (arrow) H&E, × 507* **D.** *Sweat gland deeply placed in the dermis. H&E, ×85.*

preserved, resulting in slower or incomplete re-epithelialization. In deep burns, the collagen necrosis results in formation of a slough or eschar.

A burn is a three-dimensional wound, often involving a considerable area and extending to varying depths. Jackson (1993) distinguished a central zone of coagulation surrounded by a zone of capillary stasis; in his experiments the vascular stasis led to an increase in the necrotic zone, and this is in accord with the clinical observation of progressive increase in the area of non-viable tissue 3–5 days after the burn. Surrounding the zone of stasis was a zone of inflammatory hyperaemia. Later research has amplified these findings. The hyperaemic zone is the result of the liberation of vascular mediators such as histamine, serotonin and vasoactive kinines. When the inflammatory reaction subsides, normal healing begins in the hyperaemic zone. Robson et al (1979) studied the progressive loss of dermal perfusion in the zone of vascular stasis, and demonstrated that thrombosis was the cause of the increase in the area of coagulation. This process is thought to begin with platelet thrombo-embolism which causes venous obstruction in the presence of continued pulsatile arteriolar flow. Robson et al (1979) have implicated prostaglandins, especially thromboxane A_2 as one cause of the intradermal vascular shutdown. The progress of necrosis in the zone of stasis can be partly arrested by wound cooling (Boykin & Molnar 1992), and by a biologically compatible dressing such as porcine skin (Zawacki 1974).

Electrical burns have unique characteristics. Necrosis is intense at the point of entry, but may extend deeply along tissue planes in an unpredictable way. Fig. 17.20 shows the effects of a severe electrical burn in the frontal region:necrosis of the scalp and skull, thrombosis of the underlying sagittal sinus and of smaller vessels in its vicinity, and necrosis of areas of the dura mater. In this case, it was noteworthy that the inflammatory reaction, though quite strong on the surface of viable dura mater, showed no sign of invasion of the necrotic aura nearly 4 weeks after the burn.

Superficial burns heal rapidly: the epidermis regenerates from mitosis in the basal layer, or if this is destroyed, from squamous cells in the surviving hair follicles, sebaceous glands and sweat glands (Fig. 5.3B-D); Boykin & Molnar (1992) quote work indicating that the hair follicles are the chief source of epidermal regeneration. It appears that the process is promoted by EGF and transforming growth factors. Deeper burns with destruction of the epidermal glands and hair follicles must heal by second intention (see above). Granulation tissue forms, and collagen is laid down: this contracts under the influence of myofibroblasts, and the scar is then epithelialized by migration of epidermal cells from the periphery. This process is retarded by overlying eschar. Slow healing predisposes to hypertrophic scar formation and an ugly secondary deformity often with considerable functional loss (p. 486). Modern burn management includes early tangential excision of eschar to accelerate the healing process and to minimise wound contraction.

Skin grafts

In the routine procedure of split-skin grafting, an area of normal skin is cut to give a sheet of epidermis, with a variable amount of the underlying dermis (Converse et al 1977); a medium thickness split-skin graft is 0.016 in. (0.4 mm) in depth. The graft is secured on the denuded area to be covered whether this be a granulating burn, an area of exposed pericranium or an area of cancellous bone. Survival depends initially on nutritional support by serum exuded from the capillaries in the denuded tissue. Permanent survival depends on vascular connections between the dermal elements in the graft and the host tissue, and these appear within 48 h of graft application. Capillary buds and later fibroblasts invade the deeper layers of the graft, and within 7-10 days both vascular supply and drainage are established. The nature of this critically important process of revascularization is reviewed by Converse et al (1977). The stratum corneum of the graft is usually desquamated in the second week. Klein & Rudolph (1972) showed that the collagen components of a skin graft are rapidly replaced by new collagen locally synthesised during the last 4 weeks. New collagen is laid down, and this contracts under the influence of myofibroblasts. The process of wound contraction may result in shrinkage of the grafted area and an ugly appearance; Rudolph et al (1992) have reviewed evidence showing that myofibroblastic activity is inhibited by a surviving skin graft. The success of the process of grafting depends on maintaining contact between graft and host tissue: this is lost if the graft moves, or if bleeding takes place under the graft. Infection, especially by *Strep. pyogenes* or *Pseudomonas pyocyaneus,* may also cause graft failure. Full-thickness skin grafts, denuded of fat, are less rapidly revascularized, but have the merit that they are less apt to contract or to become pigmented.

Skin allografts, whether taken from a living donor or from a cadaver, are invariably rejected, unless taken from an identical twin. The grafted skin is at first accepted and invaded by capillaries, but after 2-4 weeks the host lymphocytes recognize tissue incompatibility and the graft becomes necrotic. Cadaver skin has nevertheless been much used as a temporary dressing to cover a large area of burned skin. Alternatively, autogenous keratinocytes can be grown in tissue culture, the growth being accelerated with EGF, and may then be used to cover a large area, perhaps in conjunction with other types of skin graft. Hansbrough (1990) and Boykin & Molnar (1992) have reviewed current work on cadaver skin and other substitutes for autogenous skin grafts.

Striated muscle

If a muscle is lacerated or severely contused, the sarcolemmal sheaths of the muscle fibres are disrupted, and the fibres undergo necrosis and are removed by macrophages. Two types of reparative process may then follow. Under favourable conditions, true regeneration of new muscle fibres takes place. Long ago, Le Gros Clark (Clark 1960) noted that if the endomysial connective tissue tubes were preserved, buds of sarcolemmal proliferation might regenerate new muscle fibres. It is now known that this regeneration represents the activation of satellite cells

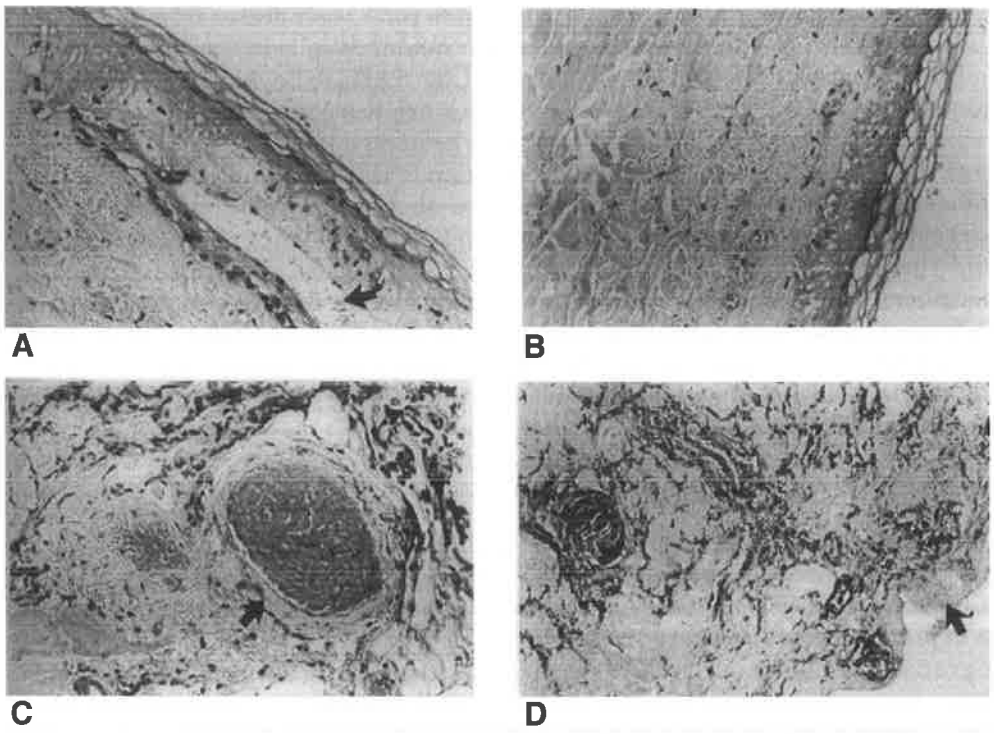


FIG. 5.4. ***Skin burns.*** *These biopsies show types of burn, several days after injury.* **A.** *Superficial burn, with blister formation (arrow); the basal epithelial layer has survived and is beginning to regenerate. H&E, × 146.* **B.** *Deeper burn, with necrosis of whole epidermis and some of the dermis. H&E, × 146.* **C.** *Necrosis of hair follicle (arrow) and adjacent glandular tissue. H&E, × 146.* **D.** *Deep burn, with necrosis of epidermis (arrow) and underlying dermis; the hair folllicles and glands are almost unrecognisable. H&E, × 146.*

lying within the muscle fibre; they are transformed into myoblasts and finally into mature contractile muscle fibres (Lehto & Järvinen 1991). However, repair by fibrous tissue is more usual. In facial muscular lacerations, the divided ends of the cut muscles are almost always separated and unite by scar tissue. Fibroblastic activity fills the gap between the muscle ends and they are joined by a collagenous scar: a reduced contractile capacity results, especially if there is also damage to the innervation of the muscle. This may affect the aesthetics of facial expression.

Adipose tissue

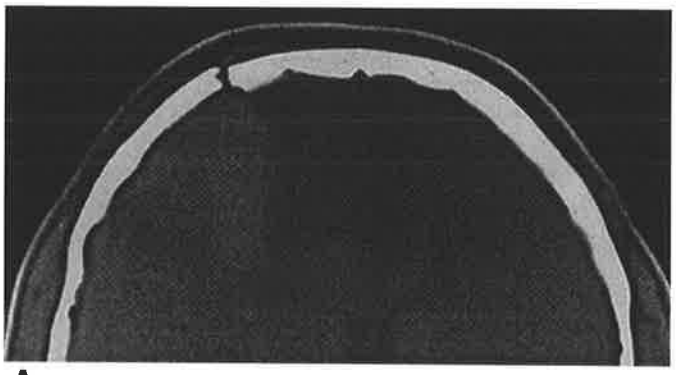
This has very limited regenerative power. Damaged fat cells liberate their contents and these are removed by phagocytosis; in orbital injuries, loss of tissue fat may result in permanent recession of the globe. The inability of fibrofatty tissue to regenerate effectively is also seen in the cheeks: damage to a cheek pad can result in loss of the normal contour of the face.

Skeletal Tissues

Bone

Fracture types

Fractures of the facial skeleton and calvaria take many forms and these can be extremely complex. However, two main fracture types are traditionally identified: linear — with or without displacement — and depressed. In a depressed fracture, an area of bone is detached and driven in below the normal anatomical surface. One can identify a third type of penetration fracture, in which a missile or sharp object pierces bone, leaving a bone defect.



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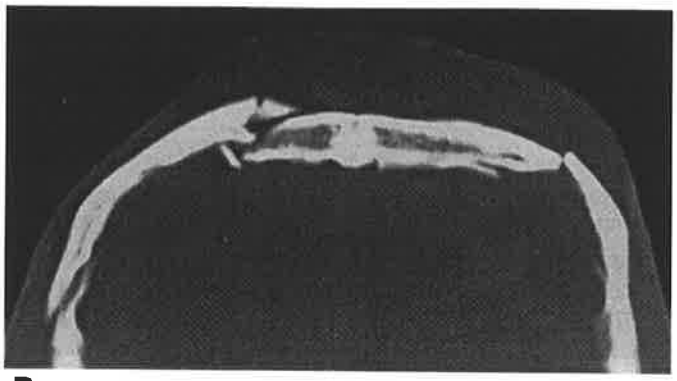

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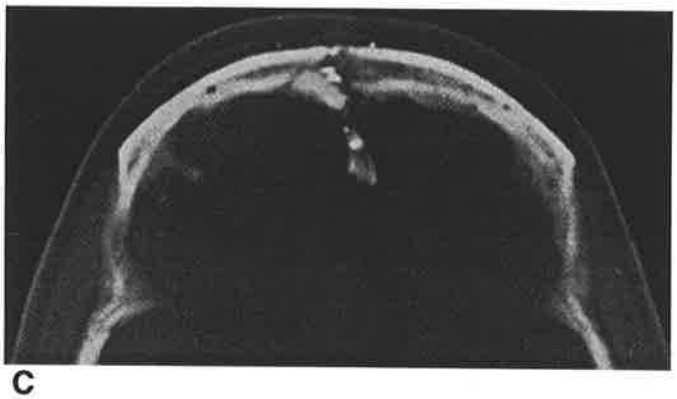


C

FIG. 5.5. ***Fracture patterns in the calvaria.*** **A.** *CT scan: linear fracture in frontal bone. There was an underlying cortical haematoma.* **B.** *CT scan: depressed fracture, with shattering of bone at medial margin of depression. There was extensive frontal contusion.* **C.** *CT scan: frontal penetrating bullet wound with indriven bone chip.*

Linear fracture

Linear fractures, usually without displacement, are often seen in the calvarial bones (Fig. 5.5A). Blunt impacts in the frontal region may crack the strong frontal bone and propagate linear fractures running into the temporal bone or the roof of the orbit; more posterior frontal impacts may diastase the coronal suture, again with linear extensions. Mandibular fractures are usually linear (Fig. 5.6A) and occur in sites of structural weakness; the action of the powerful masticatory muscles often causes displacement. Linear fractures are also often seen in the maxillary complex, running along the lines of weakness identified by Le Fort (p. 18); these are usually associated with some degree of displacement.

Depressed fracture

These fractures result from localized overload (p. 102). An area of bone is driven in, and displaced into the underlying soft tissues. Such fractures often result from impacts on the frontal bone (Fig. 5.5B), the depressed fragment(s) being

driven into the brain or the frontal air sinus. Fractures of the zygomatic bone are also usually depressed the slender arch breaks and the body of the bone is driven into the temporalis muscle, with variable infracturing of the lateral wall. Blow-out fractures of the floor of the orbit are depressed by force transmitted through the orbital contents (p. 104); depressed fractures of the orbital wall (Fig. 5.5B) or roof may be caused by buckling. Missile wounds sometimes cause depressed fractures, when the missile has a low impact velocity, or when it strikes the skull tangentially.

Penetration fracture

The penetrating or perforating wounds inflicted by bullets are characterised by a round bone defect, often not much larger than the bullet; numerous small chips of shattered bone are driven deeply into the brain or facial soft tissues - (Figs 5.5C and 5.6C,D). Very high energy missiles leave a larger bone defect, but these are usually lethal.

Bone healing

Whatever the fracture pattem, the healing process expresses the general tendency of bone to reconstitute unless there are adverse local or systemic conditions. Although most parts of the facial and calvarial skeleton differ from the long bones in embryological origin, there is no reason to think that the healing processes are qualitatively different: facial fractures tend to unite more rapidly than most long bone fractures, but this can be attributed to their small size and good local vascularity (Phillips & Rahn 1992).

Bone healing may take two forms: primary, or direct, and secondary, or healing by callus. Direct bone healing is further classified as contact and gap healing, according to the distance between the bone edges.

Primary bone healing

This is seen when the bone edges are in good apposition and immobile (Spiessl.1989). If there is less than 0.1 mm between the edges, it is usual to speak of contact healing (Fig. 5.7A); if the distance is 0.1-1 mm, the process is termed gap healing (Fig. 5.7B). Two phases are recognized: an initial inflammatory phase and a later phase of remodelling. In the first, which is analoguous to the inflammatory phase of healing of a skin wound, there is a proliferation of capillaries, osteoblasts and osteoclasts from the edges of the fractured bones: the endosteum, the haversian canals and the periosteum all contribute. Osteoblasts lay down new bone; osteoclasts remodel surviving compact and trabecular bone.

In contact healing, the process is direct: bridges of new bone extend from one bone surface into holes reamed out of the opposite face, so that continuous haversian systems are established. Rahn (Spiessl 1989) has visualized this process very elegantly in scanning electron micrographs taken after separation of the bone edges: wedges of new bone are seen projecting to engage in reciprocal pits in the opposite bone face. Contact healing is nevertheless associated with initial osteoporosis and loss of structural strength.

When the gap between the edges is too wide to allow direct union of haversian systems (osteons), the interspace is filled with osteogenic granulation tissue, in which trabeculae of bone are laid down, often in a plane parallel to the fracture line. In the second stage of bone healing, the process of remodelling, these trabeculae are converted into lamellar bone. Gap healing is slower than contact healing, but ultimately as strong.

Secondary bone healing

This is seen when the bone edges are not fully immobilized (Fig. 5.7C). Bleeding into the fracture site forms a haematoma which is invaded by blood vessels, macrophages and fibroblasts. It appears that the haematoma fluid develops

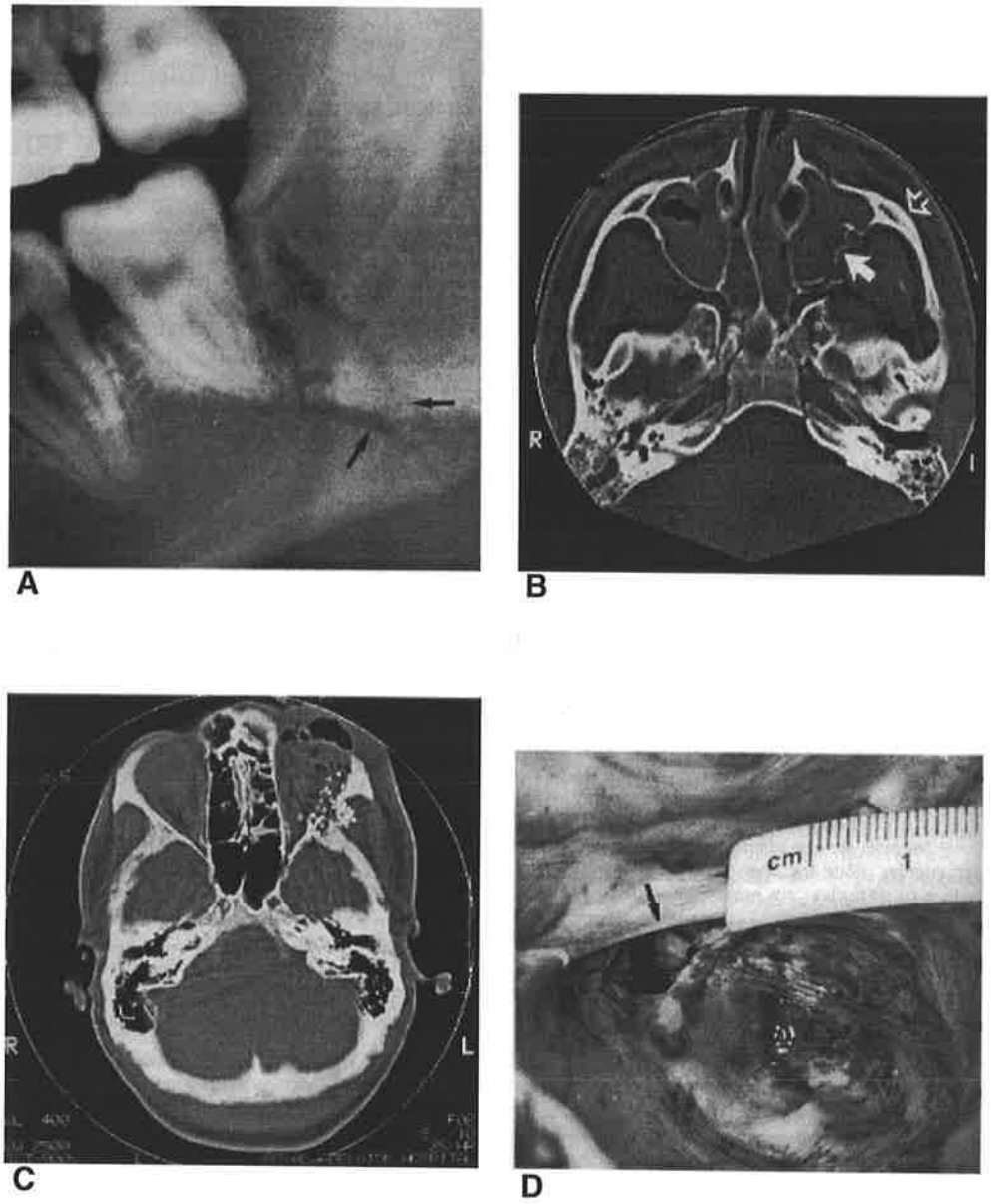


FIG. 5.6. ***Fracture patterns in the facial skeleton.*** **A.** *OPG: linear fracture (arrows) in the mandible.* **B.** *CT scan: depressed fracture (arrow) in the maxilla, buckled by a blow (open arrow) on the malar eminence.* **C.** *CT scan: penetrating wound of orbit by fragmented hunting rifle bullet.* **D.** *Autopsy (same case): the arrow points to a small fracture in the posterolateral wall of the orbit, below the sphenoidal ridge. By courtesy of Dr. C. Manock.*

osteo-inductive capacities (Mizuno et al 1990). Collagen is laid down and there is an influx of cells capable of osteoblastic and chondroblastic activity. Variable amounts of fibrocartilage and osteoid appear and glue the bone edges together. As the blood supply improves, the cartilage becomes calcified and finally ossified as a mass of callus joining the bone ends with increasing rigidity. In the stage of remodelling, the callus is converted into cortical and trabecular bone and the structure of the bone at the fracture site is restored.

Regulation of bone healing

Bone healing is apparently promoted and regulated by a cascade of growth factors; PDGR and IGF-1 are considered to be important (Lynch et al 1989b). Proteins present in bone matrix evidently stimulate local cellular activity; one of these, known as bone morphogenetic protein, is potent in inducing new bone formation (see p. 134). Bioelectric forces also play a local role in the process, while systemic parathormone appears to modulate the formation and release of growth factors (Mohan & Baylink 1990).

Bone healing in the CMF region

In this region, both types of bone healing may be seen. Calvarial fractures unite by the primary or direct process: the intact bone edges slowly generate new bone. This is accompanied by a transient increase in vascularity evident in X-ray pictures as osteoporosis. A similar process is seen in depressed calvarial fractures: pieces of detached bone undergo avascular necrosis, appearing abnormally dense, with surrounding osteoporosis in the intact bone (Fig. 5.8). Even untreated depressed fractures of the skull vault heal in this way: the depressed fragment is eventually fully incorporated, still in the depressed position, with the tables and diploe reconstituted. Similar primary union is seen in facial fractures and osteotomies: Thaller & Kawamoto (1990) have shown full reconstitution of lamellar architecture in biopsies of middle third facial fractures. Indeed, the thin well-vascularized plates of facial bone commonly unite rapidly by contact or gap union.

Only in fractures of the mandible has union after callus formation been a common finding, and this of course expresses the difficulty of achieving complete immobility of the fractured mandible. Rowe & Killey (1955) illustrated the process of secondary healing in a series of biopsies of mandibular fractures. First, the fracture haematoma was invaded by monocytes, followed by fibroblasts depositing collagen, which effected fibrous union after 3 weeks. Primary callus was evident at 6 weeks: the authors saw this as woven bone, though their beautiful micrographs suggest that some cartilage may have been present. Later remodelling created lamellar bone. Rever et al (1991) have found that experimental fractures in the rabbit zygoma show an intermediate phase of cartilaginous union before eventual complete bony union. Modern methods of miniplate fixation (p. 237) should achieve primary union in all facial fractures.

Fracture complications

These include non-union, mal-union and infection. In the CMF region, fractures of the mandible are especially likely to show these complications, but fractures in other parts of the facial and calvarial skeleton may also exhibit abnormal healing patterns.

Non-union

This may result from displacement of bone fragments and interposition of soft tissue (Fig. 5.9). This is seen especially in the roof and floor of the orbit: herniations of brain and orbital contents respectively prevent reconstruction of the skeletal anatomy. Herniations of brain and/or leptomeninges will prevent bone healing in the skull base; Linell & Robinson (1941) showed in autopsy material that this failure of healing in the vicinity of one of the paranasal air sinuses is a cause of delayed meningitis (p.147). In infants and young children, pulsating leptomeningeal herniations may actually expand a fracture line, constituting a growing fracture (p. 511). Growing fractures are usually seen in the parietal region, but have been reported in the frontal bone. Non-union of mandibular fractures, when not due to infection, may be due to poor blood supply, as in the edentulous mandible, or to failure in immobilization (Spiessl 1989).

Mal-union

This is basically the result of failures in reduction and fixation of fractures; these are discussed in Chapter 11.

Infection

This is no longer a common event in developed countries, but may always occur if wounds in the CMF area are neglected. Infected cranial fractures can be complicated by spreading osteomyelitis, and by infection in the extradural space; extradural abscess is potentially a serious condition, since it can lead to subdural

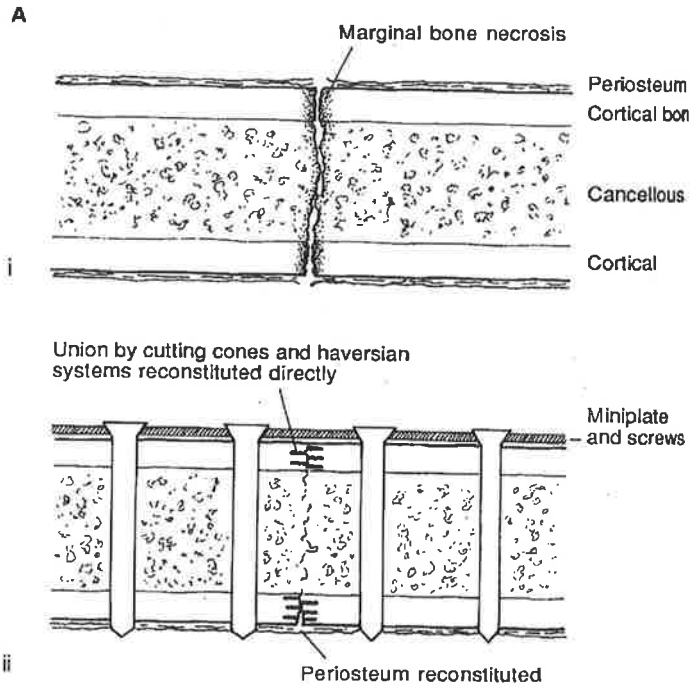


FIG. 5.7. ***Types of bone healing after fracture.*** **A.** *Contact healing:* **i.** *The bone ends are in contact. There is some bone necrosis at the margins, but little haematoma formation.* **ii.** *The fracture is immobilized by a plate. Osteoclasts ream out new haversian systems in the apposed bone surfaces, and bridges of new bone unite the fragments. The periosteum is reconstituted.*

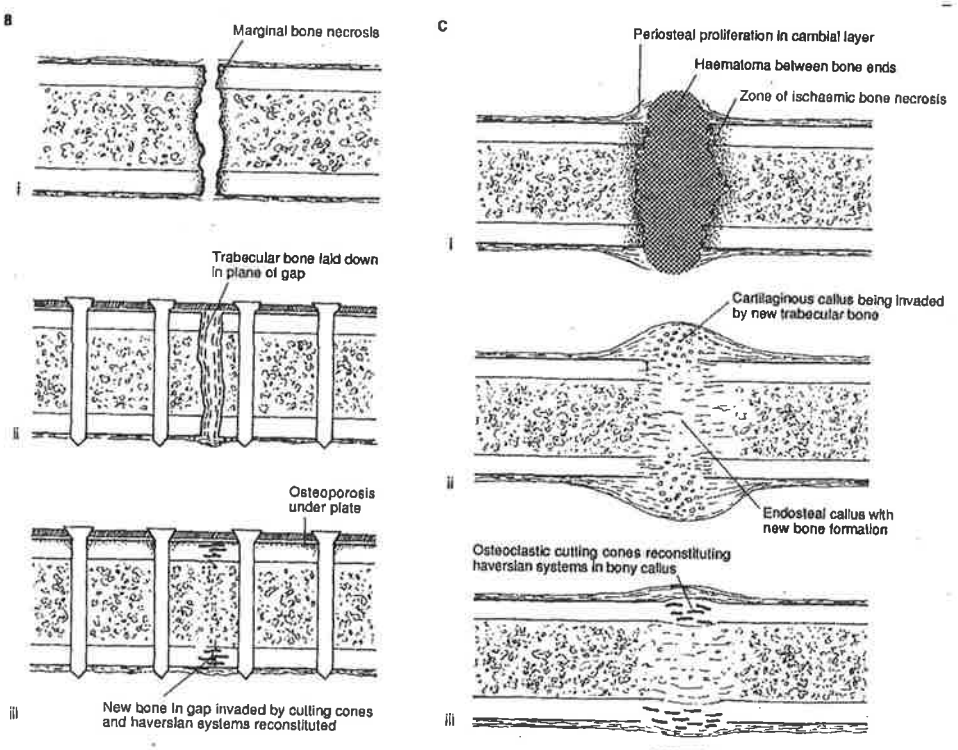


FIG. 5.7. ***Types of bone healing after fracture.*** **B.** *Gap healing:* **i.** *The bone edges are separated by a narrow gap.* **ii.** *The fracture is immobilised by a plate. The gap is filled by trabeculae of new bone, formed in osteogenic granulation tissue.* **iii.** *The new bone is remodelled into haversian systems in the cortex, and trabecular bone in the medulla or diploe.* **C.** *Healing by callus formation:* **i.** *The bone edges are separated by a haematoma. The fracture is not immobilized.* **ii.** *The haematoma is converted to granulation tissue; osteoid and cartilaginous callus forms, especially around the fracture site.* **iii.** *The callus ossifies and is remodelled into cortical and medullary bone.*

and/or cerebral suppuration (p.147). However, the intact cranial bone usually resists infection though ischaemic fragments of bone are likely to be sequestrated.

Osteomyelitis of the mandible is always a threat, since fractures of this bone are often compound into the mouth (Fig. 5.10). The older literature contains many references to such infections; Rowe & Killey (1955) identified dead teeth or tooth roots, foreign material and delayed treatment as aetiological factors. Haematoma formation has also been cited as a cause (Bochlogyros 1985). Devitalized bone is also likely to be infected; the rich blood supply of the mandible makes bone necrosis unlikely in civilian practice, though missile wounds with extensive tissue loss and devitalization may be complicated by sequestration of infected bone fragments.

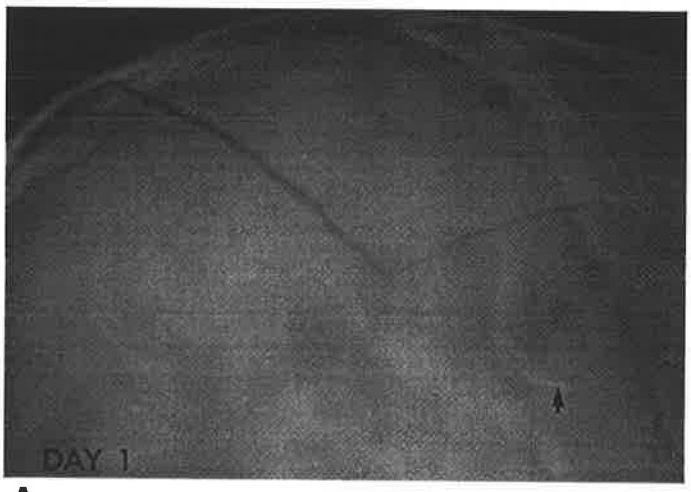
Bone grafts

Free grafts of autogenous bone are routinely used in the repair of facial fractures (p.331) and calvarial bone defects (p.548); vascularized bone transfers are increasingly used in the repair of gunshot wounds of the mandible when a large segment of tissue is lost (p. 626). The process of bone transplantation is therefore of fundamental importance in CMF trauma surgery.

We use grafts of iliac crest, rib and calvarial bone. These are as a rule composed of both cancellous and cortical bone. Cancellous bone is rapidly and reliably accepted and incorporated into the host bo' 'he process known as creeping substitution (Burchardt 1983). The transplanted bone has no blood supply, and most of its cellular elements die before the graft undergoes neovascularization, though some osteoblasts on the periphery of the graft may survive to join with cells from the host tissue — usually a fracture site or cranial defect — to form new bone. Granulation tissue from the host tissue forms around the graft, and mesenchymal cells in the granulation are induced to become the precursors of osteoblasts and osteoclasts. The process of bone induction is governed by growth factors derived from the grafted bone as well as from the host tissue; Urist et al (1983) have stressed the role of bone morphogenetic protein, which is present in demineralized bone matrix and able to induce bone and cartilage in ectopic sites. When cells with osteogenic capacity have been induced, the process known as osteoconduction begins. The marrow spaces of the transplanted cancellous bone are invaded by new blood vessels, and osteoblasts deposit appositional new bone on the trabeculae of dead graft bone. For a period, living and dead bone coexist, and the graft actually appears in radiographs to be denser than normal bone. (Fig. 5.8B shows this in a piece of replanted calvarial bone.) Later, osteoclasts remove the dead bone, and eventually the cancellous graft is entirely replaced by new bone. The process of bone conduction appears to be faster in young persons.

Cortical bone is often used to give structural strength or a smooth contour. The incorporation of cortical bone is slower. First, osteoclasts ream out the haversian canals of the graft, which are then revascularized; during this process, the graft loses density and strength. New bone is then laid down and the graft is partially replaced by creeping substitution; however, some of the dead cortical bone is incorporated and remains permanently in the reconstituted structure. Manchester (1972) showed that a large graft of cancellous and cortical iliac bone, used to replace an excised hemimandible, may survive and behave like living bone, healing when osteotomized and showing trabecular remodelling. He attributed the success of such large grafts to rapid revascularization in the absence of haematoma formation. This emphasises the importance of the state of the host bed in which the graft is placed. In the facial region, the soft tissues are normally very vascular' and in general bone grafts survive well if firmly fixed. This is not always so with bone grafts placed in calvarial defects, perhaps because the adjacent diploe is often poorly vascularized.

It is sometimes convenient to use autogenous bone preserved by freezing, as when a craniotomy flap has been removed because of brain swelling. In such



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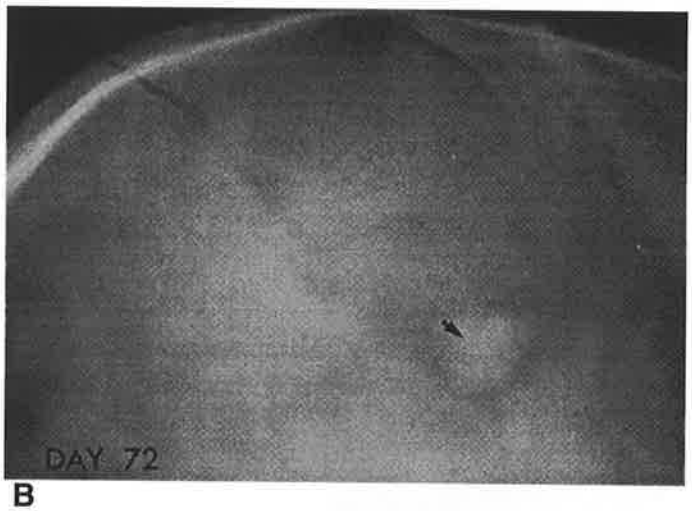

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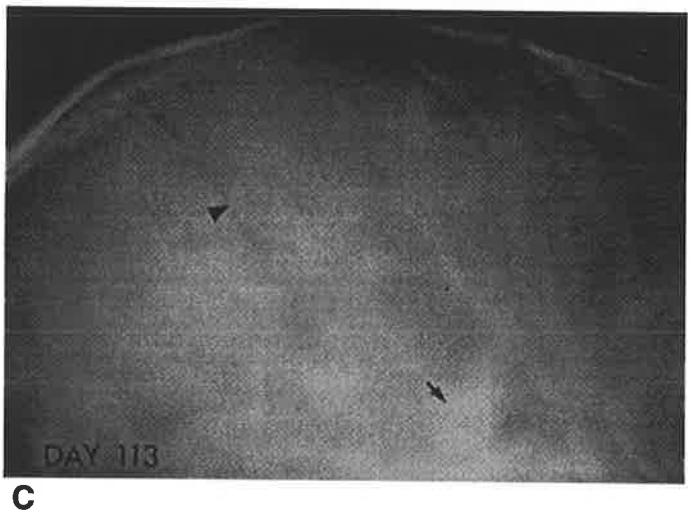

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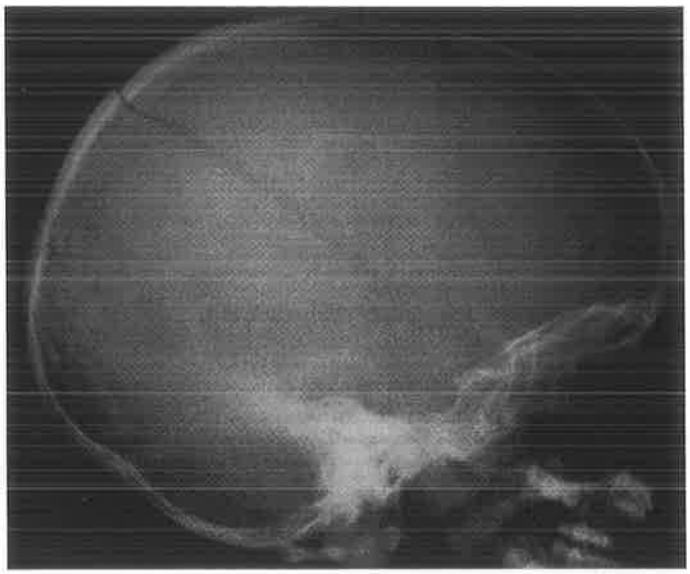
FIG. 5.8. ***Healing in a depressed calvarial fracture.*** *A child aged 8 was hit with a cricket bat; he sustained a depressed fracture in the parietal region, and a small extradural haematoma. After some delay, the fracture was elevated and the clot was evacuated. The bone fragments were replaced.* **A.** *Day I (before operation): the edge of a depressed fragment (arrow) is seen as a white line.* **B.** *Day 72: the depressed fragment (arrow) appears hyperdense, and is surrounded by hypodense bone.* **C.** *Day 113: The depressed fragment (arrow) is less dense and appears to be incorporated with the rest of the calvaria. The linear fracture (arrow head) is largely but not wholly united.*



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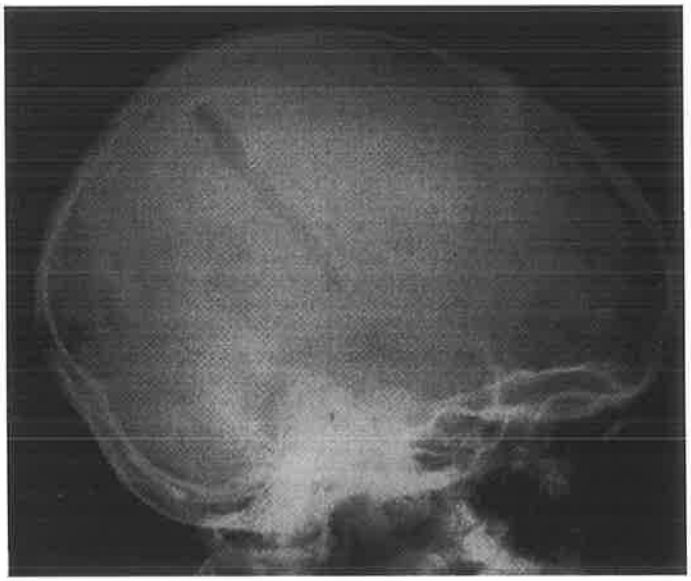


B

FIG. 5.9. ***Partial healing of a linear calvarial fracture.*** *Fracture of parietal bone in a boy injured at the age of 3 years.* **A.** *The fracture shows some separation of the edges soon after injury.* **B.** *At the age of 10 years, the fracture edges (arrow) show failure of healing and slightly increased separation. There was no palpable defect or pulsation. There was complete healing in the midline.*

grafts, the osteocytes are presumably dead, but osteo-inductive factors such as bone morphogenetic protein may survive. Frozen autografted bone is usually accepted, though we have seen resorption of such grafts under circumstances us which a fresh graft would probably have survived. At one time, we sterilized such preserved autografts by gamma irradiation: most of the grafts underwent resorption and this is in accord with the observation that bone morphogenetic protein and other bone growth factors are destroyed by irradiation (Marx & Stevens 1991).

When a bone graft is vascularized by microsurgical anastomosis, the transplanted bone survives unless the period of ischaemia is too long. Ostrup & Frederickson (1974), in their pioneering experimental study of mandibular reconstruction with a vascularized rib, found that many grafted osteocytes did die, presumably from ischaemia, but no massive bone necrosis took place and the graft formed abundant new bone. The general success of vascularized bone grafts confirms that the process of incorporation is more direct and more rapid than with free autogenous grafts.

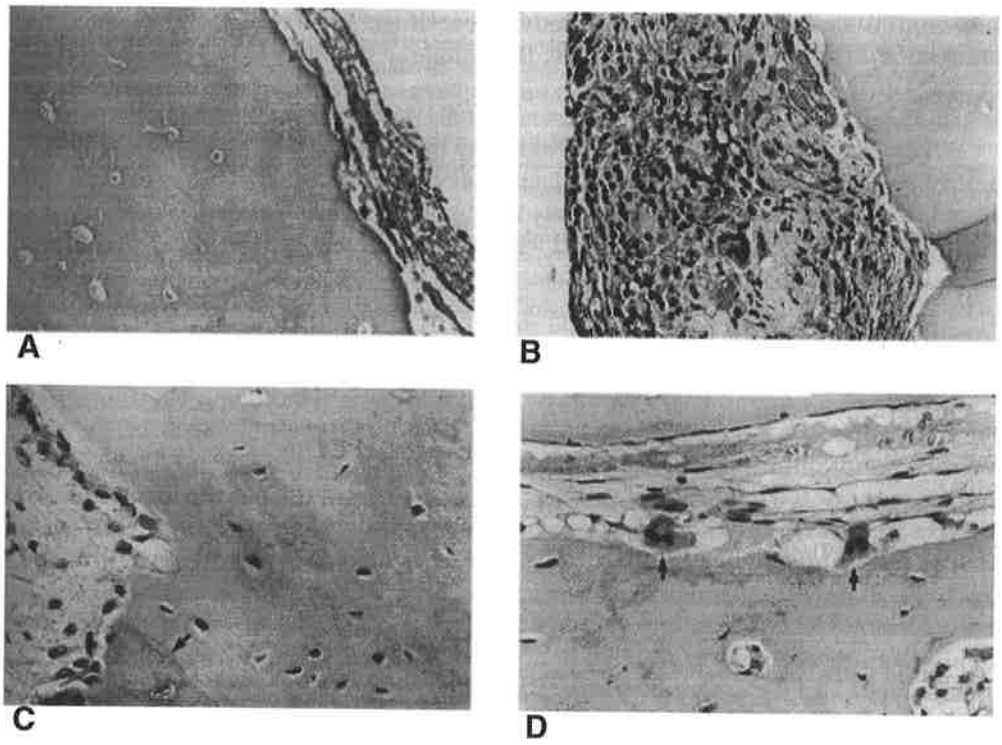


FIG. 5.10. ***Cellular activity in an infected mandibular osteotomy.*** *Biopsies show stages of bone necrosis and repair.* **A.** *Sequestrum of necrotic bone: empty lacunae containing no osteocytes; very little cellular activity on the surface of the bone. H&E, × 210.* **B.** *Chronic inflammatory granulomatous reaction H&E, × 210.* **C.** *Osteoblasts laying down new bone; arrow indicates older bone being remodelled. H&E, × 210.* **D.** *Two osteoclasts remodelling living bone. H&E, × 210.*

Since the supply of autogenous bone is limited, efforts have been made to promote bone union by cadaver bone grafts. These allografts are immunologically active and may be rejected, but the allogeneic bone can act as a resorbable supporting matrix, especially if used in conjunction with an agent able to induce new bone forrnation, such as autogenous bone or bone marrow. Marx & Stevens (1991) give an excellent account of the use of cadaver bone in conjunction with autogenous cancellous bone to repair mandibular defects, but we have not used this method. Nor have we used deproteinized animal bone for CMF injuries: the use of this material, in conjunction with autogenous marrow grafts, has been reviewed by Salama (1983). Oberg & Rosenquist (1994) have described the osteogenic effects of implants of allogeneic bone from which antigens had been extracted, combined with hydroxyapatite: exuberant bone overgrowth appeared, presumably from the action of bone morphogenetic protein. Preparations of purified bone morphogenetic protein are being used clinically to promote retention of surgical implants in the facial skeleton (Sailer & Kolb 1994).

Cartilage

Mature cartilage has very little reparative capacity and most wounds involving cartilage unite, if at all, by fibroblastic activity. On the other hand, cartilage is very tolerant of ischaemia. In the CMF region, several types of cartilage are found, and it is likely that their reparative capacities are somewhat different. Hyaline cartilage is found in the nose and nasal septum, and the external ear contains yellow elastic cartilage; incised wounds of these structures unite only by fibrous scar tissue. The articular surfaces of the temporomandibular joint (TMJ) and its disc are fibrocartilaginous; wounds of the joint may be filled with fibrous tissue from the joint capsule, and this may be transformed into cartilage to re-establish the original joint surface. Robinson (1993) has shown this in the marmoset: small lesions in the mandibular condylar cartilage were first filled with collagen fibres and then with normal-seeming cartilage. It appeared that the new chondrocytes were derived from precursor cells in the periosteum, rather

than from the adjacent uninjured condylar cartilage. This reparative capacity is not seen in other cartilaginous joints; it is unclear whether it is relevant in the recovery of the TMJ after trauma.

The cartilages of the nose and ear may be damaged in facial burns. There is only a thin layer of subdermal tissue between skin and cartilage, and the subcutaneous vasculature is intimately related to the perichondrial blood supply; thermal skin injury may be associated with necrosis of the cartilage and secondary deformity (p. 479).

Cartilage grafts

Free grafts of autogenous rib cartilage, or combined rib bone and cartilage, are used in repair of some CMF injuries, especially those involving the temporomandibular joint (p. 287). Cartilage, an avascular tissue, usually survives well, unless unable to obtain nutrition by diffusion from the surrounding tissues or exposed by skin breakdown or absence of the mucoperichondrium. However, sculpting cartilage to meet an aesthetic need may unbalance tensions within the graft, which may then warp; the literature on this is briefly reviewed by Wornom & Buchman (1992). Cartilage cadaver grafts have been used, but cartilage has some immunogenic capacity and it appears that cartilage allografts are less satisfactory than autogenous grafts (Burwell 1985).

Teeth

The teeth have limited reparative capacities, even in young persons, and certain types of localized injury may lead to destruction of the entire tooth and even to spread of infection to the underlying bone.

Dental fractures

These are common injuries, young permanent upper incisors being especially at risk; the fracture can involve the palatal, incisal or proximal surfaces of the tooth. Andreasen (1981) classified dental fractures on an anatomical basis:

1. Crown infraction: crack involving only enamel, with no loss of tooth substance.
2. Crown fracture:
   a. uncomplicated
      —involving enamel only (Fig. 5.11A)
      —involving enamel and dentine, but not the pulp (Fig. 5.11B)
   b. complicated—involving enamel, dentine and pulp (Fig. 5.11C)
3. Crown-root fracture:
   a. uncomplicated—involving enamel, dentine and cementum but not involving pulp
   b. complicated—involving enamel, dentine, cementum and pulp (Fig. 5.11D)
4. Root fractures—involving dentine, cementum and pulp (Fig. 5.11E)
5. Subluxation: tooth loose but not displaced
6. Luxation: tooth displaced into the alveolar bone (intrusive), or partially avulsed (extrusive), or displaced laterally
7. Exarticulation—tooth completely avulsed.

Fractures involving only the enamel present minimal risk to the pulp or periodontal tissues. When the fracture involves the dentinal layer, bacteria invade the dentinal tubules. The prognosis for the tooth then depends largely on the extent of the fracture: a fracture involving only a small exposure of dentin, such

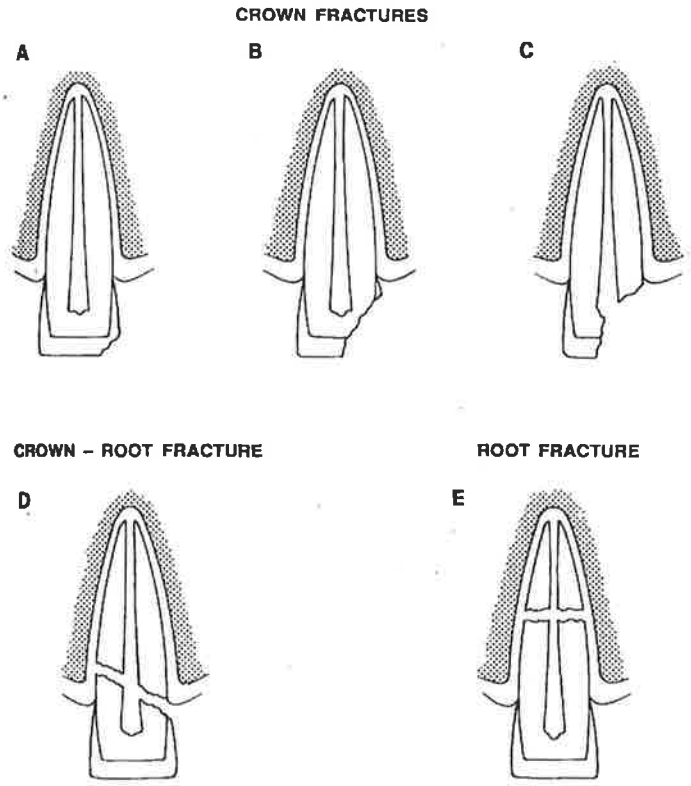


FIG. 5.11. ***Types of dental fractures.*** **A.** *Uncomplicated crown fracture, confined to the enamel.* **B.** *Uncomplicated crown fracture, involving enamel and dentin but not the pulp.* **C.** *Complicated crown fracture, involving the pulp.* **D.** *Complicated crown-root fracture, involving the pulp.* **E.** *Root fracture. Redrawn after Andreasen (1972).*

as a mesio- or disto-incisal edge fracture, is less likely to go on to necrosis than a fracture going deeply into the dentine.

After a fracture complicated by pulp exposure, blood clot, fibrin and inflammatory cells may be present on the pulpal surface (Fig. 5.12A). If treatment is delayed for a few days, the exposed pulp usually develops a hyperplastic mass of exudate rich in fibrin, cells and antibodies. This defence reaction may be followed by sealing of the exposed dentinal tubules by a calcific layer (Fig. 5.12B); secondary dentine may be formed in the pulp cavity, and the tooth may be saved. However, the formation of plaque on exposed dentinal tubules may lead to diffusion of microorganisms through these tubules into the pulpal tissue a deeper inflammatory response is now established under the surface of the fracture, which can spread to the periapical tissues and beyond as osteomyelitis of the jaw. The dental defence mechanism can be frustrated if there is excessive bleeding, separation of the pulp tissue from the pulp wall at the moment of fracture, and/or fluid build-up under pressure in the pulp chamber.

Pulp injuries

The pulp occupies the central cavity of the tooth, and is enclosed by dentine. Although the cells lining this cavity can be regarded as mesenchymal connective tissue, they carry out a number of vital functions. During the developmental period, the pulp mesenchyme has cells (odontoblasts) which are capable of producing dentine. In the adult tooth, these cells continue to produce physiological dentine, and if the tooth is traumatised, a reparative process begins, whereby secondary dentin is laid down under the site of the assault. This may go on to obliterate the pulp chamber.

The dental pulp responds to injury by manifesting all the classical signs of inflammation: dilation of blood vessels, transudation of serum and extravascular migration of leukocytes within the pulp chamber. The presence of extravascular

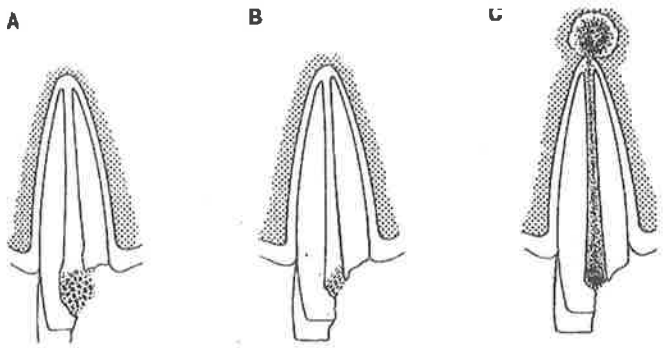


FIG. 5.12. *Complications of dental fractures.* **A.** *Granuloma formed in pulp cavity.* **B.** *Calcific budge closing the fracture and sealing the pulp cavity* **C.** *Bacterial infection in the pulp cavity, giving rise to an apical abscess. Modified after Andreasen (1972).*

exudates in the confined space of the rigid pulp chamber results in increased pressure and pain from stimulation of pulp nerves and nerve endings. If the process is mild in nature and of short duration, the pulp tissue will usually recover; in severe cases, the pulp may undergo necrosis and the tooth may be lost.

Bacterial invasion follows traumatic exposure of the pulp. The pulp tissue reacts in a protective manner, but the resulting inflammatory processes may continue and lead to pulp necrosis and apical abscess formation (Fig. 5.12C). Young teeth have a large pulp chamber which has considerable capacity to survive injury. However, it is important in dealing with injuries of young teeth having incomplete root development to ensure that pulp vitality is protected. The treatment of pulp exposure is discussed on p. 349.

Dental and alveolar resorption

Severely traumatised teeth may occasionally undergo resorption (Fig. 5.13). This peculiar dystrophy of the pulp tissue results in massive destruction of the dental hard tissues. The process begins in the pulp and spreads laterally through the dentine; in all probability, the internal resorption of the dentine, as in other hard tissue resorptions, is the work of macrophages and multinucleate giant cells, indistinguishable from osteoclasts (Fig. 5.10D). The lost dentine is replaced by chronic inflammatory tissue. The process may or may not result in loosening and loss of the affected tooth; appropriate endodontal therapy may prevent this, though in the case of Fig. 5.13 this treatment was unsuccessful.

In adults, loss of teeth by trauma or extraction results in resorption of the dental alveolus. This is especially important in the anterior maxilla following the loss of one or more teeth. This bone resorption can jeopardize aesthetic tooth replacement by fixed bridgework, and may also prevent the insertion of dental titanium implants to hold replacement tooth crowns. Bone grafting may be necessary to augment the alveolus before placing an implant (p. 637).

Glandular Tissues

Salivary glands

These glands are often contused or lacerated; the parotid gland is especially vulnerable. The chief complications of parotid injury are pseudocyst formation and salivary fistula. It appears that permanent obstruction of the parotid duct leads to atrophy of the secretory cells (Landau & Stewart 1985). Harrison & Garrett (1976) showed this in the cat: parotid duct ligation caused severe progressive glandular atrophy, but ligation of the submandibular and sublingual ducts caused less striking changes. Parasympathetic denervation also induces some degree of glandular atrophy.

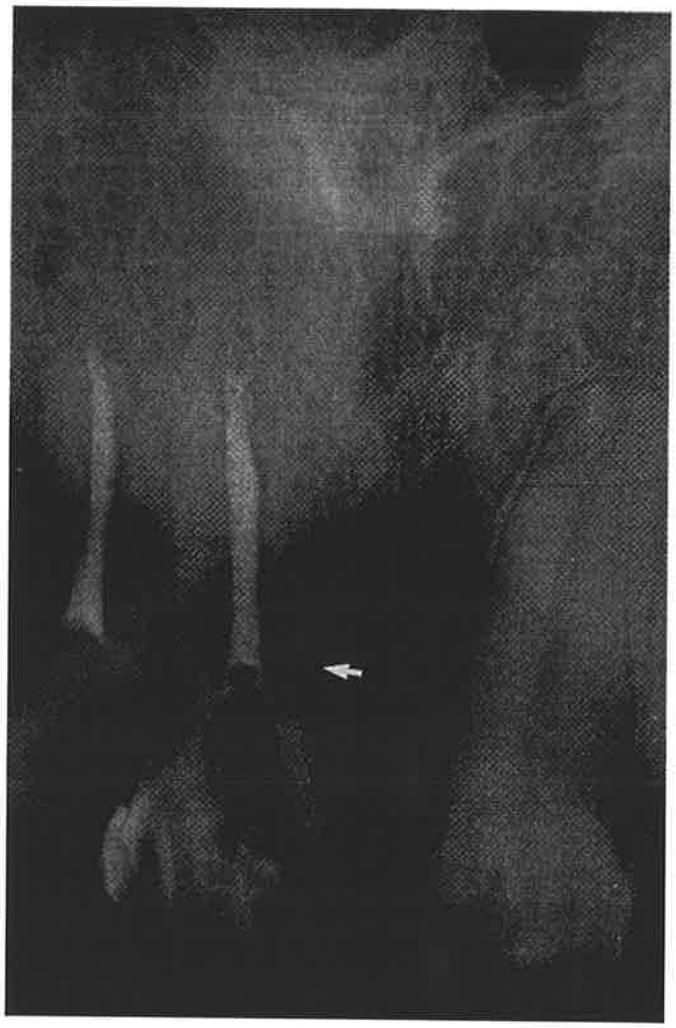


FIG. 5.13. ***Severe resorption following endodontic treatment of fractured teeth.*** *Radiograph showing maxillary incisors and canine teeth in an adult, some time after crown fractures. In the two incisor teeth, the fractures had opened the pulp; gutta percha was inserted in the hope of saving the tooth, but dramatic resorptive changes appeared in the mid root regions (white arrow) and in the alveolar bone. In the canine fracture, the pulp was not opened.*

Neural Tissues

Cranial nerves

First cranial nerve

The olfactory system is complex (p. 57). The olfactory tract and bulb are part of the central nervous system; they are often contused or lacerated in association with frontal lobe damage and have no capacity to regenerate. The olfactory filaments are peripheral nerves and are said to have some capacity to regenerate, at least in the monkey (Monti-Graziadei et al 1980); this may explain the rare but well-documented recovery of olfaction after closed head injury.

Second cranial nerve

The optic nerve is a component of the central nervous system and has no capacity to regenerate. The optic nerve and chiasm are often damaged in cases of CMF trauma; they may be lacerated, contused or rendered ischaemic. The end result of such injuries is optic atrophy and demyelination. The pathology of optic nerve trauma has been well reviewed by Walsh & Hoyt (1982-1991) and by Kline et al (1984) but is still controversial. Several surgeons have ventured to biopsy damaged nerves soon after loss of sight, and the appearances have been variously

TABLE 5.1

Classification of nerve injuries (after Seddon 1972)

Characteristics	Neurotmesis	Axonotmesis	Neurapraxia
Pathology	Continuity lost in all elements	Epi- and perineurium and Schwann preserved; axons interrupted	Nerve in continuity; selective focal demyelination of larger axons
Clicincal	Complete paralysis	Complete paralysis	Motor paralysis; often sensory sparing
Treatment	Nerve suture	Expectant	Expectant
Recovery	1-2 mm/day; likely to be imperfect	1-2 mm/day, usually perfect	Perfect

interpreted as indicative of ischaemic necrosis due to damage to the pial vascular supply of the nerve, or to axonal shear damage. Crompton (1970) found evidence of one or both processes in 37 of 84 autopsied cases, but concluded that it was impossible to distinguish between them in histological material. The clinical implications of this uncertainty are discussed in Chapter 14 (p. 420).

Nerve regeneration

The remaining cranial nerves are often injured in association with CMF trauma; they are peripheral nerves with Schwann cell sheaths, and they are capable of regeneration. Seddon (1972) and Sunderland (1978) devised classifications of peripheral nerve injury, using terms which express the loss of continuity of the components of the nerve. Table 5.1 gives the classification and nomenclature popularised by Seddon, though he did not coin the elegant names neurotmesis, axonotrnesis and neurapraxia . If the axon is transected, as in both neurotrnesis and axonotrnesis, then it degenerates distally in its whole course: the classical Wallerian degeneration. The proximal axon degenerates to the first node of Ranvier, and sometimes further if there has been severe trauma, as in a gunshot wound. The cell body of the parent neuron shows loss of Nissel substance, but usually survives and synthesizes the proteins necessary for axonal regeneration. The distal axon shows swelling and clumping of mitochondria; the axoplasmic reticulum breaks up and the neurofilaments fragment. The myelin sheath also breaks down and the degenerating fragments are removed by macrophages. The Schwann cells along the course of the degenerating axon swell and undergo mitotic division under the stimulus of a mitogen or mitogens produced by the degenerating axon or its sheath.

The Schwann cells form tubes and cords extending distally from the point of nerve section. It appears that these cells form or attract neuronotrophic substances, of which nerve growth factor (NGF) is the best studied (Taniuchi et al 1988). Under the stimulus of such chemo-attractive agents, axons sprout from the proximal stump of the nerve and form multiple small unmyelinated fibres, one of which — all being well — finds a Schwann cell tube, enters it and grows toward the target structure at a rate usually estimated as 1 mm/day. Eventually, the target structure (muscle fibre, gland or sensory terminal) is reinnervated and function is restored.

The success of this remarkable process depends on the nature of the injury: in axonotmesis, as in a crushed nerve, full regeneration is often seen, whereas in neurotrnesis recovery will be prevented if the severed ends are not apposed or if there is local sepsis. Age, the nature of the nerve and other factors also influence the degree of recovery. Not rarely, regenerating axons take aberrant paths, and reach inappropriate muscle fibres or secretory cells.

In facial injuries, the nerves likely to be injured are the facial nerve, a predominantly motor nerve, and the trigeminal nerve which is for practical purposes a sensory nerve, since its motor component is deeply placed and only

injured by penetrating trauma. The third, fourth and sixth cranial nerves are often injured in closed head injuries.

The facial nerve may be divided or less often crushed at any point in its course. It has a remarkable capacity to recover, if the ends are in continuity. But the complex anatomy of the facial nerve (p. 62) makes aberrant regeneration a common sequel of CMF injuries involving that nerve; mass facial muscular action, gustatory sweating (Frey syndrome) and lacrimation (crocodile tears) may be disfiguring consequences (p. 441). Misdirected axonal regeneration may also follow injury of the oculomotor nerve (p. 405).

The branches of the trigeminal nerve are likely to be injured when traversing bony canals or grooves: damage to the supraorbital, infraorbital and inferior dental nerves are commonly associated with facial fractures of various types. The trigeminal ganglion and its main branches are sometimes injured in skull base fractures. McGovern et al (1986) have reported a case of trigemino-abducens synkinesis after a severe CMF injury, which appeared to represent aberrant regeneration of trigeminal motor axons into the proprioceptive innervation of the abducent nerve (P. 415)

Sensory nerve recovery is often less than perfect, but may be masked by lateral ingrowth by the terminals of intact nerves supplying the neighbourhood of the denervated area. If nerve regeneration is not achieved, there will be a neuroma at the point of section, composed of axon end bulbs and proliferating Schwann cells and fibroblasts; these neuromas may be painful, but their importance as a cause of chronic post-traumatic facial pain is in doubt (p. 660).

Nerve grafts

Autogenous nerve grafts are used to repair gaps in the inferior dental nerve, usually iatrogenic, and in facial nerve repair. The grafted nerve provides tubes of living Schwann cells to guide the regenerating axons to the distal nerve trunk. To survive, the graft must be revascularized from the proximal and distal nerve stumps and from the host bed within a few days: if this does not happen, the graft undergoes ischaemic necrosis. The facial tissues are very vascular, and with good microsurgical technique excellent results are usually obtained with free grafts. However, for the long transfacial anastomoses used to reinnervate the facial nerve from the opposite side, a microvascularized graft from the saphenous nerve has been used (Karcher et al 1989) and increasing use is being made of this method of promoting regeneration.

There is an extensive literature on nerve allografts, but at present these are not recommended.

Meninges

The dura mater is of great importance in cranial trauma. The outer layer is osteogenic and participates in the repair of fractures of the calvaria and skull base; in infants and young children, large calvarial bone defects may close spontaneously if the dura is intact. There is some debate on the capacity of older subjects to achieve this spontaneous regeneration. Kent & Misiek (1991) give 6 years as the upper age limit; we have seen substantial reossification in adolescents, especially when the pericranium is intact, but agree that this is not always evident.

Haemorrhages from arteries and veins in the outer layer of the dura can give rise to extradural haematomas (see below); if these do not cause serious cerebral compression, they are invaded by fibrovascular tissue and absorbed, or less often encapsulated by fibrous tissue and even bone from the dura mater. The inner layer of the dura (p. 36) is also capable of considerable fibrovascular activity in response to subdural bleeding, forming the inner and outer membranes seen in chronic subdural haematomas (p. 524).

The dura is a valuable barrier against infection; tears or penetrating dural wounds usually require surgical closure to reconstitute the barrier. Incised wounds of the dura mater heal well if the edges are coapted. However, dural healing may be deficient if leptomeninges and brain tissue are interposed between the edges of the dural tear (see below), or if the tear is in proximity to a cerebrospinal fluid cistern. Dural defects are closed by a neomembrane formed from the adjacent soft tissues rather than from the edges of the torn dura, and this may constitute a false meningocele. The arachnoid and pia mater are almost always torn in penetrating wounds of the dura, and participate with it in forming a fibroglial meningocerebral scar.

Dural grafts

Autogenous fascia or pericranium is often used to close dural defects; in a few months the graft usually appears to have fused with the rest of the dura. Allografts of freeze-dried (Iyophilized) cadaver dura mater have been used for some 40 years (Campbell et al 1958), and with considerable success; the process of lyophilization eliminates the immunogenicity of the transplant. The transplanted collagen hyalinizes and is slowly removed and replaced by new collagen; after a few months the graft appears incorporated. However, Brown et al (1992) have reported seven cases of progressive dementia and death from Creutzfeld-Jakob disease after implants of this allograft, and we have ceased to use it. Xenografts of freeze-dried porcine dermis have been used to repair dural defects (O'Neill and Booth, 1984), and it appears that the grafted collagen was not rejected.

Brain

It is usual to distinguish between closed and open head injuries, and this distinction is especially relevant in the neuropathology of impacts in the CMF region. In closed head injuries, primary brain damage is inflicted by acute tissue deformation due either to acceleration imparted to the whole head (Fig. 4.8), or to force acting locally at the impact site (Fig. 4.1): the resulting brain lesions may be diffuse or focal. In open head injuries, localized primary brain damage may be inflicted by something that penetrates the protective capsule of the brain, the skull and meninges (Fig.4.7). Both types of injury may be complicated by secondary pathological processes, such as cerebral compression or anoxia; in open head injuries, there is the additional risk of microbial infection. In the CMF region, open brain injuries are common and often serious; they may involve the paranasal air sinuses, and may give rise to cranionasal fistulas (p. 376).

Closed brain injuries

The primary effects of blunt head impact have been well reviewed by Adams (1992). Focal effects include contusions and lacerations. Frontal impacts commonly cause haemorrhagic contusions in the frontal lobes, especially in the polar and orbital cortex; the temporal poles may also be injured. Thus, CMF injuries are likely to be complicated by frontotemporal brain damage; contrecoup occipital damage is very unusual except after impacts incompatible with survival. The probable mechanisms have been discussed (p. 106).

Contusions

Small contusions are typically collections of red blood cells, often grouped around a small vessel running at right angles to the surface of the cortex. Larger haemorrhagic contusions may extend into the white matter, and subarachnoid bleeding is also often seen. The red cells remain intact for a day or more and then show lysis; there is a macrophage reaction and deposition of bilirubin. There is neuronal death in the contused area and eventually replacement by astrocytic gliosis, often stained yellow by the bilirubin. The site of an old contusion is evident as an area of atrophy, typically at the convexity of a convolution and stained yellow or brown.

The sequential changes in a cerebral contusion are mirrored in radiological images. In computerized tomograms (CT), the haemorrhagic components of the

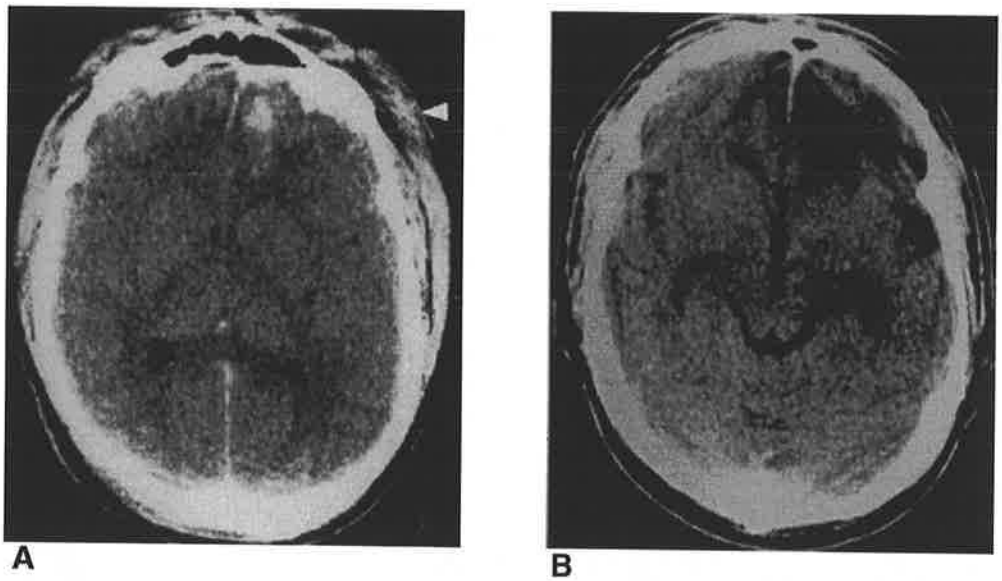


FIG. 5.14. *Contusional brain injury from frontal impact.* *A 26-year-old man fell from a slowly moving car and struck his head in the left frontal region. He sustained a left-sided linear skull fracture and was drowsy.* **A.** *CT 24 h after injury showed multiple hyperdense lesions in the left frontal lobe, presumably haemorrhagic contusions. Scalp swelling was evident at the site of impact (white arrowhead).* **B.** *CT 6 weeks later showed extensive bilateral low density lesions in the frontal white matter; these presumably represent axonal destruction and gliosis. There were permanent changes in personality.*

contusion are obvious as hyperdense masses ((Fig. 5.14A), while the associated oedema and later gliosis appear as hypodense areas (Fig. 5.14B). Magnetic resonance images (MRI) also visualize contusions well, and the progressive degradation of haemoglobin gives changing appearances that relate to the age of the lesion (Table 7.5).

Lacerations

These are areas of disruption of brain and meninges; they are seen specially in association with depressed skull fractures, but may also be caused by violent movement of the brain. The junction of the pons and the medulla is often the site of a laceration, or even a complete pontomedullary transection; this injury sometimes follows high velocity impacts in the CMF region (Simpson et al 1989) and usually results in instant death, though smaller lesions are compatible with prolonged survival (P. 212).

Diffuse brain injury

The diffuse effects of blunt head impact include damage to axons, dendrites vessels and glia. Widespread axonal damage presumably from shearing stresses, is termed diffuse axonal injury (DAI). The axonal lesions are often multifocal rather than diffuse, and there are sites of predilection, notably the corpus callosum and the superior cerebellar peduncle (Fig. 5.15). Similar impacts may cause multifocal or diffuse vascular injury (DVI), and again there are sites of predilection: these include the corpus callosum and the superior cerebellar peduncle, but also the parasagittal cortex and the basal ganglia. It is not yet known whether the distribution of DAI lesions is related to the site of the causative impact; however, it is clear that DAI is often seen after impacts in the CMF region. In life, DAI can be inferred on clinical grounds, but cannot be verified radiologically, whereas the larger (5-10mm) lesions of DVI can usually be seen in CT or MRI scans. It is often assumed that DVI ('tissue tear haemorrhage') is a marker of DAI, and that nonhaemorrhagic lesions seen in MRI scans are areas of DAI (p. 210). These assumptions are often correct, but the correlations between radiological pathology and histopathology need fuller documentation. Damage to glial cells may accompany DAI (Blumbergs & Wainright 1988); the clinical significance of diffuse glial injury remains to be explored.

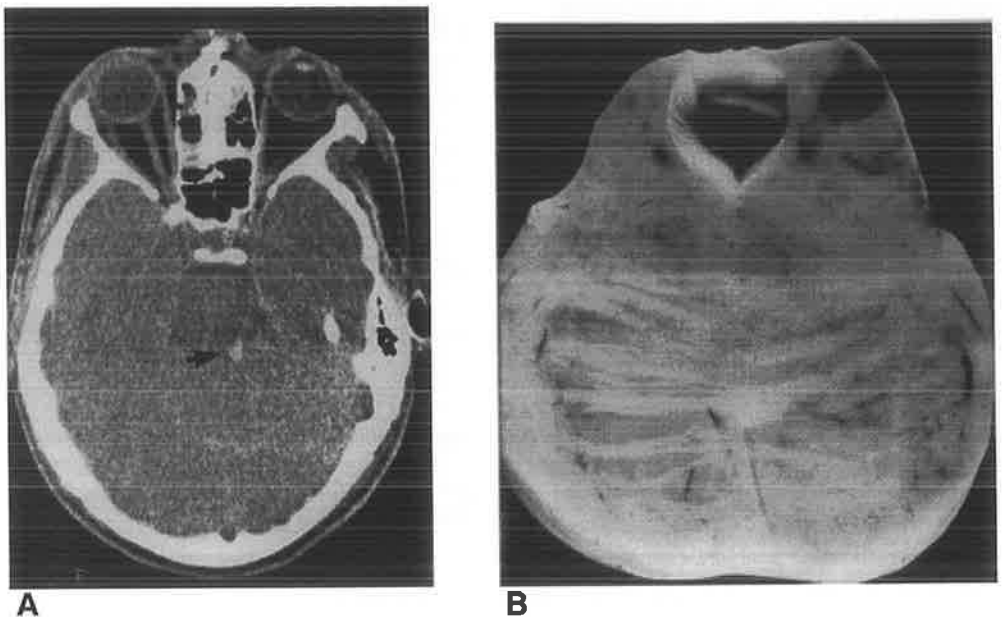


FIG. 5.15. ***Vulnerable sites: the midbrain.*** *The attachment of the superior cerebellar peduncle to the lower midbrain appears to be a vulnerable site in diffuse brain injury: both vascular and axonal damage are often seen in this area.* **A.** *CT in young adult hurt in motor cycle accident: the hyperdense lesion (arrow) is presumably a haemorrhage. Note the absence of the perimesencephalic cisterns: a sign of raised intracranial pressure, though it may also result from subarachnoid bleeding.* **B.** *Similar lesion in autopsied case.*

Minor head injuries

Clinicopathological correlation is more difficult in cases of closed head injury caused by less severe impacts since autopsy evidence is rarely available and radiological evidence of damage may be absent. The pathology of minor head injuries is still largely unknown, but minimal lesions of DAI type are demonstrable (Oppenheimer 1968), and the upper brainstem is the probable site in cases of transient loss of consciousness ('concussion'). P. Blumbergs (personal communication) has also noted a high incidence of lesions in the fornices after minor head injury.

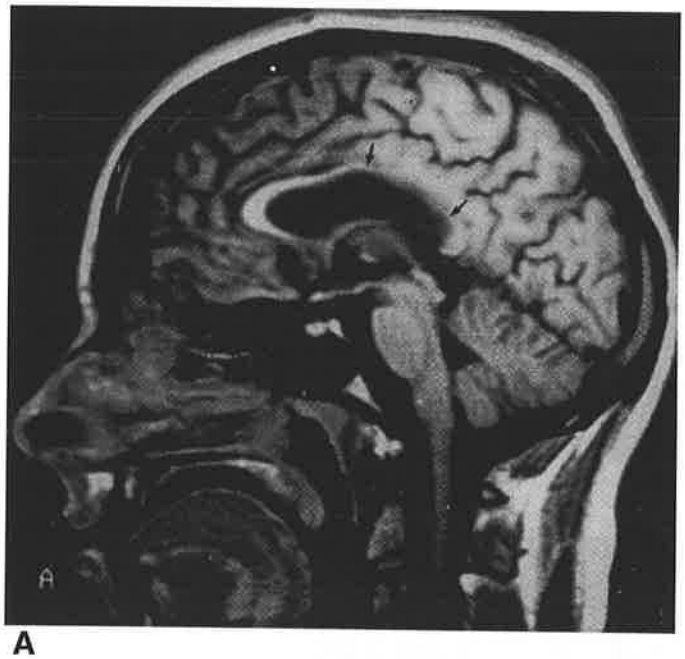
Open brain injuries

These are characterised by focal penetration of the meninges and brain, with variable remote damage. At the point of focal penetration, there is a cerebral laceration, usually with some haemorrhage.

A distinction is made between brain wounds made by depressed skull fractures, penetrating brain wounds and perforating ('through-and-through') brain wounds. This distinction expresses the size of the impacting object and its kinetic energy. A large, relatively blunt object will depress a large area of bone (Fig. 5.5B), and the underlying brain will be contused and often lacerated, but unless the impact force is very great, damage will be confined to the impact site. Smaller objects with high kinetic energy will penetrate the bone (Fig. 5.5C), and if the kinetic energy is not attenuated at the initial impact, there is likely to be deep penetration and widespread brain damage.

Missile brain wounds

If the penetrating agent is a bullet or shell fragment, there will be a track of brain destruction; if the missile has very high energy, there will be extensive local cavitation causing deformation and bleeding wide of the missile track (Fig. 5.17A). High energy missiles may also cause contusional damage in remote parts of the brain and even fractures of the skull base by transmitted force; this is seen especially in the thin bones of the orbital roofs (Strich 1976, Allen et al 1982). The autopsy on President Lincoln (p. 17) showed shattered orbits, supposedly



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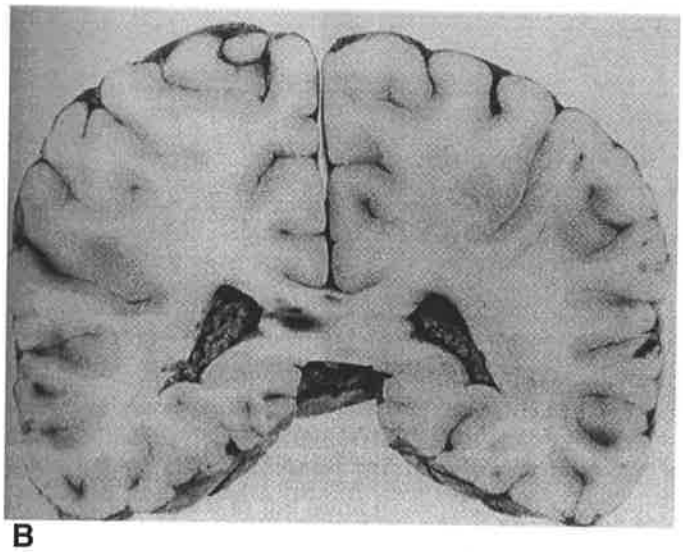
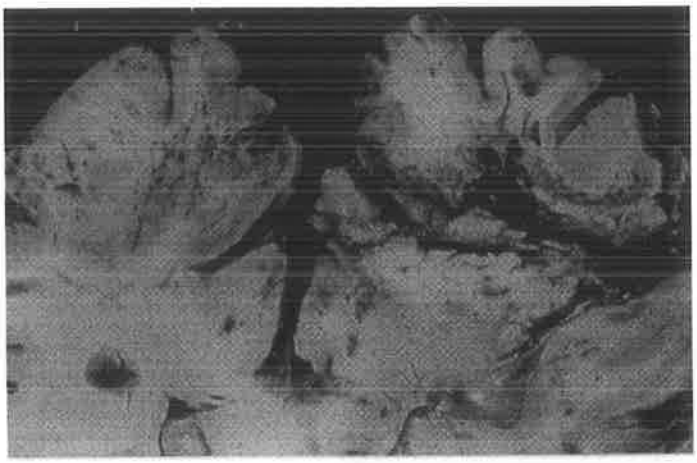


B

FIG 5.16. *Vulnerable sites: the corpus callosum.* *The corpus callosum may be injured in closed head injuries; haemorrhagic lesions and diffuse axonal damage are often seen.* **A.** *MRI 8 years after a severe closed head injury with residual intellectual and behavioural disabilities. Between the arrows, the body of the corpus callosum is barely visible; in coronal views, it was seen as a thin band.* **B.** *Haemorrhagic lesion in splenium of corpus callosum in a 12-year-old boy hit by a car; it is likely that this was a survivable head injury, death being due to an intra-abdominal haemorrhage.*

from this mechanism. The initial effects of mechanical disruption are increased by haemorrhage, oedema and loss of vascular autoregulation, combining to produce raised intracranial pressure (ICP) with all its consequences.

For surgical purposes, craniocerebral missile wounds are categorized as penetrating, perforating and tangential. In a penetrating wound, the missile penetrates the scalp, the skull and the dura, but does not leave the head (Fig. 5.18A): there is no exit wound. In a perforating wound, the missile traverses the brain ('through and through') and has both entry and exit wounds (Fig. 6.2). A missile wound is described as tangential when it grazes the skull but does not penetrate it; the skull may or may not be fractured, and the brain may be damaged by transmitted energy (Fig. 5.17B). Missile wounds may be complicated by intracerebral or subdural haemorrhage, sometimes of delayed onset; Hadas et al (1990) have reported cortical laceration and acute subdural bleeding after a tangential pistol wound without breach of the calvarial bone.



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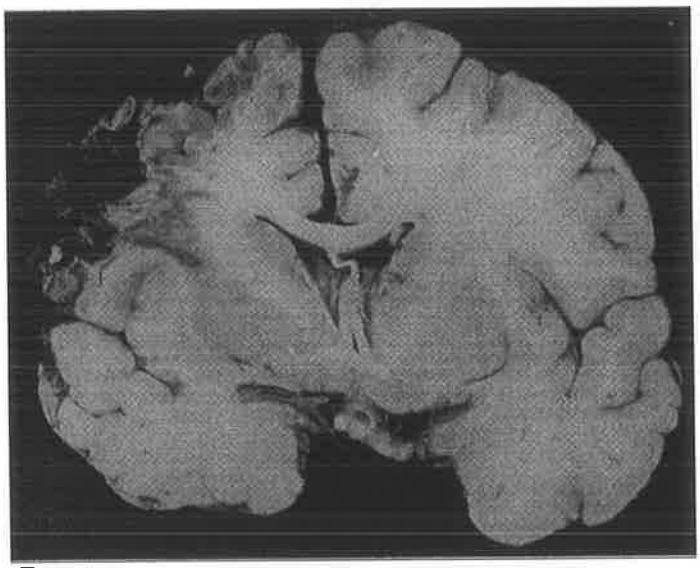


B

FIG. 5.17 ***Missile wounds of the brain.*** **A.** *Perforating frontal suicidal wound with widespread haemorrhages: the bullet traversed both frontal lobes. Many satellite haemorrhages are seen; these are not directly in the track of the bullet, and result from acute deformation.* **B.** *Tangential wound, with contusion of the cortex; bone chips were driven in from the calvaria.*

Metallic fragments and even entire bullets may remain in the brain. Lead and iron fragments are usually fixed in a chronic fibroglial scar (Fig. 5.18B,C); missiles containing copper are more toxic and evoke a local inflammatory reaction (see below). Large bullets may remain mobile in a cyst or in the ventricular system, changing position according to the posture of the head (Fig. 5.19).

Histopathology of brain injury

Whatever the cause of injury, the microscopic appearances are dominated by neuronal death and its consequences. Neurons in the central nervous system have very little reparative capacity. If an axon is severed or a neuronal cell body ruptured, then the entire neuron dies; there may even be trans-synaptic degeneration of other neurons deprived of functional stimulation. The axon undergoes degeneration in its whole length, as do the dendrites. The synaptic terminals swell and become argyrophilic. The axis cylinder becomes fragmented, and light microscopy shows globular and fusiform masses which stain pink with eosin and black with reduced silver stains (Fig. 5.20A); electron microscopy shows that these masses are electron-dense and composed of degenerating neurofibrils, mitochondria and other organelles. These axonal changes are seen by light microscopy 3 or 4 days after injury or even earlier; recent neuropathological studies in our laboratory (P.Blumbergs, personal communication) have shown

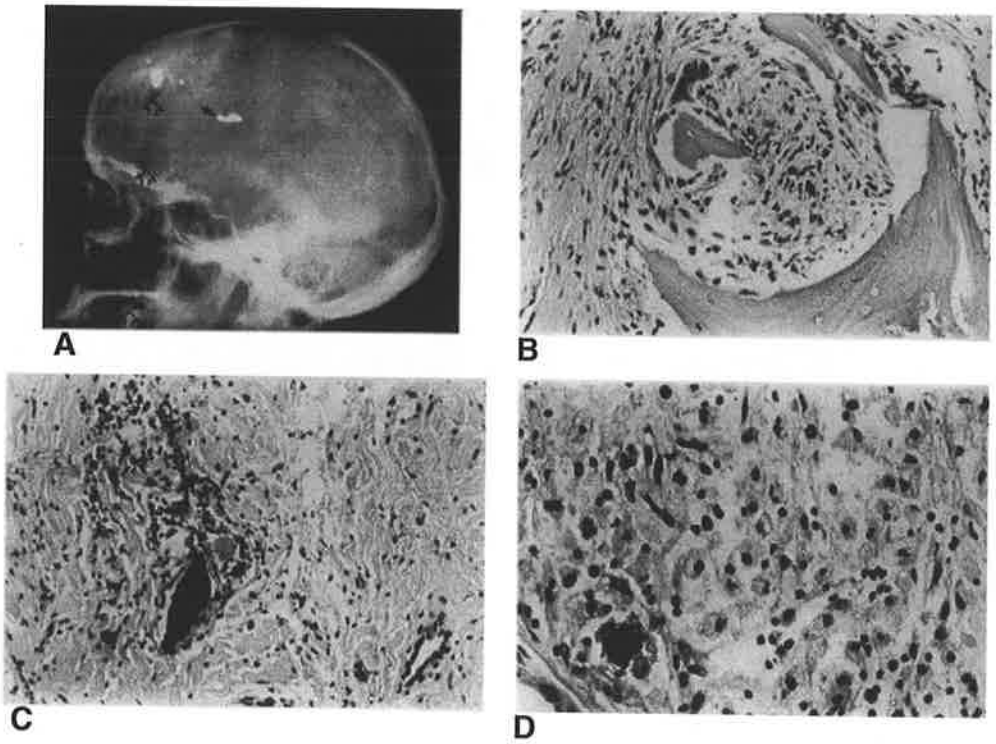


FIG. 5.18. ***Penetrating brain wound: histology of a meningocerebral scar.*** *A 39-year-old man fired a .22 in lead bullet through his chin into his brain. He survived. The missile track was explored (probably unnecessarily) 5 weeks after wounding.* **A.** *Plain skull radiograph showing missile track (open arrows) in skull, with large fragment of lead bullet lying subcutaneously. The deeper fragment (solid arrow) was embedded in the vicinity of the falx.* **B.** *Biopsy of scar tissue around the deep fragment: a bone chip with cellular reaction, including a giant cell. H&E, × 171.* **C.** *Biopsy in same area: black material, probably carbon from propellant. H&E, × 171.* **D.** *Biopsy of cerebral tissue near the deep fragment: foamy macrophages and a fragment of black material. H&E, × 342.*

that immunochemical stains for Alzheimer precursor protein (APP) show swollen axons at an earlier stage after injury than the silver stains. These may represent interference in fast axoplasmic transport. Electron microscopy (Povlishock et al 1989) has shown in experimental studies that the axon may be intact in the first few hours after injury, but later swells and fragments. (This finding raises hopes that early use of neuron protective drugs may preserve axons). Concurrently, the neuronal body loses its Nissl substance and the cytoplasm becomes homogeneously eosinophilic (Duchen, 1992). The myelin sheath at first looks unaffected, but after several weeks, it begins to fragment, at first near the point of injury and along the whole length of the fibre. The myelin changes its chemical structure and becomes stainable by the Marchi stain; later there is a further breakdown to simple lipids which can be stained by ordinary fat stains. Eventually the entire neuron usually disappears. This process of relentless degeneration is seen in the brain and also in the optic nerve and olfactory tract; it underlies the permanent neurological and opthalmological disabilities so often seen after CMF trauma. But some qualifications can be made. A neuron may survive a distal axonal injury if its functional activity is maintained through a collateral branch proximal to the point of injury A neuron deprived of its input will survive if its functional activity is maintained by other inputs; it may even obtain more inputs by the sprouting of new synapses from intact neurons, though the significance of this process in clinical recovery is still uncertain.

The inflammatory response to brain injury is very different from what is seen in other tissues. Polymorphs are seen in penetrating wounds but are inconspicuous in closed brain injuries. Macrophages appear in large numbers when there is necrotic neural tissue (Fig. 5.18D). Some of these macrophages are circulating monocytes from the circulating blood (Imamoto & Leblond 1977), and some are thought to be mobilized microglial cells, though the evidence for this metamorphosis is questionable. These cells phagocytose the products of

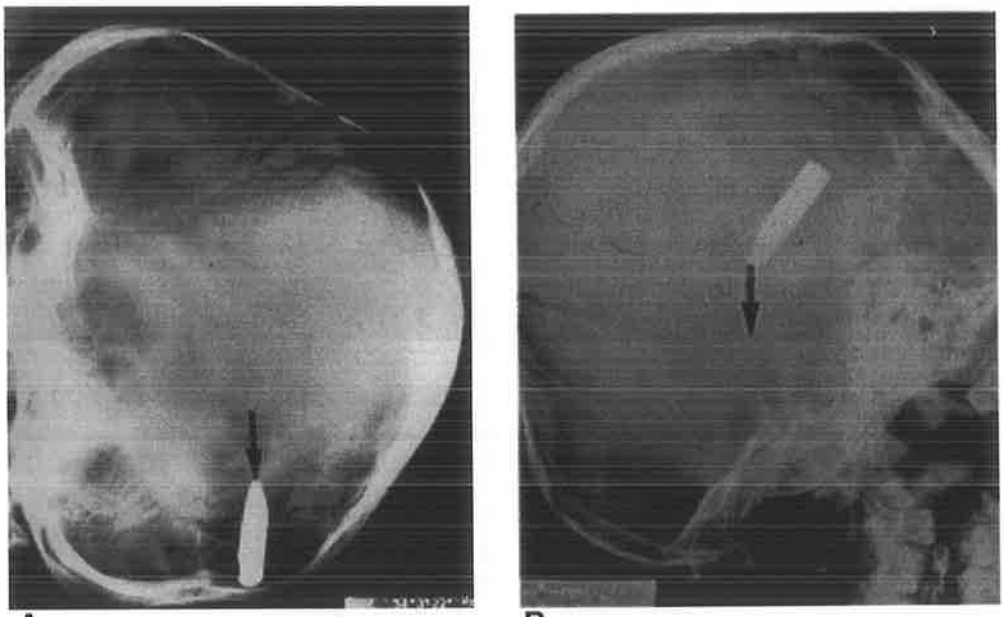


FIG. 5.19. ***Migrating bullet.*** *Military rifle bullet in the brain of a young child, hit at long range by a random shot in the occipital region.* **A.** *In the brow up position, the bullet lies near the point of entry.* **B.** *In the brow down position, the bullet has moved forward into the lateral ventricle. Arrows point down.*

degenerating myelin and other debris and soon become stuffed with lipid material. Somewhat later, astrocytes respond to the injury: they multiply by mitosis as early as 10 days after injury and lay down glial fibrils in the damaged areas. It is probable that the activity of astrocytes is stimulated and regulated by growth factors; Lipton (1989) has reviewed possible roles of these in promoting favourable interactions between glial cells and injured neurons. Eventually, after months or years, the site of injury and the course of the degenerated fibre systems are identifiable by an increased glial population as well as by absence of neurons and their processes. Fibroblasts play little part in the reparative processes of clean brain wounds, but appear in great numbers when an abscess forms: the walls of chronic abscesses are densely collagenous. Fibroblasts are also active in meningeal and meningocerebral scars.

Secondary complications of head injury

These include:

1. Intracranial haemorrhage — extradural, subdural and intracerebral
2. Brain swelling
3. Hypoxic brain damage
4. Arterial injury — traumatic thrombosis, false aneurysm formation and carotid-cavernous fistula
5. Infection — cerebral abscess, subdural abscess, pyogenic thrombophlebitis and leptomeningitis; extradural abscess and cranial osteomyelitis are less serious forms of infection
6. Obstructive hydrocephalus.

The clinical implications of these life-threatening complications are discussed in Chapter 13. Here it is appropriate to review briefly their pathology and to discuss to what extent general neuropathological descriptions apply to the complications seen after the frontal impacts which cause CMF injuries.

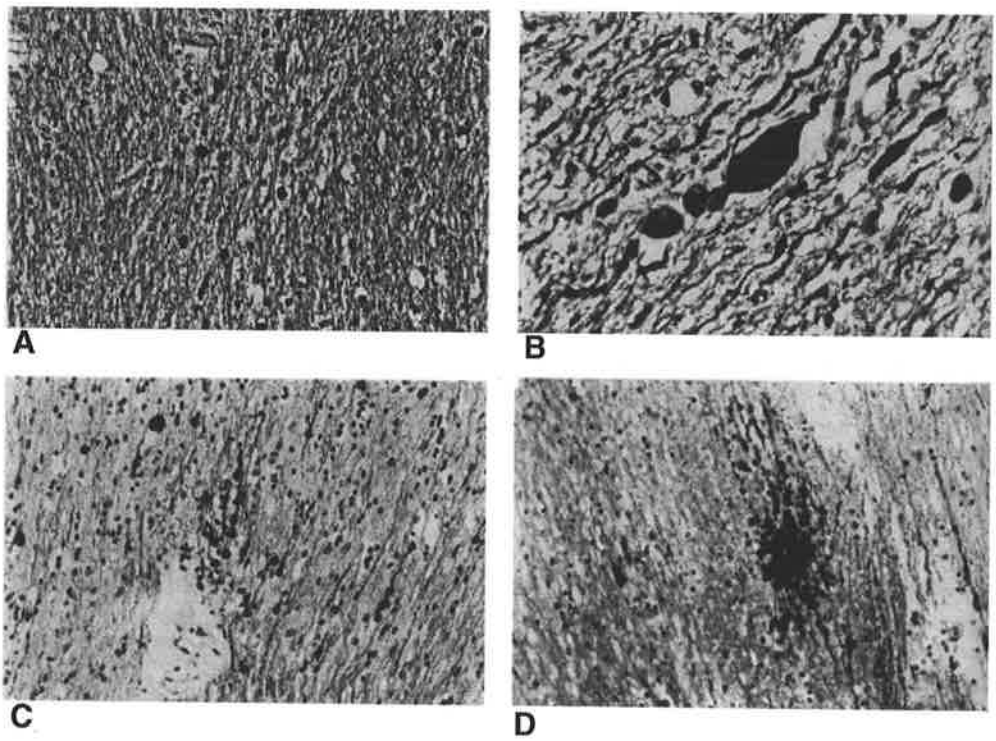


FIG. 5.20. *Diffuse axonal injury in the corpus callosum.* *A 15-year-old boy died 5 days after a road accident. Sections taken from the corpus callosum show typical axonal degeneration.* **A.** *Palmgren's silver impregnation; the dark globular masses represent degenerating axons. × 167.* **B.** *The same: higher power view showing a large fusiform mass of degenerating axon. × 342.* **C.** *Immunochemical stain for neurofibril protein (phosphorylated: NFP-P): the dark balls represent degenerating axons. × 167.* **D.** *Immunochemical stain for Amyloid precursor protein (APP): the mass of degenerating axons is well defined from the surrounding corpus callosum. × 167. All three stains show abnormal material derived from degenerating axons. In the two immunochemical stains, the degenerating axons are clearly distinguished from the remainder of the corpus callosurn: identification of the lesion is easier than in the classical silver stains. The changes demonstrated by the APP stain are evident at a relatively earlier time after injury; indeed, they may be seen in cases surviving less than 1 h.*

Extradural haemorrhage

Extradural haematomas of clinical importance usually result from injury to a meningeal artery. In our experience, a substantial minority of extradural haematomas are located in the frontal or subfrontal area. Most of these result from off-centre frontal impacts. In our series of 366 cases of extradural haematomas of all types (Jones et al 1993), 11.4% were classed as frontal and the percentage was higher in children (Molloy et al 1990). Commonly, these frontal haematomas result from tears of relatively small branches of the middle meningeal artery, and are less rapid in onset, hence less serious, than temporal or subtemporal extradural haematomas (Fig. 4.14).

Subdural haemorrhage

Subdural haematomas of clinical importance may result from injury to a bridging cortical vein, or from bleeding related to a severe cerebral contusion. Subdural haemorrhage is not especially common after frontal impacts, but can occur. The presentation may be acute or chronic; the chronic haematomas of elderly persons are rare as sequels of CMF injury (p. 524).

Intracerebral haemorrhage

Intracerebral haematomas may complicate both closed and penetrating injuries. Severe closed frontal impacts may cause deep frontal or basal ganglionic arterial bleeding; the striate arteries appear to be the usual source. Missile wounds usually cause some intracerebral bleeding, and sometimes this is severe enough to constitute a space-occupying lesion.

Brain swelling

This is a common and often lethal complication of severe brain injury. Localized swelling is a usual pathological concomitant of contusions. Diffuse hemispheric brain swelling is a common complication of acute subdural haematomas; it may also be seen in association with hyperacute extradural haemorrhage and around missile tracks (Carey et al 1990). Under the light microscope, the white matter is most obviously affected: myelin stains show pallor in the vicinity of a contusion, and the myelin sheaths are seen to be swollen vacuolated and beaded (Miller & Adams 1992). However, the histopathological examination of brain swelling is often uninformative, and the best understanding of this very important complication has come from clinical and experimental studies.

Brain swelling may represent true oedema in the sense of extravascular fluid collection, or vasodilation attributable to failure of autoregulation. North & Reilly (1990) recognize several forms of cerebral oedema:

Vasogenic oedema, where a protein-rich transudate passes into the extracellular space through a dysfunction of the blood-brain barrier in the cerebral capillaries. This process is increased by increased intravascular pressure and/or increased cerebral blood flow due to hypercarbia or hyperpyrexia. It is the most important form of post-traumatic oedema.

Cytotoxic oedema, where fluid accumulates within the cell bodies of neurons, glial cells and endothelial cells, because of failure of cell membrane mechanisms, as in anoxic or ischaemic states.

Hydrostatic oedema, where fluid transudes from the ventricles into the periventricular white matter as a result of increased intraventricular pressure — this is also called interstitial oedema. Hydrostatic oedema is also seen after the evacuation of a large intracranial clot, when increased intravascular pressure is suddenly transmitted into an unprotected, possibly damaged, capillary bed. Hyperemic vascular engorgement due to an increase in cerebral blood volume may result from loss of the autoregulatory functions of the cerebral vasculature, especially the arterioles. This is said to be especially common after head impacts in children; the evidence for this is considered in Chapter 19.

Cerebral hypoxia

This is is a common complication of severe head injury and has many causes. After CMF injuries, airway obstruction is often seen and may convert a recoverable brain injury into irreversible brain damage. Arterial hypotension may occur after a severe brain injury or as the result of blood loss from a maxillofacial wound; this may impair cerebral tissue perfusion. Raised ICP may have the same effect. The most obvious early microscopic sign of neuronal anoxia is loss of Nissl substance and eosinophilic cytoplasmic change; later, neuronal outfall is seen, typically in such vulnerable areas as the hippocampal cortex and the cerebellar Purkinje cell layer.

Arterial injury

Injury of small cerebral arteries is a common effect of closed head injury, but damage to larger arteries is relatively unusual. However, the internal carotid artery is vulnerable in its cervical course and within the skull. In both sites, the arterial injury may complicate the management of other CMF injuries. An impact in the neck may crush the internal carotid artery in its extracranial course, perhaps against the vertebral column. Giannotti & Gruen (1992) report an association between displaced mandibular fracture and damage to the underlying internal carotid artery; we have not seen this association, nor does it appear in the large series of carotid injuries reviewed by Krajewski & Hertzer (1980), though these authors mention a case associated with temporomandibular dislocation. The internal carotid artery may also be stretched or even ruptured by violent

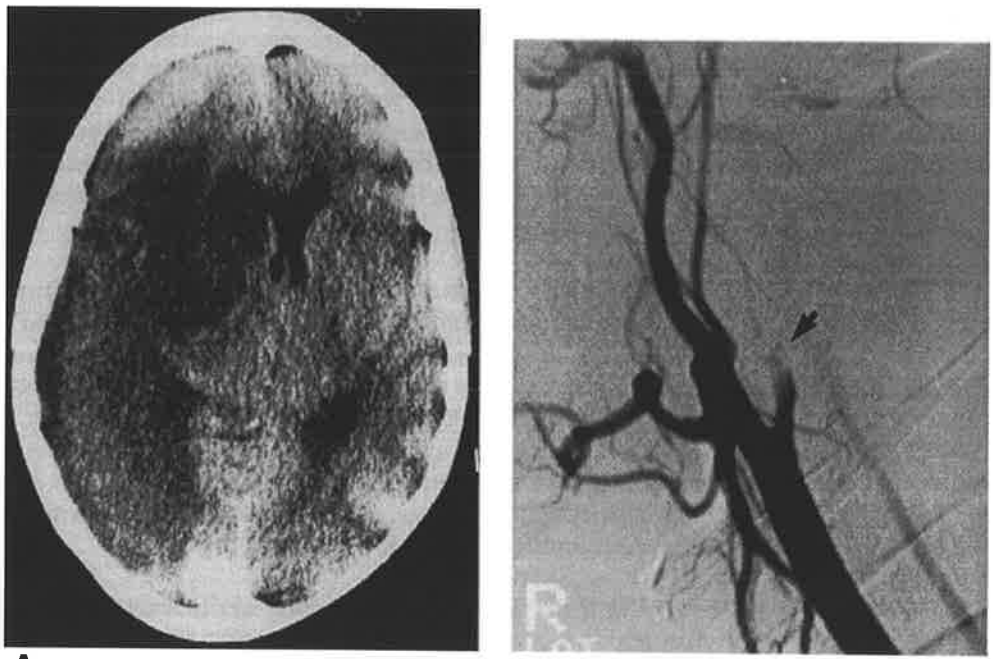


FIG. 5.21. ***Carotid thrombosis: ? a seatbelt injury.*** *A 28-year-old car driver was injured in a head-on crash. She was wearing a lap-sash belt. There was bruising in the neck but initially she appeared neurologically intact. A few hours later, she developed a left sided hemiplegia.* **A.** *CT showed extensive low density change in the right cerebral hemisphere, suggesting massive infarction.* **B.** *Angiography showed obstruction of the right internal carotid artery. She remains very severely disabled.*

hyperextension of the head, often a consequence of impact in the CMF area. In both types of arterial injury, the pathological findings include a fracture of the intima, possibly rupture of the media, and thrombosis. Occlusion of the artery, or secondary embolism from the thrombosis, may cause cerebral infarction (Fig. 5.21).

Carotid-cavernous fistula

The internal carotid artery may also be injured within the cavernous sinus, as a result of a fracture of the skull base. The artery is fixed at its point of entry into the cavernous sinus, and also where it leaves the sinus anteriorly; it is susceptible to shearing stresses between these points of fixation. Transverse fractures of the spheroid bone are especially likely to cause intracavernous arterial injury, and the injured artery may rupture into the cavernous sinus. In some reported cases, there has been a rent in the anterior part of the artery within the sinus; in others, the artery has been totally transected. Under these circumstances, there is a high-flow leak of arterial blood into the cavernous sinus. When one of the small intracavernous arterial branches of the artery is torn (p. 47), there may be a low-flow leakage from both ends of the torn artery (Parkinson 1965).

The pathological effects of a carotid-cavernous fistula can be dramatic. The regional draining veins dilate under arterial pressure, and may rupture into the nasal cavity or the subarachnoid space. This however is unusual; more often, the arteriovenous fistula produces proptosis and other visual symptoms. The visual effects of carotidcavernous fistulae include impaired extraocular muscle action, hypoxic retinopathy and secondary glaucoma Jorgenson & Gutthoff 1985). The flow dynamics and surgical management of these lesions are discussed on p. 392.

Traumatic aneurysm

Penetrating wounds may easily injure large arteries, and traumatic aneurysms may form at the site of the arterial wound. These are false aneurysms, and have been described after both missile and knife wounds (de Villiers 1975).

Cerebral abscess

A suppurative infection may result from implantation of microbes in the brain substance in a penetrating wound or by secondary spread from infection outside the brain. Thrombophlebitis is a possible mechanism of spread from a local source of injection, and haematogenous seeding can occur as a result of post-traumatic septciaemia. Implanted foreign bodies are a well-attested cause of brain abscess (Fig. 13.12).

The first response to cerebral infection is a migration of circulating polymorphs into the brain; macrophages and lymphocytes are also mobilized and an area of cerebritis develops, with necrosis of brain tissue. Macrophages ingest the products of degeneration, especially lipoid material. Continued bacterial growth in the necrotic area and continued response by leukocytes generate pus. If the infection is contained, and does not spread or burst into the lateral ventricle, granulation tissue forms around the purulent cerebritis; fibroblasts appear, presumably migrating from the adventitia of small blood vessels, and form a collagenous abscess capsule. Astrocytes also lay down a fibrillary gliosis.

Brain abscesses resulting from externally compound skull fractures are most often due to staphylococcal infection, but many other organisms have been reported, including clostridia; brain abscesses complicating fractures of the skull base may give more diverse microbial cultures (Table 5.2). The reported increase in infections by gram-negative infections may result from prophylactic antibiotic medication (Rish et al 1981).

Subdural abscess

This form of intracranial suppuration, also known as subdural empyema, is rare as a sequel of penetrating brain injury. It is an extremely dangerous infection, spreading and pocketing over the surface of the cortex and often loculating between the cerebral hemispheres in relation to the falx. Vasculitis may cause infarction of the underlying cortex and sometimes this leads to survival with devastating brain damage. The literature on post-traumatic subdural abscess is too scanty to comment on the bacteriology; in two cases of ours, a blunt frontal impact of no great severity was followed by aggressive extra and subdural infection due to a multiplicity of organisms, suggesting pre-existing paranasal sinusitis (p. 390).

Extradural abscess.

Extradural pyogenic infection is usually found in association with cranial osteomyelitis (see above), and has also been seen as a sequel of operative repair of compound frontal fractures (p. 372). The dura mater is a good barrier against the spread of infection to the leptomeninges, and extradural infection is usually far less serious than subdural infection.

Acute leptomeningitis

Bacterial meningitis is a frequent complication of fractures involving the paranasal air sinuses. Table 5.2 gives the bacteriological findings in 31 cases of meningitis in a series of 238 cases of anterior fossa fractures treated by us. In this series, the chief organism was *Strep. pneumoniae,* and the infection was often fulminating, though the overall mortality was low.

It cannot be too strongly emphasised that these infections can occur many years after injury (Simpson et al 1990). Fractures involving the skull base may never heal if a protrusion of brain has been driven into the fracture line. The poor healing of fractures in the vicinity of the paranasal air sinuses is familiar to neurosurgeons exploring the anterior fossa in cases of post-traumatic meningitis and was well shown histologically in autopsy material by Linell & Robinson (1941), but is often forgotten by persons who are reassured by the cessation of a leak of

TABLE 5.2

Meningitis and brain abscess complicating anterior fossa fractures (from Simpson et al 1990)

Organism	Meningitis abscess	Meningitis abscess	Cerebral
Streptococcus pneumoniae	22 (1 died)	1	–
Other streptococci	2	–	–
Neisseria meningitidis	1	–	–
Haemophilus influenzae	3	–	–
Staphylococcus aureus	–	–	2
Streptococcus + Bacteroides	–	–	1
Pseudomonas spp.	1	–	1
Enterobacter	1	–	–
Mixed	–	–	1 (died)
Unknown	1 (died)	–	–
Total	31	1	5

cerebrospinal fluid (CSF). In our series, the longest latent period between injury and meningitis was 10 years, but we know of longer intervals.

Cerebral thrombophlebitis and venous sinusitis

These conditions are not often seen as complications of CMF injury; Krayenbuhl (1967) reported on a series of 73 cases of cerebral and venous sinus thromboses of all kinds, and in only three was trauma seen as an aetiological factor. Nevertheless, pyogenic cortical thrombophlebitis is sometimes seen as a sequel of post-traumatic extradural or subdural infection; it may lead to cerebral abscess formation. Septic cavernous sinus thrombosis has been reported as a sequel of infected facial wounds or posttraumatic infections of the ethmoid or spheroid sinuses, and also as a complication of facial surgery (p. 391); however, even in the pre-antibiotic era, trauma does not appear to have been a frequent cause of this deadly infection. It may be associated with meningitis and cerebral venous infarction. Septic thrombosis of the superior sagittal sinus appears to be even rarer as a complication of CMF injury, though it may follow frontal sinusitis; we have seen thrombosis of this sinus in a case of deep frontal electric burn, but the histology of the sinus suggested that the thrombosis was due to the burn rather than to secondary infection (Fig. 479).

Post-traumatic hydrocephalus

Obstructive hydrocephalus is sometimes seen after severe closed head injury. It seems likely that the cause is blockage of CSF circulation by the breakdown products of traumatic bleeding into the ventricular system or the subarachnoid space; the site of blockage is presumed to be the arachnoid villi, though occasionally post-traumatic aqueduct obstruction is seen. Obstructive ventricular dilatation must be distinguished from ventricular dilatation due to cerebral atrophy.

Raised ICP and internal cerebral displacements

These are life-threatening complications of many types of CMF injury. A mass lesion complicating a frontal impact, whether acute (e.g. an extradural clot) or chronic (e.g. a frontal lobe abscess), may expand within the cranial cavity. A small expanding mass is at first accommodated by two mechanisms: CSF is displaced into the spinal canal, and venous blood is displaced from the cerebral veins into the general extracranial circulation. When the mass exceeds a critical volume, these compensatory displacements are insufficient and ICP rises. The pathophysiology of raised ICP has been reviewed by North & Reilly (1990).

Severe and sustained elevation in ICP can impair cerebral blood flow (CBF). In any vascular bed, blood flow is dependent on the gradient between arterial and venous pressures, and on the vascular resistance to flow. Since the cerebral veins are thin-walled and collapsible, venous pressure and ICP are closely related: in practice, sagittal sinus venous pressure is 1-2 mmHg lower than ICP. So the relation can be expressed by the equation:

$$\text{CBF} = \frac{\text{arterial pressure} - \text{ICP}}{\text{cerebrovascular resistance}}$$

Normally, total CBF is maintained at a fairly constant rate (about 50ml/100 g brain tissue/min) by variations in vascular resistance: these variations express the ability of the cerebral arterioles to dilate or contract in response to the local metobolic demands of the brain. This mechanism is termed autoregulation. When ICP rises, autoregulation causes arteriolar dilatation, and this, together with neurogenic reflex arterial hypertension, will maintain cerebral perfusion. But if ICP reaches an extreme height, or if autoregulation fails, then the cerebral resistance vessels dilate passively and CBF falls. Experimental studies suggest that neuronal activity ceases when CBF is than 12ml/100g brain tissue/min (Astrup 1985). With very high ICP, flow through the internal carotid artery ceases altogether and this is a recognized sign of brain death (p. 231).

Often, the effects of post-traumatic mass lesions are; due to internal cerebral displacements rather than to a: general elevation in ICP. The cranial cavity is compartmented by the falx and the tentorium. An expanding haematoma or localized area of brain swelling may displace the cingular gyrus under the falx (subfalcine herniation). More seriously, the medial temporal lobes may be displaced through the tentorial hiatus (transtentorial herniation: Fig. 2.28). The midbrain may be compressed and the quadrigeminal cistern may be obliterated — a danger sign in the CT scan (p. 209). Laterally placed mass lesions characteristically cause medial and downward displacement of the ipsilateral temporal lobe, with compression of the ipsilateral third nerve and pupillary paralysis, but the frontal haemorrhages and brain swelling more characteristic of impacts in the CMF area are likely to cause symmetrical transtentorial herniation. A rare but very serious effect of transtentorial herniation is kinking and occlusion of one or both posterior cerebral arteries: this can result in bilateral occipital infarction and cortical blindness. With more advanced downward displacement, the vessels supplying the brainstem may be stretched: areas of ischaemia and small haemorrhages may appear in the midbrain and pons. This usually means that the process is clinically irreversible. For the pathologist, these secondary effects of a mass lesion must be carefully distinguished from primary brainstem haemorrhages which represent an element in diffuse vascular injury: these haemorrhages are more likely to be in the dorsal tegmentum and in the superior cerebellar peduncles. Finally, the expansion of the intracranial contents may squeeze the cerebellar tonsils into the spinal canal: this is tonsillar herniation.

The Eye

Ocular injuries

The globe of the eye may suffer primary damage from a penetrating agent (Fig. 5.22) or from blunt violence (Fig. 5.23); the eye may also suffer secondary effects of injury to some part of the visual apparatus, such as the aqueous drainage, the blood supply of the retina or the blink mechanism and the lacrimal system. The eye may also be injured by thermal energy, radiation, or chemical agents, especially alkalis. The pathology of ocular trauma is well discussed by Iyer & Rowland (1993).

The eye is unique in possessing a transparent optical pathway which focuses light on the light-sensitive photoreceptors in the retina. To maintain the

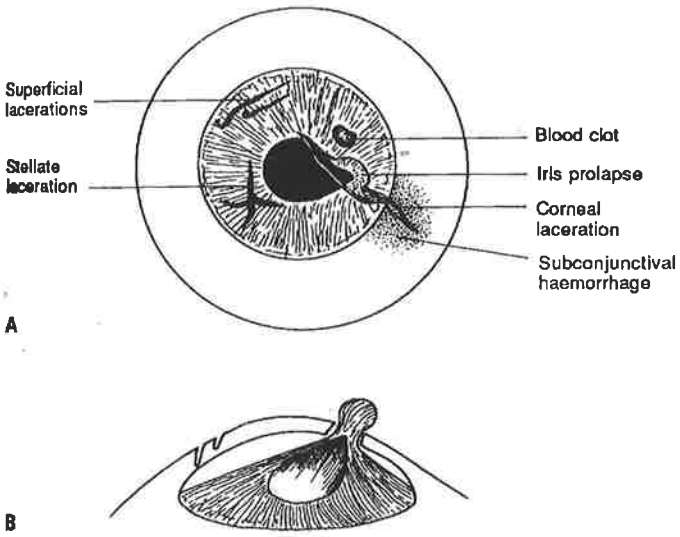


FIG. 5.22. *Corneal laceration.* A. *Composite diagram showing the effects of penetrating (sharp) trauma to the anterior segment: superficial non-perforating, shelving corneal lacerations; stellate laceration; perforating corneal laceration with iris and prolapse leading to distorted pupil, associated hyphaema (i.e. blood clot in anterior chamber) and subconjunctival haemorrhage.* **B.** *The same in section.*

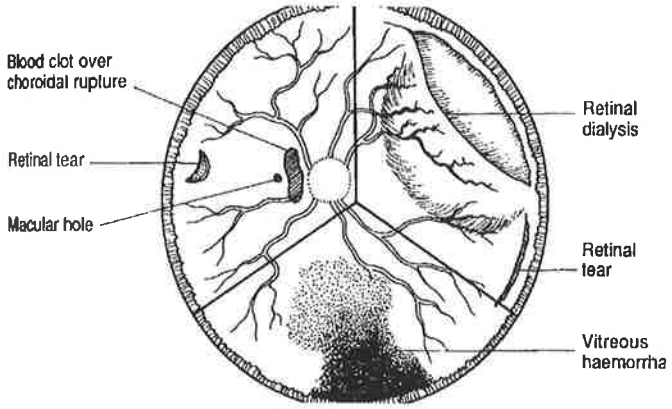


FIG. 5.23. Blunt trauma to posterior pole of the eye. *Composite diagram showing detachment of the iris from its insertion (dialysis) with a distorted pupil; blood in the anterior chamber (hyphaema); cataract formation in the lens.*

transparency of this optical pathway the structures through which light travels are predominantly avascular. The effects of injury and also the effects of healing processes will in most instances produce changes in the clarity of the optical pathway or induce alterations in the lens system (cornea and crystalline lens). The unique hypovascularity of the globe renders it excessively sensitive to infection and slows normal healing processes. Moreover, the globe is enclosed in a corneo-scleral skeletal envelope and is isolated by a physiological blood-ocular barrier: it is thus relatively sequestered from the immune reactions of the body as a whole. The neural tissues of the retina and optic nerve have very little reparative capacity: indeed the effects of injury and of healing on the ganglion cell axons which form the optic nerve are as adverse as those of the brain.

The basic healing processes of coagulation, inflammation, proliferation of fibrovascular tissue and surface cells and tissue remodelling occur in all sites of the eye after injury. These processes are modified in the avascular zones. Thus there is little in the way of haemostasis around corneal wounds; however, the composition of the aqueous in the anterior chamber undergoes secondary changes after penetrating injury, developing a high protein content and fibrin clot, and this, combined with the swelling of corneal stromal fibres, prevents excessive leakage of aqueous.

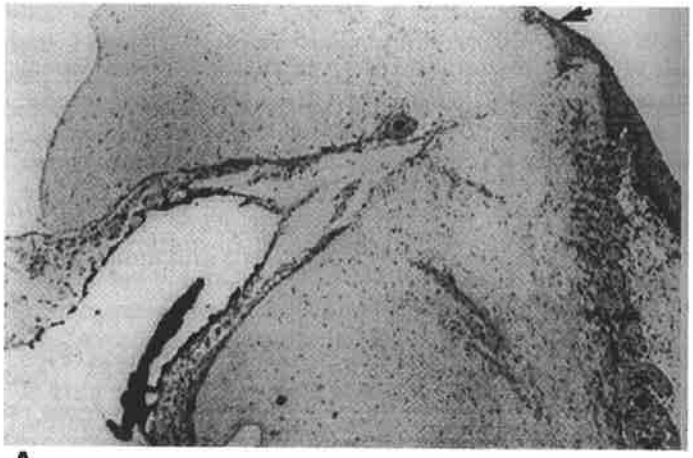
Cornea

The cornea can be damaged primarily from an impact or a burn, or secondarily from injuries of the lids, conjunctive and limbal region. Distortion of the normal lamellar arrangement of the collagen fibres in the cornea produces scarring and refractive changes such as astigmatism which may be progressive. Scarring in the optical axis of the eye will produce reduced vision or glare and light intolerance.

When the cornea is lacerated epithelial cells are damaged for a variable distance from the anterior aspect of the wound edge. Stromal keratocytes are transected and endothelial cells are destroyed or dislodged from the posterior aspect of the wound (Matsuda & Smelser 1973). The corneal epithelial layer can regenerate itself by mitotic division and cell migration over the denuded part of the cornea; regeneration is faster if the basement membrane is intact (Khodadaust et al 1968). Undamaged epithelial cells aggregate and after a brief refractory period migrate towards the wound edge (Buck 1979). The origin of these cells is the limbal stem cell population; their migration is guided by the topographical features of the epithelium and eyelids and by chemical messengers (Cameron et al 1988). Limbal stem cells are of great importance in corneal epithelial healing; ischaemia of the limbal region, often produced by alkali injury, is one of the most important factors in predicting the outcome of such injuries. Corneal healing will not be satisfactory if the limbal region is severely damaged.

The migration continues over exposed Bowman's membrane until the cells meet the physical barrier of corneal stroma, fibrin clot or healthy endothelium. When migration is complete, further mitotic cell division occurs and the corneal epithelium is reconstituted. If Bowman's membrane is intact, this process leaves no scar. If this membrane, which does not regenerate, is damaged, the epithelium is restored, but there may be a corneal nebula in the injured area. Wounds of the corneal stroma are repaired by fibroblastic proliferation. The corneal keratocytes have a long refractory period: after several days the keratocytes repopulate the wound area and begin producing new collagen which lacks the original sheathing. The new collagen is not arranged in the highly organized fashion of undamaged cornea and therefore does not provide complete clarity. The corneal endothelium has little capacity to restore itself. Defects in the endothelial layer may be covered by migration and stretching of the remaining cells. The migration extends over Descemet's membrane as a monolayer of cells and mitosis is very limited. Generally the endothelium regenerates and heals poorly and this may lead to persistent corneal oedema. In future, agents such as EGF, TGFα and –β and fibronectin may be used in promoting corneal healing (Schultz et al 1992); zinc and other trace metals as well as vitamins may also have a role in this. Perforating wounds of the cornea may be complicated by prolapse of the contents of the globe, forced out by the intraocular pressure: uveal tissue or the lens capsule may be trapped in the corneal wound, and this will prevent healing (Fig. 5.24). The prolapsed tissue may be welded to the cornea by granulation tissue invading the cornea, or corneal epithelium may migrate through the wound into the anterior chamber of the eye; if this invading epithelium obliterates the filtration angle of the anterior chamber, then aqueous drainage is blocked and glaucoma is the outcome. Penetrating wounds of the cornea may also lead to bacterial infection—a corneal abscess or hypopyon. Blunt injury of the cornea may cause internal damage to the endothelial layer, allowing aqueous to be forced into the corneal stroma, which becomes hazy.

When the cornea has been significantly damaged and there is distortion of the usual lamellar collagen fibrils as well as disturbances of the limbal anatomy, blood vessels grow into all layers of the cornea. These abnormal blood vessels contribute to the healing process but naturally have an undesirable effect on vision in that they increase the opacity of the cornea.



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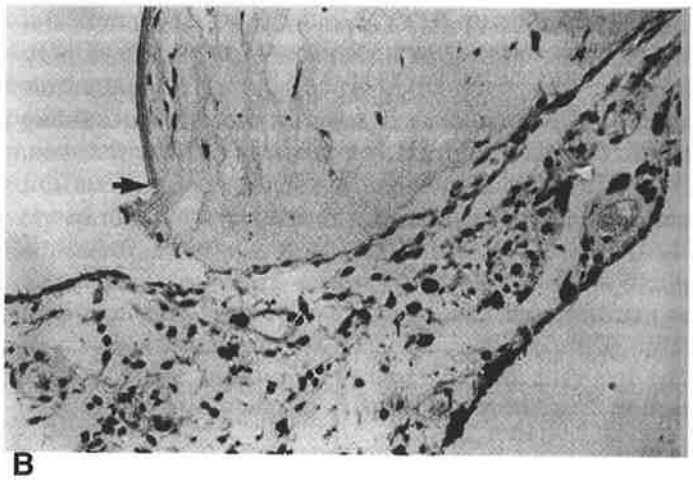

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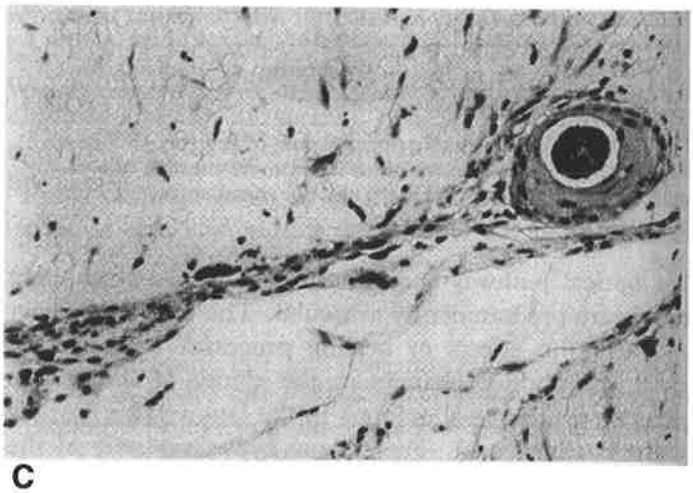

C

FIG. 5.24. *Penetrating eye injury: iris synechia.* *The iris has herniated into a traumatic cleft in the cornea in the limbic region. Healing of the cornea is prevented.* **A.** *The herniation of iris tissue extends deeply into the corneal stroma; the corneal epithelium (arrow) is intact. H&E, × c. 35.* **B.** *Iris tissue, which contains much pigment, is applied to the lacerated cornea. Descemet's membrane (arrow) shows no sign of regeneration. H&E, × c. 83.* **C.** *Island of corneal epithelium in the corneal laceration, presumably a traumatic inclusion cyst. H&E, × c. 165. By courtesy of Dr Prema Iyer.*

Sclera

Perforation of the sclera heals well unless complicated by extrusion of uveal tissue; healing involves the formation of vascular granulation from the choroid and/or the episcleral layer, followed by scar formation. Blunt violence to the globe may rupture the sclera, sometimes at the site of impact, sometimes elsewhere, perhaps at the thin equator of the globe (p. 72), perhaps at the limbus. As a rule, the eye is destroyed by an impact severe enough to cause scleral rupture. The sclera heals mostly from the fibrovascular components of the episclera and uveal tract.

Iris and ciliary body

The iris may be damaged both by penetrating and non penetrating trauma (Figs 5.22 and 5.23). Bleeding results, and the red cells may sink to the lower part of the anterior chamber as a compact mass or hyphema. This eventually absorbs, but if the hyphaema is large the cornea may be stained. Bleeding into the aqueous can lead to glaucoma, specially if the bleeding recurs.

Trauma to the iris may cause alterations in pigmentation and loss of the posterior pigmented epithelial layer leading to a translucent iris. There may also be alterations in the muscular structures of the iris producing irregularities in the size and shape of the pupil. There may be functional changes in that the pupil does not constrict normally to light (traumatic mydriasis). These changes, although relatively minor, may affect vision by including optical aberrations and by reducing the eye's tolerance of glare. Dialysis of the iris occurs if the iris is torn from its root; generally this is not of major clinical importance, though it increases sensitivity to glare and occasionally causes diplopia. Traumatic inflammatory responses may produce adhesions of the iris to the cornea or lens (anterior or posterior synechiae), and also secondary glaucoma and cataract. In severe contusive injuries the iris root and the anterior chamber angle may be cleft, producing angle recession. The fibrous reparative tissue may then impede aqueous outflow and produce secondary glaucoma.

Lens

In most instances, the lens responds to trauma by becoming opaque. This may be as a result of direct injury to the lens or may be a secondary effect of forces such as those of electrocution or shock waves, producing damage to the mitotically active lens epithelium and perhaps inducing structural changes in the lens proteins. The lens has little reparative capacity. A small perforation of the lens capsule will heal, but a larger tear admits aqueous into the lens substance: the lens fibres swell and usually fragment, and the lens becomes opaque. Blunt violence may tear the attachments of the lens to the zonule; the lens may be partially or totally dislocated.

Posterior segment trauma

The prognosis for posterior segment trauma is generally bad. Blindness frequently results from fibrovascular and glial proliferation within the vitreous cavity, causing retinal detachment and ciliary body damage leading to a blind hypotonic eye.

Blunt injury to the globe causes deformation (Delori et al 1969) This can lead to posterior pole and vitreous changes remote from the impact site (Fig. 5.25). This may be seen as retinal oedema or retinal tears. Vitreo-retinal traction may cause retinal breaks, most commonly dialyses. Horseshoe tears may occasionally be found, most frequently in the upper nasal quadrant. There may also be mechanical fragmentation of the retina.

In contusion injuries of the posterior segment there is hyperaemia and dilatation of the choroidal vessels at the impact site, with infiltration of polymorphonuclear leukocytes (Gregor & Ryan 1982); as in other inflammatory reactions, macrophages later become the predominant cell type. Subretinal haemorrhage may result from rupture of choroidal vessels; these may bleed into

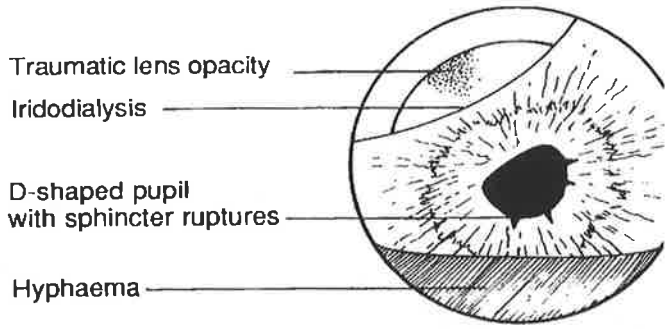

FIG. 5.25. *Blunt trauma to posterior segment.* *Composite diagram showing some of the effects of blunt trauma as seen by ophthalmoscopy: retinal tears; a traumatic hole in the macula (likely to cause loss of acuity and a central scotoma); a small blood clot obscuring a choroidal rupture through the maculopapillary bundle (likely to cause central visual loss); retinal detachment (dialysis; likely to cause flashes, floaters and a field defect); inferiorly a vitreous haemorrhage.*

the vitreous. Subretinal blood is replaced by fibrocellular membranes resulting from proliferation of the retinal pigment epithelium. Intravitreal proliferation may be stimulated by serum–derived proteins such as fibronectin, PDGF, complement and interleukin–1. These substances are chemotactic for retinal pigment epithelial cells, fibroblasts and glial cells (Campochiaro et al 1984, Glaser et al 1987).

When severe penetrating trauma produces gross tissue disruption or expulsive choroidal haemorrhage, there is little potential for visual recovery. Penetrating trauma involving the vitreous and the lens produces cataract and vitreous haemorrhage, with fibrovascular ingrowth into the vitreous which produces membranes of a contractile nature. Ultrastructural studies have revealed cells with the characteristics of myofibroblasts (p. 123) embedded in the matrix of collagen-like fibrils within the vitreous at 2-6 weeks following injury (Cleary et al 1980). These cells are contractile fibroblasts, which proliferate from the pigmented and non-pigmented cells of the ciliary epithelium. They extend into the vitreous cavity along the surface of the detached posterior vitreous.

By 4–6 weeks after the injury, multilayered epiretinal membranes occur on the posterior retina; subretinal membranes may also occur. With these membranes may be associated areas of subretinal haemorrhage and proliferation of retinal pigment epithelium. The retina heals very poorly but occasionally there may be some visual recovery: this depends on the degree and severity of retinal damage and the persistence of haemorrhage and oedema, which delay the establishment of retinal layer continuity.

The contractile properties of intravitreal membranes are responsible for the development of tractional retinal detachment. Using microstrain gauges, forces of 30–100 mg were generated by these membranes (Kirmani & Ryan 1985).

Secondary complications of eye injuries

These include:

1. Raised intraocular pressure (glaucoma)
2. Infection
3. Sympathetic ophthalmitis.

Raised intraocular pressure

Secondary glaucoma may be due to direct damage to the anterior chamber angle and trabecular meshwork with fibrovascular tissue distorting the normal aqueous outflow channels and producing a rise in pressure. This may result from anterior

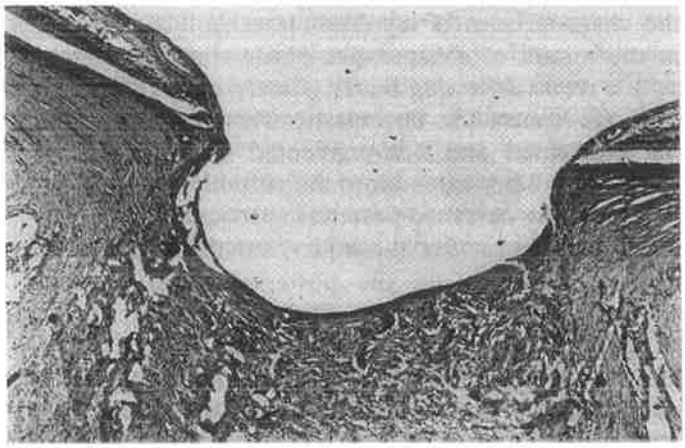

FIG. 5.26. ***Post-traumatic glaucoma.*** *The optic disc is deeply cupped; the optic nerve shows evidence of atrophy. H&E, × 50. By courtesy Dr Prema Iyer.*

chamber inflammatory reactions, debris from red blood cell breakdown, and accumulations of inflammatory and phagocytic cells in the anterior chamber angle, blocking the trabecular meshwork.

Raised intraocular pressure may also arise after lens rupture. Phakolytic and phakoanaphylactic glaucoma and uveitis occur when the lens capsule has been ruptured and cataract has formed, releasing lens proteins into the general circulation. This complication was first described by Verhoeff (Shingleton et al 1991); it is characterised by polymorphonuclear leukocytes, eosinophils and giant cells in the presence of lens matter. Lens proteins normally enjoy an immunologically sequestered position within the lens capsule: the inflammatory reaction to released lens protein may block aqueous drainage through the trabecular meshwork. Secondary glaucoma results in cupping of the optic disc and thinning of the nerve fibres, which may go on to optic atrophy (Fig. 5.26).

Infection

Microbial infection may be implanted in the eye by a penetrating wound, especially when a foreign body has been introduced. A wide range of organisms may be responsible; since the eye is immunologically isolated, its antimicrobial defences are not strong and even bacteria and fungi of low virulence can establish a severe endophthalmitis (p. 410). The infection may at first be localised, but is likely to spread throughout the eye (panophthalmitis). There is hyperaemia of the uveal tissues, with an effusion of leukocytes and serum; this tends to pool in the lower part of the anterior chamber (hypopyon). The posterior chamber may also be affected, and destruction of the retina may result.

Sympathetic ophthalmitis

This devastating complication of ocular injury can lead to complete blindness of the uninjured as well as the injured eye. It is a delayed inflammatory reaction in the uveal tract, usually seen some weeks after perforating injury of the iris, ciliary body or lens capsule. The histological features were described long ago by Fuchs (1905). The whole uveal tract, the choroid especially, is infiltrated by lymphocytes, epithelioid cells and some giant cells the epithelioid and giant cells may phagocytose pigment (Fig. 5.27). Eosinophils are also seen. Nodular aggregations form on the inner surface of the choroid (Dalén-Fuchs nodules). Lymphocytes form a mantle on the perforating posterior ciliary vessels and vortex veins. Electron microscopy and immunohistological staining show that Dalén-Fuchs nodules are transformed retinal pigment epithelial cells (Jakobiec et al 1983); mature nodules contain depigmented retinal epithelial cells, histiocytes and a few lymphocytes. As the disease advances, the immunological and ultrastructural properties of the uveal infiltrate change, the helper/inducer

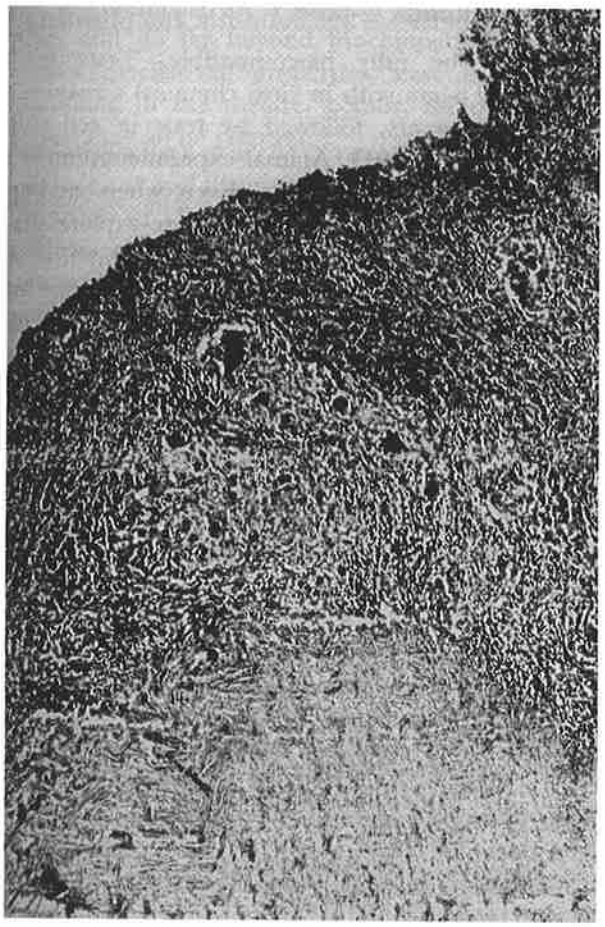

FIG. 5.27. *Sympathetic ophthalmitis.* *The choroid is expanded by a diffuse granulomatous inflammatory infiltrate containing lymphocytes, plasma cells, epithelioid cells and multinucleate giant cells. H&E, × 50. By courtesy of Dr Prema Iyer.*

subset of T lymphocytes being replaced by suppressor/cytotoxic cells (Kaplan et al 1986).

The cellular infiltration spreads out into the extraocular tissues along vascular and neural pathways. The inflammation goes on to diffuse atrophy and fibrosis of the choroid, gliotic destruction of the retina and finally complete disorganisation of the globe. The onset is insidious and the course is often remittent.

Both the histopathological appearances and the results of animal experimentation strongly suggest that sympathetic ophthalmitis is a cell-mediated hypersensitivity response to antigens liberated by damaged uveal tissue. The relatively normal lymphoid cell distribution in the peripheral blood suggests that the condition is a localized and not a systemic disease (Müller-Hermelink et al 1984). The eye, as Medawar (1948) showed, is a privileged site, exempt from many immune reactions; however, it is believed that ocular perforation allows intraocular antigens to reach regional lymph nodes, inducing immunopathological responses within the eye. This concept explains the absence of sympathetic ophthalmitis after non-penetrating trauma, however severe, and after retinal detachment or photocoagulation (Rao et al 1979).

Wacker et al (1975) have identified three distinct antigens: S and P antigens from the retina and U from the membrane of Bruch in the choroid. It is now believed that the P antigen is identical with the visual pigment rhodopsin. Experimental studies in guinea-pigs have shown that as little as 5–10 μg of the S antigen will produce a granulomatous panuveitis with histological features similar to those of sympathetic ophthalmia. HLA A-11 antigens appear to be a genetic

marker for increased susceptibilty to sympathetic ophthalmitis complicating ocular perforation (Reynard et al 1983).

Corneal grafts

Cadaver corneal allografts are widely used; because the Cornea is to a considerable extent immumologically privileged, the risks of rejection are much less than with other tissue allografts. Nevertheless, there are immunogenic cells in corneal grafts, and if these come into contact with host cells then rejection is likely. Previous corneal vascularization or infection may result in graft failure (Coster & Williams 1989); when done for post-traumatic corneal opacities the risk of rejection is not inconsiderable (p. 407).

Tissue Reactions to Foreign Material

Causes of implantation

Foreign material may be implanted by accident or surgical intention. Accidental implantation is seen when earth, road material, wood, windscreen glass, bullets or shell fragments are driven into the tissues. Such implants are often contaminated with bacteria. Surgical implants, on which there is a vast literature (see Kent & Misiek 1991), are used in many of the procedures discussed in this book.

Histopathology

Unless wholly biocompatible, implanted foreign matter excites a cellular reaction and a chronic inflammatory state may develop. It appears that the macrophage is the chief cell mobilised in response to foreign material, followed by a fibroblastic response. Macrophages can engulf small particles, but if the material is resistant to enzymic dissolution, the macrophages beome less active as phagocytes, and assume an epithelioid appearance. Multinucleated giant cells are characteristic of a foreign body reaction (Fig. 5.2A): they are formed by coalescence of macrophages. Sometimes, a giant cell may contain a fragment of silica or some other exogenous material. The histological picture may be complicated by reactions to implanted microorganisms or by an immune response. The presence of continuing low-grade microbial infection may attract polymorphs; lymphocytes and plasma cells suggest an immune response. But the typical response to an irritative foreign body is the macrophage-giant cell reaction; non-irritative foreign bodies evoke a protective fibroblastic response and become encapsulated.

Accidental metallic implants

The materials often accidentally implanted include some which are extremely irritative or even toxic.

Copper

Of the metals likely to be implanted, copper—used as a jacket in some bullets (p. 114)—is probably the worst (Wigle 1992). Copper ions pass into the tissues and form toxic salts; in the eye or the brain, this can cause severe neuronal destruction (Sights & Bye 1970).

Lead

Lead salts may be toxic, but lead in the tissues forms an oxide which is well tolerated, and lead missiles are encapsulated by fibrous tissue without much local tissue damage (Fig. 5.18). In the past, shotgun pellets were made of lead, and rare cases of systemic lead toxicity have been recorded when large numbers of pellets are implanted (Stromberg 1990).

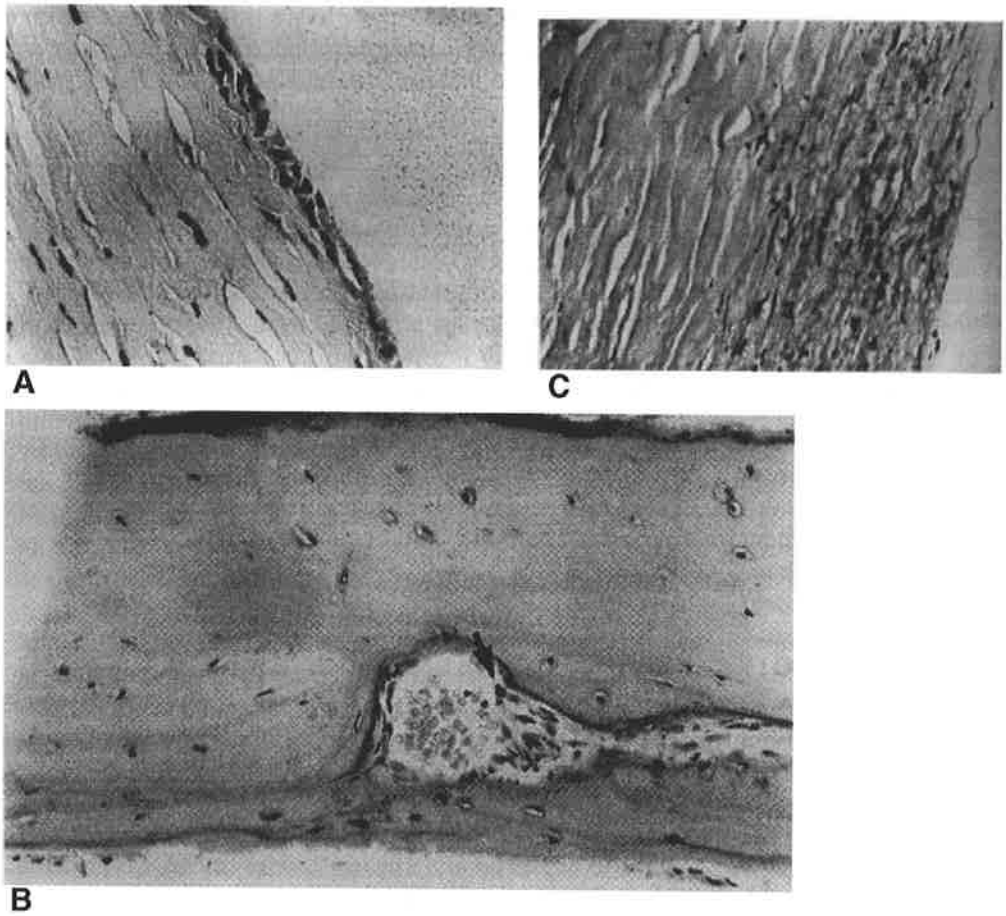

FIG 5.28. *Tissue reactions to biocompatible implants.* **A.** *A tantalum plate became infected and was removed. The biopsy, taken from an uninfected area, shows a collagenous membrane encapsulating the plate; there is a layer of fusiform or multangular cells in contact with the plate. H&E, × c. 163.* **B.** *A titanium plate was removed after 6 years because a dermal sinus formed in one of the plate holes. The plate was found to be well integrated into the skull and bone had grown over its edges: biopsy of thin bone on the outer surface of the plate showed normal appearances. Some black pigment, presumably titanium dioxide, was present in a cavity (arrow) within the new bone. H&E, × 190.* **C.** *In the same case, biopsy from the inner surface of the scalp in contact with the plate showed layers of flattened fibroblasts, with no evidence of giant cell reaction to the titanium. H&E, × c. 82.*

Iron

Iron salts are much less toxic, but iron and mild steel, used in shell casings, corrode in the tissues and can cause a local inflammatory reaction. Iron fragments in the eye may cause progressive retinal destruction (siderosis bulbi). Corrosion is increased by electrolytic action, and this is especially seen when two dissimilar metals are in proximity.

Surgical implants

These must be fully biocompatible. Tests for bio-compatibility begin with in vitro chemical screening tests for cytotoxic agents, followed by tests in cell cultures (Kent & Misiek (1991). Animal experimentation is then necessary before clinical trials. Even when an implant material is approved, surgeons who re-explore the tissues in the vicinity of an implant should obtain a biopsy if there is any suspicion of an unusual reaction, as manufacturing errors or adventitious contamination can occur. A surgical implant should not excite a prolonged macrophage/giant cell response, and is usually encapsulated in collagenous fibrous tissue with a smooth lining of flattened cells apposed to the implant surface. Fig. 5.28 shows such appearances in biopsies from the vicinity of tantalum and titanium implants: tantalum is now little used, but titanium is now employed in many CMF procedures.

The requirements for a surgical implant relate to its purpose, and go far beyond biocompatibility: strength and other mechanical properties, ease of sterilisation and handling in the theatre, colour, radiolucency, and cheapness may all be relevant.

Plastics and ceramics

Of the plastic materials used for implants in the CMF region, methyl methacrylate polymer is reasonably well accepted by the tissues, but monomer left after the process of polymerization can excite a strong reaction, often with a serous effusion. Silicone rubber is still less likely to evoke an adverse tissue response, though over time calcification may form over the implant, and occasionally materials used in the preparation of the implant may be irritative. Polyethylenes have been used in facial reconstruction for many years. Polyethylene implants evoke a fibroblastic reaction and if the implant is porous, it will be invaded and fixed by fibro-vascular proliferation. Porous high-density polyethylene (Medpor®) is now widely used to replace aesthetic deficiencies in the facial skeleton, and appears to be well tolerated (p. 563). However, polyethylenes are not recommended as load-bearing or protective implants.

Polytetrafluoroethylene (Teflon®) has been much used as an implant. It is inert chemically and is well tolerated, though the initial tissue response is inflammatory, with a giant cell reaction. Proplast® is a porous bone-like composite, in which polytetrafluoroethylene is the chief ingredient. In the original Proplast, carbon fibres were used to give the mechanical properties of bone; in later variants, aluminium hydroxide or hydroxyapatite has replaced the carbon fibre. When implanted, Proplast evokes a cellular response and its pores are invaded by granulation tissue; this is later replaced by fibrous tissue, or in favourable situations by osteoid or in the case of Proplast-hydroxyapatite by mature bone. Unfortunately, it has been found that Proplast® Teflon® may evoke a local osteolytic reaction, and the use of this substance is now in question, especially in a load-bearing structure (Feinerman & Piecuch 1993). Hydroxyapatite is the chief mineral element in bone and has therefore obvious attractions; it is now available for use in implants. Porous or dense ceramics of hydroxyapatite have been used as bone substitutes in cranioplasty and for other purposes; their clinical utility is at present uncertain. Coralline hydroxyapatite has proved satisfactory in our hands as an orbital implant after enucleation of a blind eye, unless Complicated by infection (p. 408).

Metals

Many metals have been used as implants in the surgery Of the face and calvaria. At present, the chief in use are 316L stainless steel, titanium and the cobalt-chromium-molybdenum alloy Vitallium. All are well tolerated in clinical practice. The Swiss AO/ASIF (p. 26) form of 316L steel typically contains 62.5% iron, 17.6% chromium, 14.5% nickel, 2.8% molybdenum and minor amounts of other elements (Disegi 1992). This steel is paramagnetic and does not move or become heated in the course of MRI; however, it is dense in X-ray images, as is Vitallium. Both the 316L stainless steel and Vitallium resist corrosion by forming a protective surface film of chromium oxide. Titanium has been used by us for some 30 years chiefly because it is relatively radiolucent (Simpson 1965); it is very well accepted by the tissues. In modern CMF surgery, titanium is used as mesh, plates, miniplates, screws and neurosurgical haemostatic clips. When implanted, a film of titanium dioxide forms over the surface of the metal and this makes the implant very resistant to corrosion. Usually titanium implants are encapsulated in fibrous tissue, with very little inflammatory reaction. Pigmented masses of titanium dioxide are sometimes seen in the tissues near an implant, but at present there is nothing to suggest that these are irritative or otherwise detrimental (Rosenberg et al 1993). In favourable conditions, bone grows over the implant and may incorporate it. This integration of bone and titanium has been seen with titanium cranioplasty plates (Fig. 5.28), and is the basis for the

use of titanium in osseo-integrated Implants to support external prostheses (p. 637). For some purposes titanium is too soft, but titanium-aluminum-vanadium alloys are harder and are said to be as well tolerated.

Even metals or metallic alloys showing good biocompatibility will corrode if subjected to continued abrasion, as in joint prostheses. Corrosion is also likely if the implant is in proximity to a dissimilar metal: this can happen when a surgeon ignorantly places a dissimilar screw in an implanted plate, but it may also occur if a piece of a dissimilar tool is broken off or cold-welded on the surface of the implant. We have advised the use of titanium-tipped screwdrivers to minimize this risk (Simpson 1965).

Concern must arise at the possibility of a carcinogenic reaction to a surgical implant, especially in children. Altobelli (1992) cites 11 reported cases of malignant tumours arising in relation to metallic implants, most of which contained chromium. Chromates and chromium alloys, and also nickel, have shown carcinogenicity in animal experiments. Friedman & Vernon (1983) have reported a case of oral squamous-cell carcinoma developing in relation to a mandibular staple supposedly made of titanium-aluminium-vanadium alloy, but this was a permucosal implant associated with much local irritation; so far it seems that no case associated with commercially pure titanium has been reported.

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Emergency Management

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Introduction

Craniomaxillary (CMF) injury may be an immediate threat to life. Impacts on the face may completely close the airway at the time of the accident; the obstruction may be due to shattering of the facial skeleton, or to dislodged dentures, or to collapse of the mandibular arch and retroposition of the tongue. Obstruction may appear after an interval of a few hours, as injured tissues swell or burns blister. The resulting hypoxia and hypercarbia may affect the brain, causing confusion and even coma, or worsening in the effects of an associated brain injury.

Severe CMF injuries, especially facial wounds caused by missiles, may bleed, causing hypovolaemic shock and hypotension—another threat to brain function. Associated brain injuries may be life-threatening in themselves, by causing depression in the vital protective reflexes, especially the cough and swallow reflexes. Blood and vomit may be inhaled, and this also promotes hypoxia; massive aspiration of blood is a common finding in cases of maxillofacial injury found dead at the accident site (Arajärvi et al 1986).

CMF injuries are often associated with injuries elsewhere in the body, which may cause hypotension and other disorders of cardiorespiratory function. Major chest injuries were present in 396 (37%) of 1159 fatal head injuries studied by Selecki et al (1981).

All patients with severe CMF trauma need careful cardiovascular, respiratory and cerebral monitoring. Any degree of airway obstruction, hypoxia or hypercarbia is unacceptable, and must be relieved. To achieve this, there must be appropriate emergency care at all stages from the accident site to the intensive care unit. This means skilled staff in an organised trauma service, and every community must accept the need for a trauma service appropriate to its demographic and economic circumstances.

Life Support

First aid at the accident site

Hossack (1972), on the basis of autopsy findings, thought that up to 7% of all road trauma deaths in Australia were due to asphyxia and might have been prevented had someone with basic first aid skills been present at the road side. In England, Hoffman (1982) studied 344 road deaths, half of which were at the accident site; at autopsy there was inhaled blood in many, especially in cases of skull base fracture or maxillofacial trauma, and in 9.3% this appeared to be the chief or sole cause of death. However, Ottoson (1985) studied a similar series of 158 fatalities, and doubted whether the number of preventable deaths was really significant. Whatever the percentage of preventable trauma deaths, there is no doubt that good first aid is desirable, and Canadian experience has shown that intensive community training in first aid has benefits in accident prevention as well as in better injury control (Hunt 1977). In some countries, notably Germany,

basic first aid training has been a prerequisite for a driving licence, but it has been questioned whether this is effective (Schneider & Schneider 1987) and an official German survey in 1980 suggested that only 18% of those holding driving licences felt confident to give first aid alone.

The first aider should be able:

1. To open the mouth and remove airway obstruction by dentures or other impacting body
2. To control external haemorrhage by local pressure
3. To place the victim in the lateral position to promote drainage of oral secretions or blood and to allow injured tissues to fall forward by gravity, thus opening the airway
4. To assess other injuries and to provide expired air (mouth–to–mouth) ventilation if necessary and possible: in some CMF injuries, the mouth and nose are completely disrupted
5. To arrange transport to a medical centre able to treat trauma.

These basic skills need to be taught in relation to CMF injuries as well as to other types of injury. To open the mouth and clear the airway without hyperextending the neck, two manoeuvres are useful: jaw thrust and chin lift. In the first, the mandible is thrust forward by pushing the angles on both sides from behind with the forefingers. In the second, the chin is lifted by grasping the symphysis menti with thumb and finger (Fig. 8.1). First aid teachers should prepare the class for the shock of feeling a shattered mandible. Control of haemorrhage is to be done with gentleness in facial wounds, and still more so in scalp wounds with an underlying depressed skull fracture; the use of a spring paper clip to control scalp bleeding can be demonstrated. The value of cardiopulmonary resuscitation should be taught, and the first aider should be warned of the problems arising if there is massive facial trauma. The use of the lateral or semiprone position may be life-saving in some facial fractures, especially if the tongue has lost its mandibular support.

Advanced life support at the accident site and in the ambulance

Good first aid will allow some accident victims to survive the journey to hospital and to arrive in better condition. However, more advanced forms of life support may be needed to deal with the airway obstruction and bleeding often seen in CMF injuries, especially missile injuries of the face and neck. Endotracheal intubation, venous cannulation and cricothyroidotomy or tracheostomy may be life-saving. These procedures may be carried out by a skilled paramedic or by a medical specialist.

In the USA, inspired by wartime experience, paramedic services have been developed to a high standard in many regions. Reines et al (1988) studied the management of multisystem trauma in urban and rural South Carolina: they found that paramedics were present at the accident scene in 93% of urban cases and 80% of rural cases. Life supportive procedures were not always successful: failure was recorded in 33% of attempts at endotracheal intubation and in 12% of attempts at intravenous cannulation. Nevertheless, the authors concluded that advanced life support administered by paramedics had been beneficial in some 85% of cases. In Germany, great emphasis has been placed on hospital-based medical retrieval teams brought to the accident site by helicopter or road ambulance at the request of the police or local ambulance service; Tscherne et al (1990) give a good account of this impressive system, which has been compared favourably with a similar US paramedic retrieval service (Schmidt et al 1992). Medical retrieval teams have obvious merits, and are used selectively in our practice. However, medically staffed teams are relatively expensive, and in rural accidents they are likely to arrive to the accident scene later than a locally deployed ambulance. We have often combined both systems: initial management

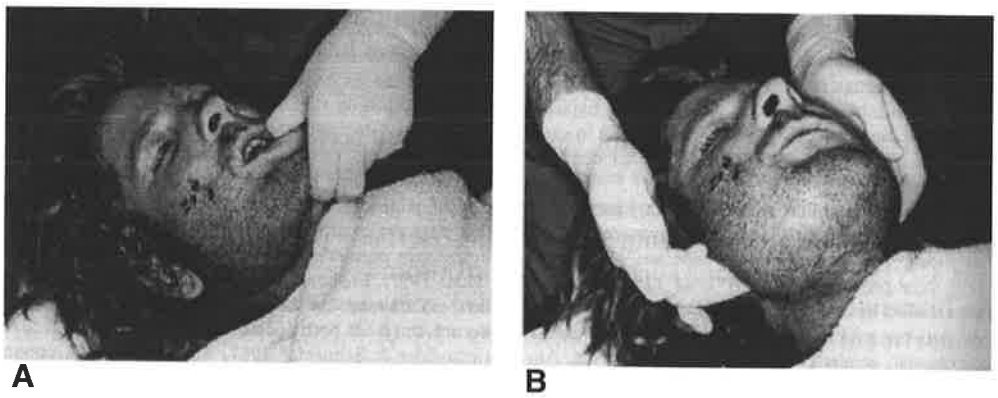

FIG. 8.1. ***Airway control in panfacial fracture.*** *A 26-year-old man was found unconscious beside his crashed car. He had panfacial fractures and required jaw support at the accident site to maintain the airway.* **A.** *Chin lift.* **B.** *Jaw thrust.*

is begun by a rural ambulance team which is joined at the site, or on the road, by the hospital-based retrieval team.

The level of advanced skills that can be developed by an ambulance service depends on the population density and geography of the region, and on its resources of health personnel. A paramedic who rarely cannulates a vein or passes an endotracheal tube should not be expected to give these services to a patient who is shocked and asphyxiated from facial injuries. The paramedic, who works in a populous North American urban area is more likely to be able to maintain these skills than is possible in a country such as Australia, where about half the major trauma occurs in isolated, thinly settled country areas: yet it is especially in places remote from a trauma centre that lives may be saved by good primary care. Throughout the world, ambulance services are having to decide whether to train their officers to 'load and go', after only basic first aid, or to provide training in more complex skills in immediate resuscitation, or to rely on medically staffed retrieval teams. The debate is sometimes acrimonious, especially when it raises issues of status and payment. Medical trauma experts should work closely with ambulance services to produce protocols, both logistic and technical, that are appropriate for their localities. Their patients will benefit.

The emergency physician, the flight nurse and the rural medical practitioner need to gain wisdom from experience and skill from practice in procedures. Special courses in Advanced Life Support (ATLS) were introduced by the American College of Surgeons in 1978, and have been very successful in many countries, including Australia; however, they have obvious limitations, and the skills they impart can be lost with the passage of time.

Resuscitation

Accident site airway control must succeed quickly to be justifiable. Endotracheal intubation is not easy at the roadside, or even in an ambulance, and unsuccessful attempts to intubate may leave the victim's airway in worse state, as well causing possibly lethal delay. Similarly, futile attempts at intravenous cannulation can be detrimental. Nevertheless, blood volume restoration before and during transport to hospital may be life-saving. The medical retrieval team can undertake this and in our practice often does; in situations where a retrieval team cannot be provided, a skilled paramedic may have an important role.

Fluid volume replacement

There has been much controversy over the relative merits Of crystalloids and colloid solutions in emergency resuscitation after trauma (Shoemaker 1976, Lamke & Liljedah 1978, Kox & Gamble 1988). Reviewing the literature, Fischer (1989) has concluded that this controversy is unlikely come to a decisive resolution, and that traumatologists should be skilled in using both types of fluid replacement according to circumstances. We favour the use of colloids — gelatin (Haemaccel),

dextran 70 (Macrodex), or 5% normal serum albumen. In experimental animals, haemodilution with colloid solutions is tolerated to a haemoglobin level as low as 4.0g/dl (Takaori & Safar 1966)

In the emergency situation, the primary need is to restore and to maintain the volume of the intravascular compartment. Colloids remain in the intravascular compartment, whereas sodium is rapidly distributed throughout the extracellular fluid because the sodium pump mechanism effectively pumps sodium ions out of cells. Crystalloid solutions such as normal saline and Hartmann's solution expand the intracellular fluid space; only one-third remains within the intravascular compartment. Hypertonic saline solutions (3% or 7.5%) are more effective in expanding the intravascular space, because they drag water from the extravascular space, giving an expansion greater than the volume of the infusion (Holcroft et al 1987).

Those fluids whose isotonicity depends wholly or partly on dextrose have no place in blood volume expansion; dextrose is rapidly metabolised, leaving only the water infused.

We believe that large volumes of crystalloids are best avoided in patients with a tendency to pulmonary oedema or with a head injury. Admittedly, blood volume expansion by as much as three times the intravascular deficit by infusion of crystalloid solutions is tolerated by young patients. However, the large volume of fluid passing into the extravascular compartment affects both the lung and the brain. In those patients, especially the elderly, in whom the left ventricle is failing as a pump, pulmonary venous pressure will rise and more fluid will be sequestered in the lung. Pulmonary capillaries may have been damaged by aspiration of acid gastric contents or by direct contusion, or later as part of multisystem organ failure. Water, electrolytes, fibrin and even red cells may pass into the pulmonary interstitial space and eventually flood the alveoli. A sticky conglomerate resembling treacle fills the alveoli, and surfactant production may fail resulting in hypoxia and microatelectasis. An increase in the interstitial and intracellular water content is also undesirable in head injuries at risk of cerebral oedema.

These issues should be clearly understood and a simple plan of management should be formulated; in emergency resuscitation, whether at the accident site or in hospital, there is no time for debate. Our preference is to use colloids, in the form of 5% normal serum albumin (sodium 140 mmol/l, chloride 125 mmol/l) or Haemaccel if available; if these are not at hand, crystalloids (normal saline or Hartmann's solution) are used. Haemodilution to 7 g/dl is quite acceptable. Hypertonic saline solution may be a good alternative, especially when there is likely to be a swollen brain. We prefer to replace losses of > 20% of the blood volume with whole blood as soon as possible.

If haemorrhage is massive, transfusion with unmatched blood may be required during transport. Ideally, O Rh– blood should be used; however, if this is not available, O Rh+ blood can be used with little increased risk. Some 50% of unimmunized Rh- individuals will develop anti–Rh antibodies if transfused with Rh+ blood, though this is less likely to happen when the transfusion reaches wash–out magnitude; the risk of later problems in transfusion and in pregnancy must be balanced against the immediate risk of death from exsanguination. But if possible, blood transfusion with whole blood or packed cells is done later after full matching of blood from donors appropriately screened for viral infection.

Monitoring of the circulatory, respiratory and neurological functions (p. 160) is begun at the accident site; the results are recorded and re-evaluated at appropriate intervals. In battle, and after civilian disasters, the accurate recording of such data is especially vital, so that deterioration may be evident to anyone seeing the injured person for the first time at a later stage in management (Fig. 8.2).

The role of the peripheral hospital

Severe CMF trauma needs the facilities of a trauma centre able to give definitive care for all the effects and complications of the injury; intensive care and neurosurgical services, CT scanning and specialized oral - faciomaxillary care are usually essential. When the accident victim cannot be admitted quickly to such a centre, there are important roles for the less well equipped hospital which is nearer to the accident site. Such hospitals should be prepared to provide blood volume replacement, surgical procedures to arrest haemorrhage, correction of airway obstruction, chest drainage and splinting of fractures. These procedures can be carried out during a short stop in the Accident and Emergency Department or even in the ambulance; patients with spinal injuries should be moved as little as possible. However, in country hospitals several hundred kilometres away from the trauma centre, a more prolonged admission may be wise, to allow fuller assessment and stabilisation. This is especially prudent when the injured person is unconscious from a head injury: a period of observation may detect a life-threatening condition such as an extradural haemorrhage or a ruptured abdominal viscus. In all circumstances, there should be telephone consultation between the peripheral hospital and the trauma centre, which should have a skilled specialist in intensive care and a neurosurgeon available for such consultations at short notice (Simpson et al 1988), There should also be a transport system able to get the injured person to the trauma centre as quickly and safely as possible: this may be by road ambulance, helicopter or fixed-wing aircraft.

Inevitably, the quality of trauma management in a peripheral hospital does not always reach the level attainable in a major trauma centre. In a prospective audit of 153 cases of severe neurotrauma referred to Adelaide from country hospitals, Simpson et al (1984) found deficiencies in management in 11 patients; these were chiefly in dealing with cardiopulmonary emergencies. Nevertheless, in Australia and in countries with similar demographic problems (Nordström et al 1989), peripheral hospitals are essential components in regional trauma services. Indeed, in countries where climatic or economic conditions make air transport less readily available, the role of the peripheral hospital may be more ambitious, and may include definitive management of a larger proportion of CMF injuries.

Transport to the trauma centre

In the choice of transport, one must carefully weigh the needs of the injured person against the hazards and costs of transport. While it is useful to have agreed guidelines, it is also important to ensure that there is good, communication between the intensivist or surgeon in them trauma centre, the ambulance or air service providing transport, and the medical officer caring for the patient in the peripheral hospital or at the accident site.

Road ambulances are cheap, safe, and capacious. Nevertheless, air transport is often the best way of bringing an injured person to the appropriate trauma centre with speed and—when this is necessary—under intensive care. Helicopters have been used with great success in war, and in populous urban areas where road transport is slow and uncertain; they are invaluable in mountain terrain and at sea (Liskiewicz 1992). We have employed helicopters both for primary transport from the accident site, and more frequently for secondary transfer from a peripheral hospital. However, helicopters are expensive and the accident rate is not inconsiderable, especially when pilots are under pressure to extricate a critically injured person from a difficult place. It is our practice to use road transport in the metropolitan area and for non-urgent transport in the country; helicopters have been used for urgent retrieval in a radius of about 50–200 km. In countries with severe urban congestion, helicopter retrieval is used over much shorter ranges. The single–engined helicopters formerly used were cramped, and unsuitable for in-flight intensive care; larger helicopters with a longer range have been available in recent years. We have found the Bell 412 helicopter very suitable for our purposes.

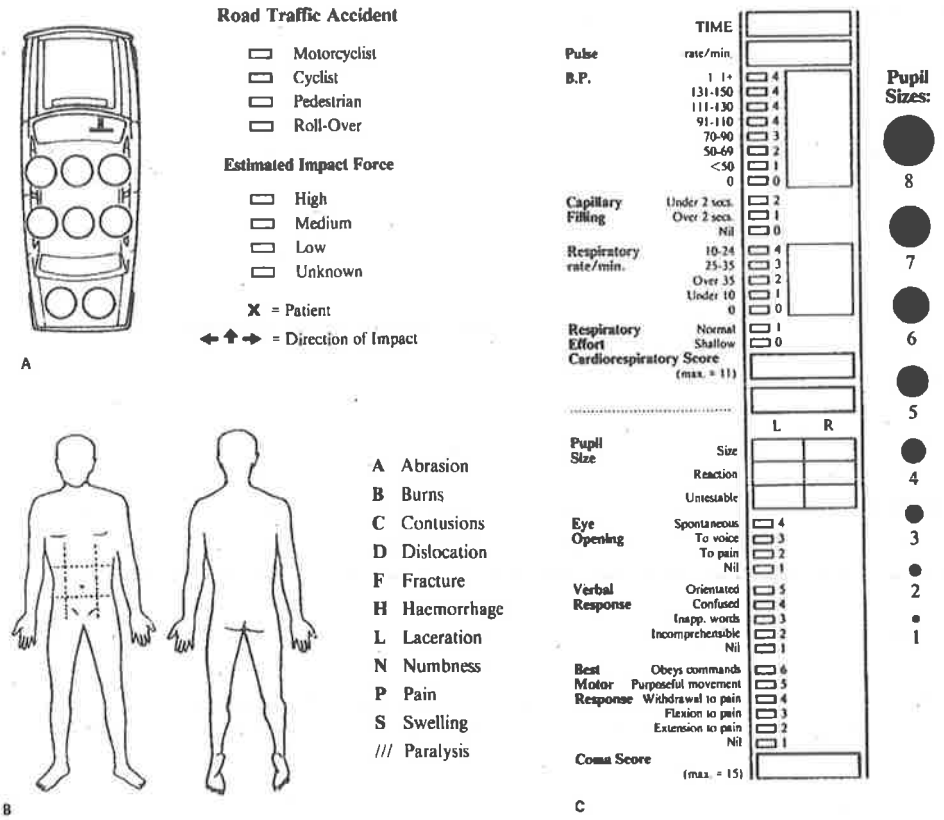

FIG. 8.2. ***Ambulance form recording accident data.*** *Information recorded by an experienced ambulance officer is of great value. Our standard record sheet includes:* **A.** *A diagram of a vehicle with seating positions and provision for identifying other types of road user, also estimates of impact force and site* **B.** *A simple body diagram to record lesions.* **C.** *Record of vital cardiorespiratory data and Glasgow Coma Scale. Pupillary diameters are in millimetres. Elsewhere on the same record sheet there is space for name and other biographical data, history of incident, ambulance times and details of resuscitation; the record is on two sheets, each with a carbon duplicate.*

Fixed-wing air ambulances find an invaluable role in secondary transfer between the peripheral hospital and the trauma centre over long distances; they are also used in international transfers, when a severely injured person is sent to another country for specialist care or to return to his/her place of origin. We have routinely used twin-engined turbopropeller-driven aeroplanes with pressurized cabins for distances in excess of 200 km in cases of head injury requiring intensive care in transit (Simpson al 1988); the aircraft at present in service are Beechcraft B200 Kingairs, with a usual ground speed of ~400 km/h. Over distances > 1500 km, economic considerations may favour the use of a commercial air liner. To transport an unconscious patient needing ventilation, we have deployed a team of one medical specialist and two expert nurses; this necessitated the use of 15 seats, but a chartered jet would have been more expensive. The need for very long range intercontinental transport is unusual, but sometimes does arise when highly specialised treatment is required, or where the injury occurred in some remote place isolated by geography or politico-military events (Spittal et al 1992). Dedicated long-range jet aircraft are ideal for such purposes, and are being used in several parts of the world. We now employ a twinjet Bae 800 for transport over long distances in Australia; this aircraft has a speed of ~800 km/h and for distances > 1500 km it is our preferred means of transport J. E. Gilligan, personal communication).

If air transport is to be used to retrieve critically injured patients it is important that the retrieval team have appropriate training and equipment. Gilligan (1990), who has been a pioneer in organizing and operating aeromedical services in our region, has emphasised the importance of stabilising the injured person's physiological state before transport by air. He has listed the ideal

requisites for any modality of transport of a severely injured person:

1. Physical safety; flying conditions or enemy action may affect this requirement
2. No abrupt movement in any axis
3. Sufficient space, with an attendant at the head end
4. Adequate supply of gases for life support and energy for the delivery systems: nickel-cadmium batteries are favoured, but must be kept charged
5. Easy embarkation and disembarkation of the injured person
6. Adequate lighting and internal climate control, including cabin pressurisation: even in so-called pressurized aircraft, the cabin pressure may be equivalent to 2000-2500 m altitude, and in some circumstances low-level flight may be requested, at the cost of a bumpy flight
7. Tolerable noise and vibration
8. Flying speed appropriate to the degree of medical urgency
9. Minimal secondary transport (e.g. transport by road to or from the airfield)
10. Good communications with the sending and receiving medical centres.

Ideally, the aircraft should be equipped as a mobile intensive care unit; a commercial plane temporarily adapted to transport an injured person is a poor substitute. Even when good in-flight facilities are available, it is best to perform procedures such as endotracheal incubation and intravenous cannulation before leaving the primary hospital. In our air ambulances, the cabins have full facilities for in-flight resuscitation and ventilation, including blood gas analysis, capnography, and oximetry. Arterial and intracranial manometry can be done, using lightweight battery-operated display systems.

Flying to higher altitudes entails lower atmospheric pressure. An eye with a penetrating wound must be presumed to contain air, and in accordance with Boyle's Law this air will expand if atmospheric pressure falls. Since the inelastic eye cannot expand, the intraocular pressure will rise; in the presence of a perforating wound, the increased pressure will force intraocular contents—aqueous, iris, lens, vitreous — out through the wound (Fig. 8.3A,B). When atmospheric pressure rises again, as when the plane comes down, the eye will collapse; blindness is the likely outcome. This lesson was first learned in the Korean War (1950-1951); it has been confirmed by primate experiments (Dieckert et al 1986). It is now recommended that penetrating wounds of the eye should not be exposed to atmospheric pressure at altitudes >650 m, from fear of promoting prolapse of intraocular contents. For penetrating eye wounds, it is wise to avoid low cabin pressures and to maintain cerebral oxygenation in flight with a high flow mask; vomiting should be prevented with an anti-emetic and the eyes should be padded (Colvin 1981). It is also considered that intracranial air could expand in a detrimental way at low environmental pressures; routine skull radiography before flight should exclude this risk, but it is again wise to keep cabin pressures at sea level during flight if an aerocele is a real possibility. The aircraft used by us permit this.

Other possible effects of high altitude flight in an unpressurized aircraft include expansion of the cuff of an endotracheal tube and accelerated flow from an intravenous line, due to expansion of gas in the drip chamber.

Emergency room

This may give the first opportunity to perform the head-to-toe examination described on p. 160, and to take a detailed history. It is often the place where definitive measures to support life are undertaken.

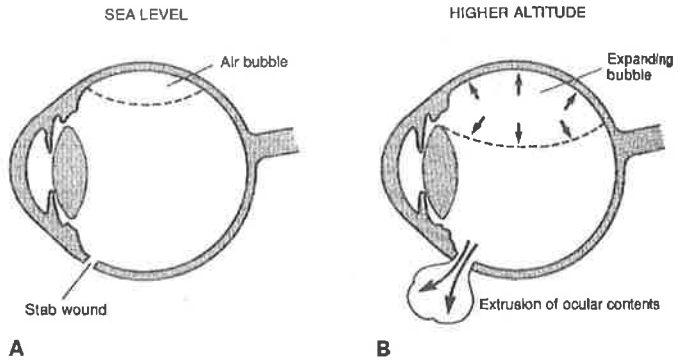

FIG. 8.3. ***Air in the eyeball: the effect of high altitude flying.*** **A.** *A penetrating wound of the globe has admitted a bubble of air into the posterior chamber.* **B.** *At low atmospheric pressure, the bubble expands, forcing vitreous and other intraocular contents out of the eye.*

Assessing the airway

Two dangerous associated injuries should be carefully looked for.

Fracture of the larynx

This contraindicates an attempt to pass an endotracheal tube (Schaefer 1991). The signs are:

- hoarseness and stridor
- local bruising and swelling
- loss of the normal laryngeal prominence
- haemoptysis
- surgical oedema
- inability to voice a high 'E'.

Attempts to pass an endotracheal tube may produce complete disruption of the larynx and airway obstruction from dissection into the tissues of the neck by the tube; even in the absence of airway obstruction, the delicate components of the larynx may be damaged. Cricothyroidotomy may have similar dangers, and tracheostomy under local anaesthesia should be considered (Nahum 1969).

Spinal injury

A fracture or dislocation of the cervical spine may be associated with CMF trauma, and attempts to secure the airway may then endanger the spinal cord. Injury to the vertebral column should be assumed until excluded by radiographs, and the neck is immobilized in all unconscious trauma victims. Therefore, if it is at all possible, a lateral X–ray picture of the neck should be taken before endotracheal intubation is attempted. The emergency room should have an overhead X–ray machine for this purpose. However, in cases of CMF injury, airway obstruction is common and cervical spinal injury is not so common, and fear of quadriplegica should not cause delay in clearing the airway if the patient is asphyxiating. Ross et al (1992) found that the chief clinical indicators of an unstable cervical spinal injury are loss of consciousness —even if brief—from head injury, spinal tenderness, and of course neurological signs; fracture or soft-tissue injury of the face did not appear to be a significant warning sign.

In assessing the X-ray picture (Fig. 7.2), care is needed to ensure that all seven cervical vertebrae are seen. In the ATLS (1993) manual, the following guidelines are given to identify significant abnormalities.

A. Alignment

1. Vertebral malalignment > 3.0 mm = dislocation
2. Anteroposterior spinal canal space < 13 mm = cord compression
3. Angulation of an intervertebral space > 11°

B Bones

1. Vertebral body
   a. Anterior height < 3 mm posterior height = compression fracture
   b. Olique lucency = tear-drop fracture
2. Lack of parallel facets of the lateral mass = possible lateral compression fracture
3. Lucency through the tip of the spinous process = avulsion fracture
4. Atlas and axis (C1 and C2)
   a. Distance between posterior aspect of C 1 to anterior surface of odontoid process > 3 mm = dislocation
   b. Lucency through the odontoid process = fracture.

C Soft-tissue space

1. Widening of the prevertebral space > 5 mm = haemorrhage accompanying spinal injury
2. Loss of prevertebral fat stripe = fracture at same level
3. Widening of space between spinous processes = torn interspinous ligaments and likely spinal fracture anteriorly.

These guidelines are not absolute indicators of trauma and the X-ray findings should be discussed with a radiologist. In a recent prospective study of 453 cases of fractures and subluxations of the cervical spine, Woodring & Lee (1993) found that 61% of unstable fractures and 36% of subluxations were incorrectly diagnosed by plain X-ray examination; indeed, in nearly one third, no abnormality was detected in the standard sequence of plain films.

Securing the airway

Difficulties may arise from:

1. Swollen lips, tongue or floor of the mouth
2. Disruption of teeth and/or jaws
3. Continuing local bleeding, especially interstitial bleeding in the neck — a very dangerous complication of a coagulopathy (Fig. 20.4)
4. Disruption of anterior cranial fossa
5. Trismus.

Trismus due to pain may relax after anaesthesia has been induced, but when the cause is a fracture of the mandibular condyle, the coronoid process, or the zygoma, then it may be impossible to open the jaw. One cannot always predict whether trismus will relax or not. Combinations of these injuries will increase the difficulties: swelling of the tongue and floor of mouth, a comminuted maxilla, gaps in the teeth, and an intact mandible make a particularly bad combination. Several methods of securing the airway are worth consideration in such situations.

Intubation without anaesthesia— 'awake' intubation

The patient may be unconscious from a cerebral injury; if not, light sedation may be given. Laryngoscopy allows damaged tissues to be lifted away from the larynx

and posterior wall of the pharynx, after which intubation is often surprisingly easy. Topical anaesthetic may be applied to the larynx; alternatively, laryngoscopy may show that it is in fact safe to induce anaesthesia with thiopentone and a muscle relaxant (suxamethonium) and intubate thereafter.

Intubation under thiopentone and muscle relaxation

This is comfortable for the patient and gives the endoscopist the best possible conditions. But what if a tube cannot be passed? Still worse, what if the paralysed patient cannot be ventilated? *This situation must and can be avoided.*

Pivotal in our thinking has been the case of a 12–yearold lad who was accidentally shot in the mouth with a .22 in. bullet, some 300 km from Adelaide. Teeth were shattered, the hard palate and the mandible were fractured and the tongue was lacerated. However, the boy was initially alert and remained so during transfer to our care by air. When he arrived in the operating theatre 8h after injury, his airway was compromised by gross swelling. It was decided to perform a tracheostomy under general anaesthesia, with gas induction. However, when placed in the supine position—he had been sitting up— the boy became distressed and uncontrollable. The options were considered and the boy was given pentothal and suxamethonium, but his tongue was so swollen that the larynx could not be seen, and an endotracheal tube was passed blindly. The surgeon then opened the neck, but met with brisk bleeding and signs of asphyxia; the cervical tissues were so swollen that the trachea and the larynx could not be located. The endotracheal tube was felt by palpation, and opened: it proved to be in the oesophagus! By the time the trachea was located and a tracheostomy performed, severe hypoxic brain damage had supervened, and the boy later died. From this case, and others, we have learned the importance of early incubation, before local swelling has developed. But this may not be possible.

Gaseous induction

If the patient can be successfully anaesthetised with O_2 and halothane, the airway can be inspected, and intubation can be accomplished with or without a muscle relaxant. But if the patient is bleeding profusely, or vomiting, gaseous induction may be difficult or impossible. If there is gross disruption of the face, it may not be possible to contain the gas within the face mask. Moreover, in patients with severely compromised airways, complete obstruction may appear when light anaesthesia is induced.

Fibreoptic intubation

It takes only a spot of blood to blind the fibrescope. The instrument is therefore unreliable in acute CMF trauma, though useful later when bleeding is absent.

Tracheostomy under local anaesthesia

This is sometimes regarded as a useful option, and we have done this with success when unwillingly disturb and massive foreign body impaling the face in the region of the temporomandibular joint (Fig. 14.1). But what if an already compromised airway becomes obstructed during the operation? There may be quite severe local bleeding under these circumstances.

Tracheostomy is easy when done electively with a secure airway and quiet ventilation. When there is asphyxia and the patient has not been incubated, the surgeon must grasp the larynx and locate the cricoid cartilage and the suprasternal notch. A midline vertical incision is made between these landmarks. In desperate cases, the midline tissues are then divided and the trachea is opened vertically or preferably with a flap, the first ring being spared. A cuffed tube is inserted, the cuff is inflated, and anaesthesia is induced. Less urgent settings allow the standard procedure of dissection, transection of the thyroid isthmus, and transverse incision of the trachea.

Percutaneous tracheostomy has been recommended as a simpler and quicker means of intubating the trachea; however, even in experienced hands, this method sometimes fails (Ivatury et al 1992) and we have not used it as an emergency procedure, though it has been very satisfactory in elective tracheostomies.

Cricothyroidotomy

This is a most useful option when it is not possible to pass an endotracheal tube, or if the necessary skill to do so is not available. A transverse incision exposes the cricothyroid membrane, which is incised. The opening is dilated and a small (5-7 mm) endotracheal tube is inserted.

The procedure should be used more often. However, there are patients in whom it is difficult to palpate the thyroid and cricoid cartilages, especially when the anatomy is distorted by swelling or haematoma. Open cricothyroidotomy is not recommended in children, in whom it is preferable to insert a large intravenous cannula through the cricothyroid membrane and insufflate oxygen intermittently.

Blind nasotracheal intubation

Blind incubation is potentially dangerous and we do not advise it in patients with CMF trauma until a fracture of the anterior fossa has been excluded: if the floor of the anterior cranial fossa is fractured, a nasal tube may pass through the fracture and enter the brain (p. 251). Moreover, in victims of acute trauma, the stomach may be full of blood and alcohol: the stimulus of pernasal intubation may induce vomiting and the vomit may be aspirated. The stomach can of course be emptied by a nasogastric tube. However, this also is dangerous if there is a fracture of the anterior cranial fossa (p. 383). Also, the stimulus may cause quite violent movement of the head and neck, perhaps endangering the spinal cord if there is an injury of the cervical spine. Any of these methods of securing the airway may fail; in fact, all may fail. Tragedies are best avoided by anticipating them. If there is any degree of airway obstruction, it must be corrected at once. If there is undue swelling, a tube should be inserted: as time passes, the situation will get worse. A misplaced tube may obstruct one bronchus (Fig. 19.1); this may cause severe pulmonary insufficiency and anoxia, especially detrimental when there is an associated cerebral injury.

Cases of CMF injury with airway obstruction require a senior anaesthetist with experience of acute trauma. Insist on that.

Control of haemorrhage

CMF injuries, especially those in the midface region, may present with exsanguinating bleeding, which must be controlled at once. The bleeding may be from superficial arteries, notably the facial and superficial temporal arteries, or from deep branches of the external carotid artery, especially the maxillary artery (p. 48). Superficial arterial bleeding may be controlled by direct pressure; this is often insufficient, and it is then necessary to explore the wound and to ligate or clip the bleeding vessel (Alexander 1989).

Epistaxis or oropharyngeal bleeding may be controlled by packing.

As a first step, the nasal cavity and nasopharynx are carefully inspected under general anaesthesia. A telescopic nasendoscopy is very useful in doing this. Any obvious bleeding point is cauterised with diathermy, and nasal tamponade is carried out with due care to ensure that the packs can be removed at a later time.

Recurrent bleeding after tamponade may need emergency operation. When the bleeding is thought to come tom the lower half of the nasal cavity, the pterygopalatine segment of the maxillary artery can be ligated through an anterior

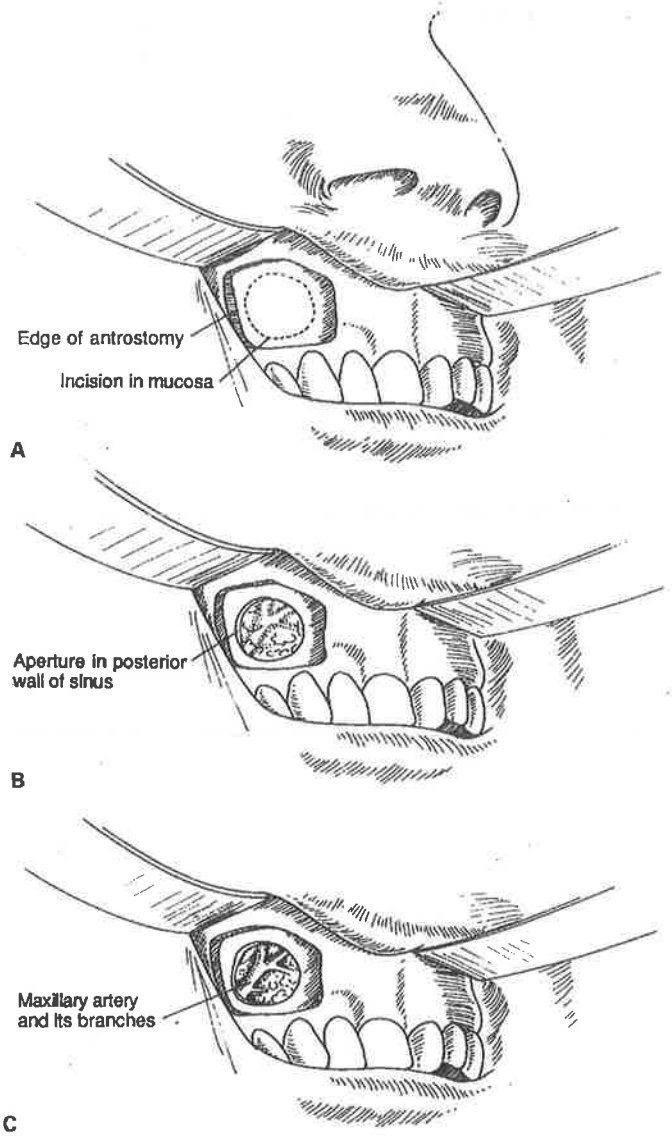

FIG. 8.4. ***Transantral exposure and ligation of the maxillary artery.*** *Under endotracheal anaesthesia (not shown), the upper lip is retracted, and a sublabial incision is made, as for a Caldwell-Luc antrostomy.* **A.** *An anterior antrostomy is made above the canine root and enlarged to allow visualisation of the posterior wall of the antrum. The posterior mucosa is incised (dotted line).* **B.** *A small bone window is made in the posterior wall of the antrum, exposing the periosteum and beyond this the pterygomaxillary fossa.* **C.** *In the space are the maxillary artery and its branches—the sphenopalatine and descending palatine arteries. They are isolated by blunt dissection and ligated with clips (see text).*

antrostomy (Friedman 1985, Spiessl 1990). A sublabial incision is made, and an opening is made in the anterior antral wall (Fig. 8.4A). The posterior wall of the antrum is removed with a diamond bur in its superomedial wall, exposing the posterior periosteum (Fig. 8.4B). An operating microscope with a 350 mm lens is then used to explore the pterygomaxillary fossa. The periosteum is opened; careful dissection with blunt hooks then exposes the maxillary artery and its descending palatine branch (Fig. 8.4C). These are then ligated with titanium or tantalum clips: three clips are essential to control bleeding in the nose and nasopharynx, on the maxillary artery proximal to the origin of the descending palatine artery, on the descending palatine artery itself (as low as possible), and on the terminal portion of the maxillary artery as near to the nose as possible. No postoperative antral pack should be needed.

It may also be necessary to ligate the anterior ethmoidal artery when the nasal bleeding appears to come from the upper part of the nasal cavity. This

should be done before the transantral attack on the maxillary artery. A skin incision is made equidistant from the medial canthus and the nasal dorsum, and curved slightly inferiorly. The periosteum is exposed and the lacrimal sac is freed from the lacrimal fossa with a blunt dissector. The periosteum is then elevated until the anterior ethmoidal artery is exposed. This artery penetrates the fronto-ethmoidal suture at the posterior limit of the anterior ethmoid cells, and it is usually found ~2.5 cm from the anterior lacrimal crest. The artery is identified and clipped.

In cases where bleeding persists, we have rarely had to perform ligation of one or both external carotid arteries, which can be exposed above or below the anterior belly of the digastric artery. However, embolization offers a good alternative, especially in cases of avulsive facial injury.

In CMF missile wounds, the carotid arteries and their branches may be severed. Surgical exploration and arterial ligation may be dangerous or impossible if the arterial wound is deeply sited. When the trajectory of the missile makes this likely, arterial embolization offers an alternative means of arresting bleeding. Dolin et al (1992) used angiography in 38 of 100 facial gunshot wounds, and coil embolization of bleeding arteries was employed in six cases. We have not yet had to employ this method of arresting bleeding in the CMF region, though it has been useful in other sites (Fig. 20.5).

It seems likely that interventional radiological measures to arrest deep bleeding will be used increasingly. Frerich et al (1991) review the options for intractable bleeding in midface fractures, and report success from selective embolization of the maxillary artery in three cases, one an 88-year-old man.

Laboratory investigations

Haemoglobin estimation, a full blood picture, serum electrolyte screen, protein screen and liver enzyme estimation are requested at once. A coagulation screen is carried out if any operation is contemplated (Table 20.2), or if bleeding has been copious. The blood group is determined, and blood matching is done if transfusion is likely to be needed. HIV and hepatitis antibody screening are requested in all cases where nursing or operative intervention will involve a risk of contamination by body fluids; in many countries, these tests require consent by the patient. In unconscious patients, the blood ethanol level is measured urgently; in our community, this is legally obligatory in all road crash victims over the age of 14.

Blood volume replacement

Estimation of blood loss is always difficult. Blood may have been lost at the accident site, or on the way to hospital. There may be much blood in the stomach, or lost from injuries elsewhere in the body. In children and young adults, intense peripheral vasoconstriction may maintain a normal pulse and systolic blood pressure until one-third of the blood volume has been lost. This does not happen in old people, who suffer marked falls in blood pressure after relatively small losses of blood; however, the elderly cardiovascular system may fail to respond by increasing the heart rate, especially if β–adrenergic; blocking drugs have been prescribed. Acceleration of the heart rate may interfere with myocardial perfusion: as the rate increases, there is an increase in the time of systole relative to diastole, and during the period of systole the myocardial perfusion virtually ceases—indeed the flow in the coronary arteries is reversed.

The central venous pressure (CVP) is most useful as a guide in giving blood replacement, which should aim to keep the CVP at 5-10 cm above the midaxillary line in young people, 10-15 cm in the old. A central venous line is therefore inserted as soon as possible. Care should be taken not to prick the pleural cavity, and a chest radiograph should be obtained after the line is inserted. A urinary catheter is also inserted to monitor urinary output. it. An arterial line is also inserted when the circulatory state is very unstable. The choice of

replacement fluid has been discussed: for reasons given above, we prefer to replace losses initially with colloid solutions and to transfuse blood to keep the haemoglobin level at ~10 g/dl.

The risk of wash-out coagulopathy arises when large volumes of blood have been replaced (p. 532). Wash-out coagulopathy does not occur until clotting factors have been reduced to 30% of the normal values; this is likely to happen after blood loss and replacement of about one blood volume.

Two units of fresh frozen plasma are therefore given when blood replacement approaches one blood volume. Blood coagulation studies are repeated, and also the platelet count, to see if specific haematological therapy, such as Prothrombinex or cryoprecipitate, is needed; platelet infusions are given if the count falls to < 60 000/ ml. Consultation with the clinical haematologist is desirable in such situations, and the possibility of pre-existing blood dyscrasia or aspirin ingestion should be kept in mind.

Control of raised intracranial pressure

Raised intracranial pressure is a surgical emergency, and progressive signs of raised pressure may call for very rapid intervention. This aspect of emergency management is discussed in Chapter 13, and here it is only necessary to note that the emergency treatment of a deteriorating head injury sometimes entails endotracheal intubation, hyperventilation and/or infusion of mannitol (0.5-1.0 g/ kg body weight as a bolus dose). Decisions on the management of such cases should be made jointly by the physician providing emergency management and the consultant neurosurgeon (Simpson et al 1988). We do not favour giving mannitol or other hyperosmolar agents as a routine measure. Steroid medication is no longer considered appropriate in the primary management of head injuries.

When a patient with CMF injury comes to the emergency room in status epilepticus, the fits should be controlled at once by intravenous diazepam (2 mg/ min to total 10 mg). If there is no response, thiopentone and possibly intubation and ventilation will be needed.

Eye protection

In any eye injury, and especially in penetrating injury, the prevention of further ocular damage is essential. This can be caused by the patient, the first aid attendant, the casualty staff, the anaesthetist, the radiographer, the nursing staff, or the surgeon. The nature of the injury should be explained to the patient; all staff members must understand the risks of the injury and how these can be prevented.

We urge that cases of suspected or verified penetrating eye injury should be transported flat with the head and neck supported. To avoid elevation of intraocular pressure, vomiting or coughing should be prevented. The eye should be protected by a lightly applied pad or shield, or by plastic film (Fig. 9.8); pressure on the globe should be avoided. If the eye has been injured by a chemical agent, the conjunctive should be irrigated thoroughly with normal saline and repeatedly from the time of injury until the patient can be assessed by an ophthalmologist.

Failure of vision is a surgical emergency, and should be discussed at once with an ophthalmologist. When caused by an increasingly tense orbital haematoma, there may be need for an emergency orbital decompression (p.422); there are other remediable causes of visual failure, and in all of them delay may be disastrous.

Implanted objects

In some cases of penetrating injury, a knife or other sharp object is left projecting out of the orbit or the skull vault. To minimize the risk of further damage to the eye or brain, it is best to leave the object in situ until the patient is anaesthetised; after appropriate radiological investigation which may include angiography, the

object can be carefully withdrawn. The possibility of bleeding after withdrawal should be kept in mind.

Pain relief

This requires care and experience; relief of pain is essential, but excessive doses of narcotics are dangerous, especially if there is a cerebral injury. In patients with severe pain, we give initially small repeated intravenous bolus doses of morphine until the patient is reasonably comfortable. The appropriate bolus dose becomes the guide to further intravenous dosage by continuous infusion. Large intramuscular doses of narcotic analgesics are to be avoided if possible. In the injured person, absorption may be delayed and variable (Masher et al 1975); the peak level may be higher than is necessary for analgesia, and the trough level may be inadequate. The result may be respiratory depression, vomiting and impaired conscious level. Where close medical supervision is not possible, as in an ambulance, self-administration of Entonox (50% nitrous oxide in oxygen) or Penthrane (methoxyflurane) have been recommended. These gases are potentially hazardous in patients with impaired consciousness; however, such persons rarely need analgesia.

Antibiotic prophylaxis

This is a contentious subject (p. 236). It is our practice to give prophylactic chemotherapy as soon as possible, for the following conditions:

Compound facial fractures

— intravenous flucloxacillin (usual adult dose 1 g 6 hourly; maximum 100 mg/kg body weight/day)

— intravenous metronidazole (usual adult dose 500 mg 12 hourly infused over 20 min; maximum 20 mg/kg body weight/day in 3 doses)

Compound calvarial fractures

— intravenous flucloxacillin only

Fractures of the skull base

— intravenous amoxycillin or ampicillin (usual adult dose 1 g 6 hourly; maximum 100 mg/kg body weight/day)

— intravenous or oral trimethoprim-sulphamethoxazole (usual adult dose two tablets or 10 ml by infusion 12 hourly; maximum 8 mg trimethoprim/kg body weight/day in 2 doses)

Penetrating eye wounds

— intravenous gentamicin (usual adult dose 120-160 mg 12 hourly)

— a third-generation cephalosporin, e.g. ceftriaxone, 1-2 g daily (maximum 50 mg/kg/day as single dose).

Our arguments for this policy of prophylaxis, and for these choices of antibiotics, are set out in Chapter 9 and elsewhere in relation to injuries of organs with specific drug barriers, such as the brain (p. 371) and the eye (p. 409); doses should be modified in cases of hepatic or renal dysfunction, and in children. These doses express our own practice, but we urge that each trauma unit should form its own policies in collaboration with a clinical pharmacologist, and our doses are not necessarily appropriate in all settings.

Special care is needed in patients with a history of drug sensitivity, especially sensitivity to the penicillins and the sulphonamides; in such cases, it may be wise to withhold prophylactic medication of any kind, since the arguments for prophylactic antibiotic medication are not unchallenged. The risk of cross hypersensitivity should be remembered; in perhaps as many as 9% of persons hypersensitive to penicillin, there is also hypersensitivity to cephalosporins (Garrod et al 1981).

Tetanus prophylaxis

This is needed unless the injured person is known to have been fully immunized within the last 10 years. If the record suggests that immunization was > 10 years ago, or if the wound seems especially prone to tetanus and immunization was 5-10 years ago, 0.5 ml adsorbed tetanus toxoid is injected. This is also given when the immunization status is unknown but the wound looks clean and not especially prone to tetanus. When the patient is not known to have been properly immunized, and the wound is associated with contamination or tissue damage, toxoid is given, and also passive immunization by human tetanus immune globulin (250 units). Full immunization by toxoid can be given later. Wounds in the CMF region are not as a rule prone to tetanus, since *Clostridium tetani* requires an anaerobic environment, but immunization is nevertheless essential. Facial burns should be regarded as prone to tetanus, since there is likely to be necrosis and ischaemia.

There is only one contraindication to tetanus immunization and that is a history of neurological or severe systemic reaction to a previous dose; a history of a local reaction need not preclude another dose (American College of Surgeons Committee on Trauma 1984).

Emergency operative intervention

In many cases of CMF trauma, definitive treatment should not be undertaken immediately: the best results are obtained by elective operation done after careful investigation and team planning. Moreover, injuries elsewhere in the body may take precedence. However, some forms of CMF injury have high priority and should be dealt with as soon as possible (Weaver 1988). These include:

1. Jaw fractures causing airway obstruction
2. Inhaled tooth fragments and dental plates
3. Open wounds of the eye
4. Open wounds, especially wounds of the lips, eyelids and face generally, and especially if bleeding (see above)
5. Incised wounds of the facial nerve
6. Open wounds involving the parotid or nasolacrimal duct
7. Craniocerebral injuries complicated by raised intracranial pressure, e.g. acute intracranial haemorrhage, intracranial aerocele
8. Open brain wounds.

These injuries are discussed in Chapters 11-15. It is necessary to emphasize that during the process of emergency assessment and resuscitation, the various injuries, are identified, and their priorities are determined (Mektubjian, 1982). In cases of multiple injuries, this process of triage is best done by a multidisciplinary team with a designated team leader.

Assessment of Viability

The team members may have to decide on the viability of the injured person, either in the emergency room or at a later stage in management. Some cases of CMF trauma are moribund when admitted; others become moribund from cardiac failure due to exsanguination, despite vigorous resuscitation; the decision to cease supportive treatment may have to be taken. More often, however, the cause of death after CMF trauma is failure of the brainstem centres as a result of irreversible cerebral swelling and/or intracranial bleeding. When brainstem death is established, further supportive therapy is not indicated. When the apparently moribund person is young (< 50 years) and previously in good health, then it is usually right to continue cardiopulmonary resuscitation and to look for a

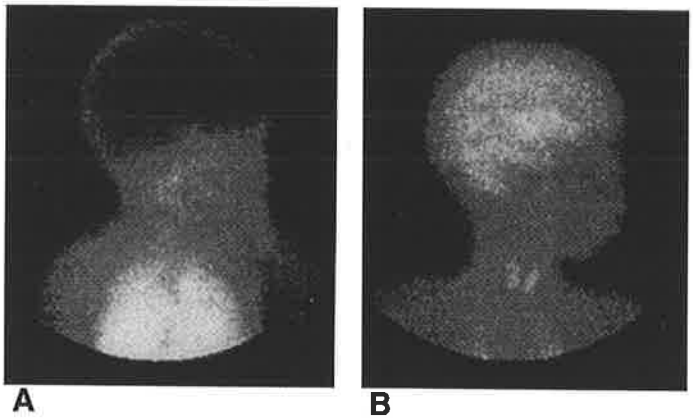

FIG. 8.5. ***Ceretec radioisotope angiogram: brain death.*** *A 2-year old child swallowed an insecticide and was admitted in coma. There was no recovery after a period of intensive care, and the criteria of brain death were evident.* **A.** *The Ceretec angiogram shows no cerebral circulation; there is a good circulation in the lungs.* **B.** *By comparison an older child with normal cerebral circulation.*

remediable condition, such as a ruptured intrathoracic or intra-abdominal viscus, or an intracranial haemorrhage. In comatose patients the possibility of a non-traumatic cause of coma (e.g. drug ingestion) should be considered. Even in older persons, efforts to restore cerebral perfusion may be justifiable, though the likelihood of a good response is much less, and the decision to cease supportive measures may be reached sooner. The previously expressed views of the victim may be helpful in this often difficult decision.

Brainstem death

The diagnosis of irreversible brainstem failure should be based on well defined criteria. In the UK, these were laid down by a joint conference representing medical surgical and anaesthetic authorities (Royal Colleges and Faculties in the United Kingdom, 1976). It is first necessary to exclude brainstem depression by drugs, metabolic abnormalities or hypothermia. A diagnosis of irremediable structural brain damage must be made. The following tests of reflex function should be made and should show no response:

— papillary light reflexes

— corneal reflexes

— vestibulo-ocular reflexes, in response to head movement and to irrigation of the external auditory canals with ice-cold water

— responses to pain in the cranial nerve territory

— gag reflex

— respiration in response to elevation of arterial carbon dioxide tension.

In our practice, the last test is given particular importance. The pCO_2 is allowed to rise to 50 mmHg while 100% oxygen is administered by catheter into the trachea and ventilation is ceased; apnoea is seen as evidence of brainstem failure if there is no evidence of a high cervical cord lesion. The tests are usually repeated independently after 30 min or more; this is always done if organ donation is intended. Reflex activity below the brainstem level need not negate the diagnosis of brain death; the term 'Lazarus sign' has been applied to such activity when seemingly integrated limb movements appear in a brain-dead patient.

The professional and legal criteria of brainstem death vary considerably in different parts of the world (Walker 1985); in some countries, the criteria include specific procedures such as cerebral angiography, to show absence of cerebral perfusion, and electroencephalograpy, to show absence of cortical electical activity. We have not used these procedures routinely, but have occasionally employed cerebral angiography in doubtful cases; it has a place when there are

confounding factors, such as a high cervical lesion or a high serum level of therapeutic barbiturate. We have also made use of nuclear scanning (Fig. 8.5). This has received greater acceptance with the advent of Ceretec ([^{99m}Tc]HMPAO), a lipophilic perfusion tracer that readily crosses the blood-brain barrier; with the aid of a bedside SPECT scan, it is easy to visualize the presence or absence of blood flow in the brain within 5-10 min after injection (Wieler et al 1993). It has however been found that in infants and young children, evidence of normal cerebral perfusion and active glucose metabolism does not exclude brain death (Medlock et al 1993). Doppler sonography has also been used but we have no personal experience of this method of verifying cerebral circulatory arrest.

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Definitive management: principles, priorities and basic techniques

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Introduction

This chapter considers the organisational structure of a craniofacial unit, and how it can be employed in the prioritization and definitive management of Craniomaxillofacial (CMF) injuries. Also considered are some of the basic policies and techniques used in combined operations for repair of complex injuries.

Organization

Facility and personnel

In the management of CMF injuries, an organisational structure should be established to allow the best use of resources and to provide appropriate levels of care. This structure is composed of two basic elements: the facility and the personnel. The term 'facility' comprehends the wards, operating theatres, diagnostic services, clinics and other hospital systems essential in the modern management it of trauma. The personnel comprise not only the committed core members of the multidisciplinary trauma team, but also the numerous less directly committed colleagues who contribute in the surgical treatment, nursing and rehabilitation of the injured patient.

Ideally, the facility should be established in a single location; if this is not possible then the locations must be geographically close to allow ease of access and intercommunication (Fig. 9.1). The facility should have a central location in relation to the main mass of the population to be served. Patients must be able to reach the facility with a minimum of delay and if possible there should be a nearby heliport or airport to enable rapid transfer of critically injured patients from outlying areas. The facility must have an adequately staffed emergency or casualty area with emergency operating rooms available 24 h a day. An intensive care unit will be needed to manage the critically injured patient. Relevant specialist services must be available immediately. These include diagnostic imaging services, medical laboratory and haematology services, and blood transfusion services, as well as the relevant clinical departments. The ideal facility for managing CMF injuries should have geographical proximity to a university campus. This will allow the very important activities of teaching and research to be undertaken with appropriate academic support and rigor. These are further discussed in Chapter 23.

Personnel with a special interest in CMF trauma bond together as a team dedicated to managing these injuries. They may be part of an extended craniofacial team already in existence. The trauma subsection of the extended craniofacial team has the following members: craniofacial surgeons (p. 683), neurosurgeons, ophthalmologists, specialist anaesthetists, ENT surgeons, and dental specialists

including dentists, orthodontists, and restorative dental experts. Psychosocial workers and specialist nurses are further important members of such a team. Working as a dedicated team, the group constitutes a structure which provides ease of communication and referral within its membership, and also an ability to collect data both for research and for regular peer reviews of all aspects of CMF injury management. Peer review is an intrinsic component of the team concept, and this presupposes an efficient record system and a good library service.

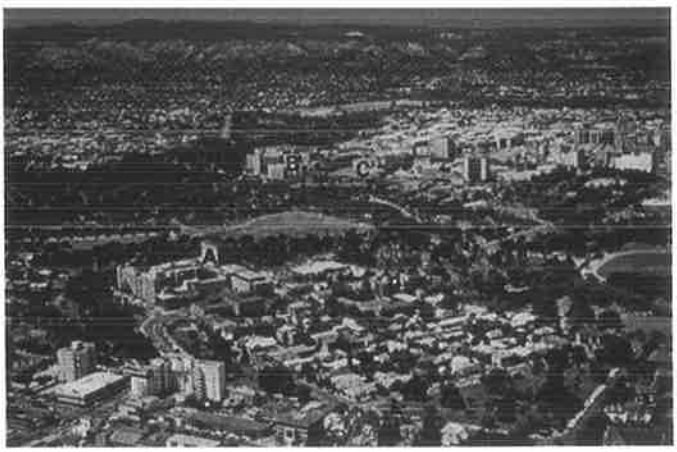


FIG. 9.1. ***Adelaide, South Australia.*** *Ariel view of the Women's and Children's Hospital* **A.** *the Royal Adelaide Hospital* **B.** *and the University of Adelaide* **C.** *Their close proximity to each other and to the city centre facilitates the management of major trauma. The sports field between the hospitals has been used for helicopter landings; direct landing on a hospital building is preferable.*

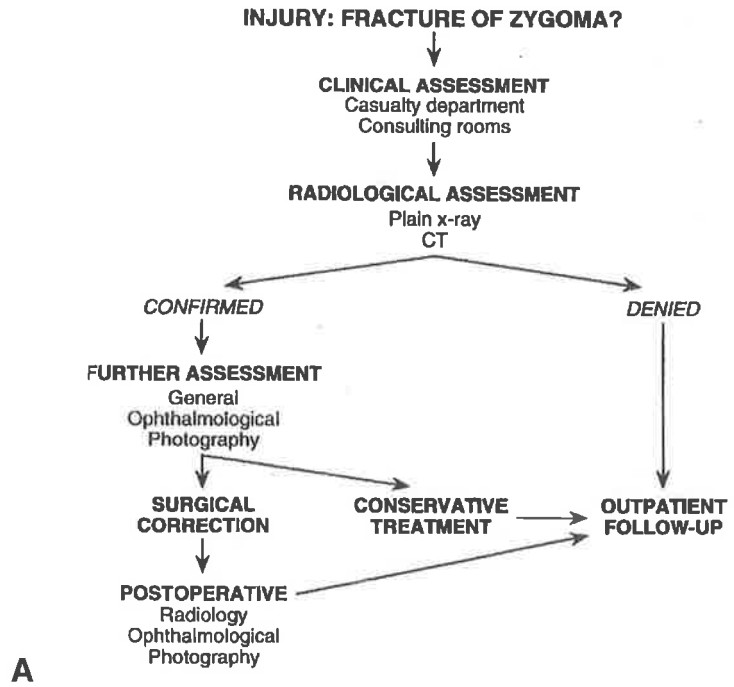
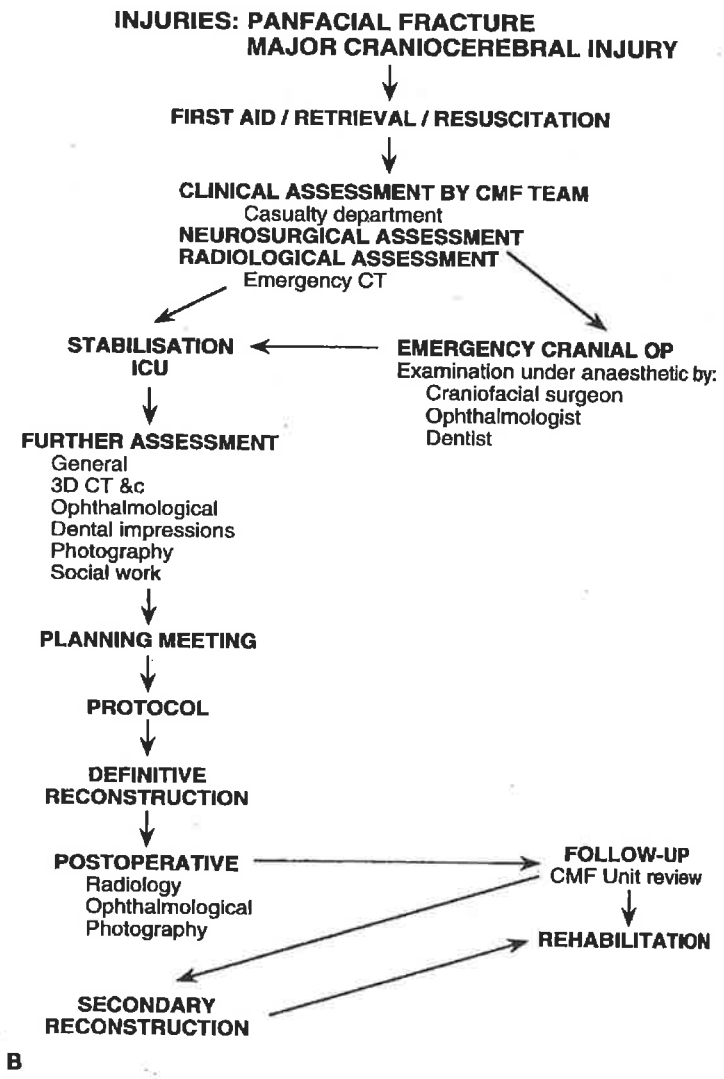


FIG. 9.2. ***Trauma management by a CMF unit.*** *The skills of a multidisciplinary unit can be deployed in trauma management in response to the pattern and severity of the injuries.* **A.** *A unidisciplinary problem, such as a possible depressed fracture of zygoma, engages the attention of one specialist, In this case a craniofacial surgeon. Investigation, diagnosis, treatment and follow-up are coordinated by the same unit. A similar scheme is applied in the management of an isolated corneal wound, or depressed skull fracture.* **B.** *A multidisciplinary problem, such as a panfacial fracture with a major craniocerebral injury, engages the attention of a number of specialists. In this case, the neurosurgeon initiates urgent treatment, but the craniofacial, ophthalmological and dental specialists are consulted as soon as possible, and the further management of the case is coordinated by the multidisciplinary team.*



B

Admission policies

After the emergency assessment and resuscitation (see Chs 6 and 8) come the allocation of immediate priorities and the decision on admission to specialized departments (Fig. 9.2). In our system of management, the patient with multiple injuries is admitted under the care of the hospital department appropriate for the most serious injury. A severe brain injury is admitted under the neurosurgery department, a proven or suspect ruptured abdominal viscus is admitted under the general surgery department and a case of spinal injury under the orthopaedic department. Facial fractures occurring in isolation or in the presence of less severe injuries are admitted to the plastic surgery department under the care of the on-call craniofacial specialist; in many trauma services, the oral surgical department has this role. Isolated eye injuries are admitted to the ophthalmological department. After admission, baseline investigations and consultations within the CMF trauma team are carried out. Data from the clinical, laboratory and radiological investigations described in Chapters 6-8 are combined in a work-up which allows the formulation of a plan of management appropriate to the unique needs of the individual patient.

Specialist clinical assessment and consultations

The craniofacial surgeon examines the patient and the X-ray pictures shortly after admission. Diagnosis of the bony and soft-tissue injury is then confirmed and recorded. All proven or suspected craniocerebral injuries are assessed by a neurosurgeon if this has not been already done in the emergency department.

All eye injuries and all orbital fractures require an ophthalmological consultation. All jaw fractures affecting the occlusion are seen by the team dental consultant, who inspects teeth for traumatic damage and takes dental impressions of both upper and lower jaws (p. 355). These impressions are used to cast plaster study models for planning surgical reduction and fixation. All consultants record their findings, diagnoses and recommended management plans in the case records.

The anaesthetist or intensivist reviews the progress of the case in the light of the serial clinical records (p. 160) and laboratory tests to confirm that the pathophysiological effects of trauma, especially hypovolaemia, anaemia and electrolyte disorders, have been corrected. The definitive anaesthetic assessment may be deferred until the operative management plan is prepared. This assessment is of great and sometimes decisive importance, especially in the elderly and where there is some significant pre-existing disease (see Chs 19 and 20).

Prevention of infection

While the multidisciplinary assessments are being made, measures are instituted to minimize the risk of infection.

Microbial infection is a threat after any open wound or any surgical operation, whether done electively or as an emergency. In the CMF region, infection may be disastrous. Cerebral infection may cause death or severe disability (p. 387). Ocular infection may cause blindness (p. 416); postoperative facial infection may lead to rejection of bone grafts and prolonged morbidity. Craniofacial reconstructions entail substantial risks of infection: they are often long, they may expose cavities that cannot readily be sterilised, and there may be postoperative dead space, especially in the anterior cranial fossa. In early reports on craniofacial surgery, high infection rates were recorded in our own initial experiences, we had an overall incidence of 6.5% infection in 170 transcranial procedures of all kinds, and the incidence was especially high in adults with pre-existing tracheostomies (David & Cooter 1987).

Given the nature of CMF trauma and the orofacial microbial environment (p.119), it seems unlikely that any surgical routine will wholly eliminate post-traumatic infection, but every effort must be made to reduce the incidence and the severity. Experience and theoretical considerations support:

1. Early wound toilet, appropriate debridement, and early primary wound closure
2. Operative planning to reduce wound exposure time, and to avoid procedures known to increase the risk of serious infection, such as tracheostomy and prolonged ventricular drainage
3. Elimination of local sources of microbial infection
4. Pre- and peroperative prophylactic antibiotics
5. Nutritional support.

It is not difficult to justify these ideals as generalisations, but in reality, many compromises must be made. Wound closure may have to be deferred because of other more urgent problems. Debridement of cerebral wounds and the removal of deeply placed foreign bodies may conflict with the paramount need to conserve cerebral function (p. 373). Bactericidal skin cleansing and prophylactic chemotherapy must be used with discrimination and at the right time.

Skin wounds must be cleaned and dressed; in our service, gauze packs moistened with saline and changed every 2 h are considered to be the most effective; the techniques used in skin lacerations and abrasions are discussed in Chapter 15.

With jaw fractures, the care of the mouth is important. Nursing staff may

have to pick away dried blood from inside the mouth; moistened probes and forceps are useful in this. An appropriate diet is prescribed with the help of the hospital dietician; this will usually need to be in a non-chew or liquid form. Oral hygiene is maintained with mouth washes of chlorhexidine or other antiseptic; attention to this after each meal is mandatory. Where eyelids are swollen, eye toilets may be needed as the poorly draining tears rapidly become mucopurulent. Eye ointments may also need to be used where eyelid closure is compromised (p. 246).

Theatre aseptic routine must be meticulous, especially where two or more disciplines are joined in a combined procedure: often, each has its own different rituals, and it is necessary to combine these, even if the resultant procedure appears cumbersome, to avoid a perception by staff members that their standards have been lowered. Preoperative 'disinfection' of the skin and the various facial cavities has limited value. Complete sterility is unattainable in the mouth, and in the skin, it is impossible to sterilize the hair follicles and skin glands (Lowbury 1981). However, it is possible to reduce the bacterial count very substantially by an appropriate bactericide; we favour povidone-iodine (Betadine®) applied with sponges a few minutes before incision. Scalp wounds are covered with Ioban®, a polymeric plastic film coated with a bactericidal iodophor.

The value of prophylactic antibiotics has been disputed, both for facial fractures and for craniocerebral wounds. It has been shown that prolonged use of antibiotics promotes the growth of resistant organisms in the pharynx (Ignelzi & Van der Ark 1975) and elsewhere. However, there is now much evidence to support the routine use of peroperative chemotherapy in elective orthopaedic and vascular surgery (Strachan 1993) and in elective neurosurgical operations (Young & Lawner 1987); we believe that this experience supports a similar policy with CMF trauma. The choice of antibiotic depends partly on the local microbiota and the likely pathogens, and partly on the capacity of the chosen antibiotic to cross the blood-brain and blood-eye barriers, and to penetrate bone.

With the exception of isolated fractures of the zygomatic arch and condylar fractures, facial fractures are usually compound through mucosal surfaces. Some authorities, relying on very early rigid fixation, do not give prophylactic antibiotics. Our plan of management does not require such early intervention as a routine, and we give prophylactic antibiotics to all patients with compound facial fractures, usually intravenously (David & Cooter 1987); in our present management plan, flucloxacillin and metronidazole as a combination are used (p. 229). This regime is continued peroperatively and for 48 h thereafter; in some cases, oral antibiotics may be continued for a further 5 days. The roles of prophylactic antibiotics in craniocerebral wounds and cerebrospinal fistulas are discussed in Chapter 13. In our practice, flucloxacillin has been given for compound skull fractures and for penetrating wounds of the brain, while patients having CSF leakage receive in addition co-trimoxazole (a sulphonamide-trimethoprim combination); this drug penetrates the blood-brain barrier. For calvarial compound fractures, metronidazole is omitted unless there is special reason to fear infection by a gram-negative organism. Hitherto, we have not given cephalosporins prophylactically from fear of promoting resistance; however, in view of its good penetration across the blood-brain barrier, ceftriaxone may prove to be a justifiable choice (Demetriades et al 1992). For open wounds of the eye, we favour a third generation cephalosporin and an aminoglycoside (p. 409).

Great care is needed in respect to antibiotic dosages, both for prophylactic and for therapeutic purposes. In this book, the dosages suggested for the various antibiotics are those recommended in our hospitals (p. 229); opinions on the choice and the dosages of many antibiotics vary, and it is wise to formulate general policies in consultation with a clinical pharmacologist and a clinical microbiologist. Similar consultations are also desirable in individual patients with unusual or life-threatening infections in any site, or when there is evidence of drug sensitivity.

Topical antibiotics, in powders, sprays, or irrigations, remain a controversial

subject; Haines (1982) found no evidence to support their use in clean neurosurgical procedures, though he saw applications in treating heavily contaminated wounds. We routinely immerse bone grafts in a solution of 0.2% flucloxacillin during facial reconstructions, and some use is made of this solution in irrigating the extradural space. All the penicillins are epileptogenic, and although small doses (e.g. 10 mg penicillin G or methicillin in 5 ml Ringer's solution) have often been used by intrathecal or intraventricular injection, we do not advise this for prophylactic purposes.

Topical antibiotic ointments and eyedrops are often used in ophthalmological injuries, but should be chosen and used with some care, especially in the presence of a penetrating eye wound. In the treatment of some eye infections, intravitreous chemotherapy may be necessary, and this should be given in precisely calculated doses and concentrations known to be within tolerated limits. Chloramphenicol eye ointments and drops have been incriminated as possible causes of blood dyscrasias, and should not be used unless there is no effective alternative (Fraunfelder et al 1993).

Multidisciplinary planning

Once all necessary assessments are completed an operative plan is produced. If other specialists are to be involved in the surgery then discussions will be held at an interdisciplinary planning meeting (Fig. 9.2B). As a result of this meeting a protocol is produced discussing the proposed airway management, the order of exposure, the method of fixation and any ancillary procedures. This protocol is circulated to operating theatre, anaesthetic and ward nursing staff, as well as to the members of the relevant departments.

Principles of Management of Facial Fractures

Priorities

When a facial injury obstructs the airway or causes massive bleeding, immediate action is needed, but the definitive treatment of facial fractures is rarely urgent and can be carried out as one or more elective procedures. In recent years, there has been a trend towards earlier intervention and to the use of combined procedures in an attempt to minimize morbidity and shorten time in hospital (p. 267). Arguments for early surgery of facial fractures include the belief that reconstruction of the bony framework should be done before the formation of contractile scar tissue (p. 123) which may prevent the correct placement of soft tissues, particularly those within the orbit (Gruss 1990). Our experience with secondary surgery indicates that this argument is not always valid. We prefer to delay surgery until the patient's general condition has stabilised, the swelling has largely settled and the dental and ophthalmological work-ups are complete. We also feel that surgery should be performed on elective operating lists and not in emergency suites at odd hours of the night. We argue that this allows the development of better operative techniques and better teaching, without detriment to the final result. There are some exceptions to this timing policy. Early bony reduction and stabilisation are carried out in massive open wounds where there has been significant soft-tissue disruption or loss (p. 450). Even in these cases, however, the bony reduction and stabilisation may be only a temporary splintage done at the time of soft-tissue repair with a view to redoing the bony reconstruction once the full work-up is completed. Minor, uncomplicated, undisplaced or minimally displaced fractures will go on the next elective operating list following completion of work-up.

Operative procedures

Reduction and fixation
The principles of surgical treatment of facial fractures have evolved rapidly over

the last 30 years (p. 27). At the beginning of this period mandibular fractures were immobilized by using the teeth as external fixateurs with various types of intermaxillary splints; they were reduced in a closed fashion and stabilized with these splints. Maxillary fractures were also reduced by closed methods and stabilized with an external frame (Fig. 1.16) or internal suspension wiring (Fig. 1.21). Orbital fractures were also reduced by closed methods, often without attempt to provide stabilisation. These methods were often satisfactory, but sometimes led to secondary deformities such as enophthalmos and foreshortening of the midface. Following the advent of craniofacial surgery in the late 1950s, emphasis moved from closed reduction to wide exposure and open reduction of all facial fractures and stabilization by interosseous wiring (Gruss et al 1985), now virtually replaced by the use of plates and screws. At the occlusal level the teeth are now of secondary importance in fracture fixation, while at the midface and orbital levels, primary bone grafting has significantly reduced problems of enophthalmos and midfacial shortening (Luce 1992).

Craniofacial instruments

It is important to have appropriate instrumentation to carry out the exposure required to visualize the facial fracture pattern and to provide stabilisation. Soft-tissue dissection is done with fine sharp craniofacial surgery instruments. A range of periosteal elevators is essential, we use the Obwegeser type. The smaller periosteal elevator is used within the orbit and in areas of sharply changing contour while the large instruments are used over broad flat surfaces. Malleable metal brain retractors of various sizes are used to retract orbital and cranial contents. Rigid blade retractors are used to retract the lips and cheeks over the jaws and face. Metal cheek retractors and metal tongue depressors are used in wiring dental arch bars to the teeth. We prefer the soft metal dental arch bars if possible as these are less damaging to the gingiva. In adults, 26 gauge stainless steel wire is used to locate the arch bars to the teeth; in children, 28 or 30 gauge stainless steel wire may be used. Where wiring is to be used for interosseous stabilisation then similar gauges of wire are used. A power drill is used to drill holes in the bone for passage of the wire; we use the Hall drill powered by compressed nitrogen. Electric drills have the advantage of being more portable.

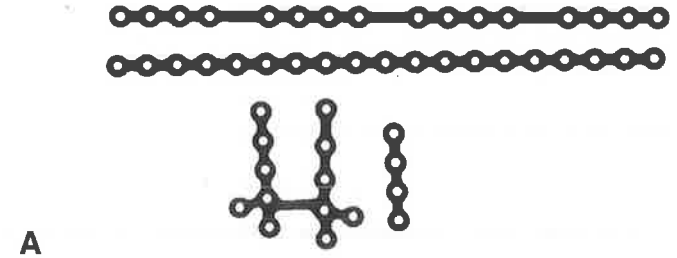
Plates

While intraosseous wiring is used successfully in many centres the advent of miniplating systems with their ability to provide three dimensional stability has been a great advance in fracture management. Plates secured by small self-tapping screws give rigid bone fixation and rapid union. Among the miniplating systems available are the Luhr Vitallium® plates, the Würzburg titanium system, and the plates designed by Champy and by the AO group (Hobar 1992) (p. 270). At our institution we use AusSystem® plates devised and produced in Adelaide (Fig. 9.3).* These plates are made of titanium and unlike the Vitallium plates of the Luhr system they do not scatter radiation which may affect postoperative computed tomography (CT) scans adversely. Furthermore the AusSystem plates have an advantage over the Wurzburg system in that they are much more malleable and easily moulded to the contour of the bone, thereby facilitating stabilisation of midface fractures and positioning of bone grafts; they can be moulded by screw pressure, and do not show 'memory'— that is, they do not tend to recoil when other forms of fixation have been released.

Miniplates may cause noticeable deformity when placed on prominent parts of the facial skeleton, in sites such as the orbital rims and the bridge of the nose, where there is relatively lithe subcutaneous tissue. The plate and screw heads usually become palpable and even visible Through the skin. This may be prevented by countersinking the miniplate and screws, or by the use of interosseous wires. Alternatively, one may use the microplate systems, developed in titanium by Leibinger and in Vitallium by Howmedica. The components of these systems are

* AusSystems Pty Ltd. 116 Melboume Street, Adelaide, SA 5006, Australia.

no higher in profile than 28 gauge wire, and unlike wire they confer three dimensional stability. It remains to be proved that microplates will stabilize fractures under high load stress, but their use in frontal and orbital rim fractures has helped to solve the problem of miniplate prominence. Special mesh-like microplates can also be used for orbital wall reconstruction, in conjunction with bone grafts) however; these are still under trial.



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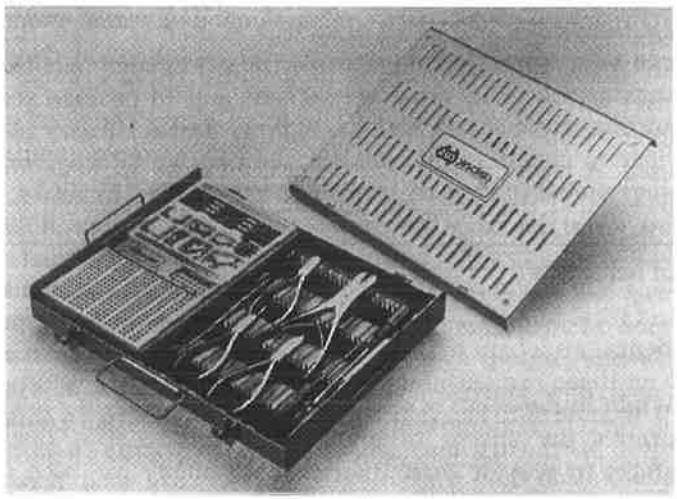


B

FIG. 9.3. ***Titanium miniplates.*** *The AusSystem® plating set.* **A.** *A range of AusSystem plates, comprising low profile screws and plates for frontal bone and orbital rim use as well as heavier but malleable plates, suitable for mandible and maxillary reconstruction* **B.** *Instrument kit for AusSystem plates.*

Craniofacial exposures

As far as possible, surgical exposure of the facial skeleton is carried out without leaving visible scars. In the upper facial skeleton this has been made possible by the use of the extended bicoronal scalp incision. The exact line of this incision may be dictated by the need to deal with cranial components of the fracture or by the site of the patient's hairline. The bicoronal flap is raised in a subgaleal plane to a horizontal level 2–4 cm above the supraorbital rim or to the site of any fractures of the frontal bone. From this level dissection proceeds subpericranially over the orbital rim and into the orbit. The supraorbital neurovascular bundle is carefully preserved; it is usually necessary to free it in the supraorbital notch by cuts with a fine chisel. Over the temporalis muscle it is important to dissect strictly against the tempera fascia or to incise through the superficial layer of the temporal fascia and dissect in the subfascial fat down to the zygomatic arch (Fig. 9.4). At the level of the zygomatic arch the periosteum must be stripped with great care particularly on the middle and posterior thirds. This may not be easy where fractures are present. Only by careful attention to these details will damage to the frontal branch of the facial nerve be avoided. Within the orbit the periorbita is dissected well back to the apex of the orbit. Where orbital fractures are present the orbital soft tissue may be trapped between the bony fragments and these must be separated to release the soft tissue. This is mandatory for fractures of the orbital roof and medial orbital wall as well as for complicated lateral orbital wall fractures. The temporomandibular joint and neck of the condyle can be approached by extending the bicoronal flap incision in the natural

contours down to the lobule of the ear. Careful dissection of the soft tissue from the lateral ligaments of the temporomandibular joint capsule visualizes the neck of the condyle and the sigmoid notch. Further access can be gained by dividing the most posterior fibres of the masseter muscle, and a subperiosteal dissection can be taken down to the angle of the mandible. Again, care should be taken to avoid excessive traction on soft tissues which may produce a temporary palsy of the frontal branch of the facial nerve (Fig. 9.4C).

A subciliary lower eyelid incision or transconjunctival incision (David 1974), with or without lateral canthotomy, produces excellent exposure of the orbital floor (Manson et al 1987; Fig. 9.5). When the lateral canthotomy is performed it can be extended up to provide exposure not only of the floor and inferior rim of the orbit, but also of the lateral orbital wall and rim as far as the frontozygomatic suture. Combined with a bicoronal approach this will allow visualisation of the entire orbit and any fractures in its vicinity.

Intraoral incisions in the upper or lower buccal sulcus provide exposures of the remaining facial skeleton with the exception of the pterygoid plate (Fig. 9.6). Care must be taken to avoid damage to branches of the infraorbital nerve in the upper buccal sulcus incision and to the inferior dental nerve in the lower buccal sulcus incision. Because of the risk of damage to the inferior dental nerve in fractures of the edentulous mandible, we prefer an external submandibular incision in such cases; this incision can also be used in the reduction and fixation of difficult mandibular angle fractures (Fig. 9.4C). When lacerations are located immediately over a fracture, they can be used in exposure.

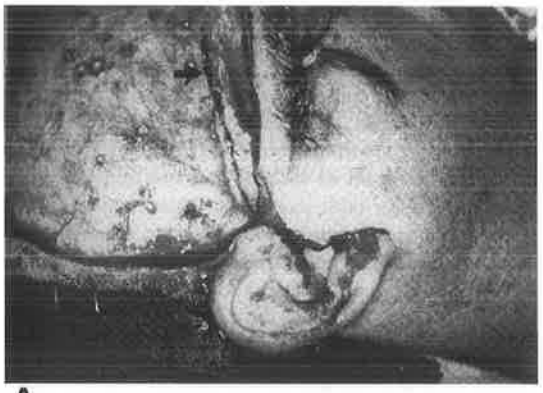
Bone grafts

The practice of wide exposure and open reduction of facial fractures has highlighted the need for bone grafting in many situations. This is particularly so when bone is found to be missing from the vertical bony pillars of the midface in association with jaw fractures involving the occlusion (Gruss & Phillips 1989). The pillars must be reconstructed to allow the correct vertical height of the midface to be restored and also to stabilize the upper alveolus during the action of chewing. The egg-shell bone of the floor and medial wall of the orbit is often lost in orbital fractures (p. 327) and for adequate reconstruction bone grafting is commonly required. Primary bone grafting is sometimes needed to reconstruct deficits in the orbital rim, or to correct the nasal bridge line where there has been severe comminution of the nasal bone and bony nasal septum resulting in total loss of support to the nasal pyramid.

Gunshot wounds may result in a significant loss of bone, especially in the mandible; when this loss is > 3 cm in length we prefer to use vascularized bone to give more certain healing. This may be a free graft of iliac bone taken on the deep circumflex iliac arterial system, or may be some other appropriate free flap (p. 622). The most commonly used sites for harvesting bone graft are the iliac crest, the rib cage and the skull vault (calvaria); Salyer & Taylor (1987) have reviewed their merits. Many factors are important in increasing the chances of survival of bone grafts, but perhaps the most important is rigid fixation followed by good soft tissue coverage preferably with a periosteal surface (Rahn 1989). Other factors promoting graft survival are discussed in Chapter 5; these include a well vascularized bed in the host tissues and prevention of haematoma formation.

Hip grafts

The wing of the ilium is perhaps the most versatile of all bone graft sites. Large pieces can be harvested, and the structure of the ilium offers a range of cortical and cancellous grafts. Cortical bone plates taken from the ilium are strong yet malleable, and are far less brittle than calvarial bone; they provide excellent reconstructive material for the orbital walls and also for mandibular reconstruction both as vascularized and non-vascularized grafts. The inner table can be harvested separately. After removal of a small amount of inner table,



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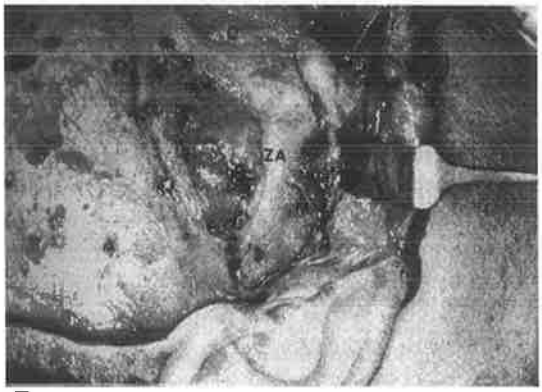

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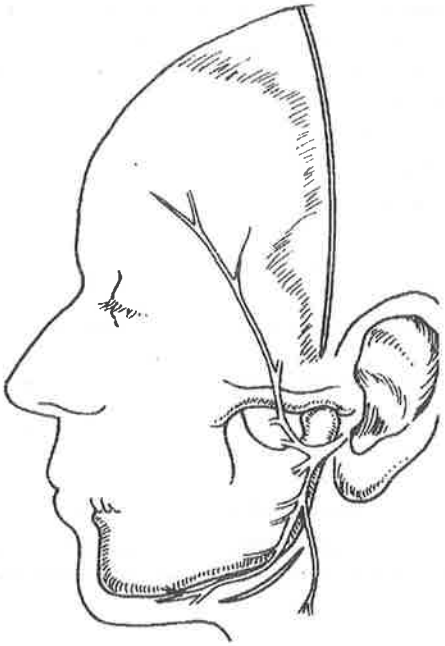
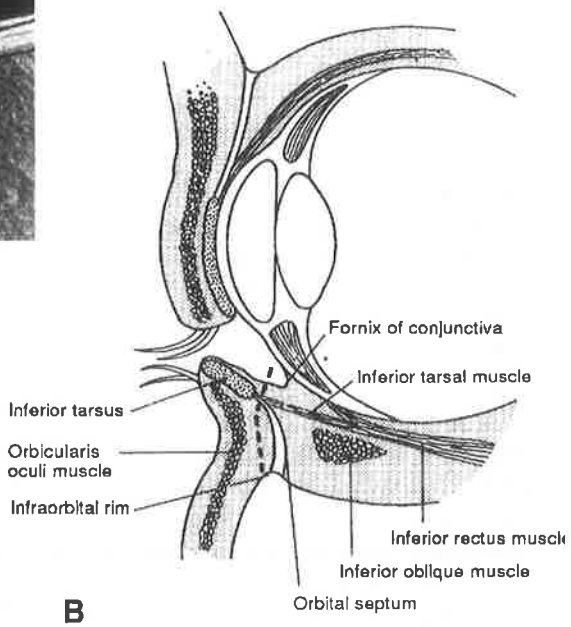


C

FIG. 9.4. ***Exposure through coronal scalp incision.*** *A. The bicoronal scalp flap is elevated over the superficial temporal fascia. On the level of the superior orbital rim (line indicated by an arrow), the fascia is incised and dissection then proceeds in a plane deep to the superficial temporal fascia down to the zygomatic body and arch. This dissection protects the frontal branch of the facial nerve. B. The zygomatic arch (ZA) and the upper part of the masseter muscle (M) are then exposed. The orbital contents (O) are also exposed. Dissection is subperiosteal over the bone and deep to fascia over the masseter. C. The diagram shows the incision in relation to the frontal branch of the facial nerve. Also shown is the relation of the submandibular incision to the mandibular branches of the facial nerve.*

A



B

FIG 9.5. ***Conjunctival incision.*** **A.** *When extended through the lateral canthus, wide exposure of the inferior and lateral orbit is obtained through this approach. The incision is taken across the conjunctive just inferior to the tarsal plate.* **B.** *Dissection (broken line) is carried deep to the orbicularis oculi muscle to the orbital rim, where the periosteum is incised. Subperiosteal incision then exposes the inferior orbital rim, the orbital floor and the lateral orbital wall and rim. Diagram by S. Cantrell*

cancellous bone may be scooped out and used alone or with cortical bone. For strong grafts, both tables can be taken. It is important not to take the crest itself, both in adults and in children. Preservation of the crest with its attached muscles preserves the contour of the hip. If the scar is well placed even the skimpiest of undergarments will conceal it. In children the iliac crest is still in part cartilaginous up to 12-13 years of age; depending on the developmental state of the child, the crest may be cartilaginous even at somewhat later ages.

FIG. 9.6. *Upper buccal sulcus incision.* *This is used to expose the anterior maxilla. Care should be taken to avoid damaging branches of the inferior orbital nerve. In this illustration, a comminuted fracture of the zygomatic buttress is seen, associated with a fractured zygoma. A large fragment of the buttress has been temporarily stabilised with an interosseous wire. Part of the anterior maxilla is missing, exposing the maxillary antrum.*

Technique. In the older child or adult the hip can be harvested by a second team working while the major craniofacial dissection is under way, thus saving time. A sandbag is placed under the hip. The skin incision is made 2 cm below the crest on the lateral side; if the skin is rolled medially the cut can then be made straight down to the bone. The dissection of the ilium proceeds according to the size of the graft to be taken. Often, only a small amount of bone is required, such as a disk for a 'bath-plug' reconstruction of the frontal region or when a small medial cortical window is opened for the harvesting of some cancellous bone. In such cases, all the muscular attachments of the crest are preserved and a cut is made down to the medial side of the periosteum which is stripped exposing the blade of the ilium directly. Alternatively the medial and lateral lips of the crest may be split free with an osteotome leaving the muscle and periosteal attachments on the mobilized segments of bone, thus exposing the ilium on one or both sides (see Fig. 9.7). At the end of the procedure, the bone segments can be reattached with very strong nylon sutures or occasionally wires. It is important to step the bone at the point of severance with the adjacent anterior spine or posteriorly; so that the reconstructed segments do not collapse after the wound is closed. If the cut is made a little more medially then the lid can be lifted off and hinged on the lateral periosteal attachments. This lid may then be replaced quite easily on the pre-cut steps. It is our custom to drain the space carefully with suction drains and we use the reciprocating power saw or the sagittal power saw to facilitate the taking of the graft.

Rib grafts

Rib grafts can be readily harvested by a submammary incision and large lengths are available. Rib has the advantage of being malleable. It is useful within the orbit, for reconstruction of the calvaria (Fig. 17.20), and as a costochondral graft in the nose or temporomandibular joint. In our practice, rib is most often used for reconstruction of the cheek bone, often as an onlay graft, and for the costochondral grafting techniques.

Technique (Fig. 21.6). The incision should be at the level of the submammary fold in women and at the equivalent in men. It is possible to harvest

ribs through relatively short (5-6 cm) incisions, and great cuts should not be made across the chest. It is important to have a good assistant and proper instruments. The initial cut is made onto the preselected rib after injection of a solution containing 0.5% adrenaline. The periosteum can be raised quite easily, and using a sharp elevator the superficial surface of the rib can be exposed. The initial dissection on the deep side of the rib may cause trouble; damage to the neurovascular bundle may occur, and occasionally damage to the pleura. Pleural damage is an unusual event and is easily repaired with a few sutures providing that the lung has not been damaged. A Doyen's rib raspatory or a similar curved elevator is then inserted in the small tunnel under the rib, and the deep periosteum stripped. One end of the rib is divided by lifting it and dividing it with rib shears; by holding it, delivering it further into the wound, and retracting firmly, a large segment can be obtained. If more than two ribs are needed, it is important to harvest two, leave one and then harvest a third, to prevent a flail chest. It is important not to leave jagged cut ends of the rib—they may cause damage to the lung and pleura—and to secure bleeding carefully. It is also important to resuture the periosteum very carefully as ribs reconstitute and may be reharvested if necessary. The quality of rib graft is variable. Sometimes the rib is so soft that it is hardly usable; in other subjects, it provides stout and useful material with good cancellous content. The rib is split on a separated table. It is held by an assistant with a bone-holding forceps. A sharp osteotome, usually without the use of a mallet, is worked from one end of the rib along its edge in a rocking motion to split the rib. In poorer quality ribs which are brittle and without much cancellous content one may have to use a mallet or even a saw.

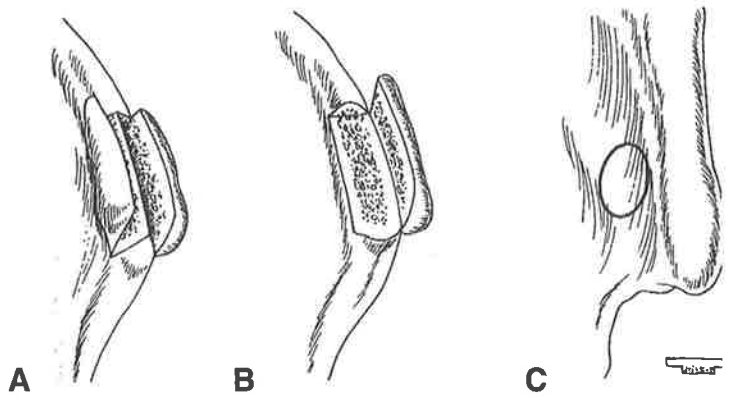


FIG. 9.7. ***Bone graft from the iliac crest.*** **A.** *Incision over the iliac crest, down to the periosteum; lids of bone and periosteum are hinged medially and/or laterally to expose the bone which can then be harvested. The crest is then reconstructed by wiring or stitching the lids back.* **B.** *Incision is made down through the periosteum over the crest and a segment of bone is lifted to medial or lateral side. The segment can be wired back into position after harvesting.* **C.** *A circular trephine graft is taken with cortex and attached cancellous bone like a bath plug (see inset).*

Calvarial bone

This is a popular source of bone grafts because of a lack of donor site morbidity; there is also a widespread but unsubstantiated view that calvarial bone is better because it is developed in membrane and hence more suitable for reconstruction of the facial bones. It does have the drawback of being more rigid than either iliac bone or rib. The services of a neurosurgeon on the craniofacial team are occasionally needed. A secondary contour defect may result if the edges are not smoothed as much as possible. It should not be necessary to point out that in harvesting calvarial bone, the sagittal sinus and its parasagittal venous tributaries should be avoided, and great care should be taken to preserve the dura mater; anecdotal accounts of dural damage abound.

It has been said that the parieto-occipital area over the nondominant hemisphere is the donor site of choice. This seems to imply that there is a significant potential for cerebral complications. This implication should be

dismissed: there should be no cerebral complications, and brain injury in this site is as reprehensible as in any other.

Technique. When a craniotomy is being performed, the inner table can be split from the bony flap raised by the neurosurgeon. In other situations, the outer table can be safely split from the skull where there is a reasonably well formed diploic space as is usual in the parietal area. It is important to view the X-ray pictures before operation as there is a wide variety of diploic anatomy and some patients have thin skulls. Where the coronal incision is being used, the posterior flap can be dissected backwards and an area of pericranium exposed. When one wishes to take only the outer table, for example to repair the orbital floor, an area of bone is defined by cutting through the pericranium around it. The pericranium is then dissected away from the proposed bone graft site, leaving a patch of pericranium overlying the bone to be taken. A trench is then cut with a large flame-shaped burr through the outer table into the cancellous bone. A thin curved osteotome is then worked around the periphery of the graft until it is freed. Sometimes the outer table is very brittle and by maintaining the pericranial cover, the bone fragments will be held together. Where calvarial bone is needed for a large cranioplasty then almost all the parietal bone should be harvested by the neurosurgeon and very carefully split using fine osteotomes and power saws. The inner table is usually replaced in the donor (parietal) region, the outer table being used in the cranioplasty. For an isolated harvesting not done through a bicoronal scalp flap, a transverse or oblique scalp incision above the ear can be used (p. 557).

Done by a proficient surgeon, this is a very safe procedure; damage to the sagittal sinus or to the brain has nevertheless been recorded, and postoperative intracranial haemorrhage is also a possible complication. Calvarial grafts should not be harvested by operators unfamiliar with the technique (Kline & Wolfe 1994), and if there are any signs of neurological impairment after such an operation, full evaluation, including CT scanning, should be done at once.

Other bone donor sites

The anterior surface of the tibia has been used in the past, but we have no experience of its use in CMF repairs.

Vascularized bone grafts have been taken from the humerus, radius, scapula, and clavicle, as well as from the ilium and the calvaria; our preferred techniques of revascularized free grafts are discussed on p. 626.

Cartilage grafts

Costal cartilage can be harvested as described above and used in small orbital floor defects which can also be repaired with cartilage harvested from the nasal septum or the concha of the ear.

Alloplastic materials

Synthetic materials, including Silastic® and Teflon® sheeting and hydroxyapatite, have all been used in attempts to reconstruct the fractured orbital floor, and as onlays in restoring facial contours. They are easy to use, do not absorb, and have no donor site morbidity, but the risk of delayed infection and movement or extrusion make them a second choice when compared to the advantages of autogenous materials. Alloplastic materials (titanium, acrylic etc.) are widely used to cover calvarial bone defects and are often very satisfactory, although delayed infection and exposure sometimes occur. In Adelaide we favour the use of titanium plates, either shaped by hydraulic pressure (Blair et al 1980) or cast in a mould modelled from CT scan data by the CAD/CAM (computer-aided design and computer-aided manufacture).

Fixation techniques

There are certain sites where secure fixation should be achieved; these include the horizontal and vertical buttresses of the face. These buttresses are the

framework or structural pillars of the face and stabilisation of properly reduced fractures in these areas will re-establish normal skeletal anatomy. Miniplating systems have greatly enhanced the ability to provide three-dimensional stabilization of bony fragments in these buttress areas.

Interosseous steel wires still have many applications in CMF fracture fixation. Wires may be used to locate bone temporarily before the application of plates and screws; this strategy is useful where all fixation points of the bone cannot be visualized at once. Fine wire may be used to stabilize the inferior orbital rim where the overlying subcutaneous tissue is very chin and where plates and screws may be palpable percutaneously. And in many parts of the world, wires may be employed because miniplate systems are unobtainable or prohibitively costly. Pre-stretched wire should be used, inserted through holes drilled with a fine burr and twisted clockwise to tighten and to achieve firm apposition of bone ends. The twisted end is cut long enough to be buried in an adjacent burr hole, yet not so long as to be palpable through the skin. Wire has been a great surgical standby, and skill in the use of wire remains essential.

Plates must be used with caution in childhood; there is ample evidence from animal experiments that plates and screws placed across sutures in a growing animal will reduce growth at that suture site (Lin et al 1991). It therefore seems reasonable to remove plates and screws in a planned fashion when the plates and screws have been placed across a growth centre, for example the mandibular condyle (p. 504).

Principles of Soft-Tissue Repair

Priorities

Primary repair of soft tissues of the face and scalp should, if possible, be carried out within 12 h of injury. But in practice, delay up to 48 h has not led to detrimental consequences, and can be accepted where there are more urgent priorities.

Management of skin wounds

Debridement
Accurate conservative debridement is the key to early primary closure. The cheeks and scalp are the only areas of the face where there is some redundancy of tissue; elsewhere, debridement must conserve vital structures such as eyelids, lip, nose and ears. Therefore an accurate repair of these structures is essential with matching of landmarks, and this should not be delayed unless there, are compelling reasons. The techniques of skin closure are discussed in Chapter 15.

Plastic procedures
Where there is such loss of skin that direct closure is not possible without distortion of a key anatomical structure, skin grafting may be the preferred primary option. In different situations, we have used split-skin grafts, full-thickness grafts and virtually all the flaps devised by plastic surgeons (p. 451). These procedures also find many applications in the management of burns (Ch. 17).

Microsurgical procedures

Microvascular tissue transfers are now routinely used repairing avulsive soft-tissue injuries (pp. 437 and 454) and in reconstructing defects in the facial skeleton by vascularized bone grafts (p. 623). The success of these depends on microvascular anastomoses by a skilled operator with appropriate equipment. In our service, the Wild M691 microscope has been chiefly used; the anastomoses are normally performed with 10/0 nylon sutures on 70 μm tapered needles. In the replantation of avulsed structures, staff and facilities for microvascular repair must be available at short notice.

In the management of CMF trauma, there are other applications for microsurgery, especially in ophthalmic and neurosurgical procedures. Each discipline has its own preferences in choice of microscope, and this can cause difficulties in combined operations.

Muscles, nerves and ducts

Careful alignment and suture of these deeper structures should also be carried out early. In most cases, use of the microscope will be required to effect accurate repairs. Where major branches of the facial nerve have been divided posterolateral to the line of the lateral canthus, careful microscopic repair is mandatory. If there is a laceration of the supraorbital nerve at the superior orbital rim then this too should be repaired to prevent troublesome numbness of the forehead. Lacerations of the inferior dental nerve where it issues from the mental foramen should also be primarily repaired. The microscope should be used for early repair of ductal structures; these include Stensen's duct and the canaliculi of the nasolacrimal drainage apparatus (pp. 429 and 439).

Principles of Dental Management

Priorities

Injuries of the teeth are not high in the list of priorities in cases of multiple trauma. Ideally, replantation of an avulsed tooth should be done within 30 min of the avulsion (p. 353), but this is rarely possible in cases of serious CMF injury. Fractures exposing the dental pulp may result in loss of an otherwise salvageable tooth and the chances of survival are increased by application of calcium hydroxide paste and appropriate endodontal therapy. Other dental injuries can be treated less urgently.

Nevertheless, the dental assessment is often of great importance, and should not be delayed in injuries involving the oral cavity. This is especially so when fractures of the facial skeleton disrupt the dental occlusion.

Dental techniques

These are discussed in Chapter 12. The skills of the prosthodontist are indispensible in the treatment and rehabilitation of fractures of the jaws. In our unit, dental specialists have led in designing and manufacturing oral and extraoral prostheses, in the use of osseointegrated implants (p. 637) and in the application of CAD/CAM techniques for fabrication of titanium and other implants. The prosthodontist has been involved increasingly in preparing ears, noses and ocular prostheses, and the role of this important discipline is likely to expand.

Principles of Brain Injury Management

Priorities

Brain injury management has some imperative priorities, which express the need to protect the brain from the secondary complications of head injury. These include:

1. Prevention of hypoxia and hypotension, especially in comatose patients
2. Control of raised intracranial pressure (ICP) and prevention or treatment of cerebral compression
3. Prevention of craniocerebral infection.

Coma management

Coma with depression of the protective reflexes is an immediate threat to life; the threat is increased when fractures of the facial skeleton compromise the airway. Coma management is discussed on p. 381; it is important here to emphasize that persisting coma may delay the definitive management of other CMF injuries. In patients remaining in coma (Glasgow Coma Scale ≤ 8) after resuscitation, we routinely institute endotracheal incubation, ventilation and respiratory paralysis as soon as possible — usually in the emergency room, sometimes at the roadside or in the retrieval ambulance. This entails many neurosurgical and metabolic dangers, and to avoid these, close 24 h collaboration with the team of intensivists is essential. CMF injuries in coma are managed in this way until the physiological state is stable and the risk of serious intracranial complications is acceptably low.

Raised intracranial pressure and cerebral compression

The patient with a craniocerebral injury complicated by raised ICP is in danger of irreversible impairments of neuronal physiology, and modern neurosurgical intensive care is planned to keep the ICP at a tolerable level: 20 mmHg is usually given as the maximum acceptable pressure, though patients have survived much higher pressures for periods of days. The need to monitor and control ICP has a high priority in victims of CMF trauma who are in coma; our methods are set out in Chapter 13.

When ICP is elevated by a mass lesion, especially an expanding intracranial clot, other considerations become dominant. The danger is not only the effect of a general elevation in ICP: there is the more immediate threat of transtentorial herniation, brainstem compression, and irreversible damage to the vital brainstem nuclei (Fig. 2.28). Modern head injury management aims to detect intracranial clots and areas of brain swelling by early and repeated CT scanning, and to a lesser extent by ICP manometry; detection by observation of the conscious level is still an essential part of the management of minor head injuries, but is excluded in comatose patients by the use of respiratory paralysis and ventilation. An extradural or subdural clot causing cerebral displacement is an imperative surgical emergency; when there is evidence of rapid deterioration or severe displacement, the patient must be rushed to the theatre. Although small haematomas may be treated conservatively, it is wise to evacuate all except the most trivial extracerebral haematomas at once by an adequate craniotomy if there is any impairment of the conscious level. Intracerebral haemorrhage, if large, may also require craniotomy, and there is sometimes justification for operating on haemorrhagic contusions constituting a mass lesion; Gudeman et al (1989) have been able to do this without increasing the eventual neurological deficit. In our experience of CMF trauma, an operation for haemorrhagic contusion is likely to end with a frontal or temporal lobectomy as an 'internal decompression', and we have avoided such mutilating operations if at all possible. External decompression, by removal of a large bone flap and enlarging the dural capacity by a fascial graft, is less detrimental, but is often ineffective. Operation for intracranial haemorrhage will usually take priority over any other procedure for CMF injury. However, in doing these urgent operations, the neurosurgeon should keep the needs of facial fracture repair in mind, especially when planning the exposure (see below); in such cases, it is helpful if a craniofacial colleague can be in the theatre to advise on this, to examine the fracture under anaesthesia, and to view the radiological images.

Intracranial infection

Prevention of intracranial infection is an important part of management of CMF trauma. Ideally, open craniocerebral wounds should be closed as soon as possible, with watertight reconstitution of the dura mater and good scalp closure. However, these injuries do not carry the highest priority. Compound depressed calvarial

fractures can be left for 24 h, or even longer, if covered with a sterile dressing and treated with systemic antibiotics. Both in peace and war, closures of penetrating missile wounds have been similarly delayed without adverse effects, though it is certainly not advisable to do so. Persisting cerebrospinal fluid leaks through the nose and other cranionasal fistulas should also be closed before, infection can supervene. However, it is highly inadvisable to attempt to close a cranionasal fistula until ICP has returned to normal. Moreover, cranionasal fistulas may heal spontaneously, and cerebrospinal rhinorrhoea is usually treated expectantly for 7-10 days in the hope of spontaneous cessation of the leak. During the waiting period, we give prophylactic antibiotics in such cases, though their value is disputed. Since fistula repair is not seen as urgent, it can be combined with elective repair of complex facial fractures (Simpson et al 1990).

Neurosurgical techniques

Instruments

For craniocerebral injuries, standard neurosurgical instruments are appropriate; microsurgical instruments, though rarely essential, are very helpful in difficult dissections in the region of the optic nerves and chiasm. Adjustable self-retaining retractors are useful, especially when used in conjunction with the rigid skull fixation given by the Mayfield head rest. When exploring war-time missile wounds of the brain without a skilled assistant, the Scoville brain spatula forceps is a most helpful substitute for a self-retaining retractor; no doubt the simple wire double retractor devised by Miyake & Ohta (1993) could be used in the same way. The operative microscope is routinely used in subfrontal explorations, especially in the vicinity of the optic chiasm; in our service, the Contraves microscope has been favoured. However, if a microscope is not available, binocular loupes and a strong headlight are usually satisfactory. Powered drills (see above) have become part of standard neurosurgical operative technique; diamond burrs are essential in opening the optic canal (see p. 425). The neurosurgeon who has to manage gunshot wounds of the head without plastic surgical assistance should be prepared to close scalp defects by split-skin grafting when the use of a rotation flap alone is insufficient to effect watertight closure without tension — always assuming that the pericranium in the secondary defect will accept a graft.

Neurosurgical exposures

The use of the standard bifrontal scalp flap is strongly advocated for frontal mass lesions and for exploration of the anterior cranial fossa, whether by a bifrontal or unifrontal bone flap (Fig. 13.11). If a lateral craniotomy is needed, the skin incision should be planned to permit a frontal flap at a later date. Occasionally, we have been able to combine operation for intracranial clot with repair if a fracture of the facial skeleton, but there are dangers in prolonged one-stage operations in severely injured patients.

Wound closure

The dura mater is always closed; at present, 4/0 braided Neurolon is used, but fine absorbable sutures have been found satisfactory. If necessary, a graft of temporalis fascia, pericranium or fascia lata is inserted. Bone flaps are replaced; in the past, these have been secured with silk or steel wire, but titanium wires or miniplates are preferable. The scalp is closed in two layers; for the galea, we use a continuous absorbable suture (e.g. Vicryl: polyglactin 910), the skin edges being apposed with staples or a continuous monofilament Prolene (polypropylene) stitch. Drainage is used as little as possible, since suction may aspirate excessive volumes of cerebrospinal fluid if there is a breach in the dura (McCulloch & Pattison 1981). But where there appears to be a risk of postoperative bleeding, low-pressure suction drainage is used.

Postoperative monitoring

All craniotomies carry the risk of postoperative bleeding, especially in the extradural space; neurosurgical nursing observations are planned to detect such complications. However, changes in the conscious level may be masked if narcotic

analgesics must be given because of painful procedures such as thoracotomy for rib grafts or exposure of the iliac crest; in such cases, small bolus doses of intravenous morphine are a reasonably safe means of giving analgesia without greatly impairing consciousness. Likewise, after exposure of the facial skeleton, the papillary light reflexes may be masked by orbital swelling. The neurosurgeon must then rely on ICP monitoring (p. 369) and on repeated CT scanning; monitoring of vision and other cerebral functions by computer-averaged records of evoked potentials has not played a large part in our practice (p. 248) but may be used as an additional safeguard.

Principles of Management of Eye Injuries

Priorities

Penetrating eye injuries should take priority over all other CMF injuries save those that are immediately life-threatening. In children, there is special urgency, since a child may aggravate the eye damage by rubbing or picking at the wounded eye. In adults with such injuries, it may be justifiable to delay the definitive repair overnight, or for whatever time is needed to transfer the injured person to a centre that can offer more expert treatment. If this is done, precautions should be taken to prevent expulsion of the contents of the eye or infection.

Remediable conditions imperilling the blood supply of the retina or optic nerve also deserve high priority. These include:

1. Optic nerve compression
2. Intraorbital haematoma
3. Occlusion of the central retinal artery
4. Acute glaucoma.

These conditions are discussed in Chapter 14; in practice, urgent surgical treatment of these conditions is rarely indicated and still less often is it successful.

The timing of secondary eye surgery is important. Vitrectomy may be needed in wounds involving the posterior segment of the eye (p. 405); this operation is best done 7–10 days after injury, and no later than 2 weeks. Lens extraction may also be needed, and this is best delayed until anterior chamber haemorrhage has resorbed, and any corneal wound has healed, so that there is a good operative view of the intraocular structures. Moreover, if an intraocular lens is to be implanted, preoperative measurements by keratometry and A-scan ultrasound will be needed; these need careful and unhurried measurement. The timing of lens implantation depends on the patient's age: in a child in the age where there is a risk of amblyopia, operation is done at 2–6 weeks, in an adult at 2–12 weeks.

Organization

Some eye injuries, especially those in the posterior segment of the eye, demand much diagnostic and operative skill. In planning a trauma service, it is important to ensure that an ophthalmologist with these skills is always available at short notice. It is equally important to ensure that all members of the trauma team are alert to detect eye injuries, and sufficiently familiar with the management of ocular trauma to participate in assigning priorities in cases of multiple injury with eye involvement. It is also important that medical and nursing staff should be trained to detect delayed visual failure.

It is not always easy to combine ophthalmological procedures in a single-stage multidisciplinary operation, as is often done with conditions requiring

plastic surgery, neurosurgery, or oral surgery. Eye surgeons are accustomed to use dedicated theatres, special equipment; and nursing staff with special skills. However, many less demanding ophthalmological procedures can and should be done as part of a definitive multidisciplinary procedure.

This is often true of enucleation or evisceration of a severely damaged eye; it is also true of tarsorrhaphy, which has many applications in CMF trauma.

Ophthalmological techniques

These are discussed in Chapter 14, in relation to the various types of eye injury. Penetrating eye wounds may require vitrectomy. Simple removal (abscission) of prolapsed vitreous can be done with scissors and cellulose sponges at the time of the primary operation, and any ophthalmologist involved in traumatic eye surgery is competent to do this. But more complex vitrectomies may be necessary for injuries involving the posterior segment of the eye, to remove vitreous haemorrhage, to deal with vitreous traction bands, to remove deeply sited foreign bodies, and for retinal detachments. These exacting procedures need special instruments and the special skills of an ophthalmologist who is dedicated to this field of surgery. The techniques of vitrectomy are beyond the scope of this book, but a general account is given in Chapter 14. Optic nerve decompression is another procedure requiring specialised instruments and skills, and the craniofacial unit should be able to provide these at short notice: the swing away from transfrontal neurosurgical decompression to transethmoid decompression may enlist the ENT surgeon in these still debatable procedures (p. 426).

Protection of vision

In the course of the management of CMF trauma, vision may be imperilled in many ways, and it is necessary to adopt routine measures designed to minimize the known risks. Morax (1984) has given an interesting review of peroperative visual dangers, and there are also risks in the intensive care unit and in the ward.

There are many situations in which a previously normal cornea may be injured, or a recoverable corneal injury may become irreversible. These include:

1. Accidents in emergency management or in the operating theatre, resulting in mechanical or chemical trauma
2. Later injury from impaired lid closure, proptosis, exposure, impaired tear production or a combination of these.

The prevention of corneal injury is an immediate responsibility for many members of the CMF team. Measures for eye protection in emergency management are discussed on p. 229. In the operating theatre, great care is needed to avoid accidental contact with the exposed eye. If possible, the eye is protected by the anaesthetist after induction, by covering the cornea with a suitable ointment (e.g. white paraffin ointment) and sealing the eyelids with a waterproof adhesive film or strapping. However, this often impossible; in many operations in the orbital region, the eye is necessarily part of the sterile field. Strong antiseptic solutions should be avoided: we have seen permanent blindness when a skin preparation of 70% alcohol was inadvertently placed in contact with the cornea. A half-strength aqueous solution of povidone-iodine is well tolerated; some surgeons prefer to use 0.2% aqueous chlorhexidine. Throughout the operation, care is taken to avoid mechanical contact or desiccation. We place discs of absorbent material covered with plastic (e.g. Telfa® dressings) over the eye, irrigating them from time to time; Morax (1984) mentions the use of coloured contact lenses to cover the cornea during surgery. The cornea is endangered if there is impaired lid closure. Initially, it is usually appropriate to employ temporary measures to close the lids and to prevent desiccation These include:

1. Copious use of lubricants, artificial tears and lid massage

2. Application of clear plastic film (Fig. 9.8A); this has largely replace the use of a watch glass taped to the face (Fig. 9.8B)
3. Taping the lids together, or sealing them with tissue adhesive
4. Inserting a 4/0 silk suture through the upper (or lower) lid, and taping this to the cheek (or eye brow); a version of this procedure is shown in Fig. 9.8C.

Tarsorrhaphy

These measures may fail, and are of course unsuitable for long-term protection; a tarsorrhaphy may then by necessary. In some situations, tarsorrhaphy may indeed be indicated forthwith. The chief of these is a combination of facial and trigeminal paralysis, with abolition of both the motor and sensory arcs of the corneal reflex. This is unusual as a consequence of an impact in the CMF region, but we have seen it after a crushing injury of the skull, and it is very likely to lead to severe keratitis.

Temporary tarsorrhaphy is appropriate when the need for eye closure is expected to be over in 3-6 weeks at most; this is so when the cause is postoperative orbital swelling and proptosis. The lids are sutured together without removal of any lid tissue; when the sutures are removed, the eye will open. We use 4/0 silk or 3/0 Nylon sutures, passed through the upper tarsal plate to emerge in the line between the orifices of the Meibomian glands and the line of the lashes, and then through the lower plate in the reverse direction; on each side of the palpebral fissure and the line of eyelashes, the stitch is passed through a short (5–10mm) length of rubber tubing to act as a bolster. Usually two of these mattress sutures will suffice.

In a permanent tarsorrhaphy, a strip of tissue is removed to allow the lids to fuse together by tissue union. To effect this, a strip of lid may be excised between the lashes and the orifices of the Meibomian glands on both the upper and lower lids; alternatively the whole of the inner lid margin may be excised. The lids are then sutured together with 4/0 silk or 5/0 Nylon stitches passed into the lid and out again as mattress sutures: they are tied on rubber bolsters, as described above. If bolsters are not used, then it is likely that the stitches will cut through the delicate lid tissue, especially in a young patient. This may lead to scarring, distortion of the lid margins and impairment of the lid function of tear spreading. In temporary tarsorrhaphy, it is important to preserve the lid margins, and in permanent tarsorrhaphy it is important to ensure that the direction of the lashes is not disturbed. If a permanent tarsorrhaphy leaves lashes misdirected inwards, the cornea and the conjunctive may be irritated.

Tarsorrhaphy a simple procedure, but it should be done carefully by a capable surgeon to ensure a satisfactory outcome. If a partial tarsorrhaphy is required (Fig. 9.8D), and this is often appropriate in cases of facial paralysis, then it is wise to make the tarsorrhaphy more extensive than seems necessary, since it is easier to open an over-large tarsorrhaphy than to resuture one that is too small.

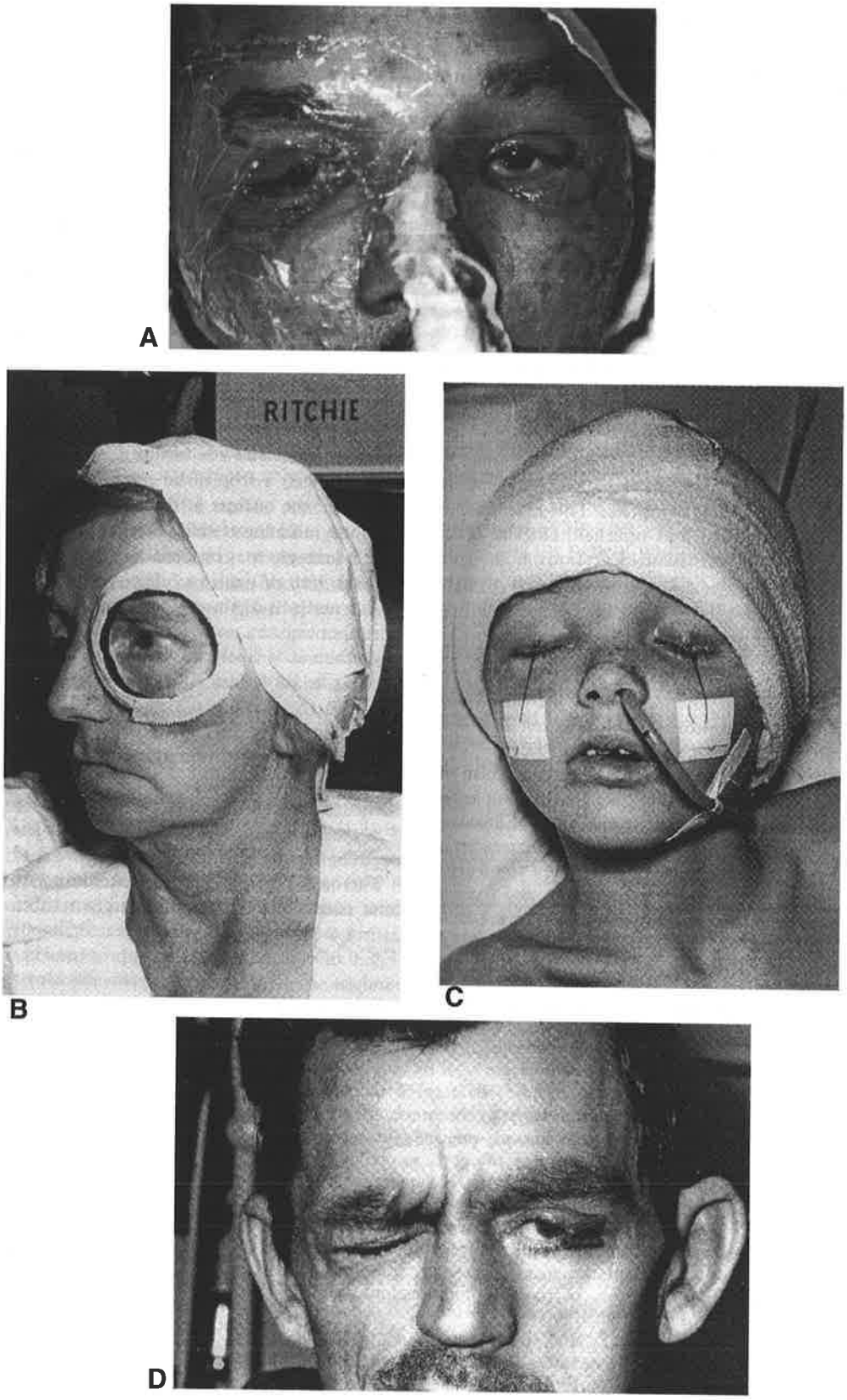


FIG. 9.8. ***Protection of the cornea.*** **A.** *The eye can be covered with clear polyethylene film (Glad®-wrap). This is now in routine use as an initial protective measure.* **B.** *A watch glass or plastic cover can be taped to the skin; this allows use of the eye. Little use is made of this now, but it has a place when the cornea is anaesthetic, as it makes it difficult for the patient to rub or scratch the eye.* **C.** *A suture can be passed through the eyelid and taped to the cheek: the suture is used to open the eye when the pupils are tested. In practice, this method is often unsuccessful because attachment of the taped suture soon becomes loose. We no longer advise this, but it has its advocates.* **D.** *Tarsorrhaphy is a versatile and usually safe method of securing eye closure. The figure shows a permanent partial tarsorrhaphy done for facial paralysis; rubber tubing is used to prevent sutures from cutting through the lid margins.*

Optic nerve protection: visual evoked responses
The optic nerve may be injured during operation, by manipulation or by the injection of an agent able to cause ischaemia or nerve damage. It may be injured after operation from compression by haematoma formation. In all orbital procedures and especially in craniofacial operations requiring correction of orbital dystopias (p. 573), it is necessary to avoid any manoeuvre likely to cause traction or compression of the optic nerves. At the close of any operation of this type, the pupils must be inspected and the light reflexes must be tested. If vision is lost, under circumstances suggesting optic nerve compression, the area of possible compression should be explored immediately.

Harding (1991) and others have used the elicitation of visual evoked responses (VER) to monitor optic nerve function during and after craniofacial surgery, and we have made some use of this diagnostic procedure. We employ a 44 mm hand-held flash stimulator which easily penetrates a closed eyelid; the stimulator contains an array of 19 light-emitting diodes. Recordings are taken from an electroencephalographic scalp electrode placed over the midoccipital cortex and about 3 cm above the inion, with a reference electrode on the ear or forehead. The evoked potentials are filtered in the range 1.6-3000 Hz, and averaged until a stable and reproducible response is recognised. Usually 25-100 averages, repeated 1-3 times, are sufficient either to confirm optic nerve transmission or to raise concern about the possibility of damage. However, this is not a sensitive test of optic nerve function, and is used only to detect gross failure of vision.

Our use of VER monitoring in practice has been limited, chiefly to cases where there is already visual impairment. In elective operations known to place the optic nerve in some jeopardy, such as extensive orbital dissections, we have routinely performed a baseline VER measurement before operation. We do not perform the investigation during operation, because the VER is affected by anaesthetic agents, especially halothane, but if there is postoperative orbital swelling preventing tests of the pupillary light reflex, then a normal recording is very reassuring. Our chief use for VER recording has been in traumatic optic neuropathy, especially where the patient is uncooperative or where there is orbital swelling (p. 424).

Priorities and Staging of Procedures

In isolated injuries, there are usually no difficulties from conflicting priorities. Multiple injuries always present the need to put first things first. In several possible scenarios it may necessary to plan a staged sequence of operations on the basis of the clinical priorities. The high priority of the major neurosurgical and ophthalmological emergencies is indisputable. Soft-tissue lacerations, both of the skin and the oral mucosa, should be closed as soon as possible; this will lessen the risk of infection and further tissue loss. At the same time, repair of vital structures such as eyelids, nerves and ducts can be undertaken; these procedures are discussed in Chapters 14-16. Urgent operative intervention may be needed for life-threatening injuries of other body symptoms within the first 24 h after injury, e.g. ruptured thoracic or abdominal viscera.

On the other hand, decision on the management of facial skeletal injury should usually wait until completion of the full work-up. When the definitive plan of management of facial injury is prepared, less urgent treatment of injuries in other systems may be scheduled to be done at the same time as the operative reduction and fixation of the facial fractures. An example would be the simultaneous management of the facial and craniocerebral components of a CMF injury (e.g. a facial fracture and a closed depressed frontal bone fracture or dural repair of the anterior cranial fossa) + enucleation of a ruptured eye + some elective extracranial orthopaedic procedures.

Principles of Counselling

When the management plan is completed, it must be explained to the patient, and usually also to the relatives. This is essential in helping the patient to make a wise choice, whether between operative and non-operative management, or between a range of operative procedures. The explanation is the responsibility, often onerous, of the surgeon(s) chiefly involved; it is easier if there are already good communications between the team members, the patient and the family. Mutual confidence is always desirable, and never more so than when the initial injury assessment points to the likelihood of some permanent deformity or loss of function. When the patient is a child, the legal guardian must give informed consent to surgery; however, it is important to explain to children, in whatever depth is possible, what has to be done and why. For elective procedures, time can usefully be spent in games that illustrate the management plan in ways that are reassuring to the child.

Informed consent

Possible complications must be explained by the surgical specialist who will conduct, or at least supervise, the recommended operative procedure(s); the risks of anaesthesia are explained by or on behalf of the anaesthetist who will give the anaesthetic. It is well when counselling can be done in an unhurried way, giving the patient time to think and take advice, or to request a second opinion in doubtful cases. In explaining possible adverse outcomes, the counsellor has to be frank yet should avoid frightening the patient unduly.

The explanation has medicolegal implications. Throughout the world, but especially in jurisdictions based on some variant of English Common Law (the UK, the USA and many countries formerly in the British Empire), surgeons are increasingly being sued by patients disappointed by the results of treatment. Among the grounds for such legal actions has been alleged failure to explain the possible complications, both those which are relatively frequent and those which are known to be very rare. It has been our practice to explain the obvious risks inherent in the treatment of a particular pattern of CMF injuries, which almost always include death and often include blindness, and to offer to list the rarer risks, while stressing their rarity. This can be done in a reassuring way. In our experience, few patients wish to know more than the major risks, and many indeed are satisfied by the statement: 'I would want this operation if I was in your position'. Nevertheless, it is the duty of the counselling surgeon to make sure that the anxieties of the individual patient are fully brought out, and that he or she is given an explanation of the extent to which these are well grounded. In the USA, some surgeons provide a lengthy printed list of possible complications; we believe that this could become a perfunctory ritual, and might actually hinder a proper explanation. It is agreed that the surgeon has a duty to give an explanation of the risks in a proposed course of action, but different jurisdictions have given different opinions on what standards should be used in deciding whether this has been done to a reasonable extent. In the case of Sidaway v The Bethlem Royal Hospital (1985), the highest British appellate court held that the adequacy of the explanation should be judged by the standard of medical practice at the time, presented as expert testimony. Nevertheless, the same court emphasized that there could be exceptions to this rule, and in a recent judgement, the full Australian High Court held that the standard must be the general concept of reasonable behaviour (Rogers v Whittaker 1992). This case is of particular interest to surgeons working with CMF injuries because it related to postoperative sympathetic ophthalmitis resulting in bilateral blindness. Medical opinion confirmed that the risk of this devastating complication was very low, but the court held that in the context of a largely cosmetic operation, it would have been reasonable to mention this possibility. In our practice, few difficulties have arisen in the early counselling of the victims of CMF trauma, and in particular the giving of informed consent has rarely caused distress or delay. Written forms of consent, signed by the surgeon and the patient, are routinely used, though such forms would not be a valid defence if the explanation given were held to be inadequate.

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Facial Fractures

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Introduction

Facial fractures vary greatly in severity and significance. Some are fractures of a single bone or small bony complex, caused by a relatively low level of force impacting on a salient part of the face. Fractures of the nose, the commonest facial fracture, are usually the result of such impacts: as a rule, a broken nose is an aesthetic problem, with only minor functional inconveniences. At the other end of the spectrum of severity, panfacial fractures are the result of very severe force impacting on the entire face and causing gross deformity and functional impairment, sometimes even a threat to life (p. 219). Facial fractures are very common, and their causes reflect the social environment in which the face is struck. The aetiology of facial injuries is discussed in Chapter 3, and our own recent experience is set out in Table 3.1

This chapter deals with the management of fractures and dislocations of the adult facial skeleton caused by blunt impacts or relatively low velocity missiles; in Chapter 16, the massive facial injuries caused by high velocity missiles and other avulsive agents are discussed, and the modifying effects of immaturity and senescence are considered in Chapter 19.

General classification of facial fractures

Facial fracture patterns express the complex anatomy of the facial skeleton, and especially the lines of strength and weakness described in Chapter 2. Some fractures are named after their discoverers (p. 18) or by the causative mechanism (p. 104). However, an anatomical nomenclature is more specific. Cooter & David (1989) have devised an alphanumeric system in which major and minor components of the entire cranium are identified by letters (see Tables 2.1 and 2.2), while bony injury is assessed on a scale of 0–3 (see Table 2.3):

0 = no fracture

1 = undisplaced fracture

2 = obviously displaced fracture

3 = comminuted and/or compound fracture.

A numerical value for the severity of injury is given for each major anatomical zone; this is derived from the sum of the injury scores for each minor zone in that major zone, to a maximum score of 5. Since there are 10 major anatomical zones, and each of these is bilateral, the summation of the scores for the 20 zones gives a value of 100 for maximal craniomaxillofacial (CMF) disruption, lesser degrees of injury being expressed as percentages. The percentage for a given case is termed the Craniofacial Disruption Score. Standard charts are used for recording the fractures as located by X-ray and/or operation. The system is designed for easy coding and computer storage, rather than for verbal description of a clinical case.

For the present purposes (Fig. 11.1), facial fractures are considered in functional groupings:

1. Fractures primarily affecting occlusion — fractures of the jaws and dento-alveolar fractures
2. Fractures primarily affecting the orbit — fractures of the naso-ethmoid region, zygoma, orbital walls and orbital roof
3. Fractures primarily affecting the nasal airway— fractures of the nasal bones and cartilages
4. Fractures primarily affecting the skull base; fractures of the anterior cranial fossa are especially important and these are considered in Chapter 13
5. Fractures affecting more than one of the above functional areas—panfacial fractures, fractures involving both the orbit and the occlusion etc.

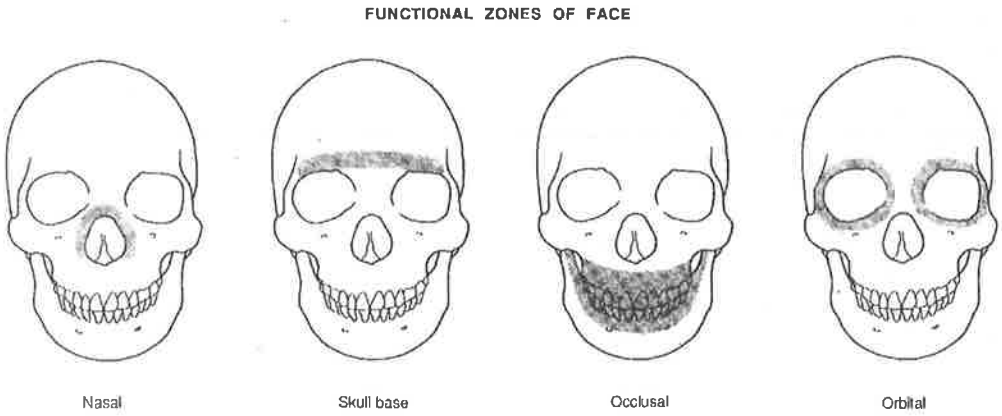


FIG. 11.1. ***Functional groupings of facial fractures.*** *Shaded areas represent the functional zones of the face. Restoration of these areas in three dimensions following fractures is mandatory for return of normal function and form.*

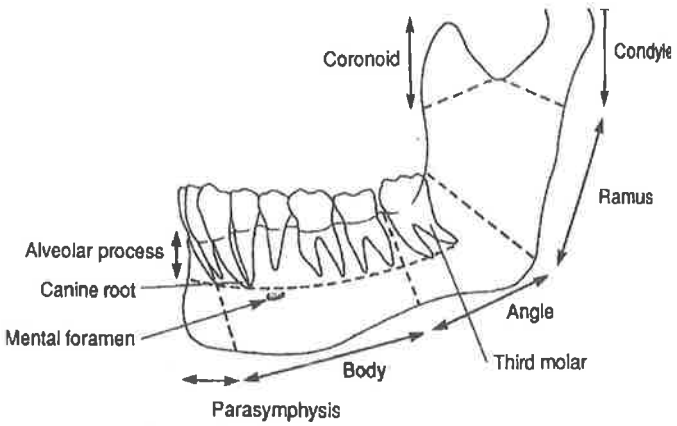


FIG. 11.2. ***Common fracture sites of mandible.*** *Commonly used descriptive terms for the anatomy of the mandible and the anatomical classification of fractures are also given.*

Fractures Affecting Occulsion

Fractures of the Ramus, Angle and Body of the Mandible

Surgical pathology

The anatomy of the mandible has been discussed in Chapter 2. It is a very strong bone (p. 105), designed to withstand the forces applied in mastication, but it has areas of weakness. These include the condylar process, the angle, and the anterior body in relation to the deep root of the canine tooth; the angle is especially weak if there is an unerupted third molar tooth. Fig. 11.2 shows the common fracture sites relating to these points of weakness; these fractures may be unilateral, bilateral or combined in any permutation.

Classification

Mandibular fractures may be classified simply on the anatomical site of the fracture. They may be linear or comminuted; comminution may be slight or very extensive. Fragment size is important: large fragments may lose soft-tissue attachments and then behave as a free graft. The fracture may be displaced or undisplaced, and this is relevant in selecting a treatment strategy: almost always a displaced fracture of angle or body in an adult requires reduction and stabilization for 6 weeks, whereas undisplaced fractures of the ramus or condyle are treated conservatively.

The distinction between compound and non-compound mandibular fractures is important. Any displaced fracture through a tooth root is inevitably compound; fractures of the ramus and condylar region are non-compound unless caused by a penetrating agent, e.g. gunshot. The presence or absence of teeth on either side of the fracture line is also important from the viewpoint of treatment by inter-maxillary fixation, and this has justified a classification:

Class I: teeth on either side of the fracture line

Class II: teeth on one side only

Class III: edentulous.

Fractures of the young child's mandible and of the edentulous mandible are discussed in Chapter 19. Special problems arise when mandibular fractures are associated with avulsive tissue loss: these are discussed in Chapter 16 and the resultant deformities in Chapter 21. Pathological fractures of the mandible, expressing local or general weakening of the bone by disease, are discussed in Chapter 20.

Clinical Assessment

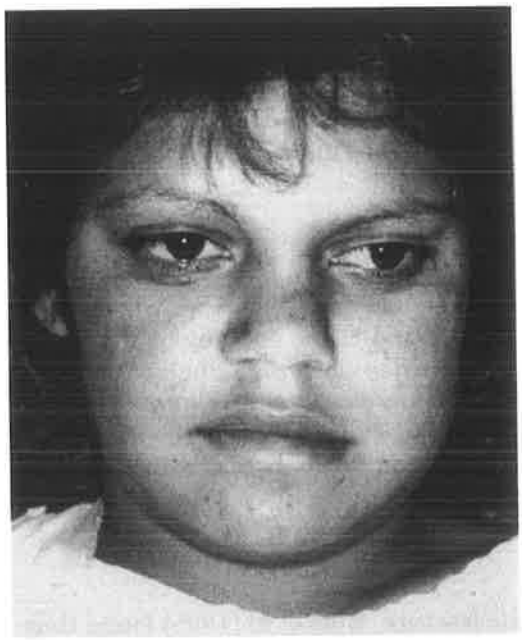
The history points to the likely site of the fracture. Ellis et al (1985) found that the incidence of angle fractures was higher in victims of assault (30.6%) than in those hurt in falls (17.2%) or in road crashes (10.9%); conversely, condylar fractures were less often due to assault: 24.3% as compared with 36.3% in the victims of falls and 34.1% after road crashes. This may reflect a higher incidence of assaults causing lateral jaw impacts, rather than impact on the point of the chin. Interrogation should also establish whether dentures were worn at the time of the accident, and whether there had been any relevant previous disease.

In cases of severe multiple trauma, resuscitation will have priority. But it is of vital importance, even in cases with life-threatening injuries, to make a preliminary assessment by palpating the facial skeleton and to examine the interior of the mouth, with a good light, using cheek retractors where necessary.

When circumstances allow, a more systematic examination of the mandible is carried out (p. 163). The patient and the examiner sit comfortably facing each other. The patient is asked to point to any areas of pain or tenderness, and to run the tongue along the teeth to detect dental irregularities, chipping, loss of a crown; the tongue is very sensitive to these and also to loss of oral sensation. The patient is asked to map out any area of numbness. Swelling and/or bruising are noted and the patient is asked to open the mouth (Fig. 11.3). Chin lacerations may give a clue to a condylar fracture (Fig. 11.4). Intraoral inspection may then be carried out, with special attention to irregularities in the teeth and steps in the dental arch: these may indicate a fracture, as may local swelling, bruising of gingival tissues or laceration of the mucosa (Fig. 11.5).

The patient is next asked to bite the teeth together, and to say whether the occlusion feels normal; the nature of the occlusion is noted. If the patient is edentulous, the dentures are inspected for signs of injury (p. 520).

Finally the mandible is palpated through the overlying soft tissues. Initially the examiner stands behind the seated patient. Bilateral palpation begins over the condylar heads and moves down to the angle and forward along the lower border to the symphysis (Fig. 6.5). Tenderness, swelling and deformity support the diagnosis of a fracture; compression on both angles will usually cause pain in an otherwise occult fracture. Neurological examination may show loss in the lower lip, from damage to the inferior dental nerve, even in the absence of a complaint of numbness. Finally, intraoral palpation with a gloved finger may be revealing.



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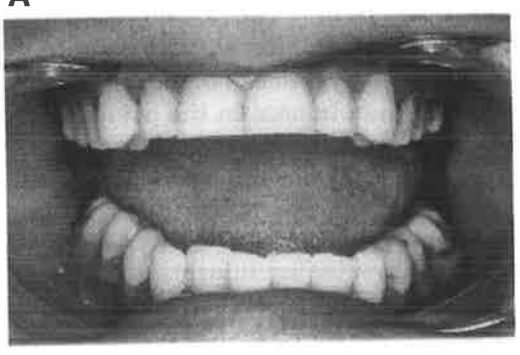

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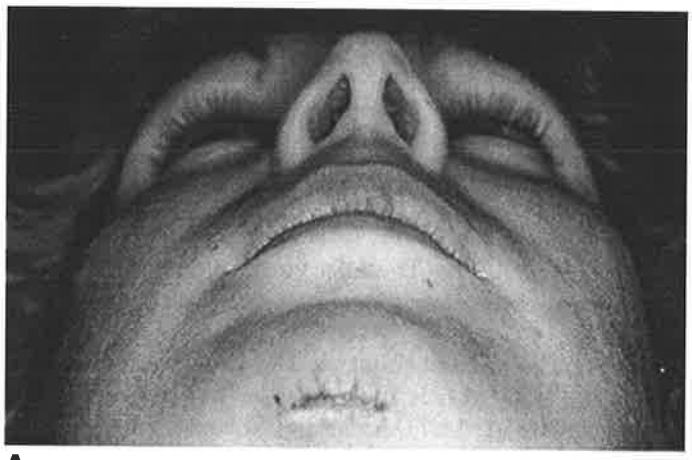
FIG. 11.3. ***Clinical signs of mandibular fractures: the angle.*** **A.** *Minimal swelling of the right cheek seen in a young woman with an undisplaced right angle fracture of the mandible.* **B.** *Bruising of the right masseter and medial pterygoid muscles result in restricted mouth opening on the affected side.*



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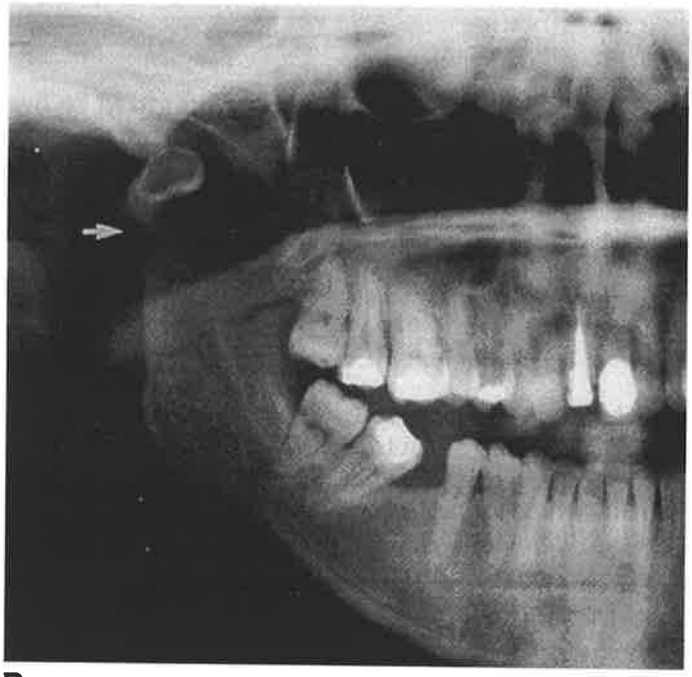


B

FIG. 11.4. ***Clinical signs of mandibular fractures: the condyle.*** **A.** *This patient fell from a bicycle onto the point of her chin. The accompanying chin laceration has been sutured.* **B.** *The right condyle fracture associated with this injury (arrow).*

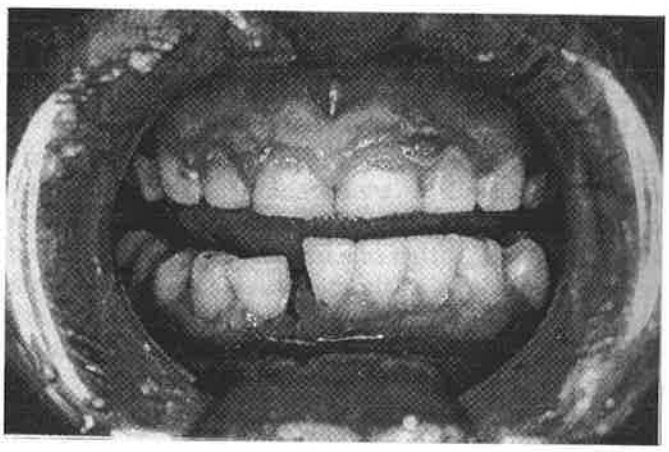


FIG. 11.5. ***Clinical signs of mandibular fractures: the body.*** *Separation between right lower central and right lower lateral incisors with a step in the occlusal plane associated with a parasymphyseal fracture of the mandible.*

Radiological assessment

If the clinical findings suggest that the mandible alone is injured, the X-ray examinations of choice are the orthopantomogram (OPG) and the Towne's anteroposterior view (p. 176). The OPG allows inspection of the entire mandible and mandibular dentition on a single film, being a composite of two lateral views. However, the tomographic process entails blurring in the symphyseal region and fractures here may be missed; moreover, lateral displacement at the angle may not be evident (Fig.11.6A,B). The Towne's view is a useful complementary examination to demonstrate lateral displacement and fractures at the symphysis.

To obtain an OPG, the patient must sit and this is impossible in severely injured or uncooperative patients. It may then be necessary to obtain lateral oblique view. on each side (Fig. 11.7). A computed tomographic (CT) scan may give useful information on the integrity of the buccal and lingual plates, which are sometimes separated or fractured in isolation (Fig. 11.8).

Early management

Patients with compound mandibular fractures are admitted to hospital and intravenous antibiotics are given: we favour flucloxacillin and metronidazole. The rationale for this choice and the dosages are discussed on p. 236. Pain relief may be needed, but narcotics are if possible withheld, especially if there is a cerebral injury. Clear fluids are allowed and later a liquid diet is given. Oral hygiene is maintained by cleansing the mouth with dressed probes, soft toothbrushes and irrigation with 0.2% chlorhexidine solution.

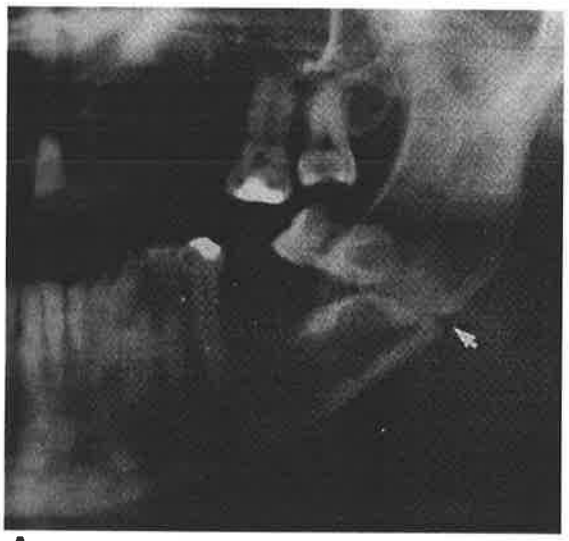
Dental assessment

This is described in Chapter 12. In dentate patients with a displaced fracture, dental impressions are taken to provide plaster casts of the occlusion in the position resulting from the injury. These casts are then cut at the fracture site and mounted on an articulator in the predicted pretraumatic position so that they can be used as models in planning how to restore the pretraumatic occlusion as accurately as possible. An acrylic wafer is made from the articulated dental model for use in the surgical reduction and fixation of the fracture.

Timing of definitive treatment

This is decided as soon as the clinical priorities of other injuries have been established. Soft-tissue lacerations, if significant, are debrided and sutured within 12 h of the injury (p. 433).

Many authorities favour early surgical reduction and immobilisation of compound fractures of the mandible. Maloney et al (1991) state that this policy has reduced postoperative infection from 4% to < 1%, and Champy et al (1986) suggest that fractures should be plated within 12h of injury for the same reason. We emphasize the importance of a detailed dental assessment and stabilization of the general condition before operation, and therefore our procedures are usually done electively rather than as emergencies, though at times a definitive mandibular operation is done early under a general anaesthetic needed for some urgent procedure, such as fixation of a limb fracture. We believe that results are better if operations are done with the best available instruments and with experienced theatre staff; these may not be provided by an emergency operating service. In a series of 50 cases of angle fractures of the mandible treated by delayed fixation, at an average time of 4 days after injury, we found only one case of infection (Moore et al 1990). However, this policy of elective operation after full dental evaluation requires cost-benefit analysis, and we do not deny that good results have been achieved by early intervention.



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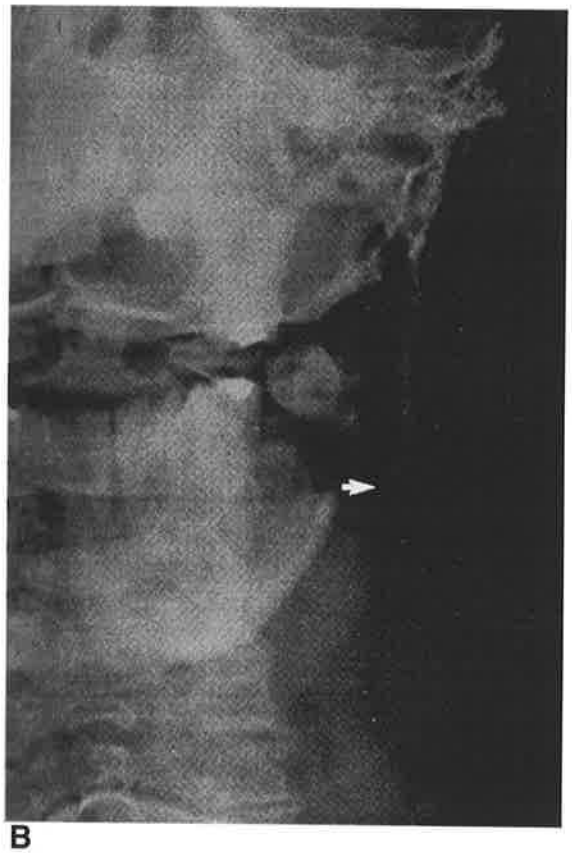

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FIG. 11.6. ***Radiology of mandibular fractures: the angle.***
A. *Fracture of the left angle of mandible seen in orthopantomogram (arrow) Anteroposterior and some superior displacement is seen in this review* **B.** *Anteroposterior Townes view of the same fracture demonstrating more significant lateral displacement not visible in the orthopantomogram.*

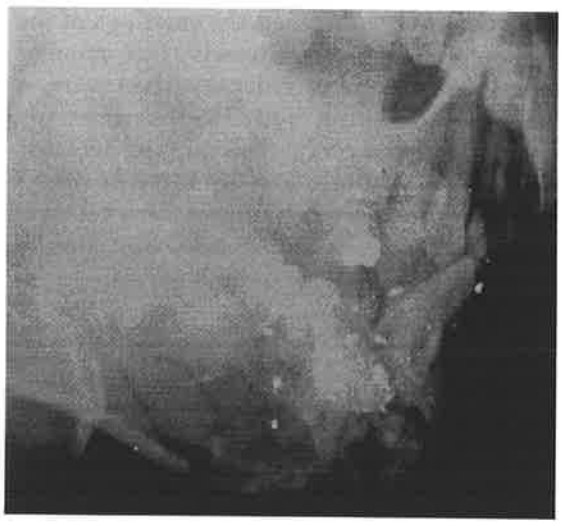


FIG. 11.7. ***Radiology of mandibular fractures: the body.***
Comminuted fracture of the left body of mandible demonstrated on an oblique lateral X-ray.

Conservative management

Stable fractures without displacement are best treated conservatively, by a liquid non-chew diet and analgesics; a soft padded neck collar may be worn to support the chin (Fig. 11.9). This conservative regimen is appropriate. for many unilateral fractures of the angle and body especially in the elderly edentulous patient, and for the greenstick fractures of children (p. 503). Most ramus fractures are securely splinted by the masseter muscle laterally and the medial pterygoid muscle medially; displacement is rare and these fractures can usually be treated conservatively. In a young adult with an undisplaced hairline fracture of the body or angle which extends to a dental root, decision may be more difficult. If the patient is judged to be sensible and likely to comply with the programme, then conservative treatment should have a good chance of success. If there is doubt about compliance, then operative treatment should be considered.

Principles of operative fixation

Until the advent of internal osteosynthesis with miniplates (pp. 26 and 237), external intermaxillary fixation was the main surgical strategy in the management of mandibular fractures—a strategy of some antiquity (p. 16). But the arguments against closed reduction and intermaxillary fixation are numerous and cogent (Thaller et al 1990).

Closed reduction demands a correct evaluation the pretraumatic occlusion, and it may be impossible to achieve this if the dentition was incomplete or grossly diseased. Intermaxillary fixation must usually be maintained for 6 weeks, to allow callus to establish a stable secondary union (p. 129), and this is an ordeal for the patient. Hygiene and feeding present problems. There is often buccal and labial irritation and even ulceration of the mucosal surfaces. It is impossible for the tongue to be used to clean the teeth and gums; manual cleaning is made difficult by the presence of wires and the wax often used to reduce their irritative effects. Plaque may build up on the teeth, causing gingivitis with potential long-term dental problems. Liquid diets delivered by syringe are tedious and unappetising; weight loss is common and sometimes serious. For all these reasons, some patients Will not endure intermaxillary fixation: they remove the wires, sometimes with disastrous results. Moreover, the teeth may move or even loosen. When arch bars are applied to premolars and molars, this is unlikely, but it is a real danger if canine or incisor teeth are ligated; if possible, these teeth should not be ligated, and if an arch bar must be supported in the central region, then this should be done by circummandibular and/or transpalatal wiring. Intermaxillary fixation is also a potential airway hazard, especially if there is a risk of sudden loss of consciousness, as in epileptics and diabetics.

Finally, there is experimental evidence to show that prolonged mandibular immobilisation causes atrophy of the muscles of mastication, fibrosis of the periarticular connective tissues and degenerative changes in the cartilage of the mandibular condyle; these changes can reduce the range of passive bite opening (Ellis & Carlson 1989). J. O. Andreasen (personal communication) has warned that teeth involved in fractures can be devitalized when ligated to the commonly used Erich bar. Brown et al (1991) compared the costs of managing isolated fractures of the mandible by miniplate osteosynthesis and by intermaxillary fixation. Miniplate fixation was considerably cheaper when account was taken of the duration of hospital stay and the service charges incurred in feeding, Dental hygiene and nursing; the complication rates were similar in both series.

We are therefore strong advocates for internal fixation. There is, however, one situation in which intermaxillary fixation is still the treatment of choice. This is in the child with mixed dentition, where a fracture line runs through multiple unerupted tooth buds, with thin bone plates unsuitable for screw fixation. The management of such fractures is discussed in Chapter 19 (p. 504).

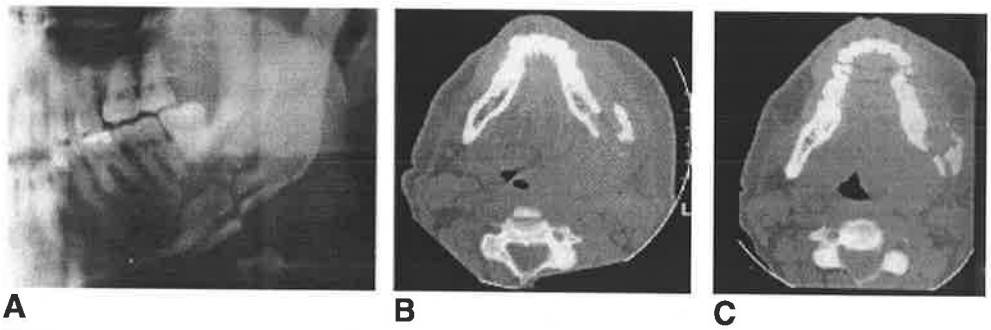


FIG. 11.8. ***Radiology of mandibular fractures.*** **A.** *Orthopantomogram of a comminuted fracture of the left angle of mandible. In this view it was not possible to tell whether the lingual cortex was intact.* **B.** *CT scan of this patient revealed a displaced fracture of the lingual cortex at the lower border of the mandible.* **C.** *CT scan of the upper border of the mandible in this patient also revealed a fracture of the lingual plate, confirming the need for surgical stabilization of the fracture.*

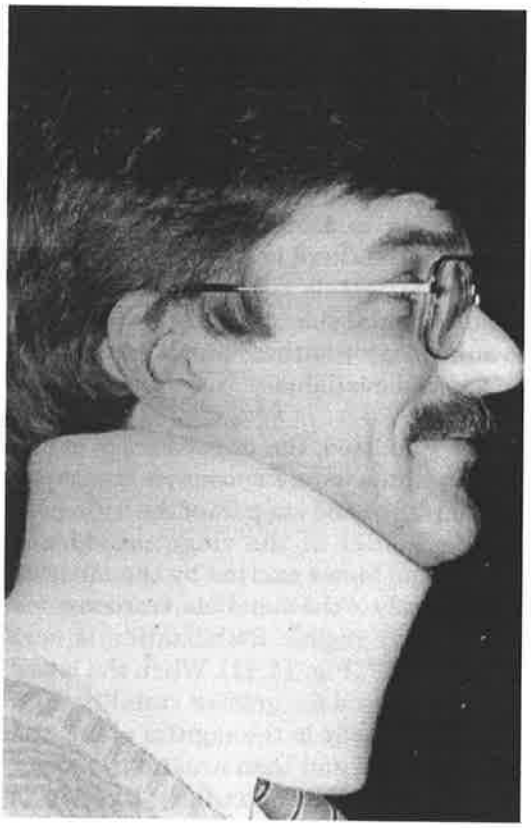


FIG. 11.9. ***Conservative treatment.*** *A soft, padded (e.g. polyurethane foam) neck collar may be worn to provide symptomatic relief via chin support.*

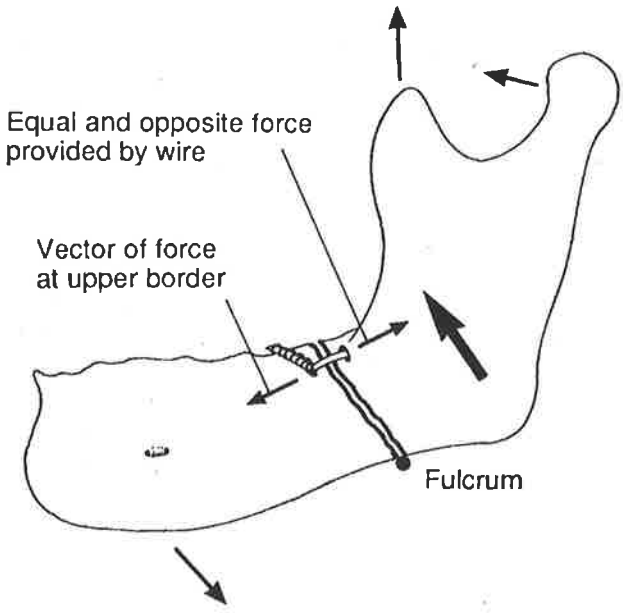


FIG. 11.10. ***Interosseous wiring.*** *Placing an interosseous wire at the upper border of an angle fracture counteracts the tensile forces produced by the muscles of mastication (arrows) around the lower border of the fracture which are acting as a fulcrum. In addition to the wire, intermaxillary fixation is required.*

Teeth in fracture line

When the fracture goes into a dental root, it has been argued whether it is necessary to extract the tooth to lessen the risk of subsequent infection. This issue was particularly important when the teeth were used in intermaxillary fixation. When miniplate fixation is the primary treatment of choice, the teeth have virtually no role in stablizing the fracture, but are important as indicators to guide accurate reduction. Our policy has been to retain teeth in the fracture line where these teeth have potential function either in the occlusion or as fixation posts for subsequent prosthodontic work — always assuming that the teeth appear likely to survive. Should the tooth lose vitality, then early restorative dentistry is undertaken (p. 358). When the tooth root is fractured, with comminution of the alveolar bone around it, then infection is almost certain and the tooth should be extracted, with removal of loose bone fragments. When an impacted third molar is involved in an angle fracture, we extract it because this tooth is rarely a functional part of the occlusion, and because we have seen chronic pain, with or without infection, develop when the tooth is not extracted. However, clinical studies by Rubin et al (1990) have shown that incompletely erupted molars may be left in the fracture line without increasing the risk of complications.

Interosseous wiring

This is indicated when intermaxillary fixation is used as the mainstay of treatment, in fractures with displacement or when there is a tooth on only one side of the fracture (type II fracture: see above). Wiring is used in conjunction with arch bars, interdental eyelet wires and/or dental splints (see below). We see this mode of treatment as inferior to miniplate fixation, but it should be remembered since miniplates may not always be available.

Where a fracture at the angle is to be stabilised, the upper border of the external oblique ridge is the obvious site to insert the wire. Exposure is transoral, allowing extraction of the third molar if it is impacted and not part of the occlusion. Placing a transosseous wire at the upper border of the ridge provides a one-dimensional stabilization against the tensile forces exerted by the muscles of mastication (Fig. 11.10). In other areas of the body of the mandible, transosseous wires are placed at the lower border or midbody region. Stabilization is best when the wire crosses the line of the fracture at 90° (Fig. 11.11). When the lower border is exposed, figure-of-eight wiring may be used for greater stability (Fig. 11.12). Alternatively, when the fracture splits the body in the sagittal plane, the wire may be passed through the two plates of bone and then around the lower border of the mandible (Fig. 11.13). After wiring, intermaxillary fixation is maintained for 4-6 weeks.

In edentulous cases, the approach is extraoral, as dental splints will be needed for intermaxillary fixation, again for up to 6 weeks, and the presence of a sutured mucosal incision and the subsequent scar line may impede both the splints and the later use of dentures.

Dental splints

Moulded acrylic dental splints can be stabilized by circumosseous wires and have been advocated for many purposes. We see two specific clinical indications. The first is in the management of dent-alveolar injuries and extruded teeth. Various types of splints can be etched to the crowns of teeth and connected to adjacent sound teeth by rods or bars: these are purpose-made appliances and will promote survival of endangered teeth: The other use for dental splints is in the management of the fractured mandible in very young children: this is discussed on p. 504.

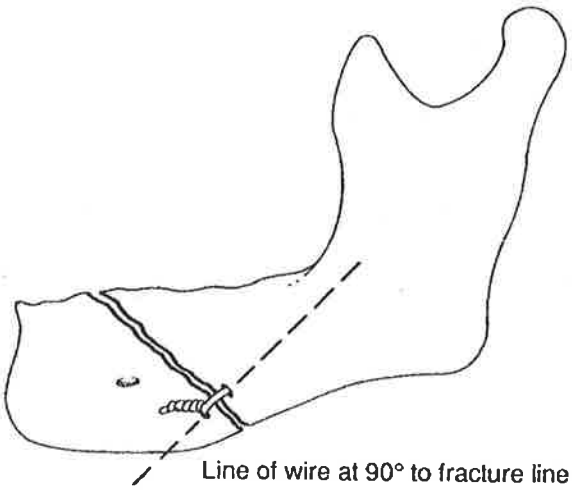


FIG. 11.11. ***Interosseous wiring.*** *Interosseous wire is placed at 90° to the line of fracture to avoid displacing the fracture along its length as the wire is tightened. In the body area of a dentate mandible, wires must be placed inferiorly to avoid damage to dental roots and the inferior dental nerve.*

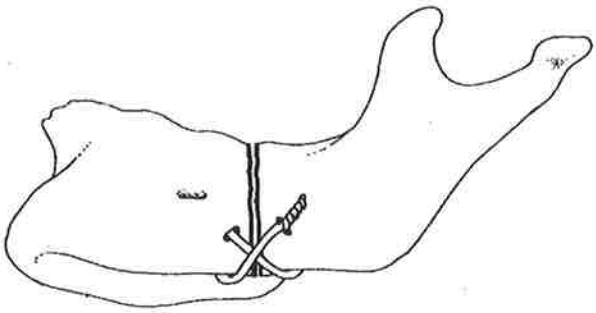


FIG. 11.12. ***Interosseous wiring.*** *Figure-of-8 technique may provide additional vertical stability at the lower border of the mandible.*

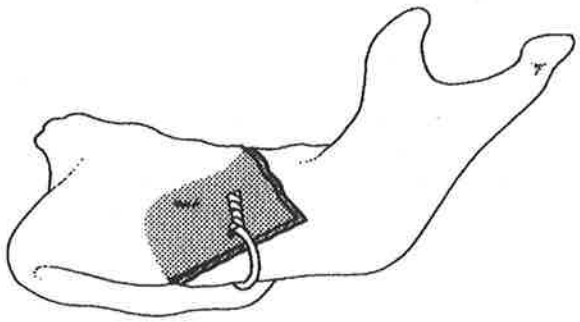


FIG. 11.13. ***Interosseous wiring.*** *Where the plane of fracture is parasagittal there may be considerable overlap of the lingual and buccal cortical bone plates (shaded area). A 'lag' wire passed across the fracture and across the bony plates will provide a compressive force between the plates, further enhancing stabilisation.*

Miniplate osteosynthesis

In 1958, the Swiss Arbeitsgemeinschaft für Osteosynthesefragen formed a study group to consider the principles of internal fixation. This now famous body, the AO/ASIF, showed the value of rigid immobilization and coaption of bone ends in securing primary bone union (p. 129); compression was seen as advantageous and compression miniplates were designed for use in the craniofacial area. Initially, stainless steel was used, but this metal has some disadvantages, being radio-opaque and also more liable to corrosion than titanium. Luhr of Würzburg developed miniplates and microplates for use in all parts of the CMF region: for the mandible, his system employed compression by bicortical screws, which had to be inserted in the lower border to avoid damage to the inferior dental nerve

and to the tooth roots. Luhr used Vitallium, but more recently titanium has been preferred because of its malleability, radiolucency and high biocompatibility (p. 155).

Success in plating fractures of the mandible depends on an understanding of the mechanical forces acting through the mandible in mastication. The mandible is normally subjected to tension forces at its upper border and to compression forces at its lower border. Unless the fracture is inherently self-stabilizing, there will therefore be distraction of the upper or alveolar end of the fracture line. The plating system must counteract this distraction.

Champy et al (1986), following Michelet et al (1973) of Strasbourg, described an ideal line for the siting of the plate (Fig. 11.14). Where a force F_2 is needed to counteract the distracting force F_1. it will be equal to the downward force in the symphyseal region multiplied by the distance L between the downward force and the fracture itself, and divided by the height H between the point of tension banding and the fulcrum or lower border of the mandible. It can be seen that the greater the value of H. the less force will be needed to control the fracture. It is also clear that the distance L will be greatest in fractures of the angle, and stronger plating will be needed for fractures in this area (Fig. 11.15). Champy also demonstrated the presence of rotational torsion forces in the anterior mandible, which is a three dimensional structure upon which bilateral muscular groups act. He recommended lower border plates as well as upper border plates to give better control of these torsional forces (Fig. 11.16.) In the tooth-bearing alveolar area, the thickness of bone is variable, and unsuitable for screw fixation; the upper border of the mandible is therefore the ideal site. Champy showed that the average thickness of the outer cortex of the mandible is 5 mm, but in some areas the cortex is <3 mm thick: there is therefore some danger of injuring the inferior dental nerve (Fig. 2.6). Care should therefore be taken to drill only through the outer cortex, and to use only 5 mm screws.

In certain situations, the usual tension-compression relations can be reversed. This may be the case when a bite load is placed just anterior to an angle fracture, and the Contralateral muscle sling is functioning normally but the ipsilateral sling is ineffective from traumatic oedema and bruising (Rudderman & Mullen 1992). In addition to this theoretical concept of intermittent reversal of the normal tension-compression zones, there is also some Clinical evidence of unfavourable results with angle fractures treated by a single plate sited in Champy's ideal osteosynthesis line. Levy et al (1991) recently reported on 61 patients with 63 angle fractures, treated according to Champy's principle of monocortical non-compression. When only one plate was used for osteosynthesis at the mandibular angle, complications were seen in 5/19 patients; when two miniplates were used, only one of 32 patients experienced complications. Although Levy and co-authors do not state exactly where the second plate was placed, one of their illustrations shows the second plate to be close to the lower border of the mandible and therefore able to act as a compression band against the intermittent reversal of tension-compression zones (see above).

Our experience with 50 cases of mandibular angle fractures treated according to Champy's principles with single upper-border non-compression plates has been satisfactory, with only one adverse result due to infection (Moore et al 1990). However, there are two clinical situations in which an additional lower-border plate is needed. First, the accuracy of reduction of an angle fracture by the intraoral route may be uncertain, because the lower border of the mandible cannot be seen. It may then be necessary to expose this area by an external incision and to achieve accurate reduction and fixation with a second miniplate to reinforce the plate inserted by the intraoral route (Fig. 11.17). Second, if it is uncertain whether the patient will obey the injunction not to chew, it may be wise to insert a second plate.

In summary, the Champy technique of upper-border single-plate osteosynthesis will be successful in most cases of angle fractures, provided that

the patient does not chew and the reduction is accurate. Failure in these respects may lead to an unusual bite force, reversal of the normal tension-compression zones, and movement at the fracture site, with greater risk of infection. In muscular male adults with angle fractures, the mechanical considerations summarized above mean that a greater banding force may be needed: this may justify the use of 6-hole plates rather than the usual 4-hole plates, and longer screws may be inserted in the proximal fragment (Fig. 11.18). Supplementary intermaxillary fixation remains as a resort when internal fixation seems likely to be insufficient alone.

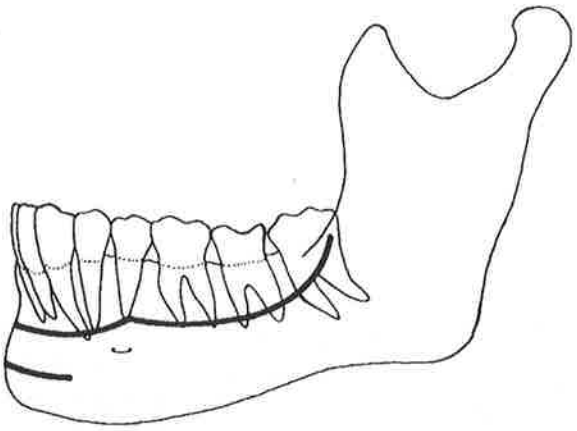
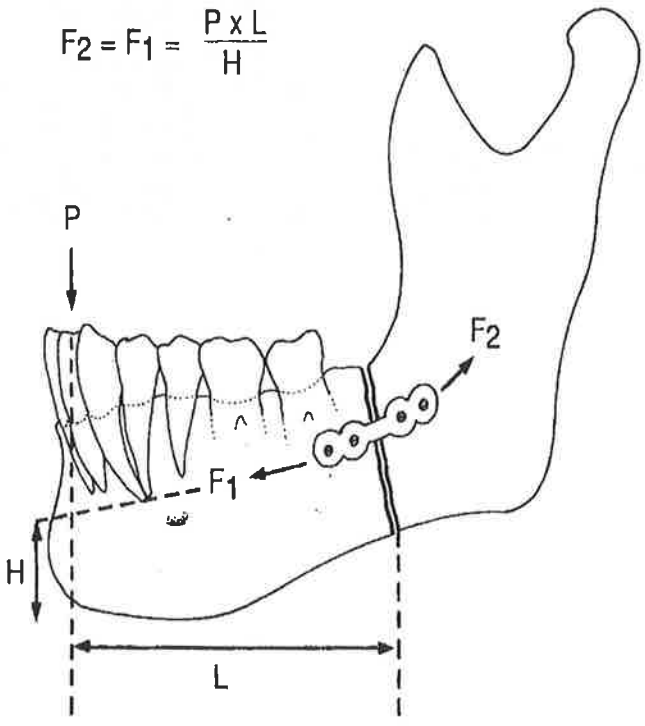


FIG. 11.14. ***Mandibular tension line.*** *The mandibular tension line is the junction of dento-alveolar and body bone (after Champy).*



CALCULATION OF MOMENT OF FORCE

P = Applied force

L = Horizontal distance from applied force to fracture

H = Distance from line of plate to lower border

FIG. 11.15. *The equation of forces of a mandibular angle fracture (after Champy).*

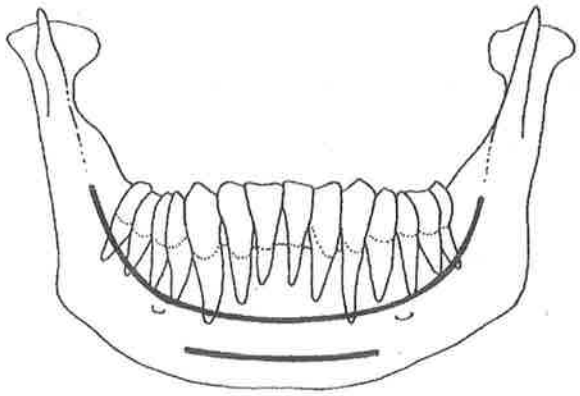


FIG. 11.16. ***Anterior body reverse-tension line.*** *The anterior lower borderline is the site for lower-border plates in conjunction with upper-border plates when fractures transgress this area (after Champy).*

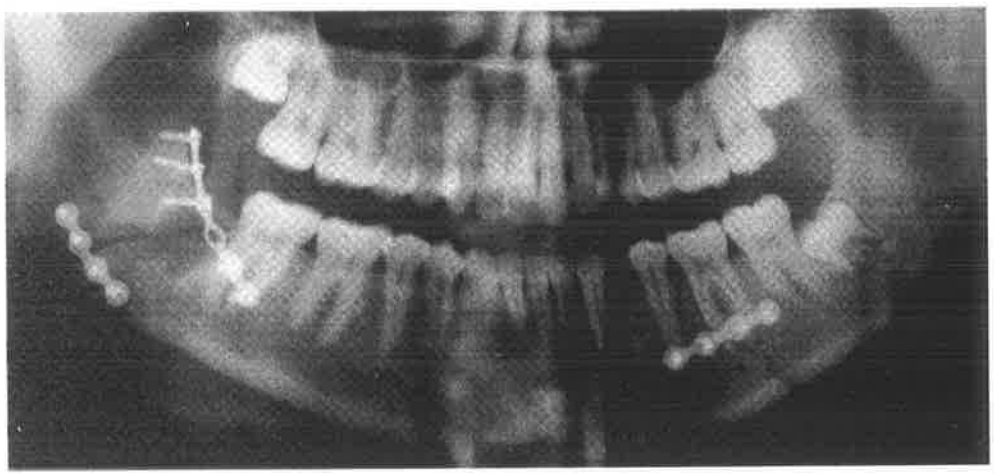


FIG. 11.17. ***Miniplating mandibular fractures.*** *Orthopantomogram following miniplate stabilisation of fractured left body and right angle of mandible. Difficulty was found in obtaining accurate reduction of the inferior part of the fracture at the angle via an intraoral exposure. Accordingly an external submandibular approach was made following application of the upper-border miniplate and screws. The lower border was reduced under direct vision and stabilised with a 4-hole miniplate and screws as shown.*

Compression plates

As stated, these require bicortical screws, and must therefore be inserted into the lower border of the mandible to control the upper-border tension zone, the plates must be strong and rigid, and the screws must be large. A modified form of compression plate has been designed for mandibular fractures, in which the outer plate holes provide compression at the inferior border, while the inner holes of the plate provide a downward movement of the fracture to counteract the distracting tension at the upper border (Hobar 1992).

The merits of compression and non-compression plates remain under debate. Compression plates increase bone contact at the fracture site, and provide rigid stabilization; this promotes primary bone union with no callus formation. However, in practice it appears that healing is equally good with non-compression systems, despite the transient appearance of callus. Compression plating systems have one potential drawback: as compression it applied across the buccal plate of bone, there is a potential risk that the fracture line may be opened on the lingual side. A small difference in reduction between lingual and buccal or labial bone can lead to a significant crossbite and potentially to a post-traumatic malocclusion. To prevent this, the flexible miniplates must be shaped to conform to the bone surface accurately: if anything, they should be overcurved, so that compression will affect both buccal and lingual surfaces equally (Fig. 11.19; Calloway et al 1992).

Lag screws

The principle of lag screwing is to overdrill the hole made in the outer plate of bone, so that the screw will engage only in the smaller hole in the inner cortex; when the screw is tightened, there will be compression between the outer and inner plates. For maximum compression, the axis of the screw must be close to 90° to the plane of the fracture, without displacing or distracting the fragments.

When a fracture shears between the lingual and buccal plates, the wide surfaces of opposed cortical bone offer ideal conditions for lag screw fixation, though this must be done where there is no risk of injuring the inferior dental nerve or dental roots (Fig. 11.20). The fragments to be screwed must not be comminuted.

When the fracture is behind the last tooth, screws can be inserted in the upper border. Niederdellman & Shetty (1987) have proposed a method of lag screw fixation of angle fractures, and report good results; Ellis & Ghali (1991) also report favourably on lag screw fixation for anterior mandibular fractures. This method may be technically demanding, and requires a good deal of practice and competence to give consistent results.

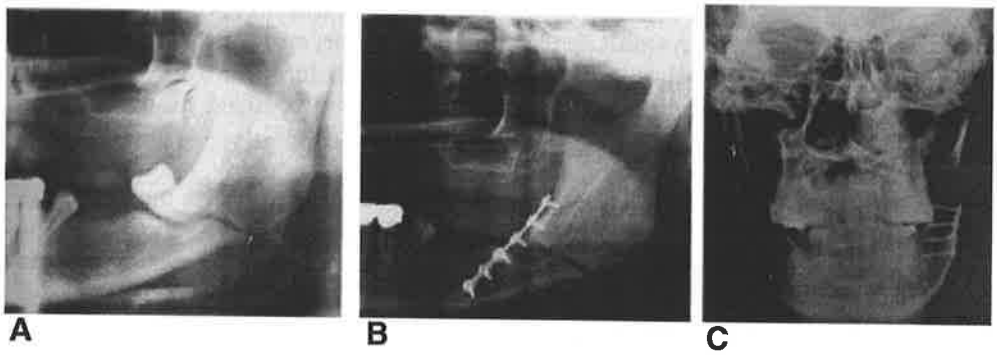


FIG. 11.18. ***Miniplating mandibular fractures.*** **A.** *Left angle fracture in a patient who was believed to be unreliable in complying with dietary restrictions.* **B.** *Stabilization of fracture with 6-hole plate and screws for greater banding force.* **C.** *13 mm screws may give greater purchase via bicortical fixation.*

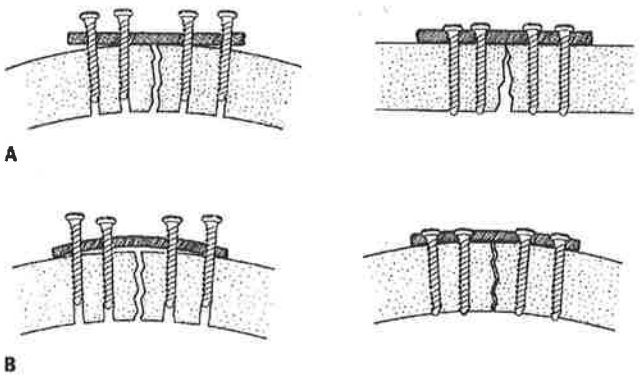


FIG. 11.19. ***Compression plating mandibular fractures.*** **A.** *Where compression plate does not exactly conform to the curvature of the underlying bone, compression will result in opening of the lingual cortex fracture line.* **B.** *To prevent this, the plate must be exactly shaped to the curve of the underlying buccal cortex or even slightly overcurved.*

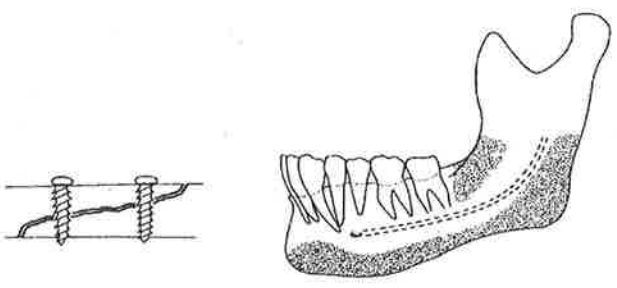


FIG. 11.20. ***Lag screwing mandibular fractures.*** *Lag screw fixation is best done where there is overlap of cortical bone plates. Screws can only be applied in areas where damage to dental roots or the inferior dental nerve will not ensue (shaded).*

Operative management (Champy technique)

Operation is done under general anaesthesia; a nasotracheal tube is passed and sutured to the base of the nostril (Fig. 10.1). The incision sites are infiltrated with 2% lignocaine and 1/80 000 adrenaline. Schuchardt or Erich arch bars are ligated to the premolar and first molar teeth with 26 gauge stainless steel wire; if a centre of support is needed for the arch bars in the incisor-canine region, the bars are anchored with circummandibular or transpalatal wires as described above. The teeth are cleaned with a toothbrush and dilute (half-strength) Betadine solution. Irreparably damaged teeth or dental roots are extracted.

The operator may use a headlight to give good illumination when working in the angle area. The incision to explore an angle fracture is placed just lateral to the anterior border of the ramus, and runs down beyond the third molar just lateral to the inferior buccal sulcus. With scalpel or cutting diathermy, the incision is deepened to the bone and the fracture line is then exposed by subperiosteal dissection down to the inferior-border of the mandible. A posterior border retractor, combined with a notched anterior border retractor, will give good retraction of the soft tissues. If the third molar is to be extracted, this is now done, care being taken not to fracture the external oblique ridge of bone. The socket is cleaned and irrigated; care is taken not to injure the inferior dental nerve, which is sometimes exposed by the fracture. The fracture edges are cleared of granulation tissue with a small sharp angled dissector, and the area is irrigated with weak antibiotic solution (e.g. 0.2 % flucloxacillin in saline) to wash away debris and reduce contamination. The fracture is now reduced manually into correct anatomical position.

The incision to explore a fracture of the body is placed to the buccal or the labial side of the inferior sulcus. To preserve the mental nerve, the scalpel is angled back sharply, under this sulcus, down to the bone. The periosteum is incised; the fracture is then exposed by subperiosteal dissection in the manner described above. If the fracture is laterally placed, the mental nerve and its foramen may also be exposed (Fig. 11.21).

When the fracture has been manually reduced, the occlusion is established and intermaxillary fixation is applied. The pretraumatic position of occlusion is established by the dental wafer and the plaster study models; however, on very rare occasions, even with the occlusion stabilised by the wafer, anatomical reduction is not achieved. When this is so, and assuming that there is no other fracture, then there must have been some error in model planning and wafer construction; the operative impression of anatomical reduction must therefore be accepted.

When the jaws are thus secured by intermaxillary wiring, and the fracture(s) reduced anatomically, it is relatively easy to achieve fixation with a miniplate applied in accord with Champy's principles. We apply a 4-hole AusSystem® plate to the upper border of the body, placing two monocortical 5 mm screws on either side of the fracture line; a similar plate is applied to the lower border in anterior body fractures. At the upper level of the body, where it joins with the alveolar bone, care is taken to drill only the outer cortex, the drill being kept at 90° to the surface; copious irrigation is used. The plates are bent as accurately as possible to conform with the contours of the mandible; however, this is less important when using these malleable titanium plates than when steel or vitallium compression systems are employed (Figs 11.22 and 21.23).

Modifications in this standard method may be appropriate in fractures in the angle area, on the external oblique ridge. A 6-hole plate may be preferred, and Mongers screws may be used in the ramus, to give better purchase in the bone (Fig. 11.18). If a second lower-border plate is needed for an angle fracture, it may be attached by an intraoral approach combined with percutaneous drilling and screwing through a cannula. But if there is doubt about the reduction of the

inferior border, there need be no reluctance to expose the area by an external incision. This is made about 1 cm below the inferior border of the mandible, below the course of the mandibular ramus of the facial nerve (Fig. 2.30). The incision is usually about 3–5 cm long, and is deepened through fat and platysma , to the fascia of the submandibular gland (Fig. 9.4C). This is incised, and the dissection is then taken upwards, so that the mandibular branch of the facial nerve remains in a superficial plane and need not be identified. Large branches of the facial artery and vein will be encountered running over the top of the submandibular gland and these should be ligated and cut. Sharp dissection through the masseter and the periosteum will then expose the fracture site. Since there is already stabilisation by a miniplate at the upper border, the fracture edges can easily be manipulated into anatomical reduction and fixed by a 4-hole non-compression plate (Fig. 11.17). The skin wound is then irrigated with antibiotic solution and closed in two layers, drainage being used only if there is much oozing. When it is not necessary to visualize the lower border of the mandible, the plate can be fixed to the lower border percutaneously by drilling and screwing through a cannula.

Intraoral wounds are closed with a running 3/0 chromicized catgut stitch, taking bites of mucosa and muscle. The temporary intermaxillary fixation is then removed and the excursions of the mandible are checked, with special attention to its ability to bite into occlusion. The arch bars are usually left in situ; if the patient readily settles into normal occlusion, the bars are removed on the second or third day, but if this does not happen then the bars can be used to allow light rubber elastic traction to help to overcome the imbalance in the masticatory muscles caused by injury and swelling. This elastic traction may be maintained for 7 days, being removed when the patient settles into normal occlusion.

Postoperative management includes intravenous antibiotics for 48 h. Clear fluids are given for 12–24 h, then a liquid or non-chew diet. Oral hygiene is maintained with mouth washes and small tooth brushes; this becomes easier when the arch bars are removed. Pain relief is rarely needed after 48h. X-ray pictures (OPG and Towne's view) are obtained on the first day after operation. If these are satisfactory, and if mandibular mobilisation is going well, the patient is discharged, usually after 2 or 3 days. External stitches are removed 1 week after operation, when oral hygiene and mobilisation of the jaw are checked. There are normally further reviews at 3 and 6 weeks, after which there should be a 4 cm opening between the incisor teeth and little local swelling. A dental assessment is then made, with special attention to oral hygiene and occlusion, as well as estimation of the viability of teeth in the fracture line. A non-chew diet is taken for 6 weeks; thereafter a normal diet is allowed. Between 6 weeks and 12 months, the patient is seen if there are clinical reasons; the healing of scars, the recovery of any areas of sensory loss and the return of normal mastication are verified. Restorative dentistry is sometimes needed.

Comminuted Mandibular Body Fractures

Management

When there is a moderate degree of comminution and little or no bone loss, as is sometimes seen after wounds from small bore weapons, the fractures may be treated by the regimen just described, with some modifications.

Debridement is performed first, small fragments of bone being removed. If the alveolus is comminuted, the shattered bone and associated teeth, and any blood clot, are removed, and the injured area is irrigated with weak antibiotic solution.

The mandible is then stabilised. After manual reduction, the teeth are held in occlusion by intermaxillary fixation with arch bars and the wafer. It may

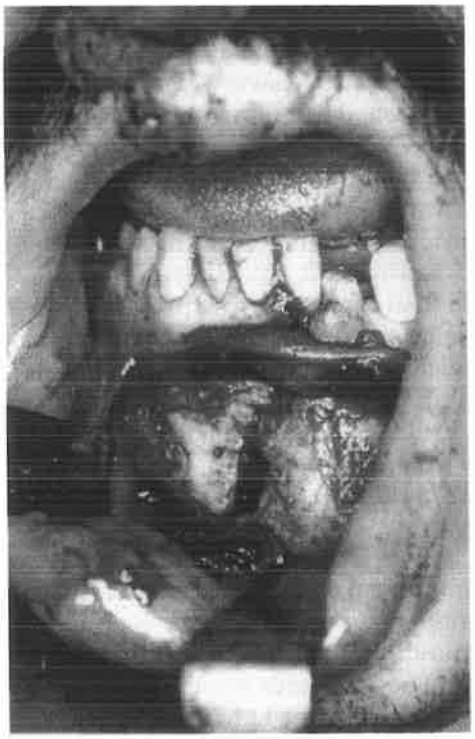
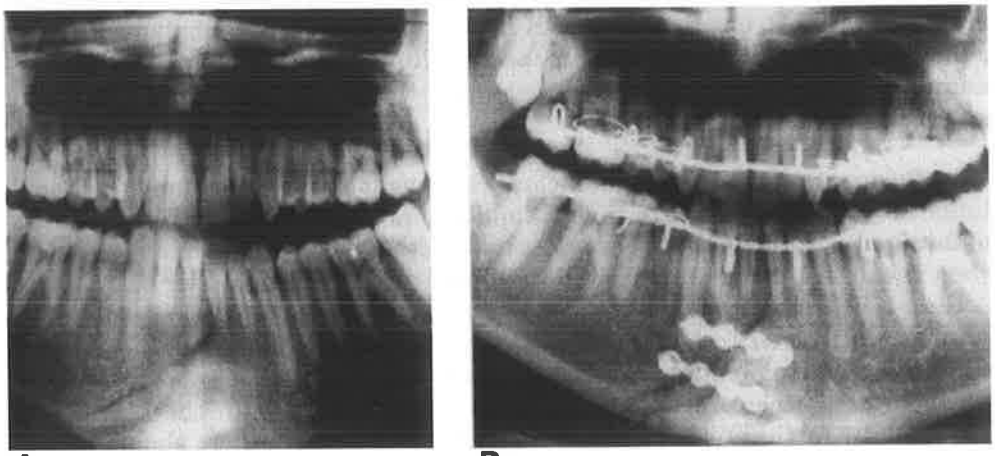
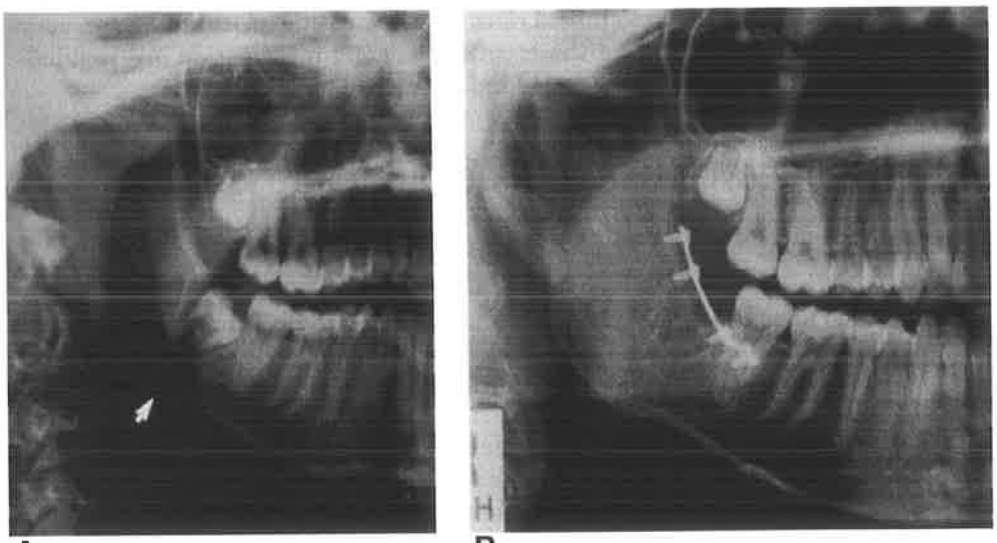


FIG. 11.21. *Intraoral exposure.* *Inferior labial sulcus incision to expose a parasymphyseal fracture of the mandible. Care is taken to protect the fibres of the mental nerve as they traverse the muscle of the lower lip.*



A B

FIG. 11.22. *Miniplating mandibular fractures: parasymphyseal.* **A.** *and* **B.** *X-rays pre- and post-surgical treatment of a parasymphyseal mandibular fracture using 4-hole miniplates applied in accordance with Champy's principles.*



A B

FIG. 11.23. *Miniplating mandibular fractures: angle.* **A.** *and* **B.** *X-rays pre- and post-treatment of a mandibular angle fracture using a contoured miniplate with four screws in accordance with Champy's principles In this case the post-treatment X-ray was taken after 6 months and no sign of fracture was seen.*

then be appropriate to stabilize the bone fragments by inserting upper and lower border plates, with at least two screws gripping the chief proximal and distal bone fragments. If there is insufficient bone to hold an upper-border plate, then it may be best to use a Luhr type of compression plate, with bicortical screws on the lower border. Where there is doubt about the reduction of the lower border, it may be wise to expose the area by an extraoral approach. If the stability of the fixation given by the miniplates is in doubt, the intermaxillary fixation should be maintained for 4-6 weeks (Fig. 11.24).

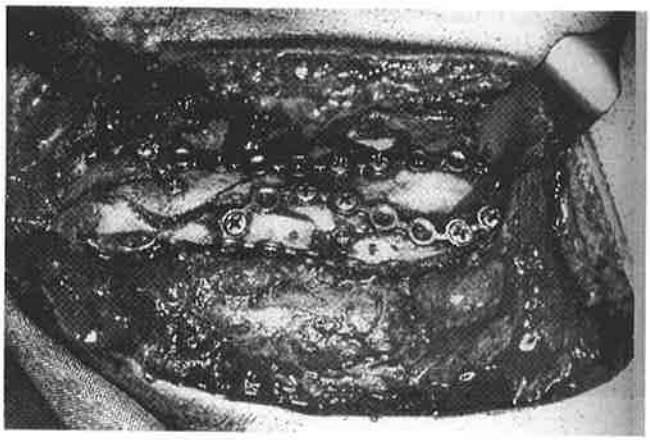


FIG. 11.24. ***Miniplating and lag screwing mandibular fractures.*** *Use of multiple plates and screws to stabilise the comminuted left body fracture seen in Fig. 11.7. This was combined with intermaxillary fixation for 6 weeks.*

Complications of fractures of the body and angle of the mandible

These include:

- Infection
- Non-union
- Malocclusion and malunion
- Plate exposure
- Nerve injury and/or dental injury due to drilling the body of the mandible.

Infection

The incidence varies in different reports and the causes are disputed. Bochlogyrus (1985) reported abscess, cellulitis or osteomyelitis in 61 of 853 patients (7%); various methods of reduction and fixation were used, but it appeared that the incidence of infection was not significantly greater after open operations. Moore et al (1985) found an incidence of 16% in a study of 100 fractures in 56 patients, with osteomyelitis in 3%. Maloney et al (1991) found no infections at all when the fractures were "reduced by the closed technique and immobilised within 72 h of injury; open reduction within 72 h had an infection rate of 2%. Iizuka et al (1991a) reported infections in 6% of patients stabilized with compression plates, all save one being fractures at the angle. Champy et al (1986) reported an incidence of 3% in cases treated by his principles; he urged treatment within 12 h of injury. Anderson & Alpert (1992) reported the high incidence of 16% infection in 75 plated fractures; however, there was only one case of frank osteomyelitis, and the authors believed that preventable errors in technique were often responsible. Levy et al (1991) gave special attention to fractures of the mandibular angle: when only one miniplate was used, without intermaxillary fixation, the infection rate was 22%, but when two miniplates were used, no infections were seen. In our report on 50 fractures at the angle (Moore et al 1990) only one infection was seen.

When infection is diagnosed, a wound culture is taken and intravenous flucloxacillin and metronidazole are at once given. X-ray pictures of the mandible are taken, to exclude fracture displacement, bone sequestrum or any complication

relating to the plate, such as fracture or dislodgement. If there is non-union and/or osteomyelitis, the fracture is explored and necrotic material is excised, together with loose fragments of bone. Teeth in the fracture line are extracted and all plates and screws are removed. The occlusion is then established and intermaxillary fixation is reinstituted. If there is bony contact at the fracture site, a heavy mandibular compression plate is immediately inserted on the lower border of the mandible, with 4–6 bicortical screws. If there is discontinuity of bone, a bone graft is inserted; intermaxillary fixation is maintained for 6 weeks (Fig. 11.25A-C). With rigid fixation and adequate drainage, osteomyelitis usually subsides (Koury & Ellis 1992).

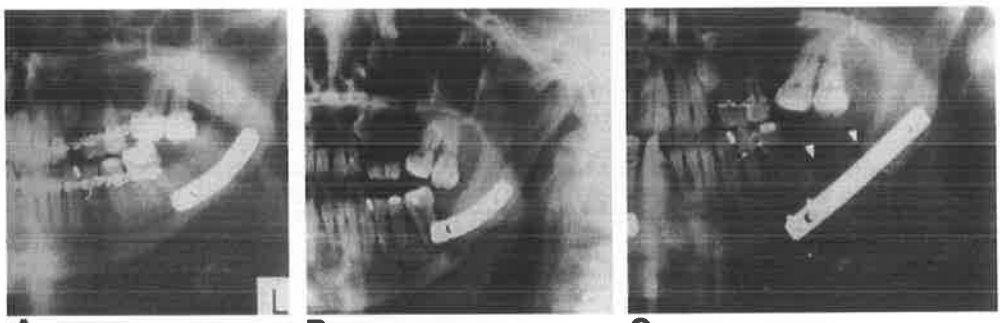


FIG. 11.25. ***Management of an infected angle fracture.*** **A.** *A fracture of the left angle of mandible following an assault to a 29-year-old male. The fracture was treated using a heavy lower-border compression plate and bicortical screws.* **B.** *After 18 months lost to follow-up, the patient returned with clinical signs of infection and non-union. The orthopantomogram showed non-union and loss of bone around first molar.* **C.** *Following debridement of necrotic bone and the first molar tooth, intermaxillary fixation, iliac bone grafting to the gap and stabilisation with heavy lower tar compression plate using eight bicortical screws, he went on to uneventful healing. The bone graft is located between the arrow heads.*

Non-union

This is clinically indistinguishable from infection, and in principle the treatment is the same.

Malocclusion and malunion

These can result from inadequate or inappropriate reduction of the fracture, but may also be the consequence of orthodontic movement of teeth which have been wired to arch bars, or the result of imbalance of masticatory muscles. Unilateral muscle injury forces the mandible into the position of greatest comfort, which may entail crossbite or overbite even when the reduction is anatomically correct. If this is the case, the malocclusion can be managed by appropriate dental methods — or more simply by rest and patience, since the complaint usually subsides with time. If the cause of malocclusion is shown by X-ray to be due to inadequate reduction of the fracture, two possibilities arise. First, the operative procedure may have been at fault: malunion from this cause should be detected by postoperative X-ray examination (Fig. 11.26A,B). Second, the patient may have been at fault, in failing to comply with the dietary programme: chewing on solid food may have fractured the plate itself, or fractured the bone around the screws. Reoperation is necessary in either case, to reduce the fracture again and to fix it more strongly. If the bone has already united — malunion in the true sense — an osteotomy will be needed to give correct occlusion; this is usually done at the level of the angle or through the body with preservation of the neurovascular bundle. The corrected occlusion is maintained by intermaxillary fixation, with either lag screws or plates to stabilize the reduction.

Plate exposure

Miniplate exposure may be due to poor operative technique in suturing the mucosa or to postoperative trauma, as from excessive zeal in using a tooth brush before the mucosa is fully healed. If there are no other problems, it is safe to leave the plate exposed until healing is complete, in 6 weeks, after which the plate can be removed without compromising the result.

Nerve injury and/or dental injury due to drilling the body of the mandible
Injuries of the inferior dental nerve, dental roots and/or tooth buds may occur when an inexperienced operator drills the outer cortex, or from the use of unduly long screws. If there is clinical and X-ray evidence that a screw is impinging on any of the listed structures, then the screw, or screws, should come out at once. It will then be necessary to follow the patient's progress for signs of loss of tooth vitality, failure of teeth to erupt, or sensory loss in the mental nerve territory. Sensory recovery, or failure of recovery, should be watched for over 12–18 months.

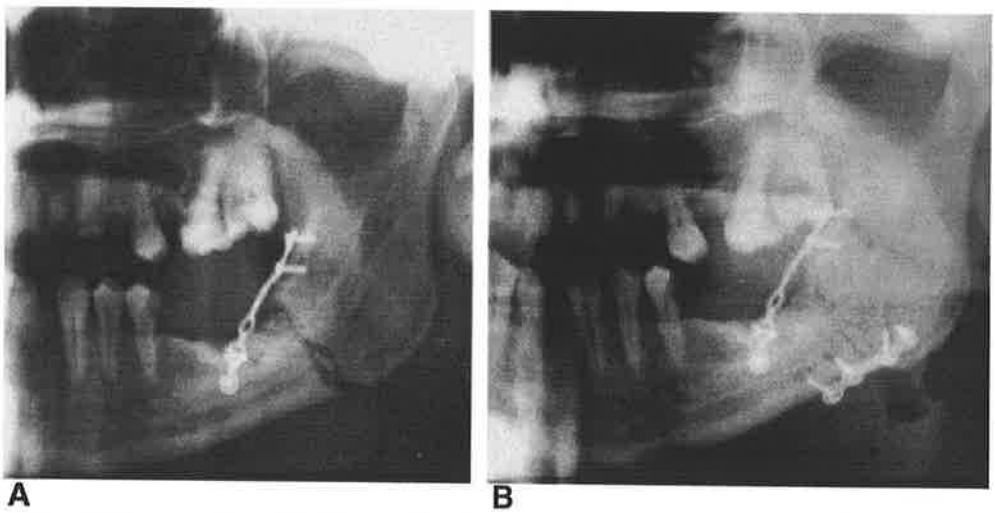


FIG. 11.26. ***Faulty operative reduction.*** **A.** *A left angle fracture of the mandible, inadequately reduced. This mistake was revealed in the X-ray pictures taken immediately after surgery.* **B.** *The fracture was re-explored via an external submandibular incision. This enabled satisfactory reduction with added stablization using a lower border miniplate*

Dentoalveolar Fractures

Surgical pathology

These may occur in isolation, either when a segment of two or more teeth is displaced en bloc, or as an area of gross comminution.

Management

When the displaced fragment of bone and teeth retains a good mucosal attachment on the lingual side, it can be preserved. The displacement is reduced, the mucosa laceration is repaired if necessary, and the reductions stabilized: this is done by etching pillars on to the crowns of both the involved and the adjacent unaffected teeth, to which an acrylic rod or bar is then fixed (Fig. 11.27). But when there is gross comminution, it is best to remove the teeth and shattered bone and to suture the mucosa over the area of denuded bone.

Fractures of the Coronoid Process

Surgical pathology

The coronoid process is a thin plate of bone, serving as the insertion of the temporalis muscle onto the ramus: as such, it is stressed to withstand vertical tension but no force applied to its lateral aspect. The process is therefore most vulnerable to a lateral impact when the mouth is open; it may also be injured by sudden contraction of the temporalis muscle at the time of an impact, or by a penetrating agent. Nevertheless, solitary fractures of the coronoid process are unusual, because the process is well protected by the temporalis and masseter muscles and by the zygomatic arch. In our retrospective review of facial fractures

treated over a 3 year period (p. 87), there was only one coronoid fracture among 324 cases of mandibular fracture — an incidence of 0.3%. Rapidis et al (1985) found that coronoid process fractures accounted for 0.6–4.7% of all facial fractures in several reported series reviewed by them. In an analysis of 52 cases of coronoid fractures, they found fractures in other parts of the facial skeleton in 77%; in 44% there were fractures elsewhere in the mandible, these being evenly distributed between symphysis, body, angle and condyle, and in 19% there was an associated fracture of the zygoma, while in only 4% were there fractures of the maxilla.

Clinical assessment

The signs may include trismus and crossbite due to injury of the ipsilateral masticatory muscles; there may be intraoral swelling and bruising in the upper retromolar region. However, these signs may be masked by the effects of associated injuries.

Radiological assessment

The fracture may be seen in the standard OPG and Towne's views; if not, it will be readily demonstrated by CT, which will also show the proximity of a depressed fracture of the zygomatic arch and hence the potential for bony fusion between coronoid and zygoma (Fig. 11.28).

Management

This is essentially the treatment of the associated injuries, followed by early mandibular mobilisation. There is no indication for open reduction and fixation of the fractured coronoid process, provided that the normal transverse space between the squamous temporal bone and the zygomatic arch has been restored.

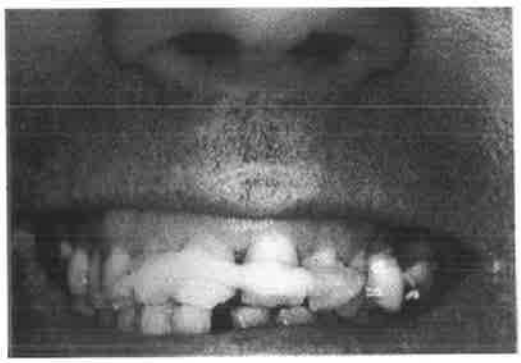


FIG. 11.27. ***Stabilization of dento-alveolar fractures.*** *An acrylic rod is etched onto the crowns of upper central teeth to stabilise a dento-alveolar fracture.*

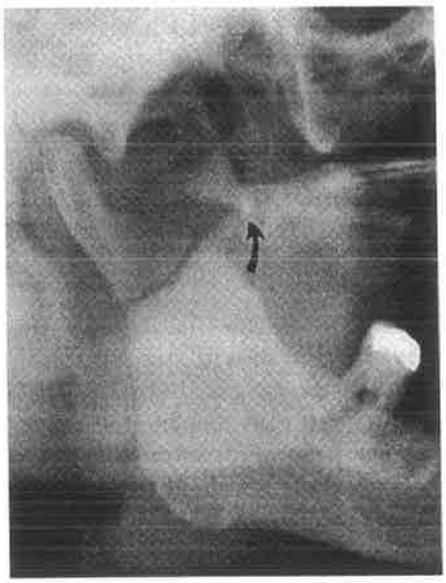


FIG. 11.28. ***Radiology of coronoid fractures.*** *X-ray demonstrating a coronoid fracture associated with a condylar fracture. The coronoid process is displaced superiorly and posteriorly.*

Fractures and Dislocations of the Condyle

Surgical pathology

The condyle is that part of the mandible which passes vertically up from the posterior border above the sigmoid notch to the glenoid fossa of the temporal bone. Fractures may occur at any level in the sigmoid notch, thus including a variable amount of the ramus in the condylar fragment (Figs 11.29 and 11.30). Rarely, there may be a sagittal fracture of the condylar head without involvement of the sigmoid notch (Fig. 11.31). There may be dislocation of the fractured condyle, either posterior, anterior, medial or lateral; rarely there is vertical dislocation into the middle cranial fossa (p. 289).

Classification

There are many classifications of condylar fractures, based on the level, the degree of obliquity, the presence of comminution or compound injury, the displacement, and the presence or absence of dislocation of the head from the glenoid fossa. Lund's (1974) classification has merit because of its simplicity (Fig. 11.32):

- Type I: enlocated head with or without displacement at the fracture site, and with no more than 60° angulation
- Type II: dislocated head, with 90° or more angulation.

Lund also described these fractures as high, involving the head or neck, and low, at the base of the condylar process.

Clinical assessment

Pain in the region of the temporomandibular joint (TMJ) may be a symptom, and trismus is a common sign. When the patient attempts to open the mouth, there will be pain and restriction of movement: this may be bilateral, but if unilateral, there may be deviation of the chin point towards the affected side. This sign is not specific: it may express any injury causing unilateral contusion of the masticatory muscles, e.g. fracture of the zygomatic arch, coronoid process, pterygoid plates or temporal bone. Indeed, a sprain of the TMJ may produce this sign. Other clinical findings may help to differentiate these types of injury, but in a severe panfacial fracture only radiology will determine the state of the condylar region. Otoscopic examination of the external auditory meatus may show bleeding from a tear in the lining of the canal: posterior displacement of the condylar head may indeed split the cartilaginous part of the canal or fracture the tympanic plate, and hearing should be tested.

Bimanual palpation of the condyles during opening and closing of the mouth may detect swelling, tenderness and perhaps absence of the normal condylar head. This can be done either by placing the fingers over the TMJ just in front of the tragus, or by inserting the finger tip gently into the external auditory canal on each side. Systematic palpation of the rest of the mandible may reveal another coexisting fracture or fractures.

Neurological examination is usually unremarkable: injury to branches of the mandibular division of the trigeminal nerve is very rare in condylar fractures. Damage to the facial nerve is also unusual, but may occur, especially if there is lateral dislocation of the condylar head.

The mouth is then inspected and the occlusion is examined. Typically, bilateral condylar fractures cause an anterior open bite not present before injury, while a unilateral fracture causes a lateral crossbite. The teeth and alveoli must be examined: there is a significant association between injuries in these structures and condylar fractures. Indeed, Lindahl (1977a) found injured teeth in 46 (37.4%) of 123 cases of condylar fracture, the association being most frequent in bilateral fractures and when the condylar head was involved.

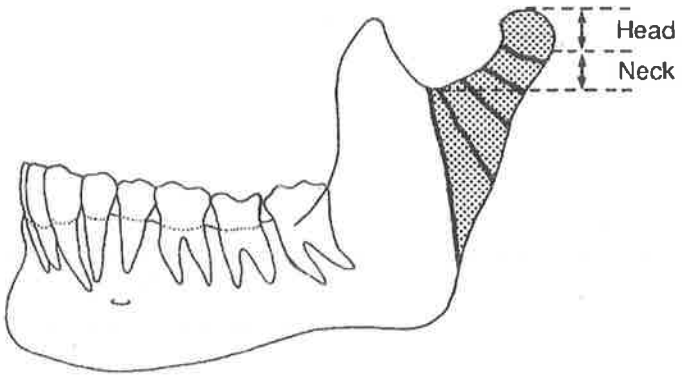


FIG. 11.29. ***Anatomy of condylar fractures.*** *A condylar fracture is any fracture which separates the condylar head from the corpus of the mandible but excludes the coronoid (see shaded area). All pass through the sigmoid notch and, depending on the level, are high or low.*

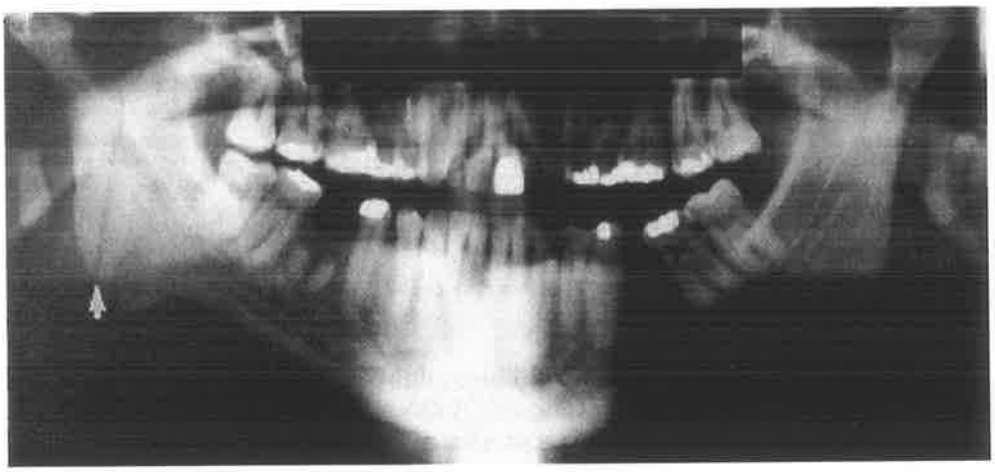


FIG. 11.30. ***Anatomy of condylar fractures.*** *A low condylar fracture extending to inferior border of ramus. To be compared with higher level in Fig. 11.28.*

Radiological assessment

The standard OPG and Towne's views will usually show fractures of the condyle. The OPG is especially helpful in showing the site of the fracture; for surgical planning, it is important to know whether the condylar fragment is large enough to allow fixation by a miniplate (Fig. 11.33).

This view also shows anteroposterior or vertical displacement of the condylar head as well as anteroposterior displacement and/or overriding at the fracture site. The Towne's view is complementary in showing medial or lateral displacement of the head or the fracture fragments (Fig. 11.34). CT scan may be helpful in showing a sagittal split, or even comminution, of the condylar head: such fractures may lead to ankylosis and need early recognition and treatment (Fig. 11.35).

Dental assessment

This is done along the lines described above; the preparation of plaster study models showing the desired final occlusion, and also an acrylic dental wafer, are necessary preludes to surgical treatment. The dentist will also plan treatment of any dental injuries. The dental findings are then discussed, together with the clinical and radiological findings, and treatment is then planned according to a formal protocol which is discussed below.

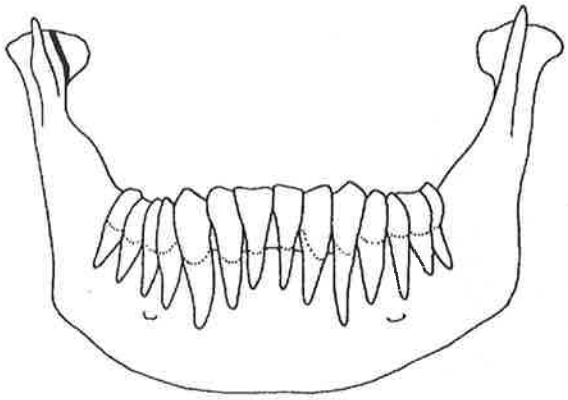


FIG. 11.31. ***Anatomy of condylar fractures.*** *A sagittal fracture of the head of the condyle may be associated with an intact sigmoid notch. Such fracture may occur in isolation or as a part of a comminuted pattern of condylar fracture.*

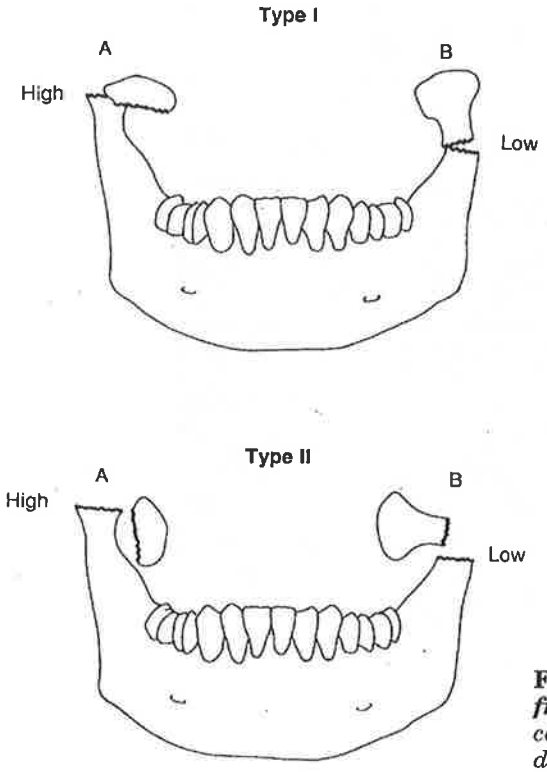


FIG. 11.32. ***Classification of condylar fractures.*** *Lund's 1974 classification of condylar fractures. Subtypes A and B denote high or low fractures respectively.*

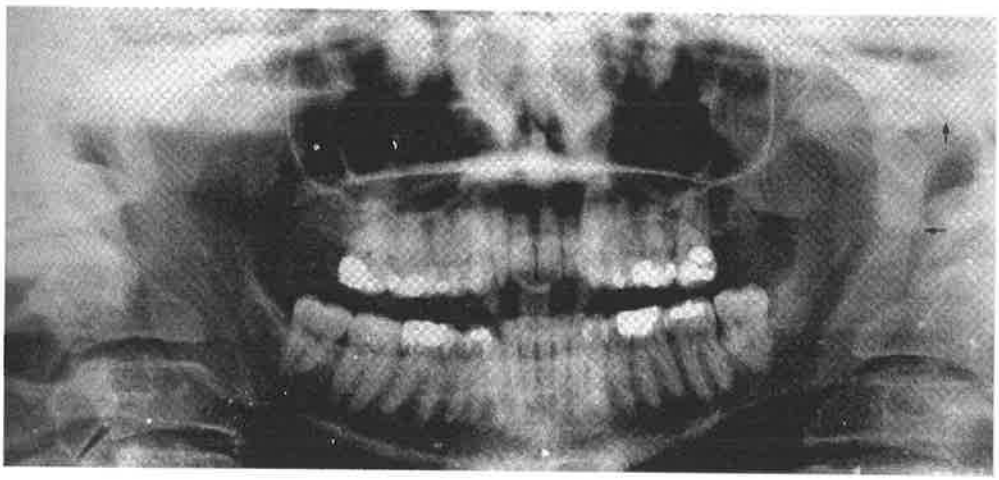


FIG. 11.33. ***Radiology of condylar fractures.*** *Orthopantomogram demonstrating medium to low level fracture of left condyle in a 14-year-old. The level of the fracture is placed above the lowest point of the sigmoid notch and below the head of the condyle.*

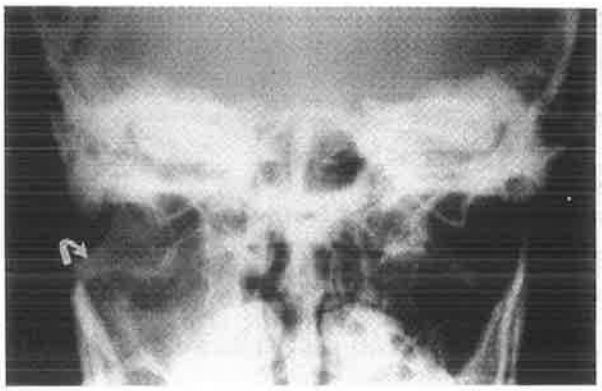


FIG. 11.34. ***Radiology of condylar fractures.*** *A Townes view demonstrating medial dislocation of the condylar head from the glenoid fossa. Angulation at the fracture site is approximately 90°.*

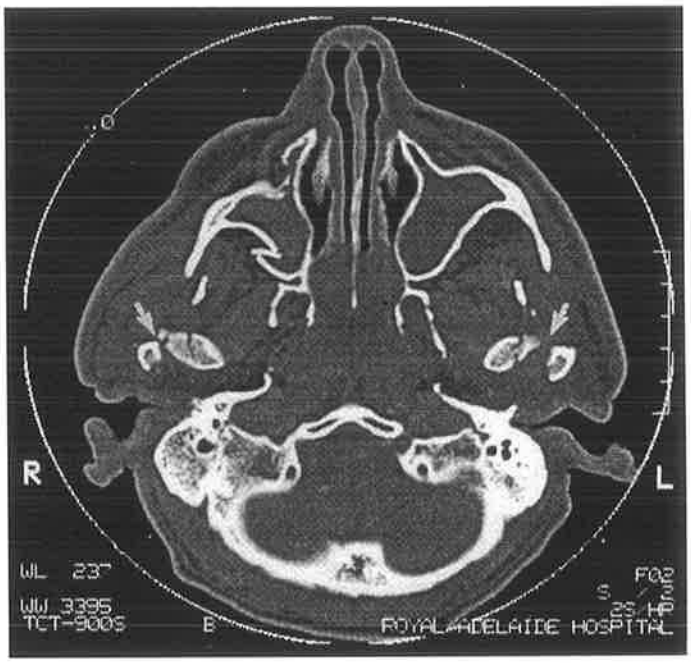


FIG. 11.35. ***Radiology of condylar fracture.*** *CT scan demonstrating bilateral sagittal fractures of the condylar heads in association with a midface fracture.*

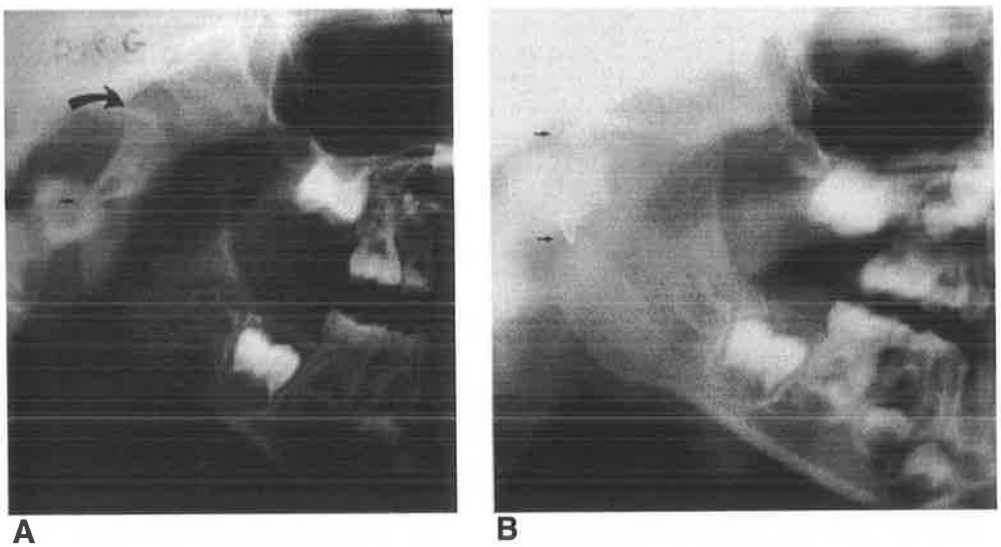


FIG. 11.36. ***Attempted wire stabilisation of condylar fracture.*** **A.** *Displaced and dislocated condylar fracture in a 7-year-old. The large arrow indicates the dislocation of the condylar head, and the small arrow indicates the angulation at the fracture site.* **B.** *1 year following reduction and stabilisation with an interosseous wire, there has been relapse followed by malunion. Remodelling did not take place, probably because of the invasive nature of the surgery (all muscle was stripped from the condyle) and inadequate stabilisation (wire only). Arrows point to condylar remnant.*

Principles of treatment

The management of these injuries is controversial, chiefly because there is uncertainty over the natural history of the unreduced fracture in children and in adults.

Lund (1974), Lindahl (1977a–c) and Lindahl & Hollender (1977) published studies on restitutional remodelling of the displaced condylar fracture in childhood, and the associated compensatory growth, which tend to support a conservative plan of management; indeed, Lund found that remodelling could result in a normal condyle. However, this process was significantly less effective when growth was nearing completion or completed. Remodelling was also incomplete when the condylar head was dislocated from the glenoid fossa, and when the fracture was low on the condylar neck. Compensatory growth of the mandible on the affected side was observed only if body growth was still occurring. In unilateral cases, compensatory growth was evident in 78% of the studied cases; in 30% this compensatory growth was excessive, causing deviation of the chin point to the unaffected side. Compensatory growth was found to be most pronounced when fractures occurred at puberty, and when the condylar head was not dislocated. Lund found no relationship between the degree of compensatory growth and the height of the fracture.

Lindahl treated 123 patients in the Maxillofacial Unit School of Dentistry in Gothenburg over a 4 year period. He noted that all the children in the series developed a satisfactory occlusion. He did not say whether this was spontaneous or the result of orthodontic treatment, nor did he relate any need for orthodontic treatment to the type of fracture, the degree of remodelling and compensatory growth or the patient age. Nor was a time-cost analysis of the achievement of satisfactory occlusion given for the various fracture types. However, Lindahl did look at the long-term effects of conservative management on mastication. He found that in children, the asymmetry of mandibular movements usually disappeared in 2 years. But in adults, the signs of asymmetrical movement persisted or became worse. Moreover, symptoms such as clicking tenderness or pain, rare in children, were frequent in adults (Lindahl 1977a,b)

Other authors have questioned whether conservative treatment will give a satisfactory, albeit adjusted, occlusion (Hinds & Parnes 1966, Robinson & Rowe 1971). It seems clear that while the results of conservative treatment may be acceptable, they are far from perfect in respect to chin deviation and to TMJ function in adults. Moreover, the so-called satisfactory results may require protracted and expensive orthodontic treatment.

In some centres, aggressive strategies of treatment have been proposed. Schettler & Rehrmann (1975) in Dusseldorf employed elastic traction from a long bridle attached to a plaster head cap: this was applied to the mandible through a bone hook inserted into the chin. Fey reported complete restoration of articular function in all treated cases; however, patients had to wear this elaborate appliance for an average period of 3.5 weeks. Open anatomical reduction of the condylar fracture has been also been favoured (Brown & Obeid 1984), but hitherto this has not gained universal acceptance and there are several reasons for this. First, methods of stabilisation have been imperfect, and have allowed malunion in one or other plane, or even complete relapse (Fig. 11.36A,B). Second, methods of exposure have not always been safe, or even effective in all types of fracture. Some reports have given no emphasis on the importance of preserving muscle attachments, such as that of the lateral pterygoid to the proximal fragment, or to the maintenance of the capsular attachments to the condyle (Takenoshita et al 1989). Moreover, operative techniques have often been reported without long-term results. For all these reasons, operative intervention has been suspect. Several criteria should be filled for operative procedures to-be acceptable:

1. The operative procedure must provide for restoration of normal function and occlusion within 6–12 weeks

2. The fracture must be reduced so that the muscles which act on the condyle regain normal tension and position
3. The displaced fragment must be large enough to be reduced and stabilized in three dimensions
4. The stabilisation must be maintainable until bone union occurs
5. The operation must not damage the attachments of the lateral pterygoid muscle to the condylar head
6. There must be no operative morbidity from injury to nerves or other important structures, or to mandibular growth

If these criteria can be met, operation offers the prospect of rapid restoration of function and of occlusion, by achieving an anatomical reduction of the fracture and of the TMJ joint, followed by early mobilisation.

Conservative management

In most cases this is principally the encouragement of early mobilization by active jaw movement. With adequate pain relief and a liquid diet, the patient will usually bite into the pretraumatic occlusion within a couple of days. Active encouragement continues until an opening close to 40 mm between the upper and lower incisors is achieved. If the patient cannot regain the pretraumatic occlusion, arch bars are applied and occlusion is established and maintained for 2 weeks by intermaxillary wire fixation.

By this time, the pain should have ceased and the intermaxillary wires can be replaced by elastic bands—light by day, heavy by night. The bands are intended to counteract the usual tendency to deviation of the chin to the affected side (in unilateral injuries) or to anterior open bite (in bilateral injuries); the light bands allow active movement, which is encouraged. This regimen is continued for another 6-8 weeks and is ceased only when the patient can maintain the pretraumatic occlusion. It may then be necessary to encourage wider opening of the mouth by an exercise programme. After eating, the patient spends 5 min three times a day before a mirror, opening -protruding - retracting - and laterally deviating the lower jaw. The patient, after these exercises, is given a stack of wooden spatulas and inserts each day an increasing number of these between the incisor teeth, until a 40 mm interincisal opening is achieved. Pain is to be expected, and the patient is reassured that this is a necessary result of stretching scarred tissues.

The best reported results from conservative management are those of Lindahl, discussed above, and they are far from perfect. In adults with fracture-dislocation of the condyle, conservative management may lead to malocclusion, loss of teeth, and pain in the TMJ (Robinson & Rowe 1971). Nevertheless, conservative treatment is indicated if there is little or no displacement, or if the fracture line is so high that surgical stabilisation is impossible.

Operative management

Zide & Kent (1983) have given as absolute indications for operation:

1. Fracture dislocation of the head into the middle fossa, or into the temporal fossa if associated with clinical disability
2. Intra-articular foreign body
3. Lateral extracapsular dislocation of the condyle
4. Inability to open the mouth or to bring the mandible into occlusion after 1 week with evidence of a bony block
5. Compound injuries, e.g. gunshot wounds.

Zide's relative indications include:

6. Displaced condylar fracture, where the fracture line is near the lower border of the sigmoid notch and the head is dislocated from the glenoid fossa
7. Condylar fracture, where vertical displacement is associated with displaced fracture(s) of the upper jaw.

We endorse these indications, and believe that in adults, when there is displacement of the fracture and where the proximal fragment has sufficient length to accept two screws to hold a plate, then anatomical reduction and rigid plate fixation will allow immediate mobilisation and good prospects of a satisfactory functional and aesthetic outcome. The scope of operative intervention in Children is considered in Chapter 19.

The condyle may be approached through an intraoral incision, or through various skin incisions discussed by Ellis & Dean (1993). Whichever route is chosen, the approach must give good exposure of the fracture site, must allow reduction without damage to the joint capsule or disk, and must not interfere with the attachment of the lateral pterygoid muscle. It must enable the surgeon to establish three-dimensional stability after reduction.

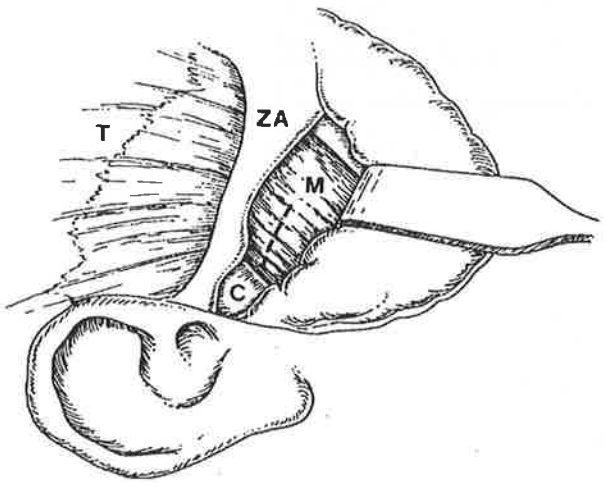


FIG. 11.37. *Surgical exposure of condylar fractures.* *Anatomy on display after turning down the bicoronal flap. T, temporalis muscle; M, masseter muscle; C, capsule of temporomandibular joint; ZA, zygomatic arch.*

We have found these aims achievable through the bicoronal scalp incision described on p. 238, but extended in front of the ear to the level of the ear lobe. It is often unnecessary to make the full coronal incision: one may spare the contralateral side. The incision is run along the natural contour of the helix and the tragus to be inconspicuous when healed; the temporal part of the incision is likewise designed to be hidden by the fall of the hair The scalp flap is turned down in the subgaleal plane to a level 1-2 cm above the superior orbital margin, and thereafter is taken subperiosteally to the orbital rim on the affected side. The superficial temporal fascia is then incised, and the dissection proceeds through the fat to expose the zygomatic arch. This method of dissection spares the frontal branch of the facial nerve. The masseter is next exposed a distance at least 2 cm below the zygomatic arch, and the capsule of the TMJ is seen (Fig. 11.37). The parotid gland is separated from the cartilaginous external auditory canal, but no further, and the facial nerve is kept covered by soft tissue. The masseter is then divided 1-2 cm below the arch of the zygoma, the cut being taken from the posterior border forward, care being taken to avoid injury to the nerves and vessels passing through the sigmoid notch. It is now possible to expose the neck of the condyle, the upper ramus, and the sigmoid notch: the fracture site is on view and can be fully exposed with only light retraction (Fig. 11.38).

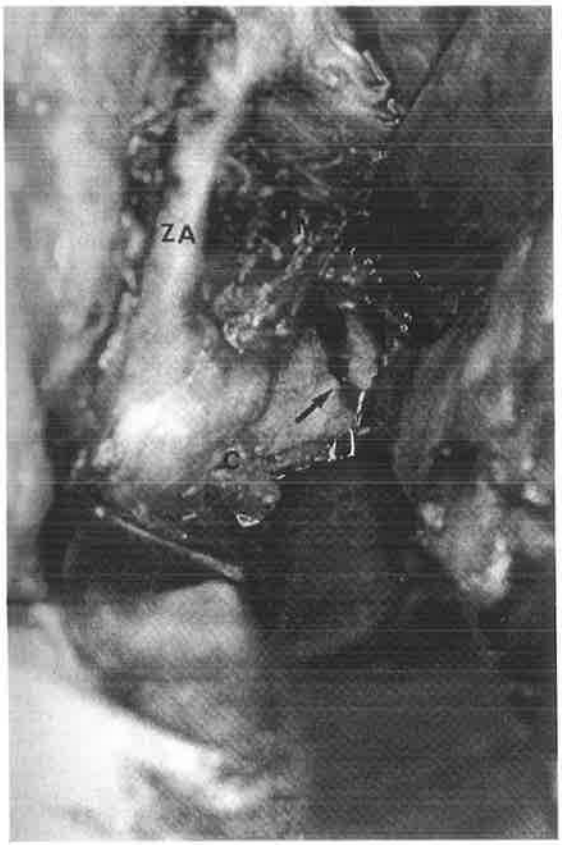


FIG. 11.38. *Surgical exposure of condylar fractures.* *Operative view of condylar fracture (arrowed). The posterior masseter has been cut to explore the fracture. M, masseter muscle; C, capsule of temporomandibular joint; ZA, zygomatic arch.*

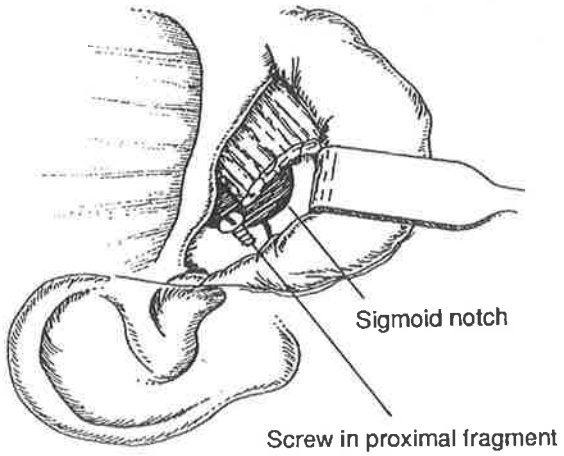
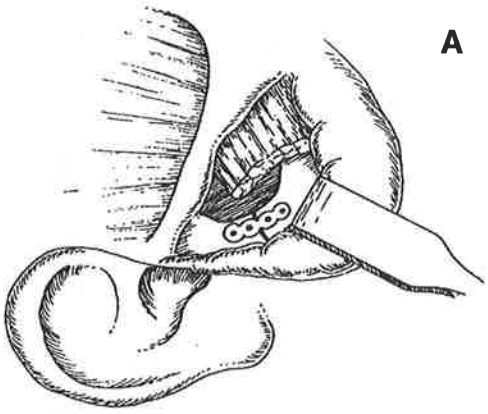


FIG. 11.39. *Surgical exposure of condylar fractures.* *Via a myotomy of the posterior masseter muscle, a hole has been drilled in the accessible proximal fragment and a screw inserted. The latter acts as a 'handle' on the proximal fragment, allowing its reduction without further trauma to its capsular or muscular attachments.*

A drill hole is then placed in the proximal fragment, and a 13 mm screw is inserted a distance of 9 mm (Fig. 11.39). This screw is then used as a handle: it is grasped with strong artery forceps and the proximal fragment is manipulated into the position of anatomical reduction: this reduction is aided by downward pressure on the ramus via the sigmoid notch. At no time is the joint capsule or the lateral pterygoid muscle injured or dissected. When reduction is effected, a 4-hole miniplate is bent to conform with the lateral concavity of the bone and fitted with four screws (Figs. 11.40 and 11.41). The screw used in manipulation is then removed. The masseter and superficial temporal fascia are repaired with 3/0 Vicryl sutures. The scalp wound is closed in two layers, 6/0 Vicryl being used in the pre-auricular area. Drainage is rarely needed. Arch bars and intermaxillary fixation are not used, except when there is a concurrent fracture elsewhere in the mandible or the upper jaw: in such cases, intermaxillary fixation is left until the scalp is healed. Postoperative care is as described for fractures of the mandibular body. The jaw is mobilised in the manner described as conservative management. At 6 weeks, mobilization is usually complete and the patient may take a normal diet.



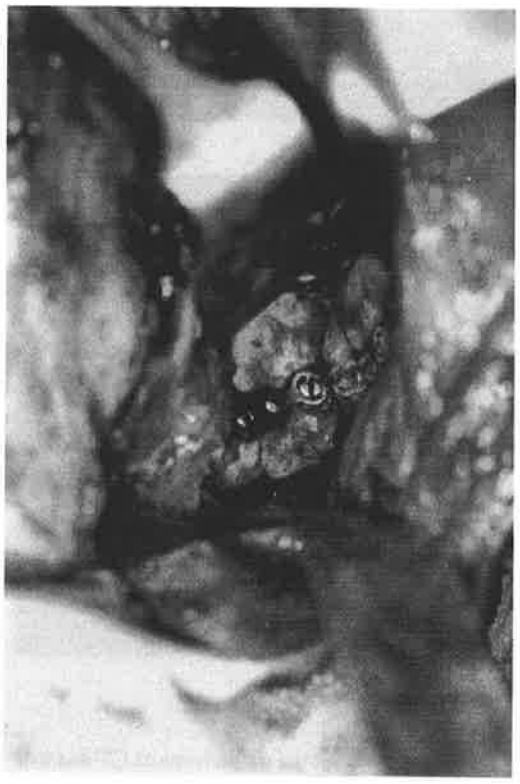


FIG. 11.40. ***Surgical exposure of condylar Fractures.*** **A.** *At least two screws through holes in a plate on each side of the fracture line provides Three dimensional stabilisation, allowing early return of function.* **B.** *Operative view.*

Steinhauser (1964) described an intraoral approach to the fractured condyle and Lachner et al (1991) have used this exposure for miniplate fixation in 14 cases, with satisfactory results; they did not indicate what degree of displacement was an indication for open reduction. We see this exposure as adequate in very low oblique condylar fractures, but not in more horizontal higher fractures (Fig. 11.42A,B). Indeed, Habel et al (1990) found it necessary to osteotomize the coronoid process to gain access to the proximal fragment when using an intraoral incision. Other external approaches to the condyle have been proposed: in the pre-auricular area (Al-Kayat & Bramley 1980), through the postauricular area (Hoopes et al 1970) and through various submandibular incisions (Fig. 11.43). But we favour the extended coronal scalp incision described here, because it gives the surgeon direct exposure of the fracture without heavy soft tissue retraction and risks of damage to the facial nerve (Fig. 11.44).

Brown & Obeid (1984) have reviewed the methods of stabilising the fracture when it has been exposed and reduced. Surgeons have used various types of interosseous wiring, Steinmann's pins with or without wiring, external fixateurs, and miniplates. Miniplates have the great advantage of giving three-dimensional stabilisation while allowing mobilisation and early return to normal function. With the exception of external fixateurs, the other methods listed do not give stability, and the jaws must be immobilised by intermaxillary fixation, which is detrimental to early functional recovery. External fixateurs, by their nature, must totally prevent mobilisation.

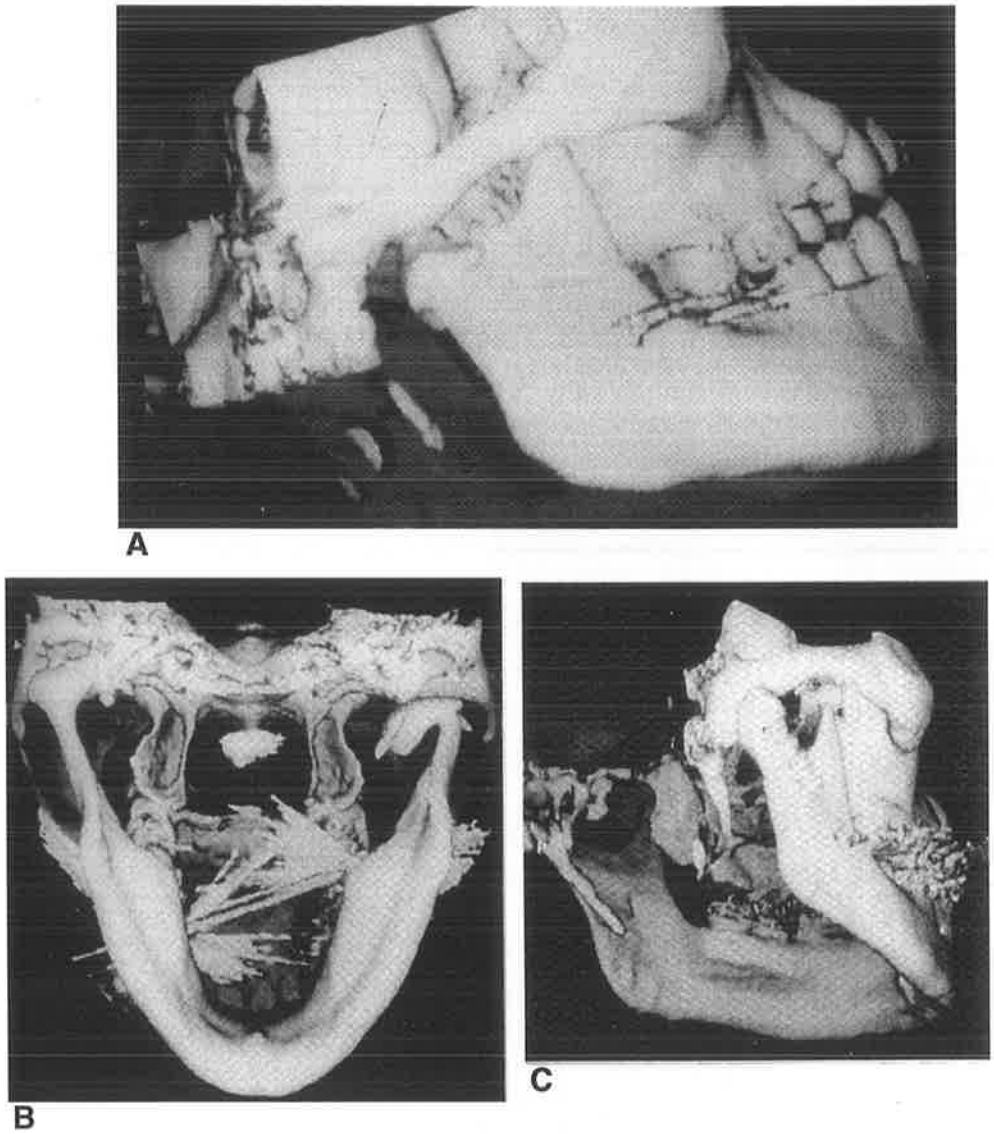
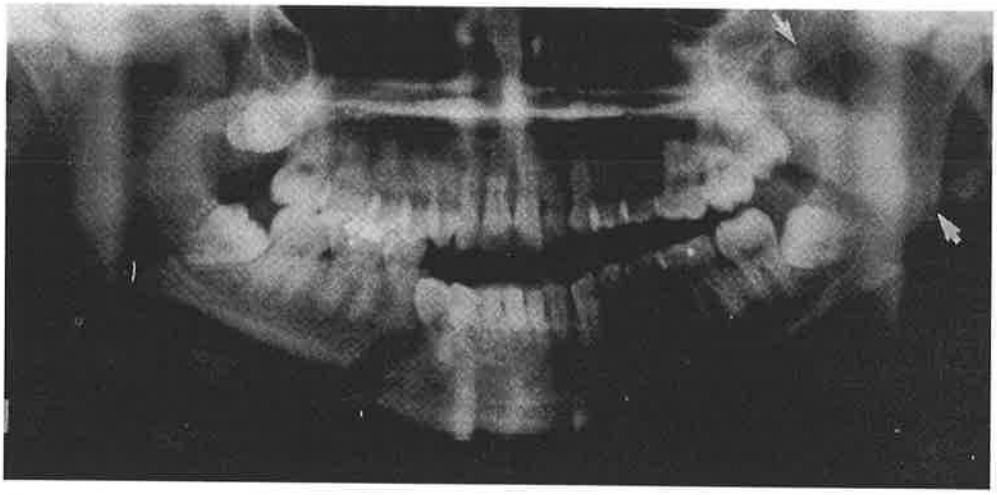
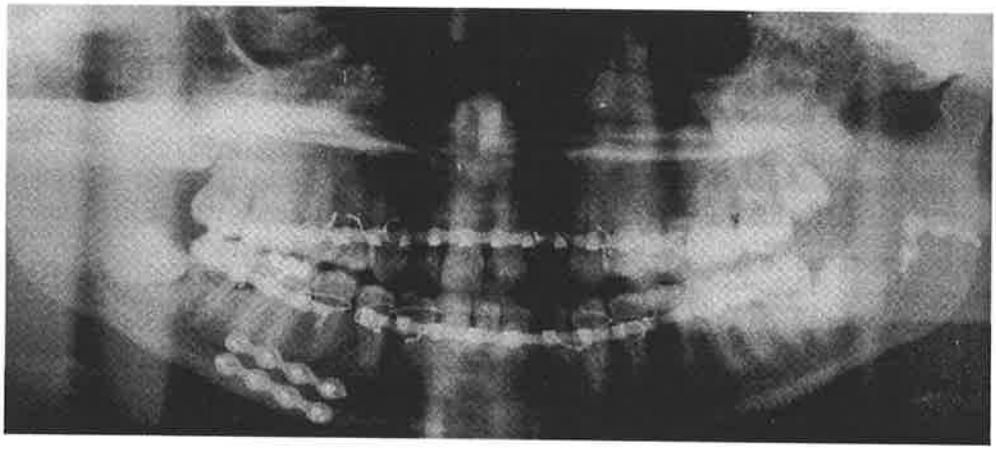


FIG. 11.41. ***Surgical exposure of condylar fractures.*** **A.** *Three-dimensional CT scan of a condylar fracture with medial dislocation of the head.* **B.** *Three-dimensional CT scan from below, showing the head displaced into the medial pterygoid region.* **C.** *CT scan after reduction and stabilisation via an extended bi-coronal approach with myotomy of the posterior fibres of the masseter muscle. The four-hole plate and screw heads can be readily seen.*



A



B

FIG. 11.42. ***Surgical exposure of condylar fractures.*** **A.** *Orthopantomogram of condylar fracture low enough for intraoral reduction (arrow).* **B.** *After reduction and stabilisation using an intraoral approach.*

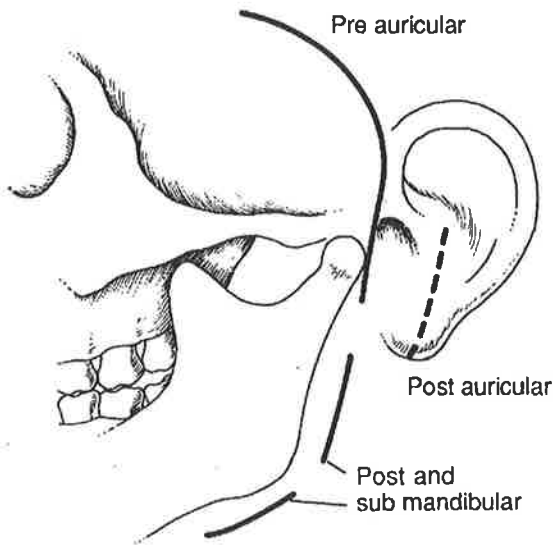


FIG 11.43. ***External incisions for exposure of condylar fractures.*** *External skin incisions described for access to condylar fractures.*

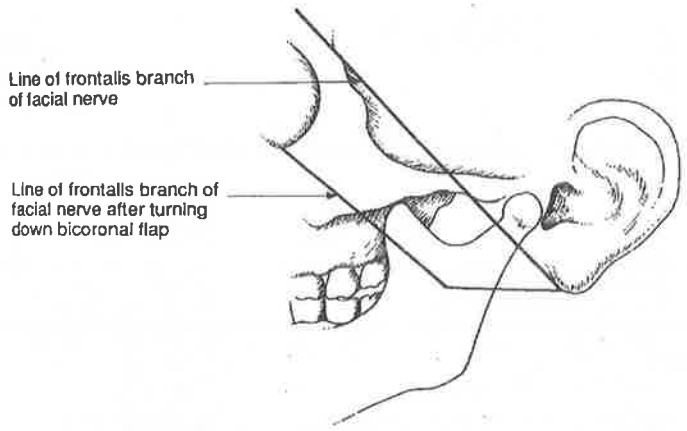


FIG. 11.44. ***Bicoronal flap 'relocation' of the frontalis nerve.*** *The act of turning down the bicoronal flap including frontalis muscle locates the frontalis branch of the facial nerve below the sigmoid notch of the mandible. Retraction on the nerve is extremely light as a result.*

Complications of condylar fractures

The condyle is isolated from the teeth by vascular soft tissues, and condylar fractures usually heal well. However. complications can occur. These include:

- Growth disturbance
- Avascular necrosis
- Ankylosis of the TMJ
- Malunion
- Other TMJ dysfunctions.

Growth disturbance
This problem is peculiar to childhood and is discussed in Chapters 19 and 21.

Avascular necrosis

This is sometimes seen when an intracapsular fracture detaches a fragment of the head of the condyle which is denuded of its blood supply; the cortical surface of the condylar head is eroded and the surface becomes rough and flattened. This complication is well described by Sanders et al (1977). The patient complains of pain and limitation of movement; changes in the condylar surface may be demonstrated by X-ray. Sanders advised removal of the avascular fragment of the head of the condyle and smoothing of the remaining bone. Avascular necrosis can also result from damage to the blood supply in the course of an open operation on the TMJ (Iizuka et al 1991b). When it is necessary to stabilize the condyle by open surgery, we believe that it is important to retain all residual capsular attachments of the proximal fragment, and also the insertion of the lateral pterygoid muscle. Some surgeons indeed have completely isolated the proximal fragment by dividing all soft tissue attachments prior to reduction and fixation, and have reported no problems from this (Takenoshita 1989), but we believe that avascular necrosis can occur under these circumstances, and we have seen an example of this (Fig. 11.36).

Ankylosis of the TMJ
TMJ ankylosis is more likely to develop when there has been tearing or disruption of the articular disk, associated with comminution of the condylar articular surface, as in intracapsular fractures. In such cases, early zealous mobilization of the jaw is mandatory, with close supervision and careful follow-up. If there

are associated fractures of other parts of the mandible, it is important to avoid treating these by intermaxillary fixation: miniplate osteosynthesis is the treatment of choice, followed by early progressive mobilisation. This regimen requires patient compliance, and this will as a rule be lacking in patients who are in coma or disturbed consciousness from head injuries or who are demented, psychotic, addicted to drugs, or intellectually disabled.

TMJ ankylosis has many implications: it causes difficulties in feeding, and may result in poor dental hygiene and consequent oral sepsis. The condition may also be a potentially dangerous factor in inducing anaesthesia; the anaesthetist should always be warned of possible difficulty in intubation and a flexible fibroscope may be useful.

In an established case, treatment can be offered. If compliance is assured, an arthroplasty is possible. The scope of the arthroplasty to be undertaken ranges from simple freeing of soft tissues and interposition of cartilage or muscle, through to total joint reconstruction, which is indicated when there is a significant bony ankylosis. Various reconstructive materials have been recommended (p. 602). Our experience is chiefly with costochondral grafts: we have used onlay rib grafts, secured to the ramus with lag screws or plates, at least 1.5–2 cm of the attached cartilage being inserted into the glenoid fossa. Our operative techniques are discussed in Chapter 21 (p. 601).

Malunion; other TMJ dysfunctions
Malunion can be tolerated unless occlusion is deranged. If after 12 weeks of conservative therapy and dental occlusal adjustment, there is still a significant open bite, with or without an occlusion likely to cause impairment of the vitality of the teeth, then there is a strong case for surgical correction of the malocclusion forthwith (Hinds & Parnes 1966). Failure to do so may result in tooth loss and/or TMJ dysfunction. Standard osteotomies through the ramus will easily correct buccal anterior crossbites. Vertical subsigmoid osteotomy with plate fixation (p. 612) and sagittal split osteotomy with lag screw fixation (p. 609) will allow early mobilization; these procedures should be done after intensive physiotherapy has given the fullest possible range of jaw opening.

Dislocations of the Condyle

Classification and surgical pathology

While dislocation of the condyle from the glenoid fossa is often associated with a fracture of the condylar process, simple dislocations of the condyle do occur in the absence of such a fracture. They can be classified as:

1. Anteromedial or anterior—the common form
2. Posterior—relatively uncommon
3. Medial—relatively uncommon
4. Lateral—relatively uncommon
5. Vertical (upward) or central dislocation through a fracture in the glenoid fossa—rare (Pieritz & Schmidseder 1981, Worthington 1982).

Clinical assessment

In the common anteromedial dislocation, which is likely to be bilateral, there is usually pain in the region of the affected joint(s), prognathism, an open mouth and an inability to close into normal occlusion. It is said that this dislocation is typically caused by a blow to the chin, especially when the mouth is open. There may be a history of previous dislocation.

The less common dislocations are likely to be associated with fracture of the body of the mandible. In lateral dislocation, there will be obvious deformity and often an ipsilateral facial paralysis, a rare finding in other mandibular dislocations and fractures. In posterior dislocation, the external auditory canal is likely to be bruised or even obstructed by the dislocated condyle.

Radiological assessment

It is necessary to exclude other fractures as well as to locate the position of the condyle in relation to the glenoid fossa; CT is especially useful in this.

Management

The treatment of anterior dislocation, whether unilateral or bilateral, has not changed much since the time of Hippocrates (p. 9). Pain relief is given, and under either intravenous sedation or general anaesthesia, the dislocation is reduced. To do this, a downward pull is applied with the thumbs on the retromolar area; when the muscular resistance is overcome the ramus comes down and the condylar head can be levered back over the articular eminence into the glenoid fossa. It may be appropriate to rest the jaws by intermaxillary fixation, using light elastic band traction, for several days at least, and then to mobilize the jaw slowly. Recurrent anterior and anteromedial dislocation may require a surgical procedure to block excessive forward movement over the articular eminence. To do this we have used the procedure reported by Dautrey & Pepersack (1982). In this, a posterior osteotomy of the zygomatic arch is performed, allowing downward fracture of the arch to form a block against dislocation of the condylar head (Fig. 11.45).

Medial and lateral dislocations are more difficult to reduce (Ferguson et al 1989, Worthington 1982). The induction of anaesthesia can be dangerous: the condyle can be tightly wedged in the temporal fossa, and the displacement of the jaw may prevent the anaesthetist from visualising the larynx. The anaesthetist must know of this hazard, and the surgeon must be ready to perform an emergency tracheotomy.

Medial dislocation has been reduced by cutting and temporarily displacing a segment of the zygomatic arch to permit strong downward traction of the ramus. Lateral dislocation has been reduced by traction exerted through a wire passed through the ramus. Exposure through the extended coronal scalp flap described above allows visualization of these difficult dislocations, which can then be reduced with or without osteotomy of the zygomatic arch (Fig. 11.46A,B). Because these dislocations entail damage to the capsule, disc and muscle attachments, early mobilization after a short period of rest is desirable: to allow this, any associated mandibular fractures should be plated.

Patterns of mandibular fracture

In our retrospective study of adults with facial fractures treated in a 3-year period (p. 87), there were 324 patients with 491 fractures of mandible. Table 11.1 shows the anatomical distribution of these fractures, in comparison with some other reported series. It is noteworthy that in nearly half of our cases, the mandible was broken in more than one place: in 152 (46.9%) there were two fractures, and in eight (2.5%) there were three.

Of the cases with double fractures, the commonest combination was angle + body (235%). In descending order of frequency came condyle + body (14.5%), bilateral body (3.5%), bilateral angle (3%), and condyle angle (2.4%). Of the small group with triple fractures, all had combined fractures of condyle + body + angle, in three of these, both condyles were fractured. Examples of patterns of multiple mandibular fracture are shown Figs 11.47–11.51.

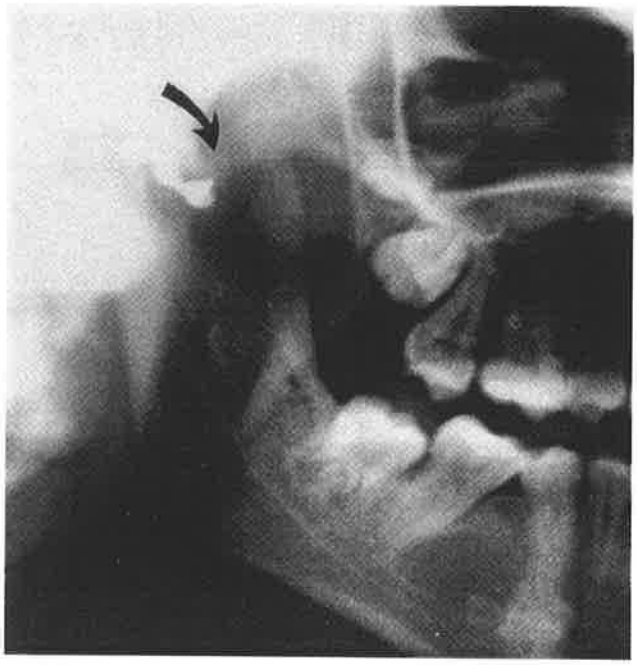


FIG. 11.45. *Surgical 'blocking' of anterior dislocation.* *OPG of Dautrey procedure. The zygomatic arch has been down fractured in the region of the articular eminence (arrow) and stabilised with a miniplate. This acts to block excessive forward translation of the condylar head.*

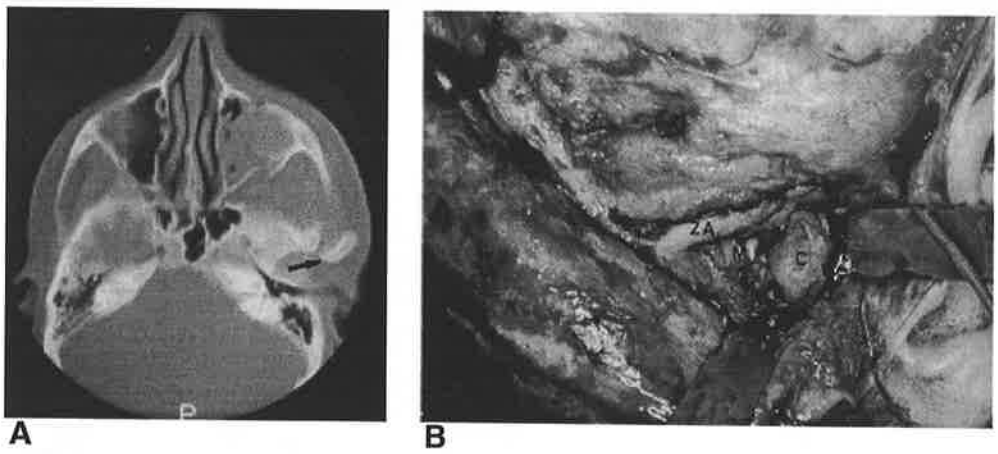


FIG. 11.46. *Management of lateral dislocation of condyle.* **A.** *CT scan of lateral dislocation of left condyle (arrow). This was associated with a left body of mandible fracture, right condylar fracture and upper jaw fractures.* **B.** *Operative appearance via extended bicoronal flap exposure and myotomy of masseter. The condylar head can be seen lateral to the zygomatic arch. C, condylar head; M, masseter muscle; ZA, zygomatic arch.*

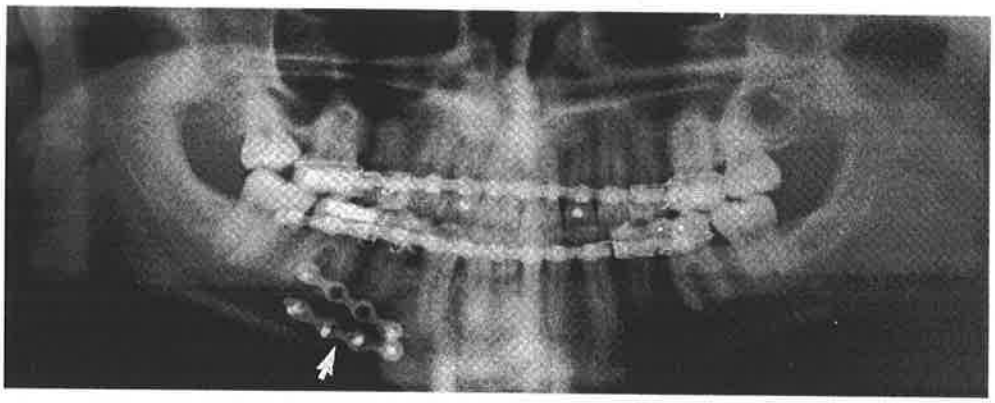


FIG. 11.47. *Mandibular fracture patterns.* *Post-fixation orthopantomogram of body fracture through the bicuspid/first molar area (arrow). This is relatively rare.*

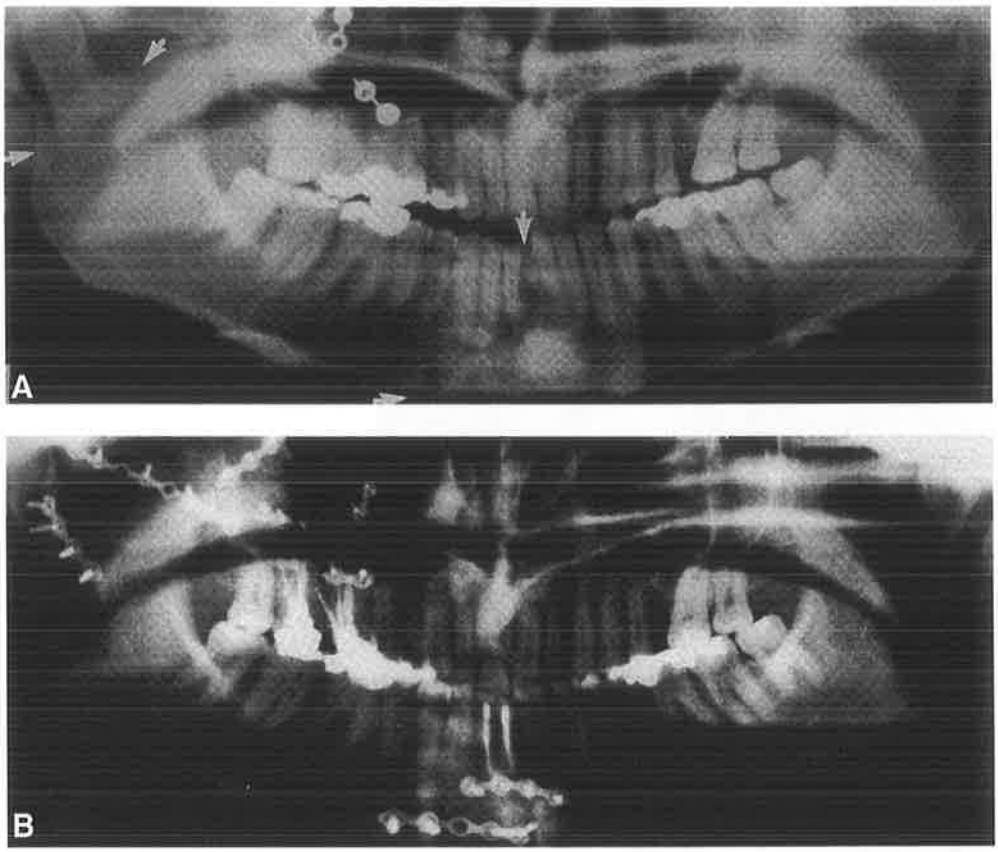


FIG. 11.48. ***Mandibular Macabre patterns.*** **A.** *Pre-operative orthopantomogram of right condylar fracture associated with Symphyseal fracture (arrows). This is a relatively common problem. However, this case also included fractured zygoma and maxillary dento-alveolar fracture.* **B.** *Post-surgical reduction and plating of mandibular and midface fractures.*

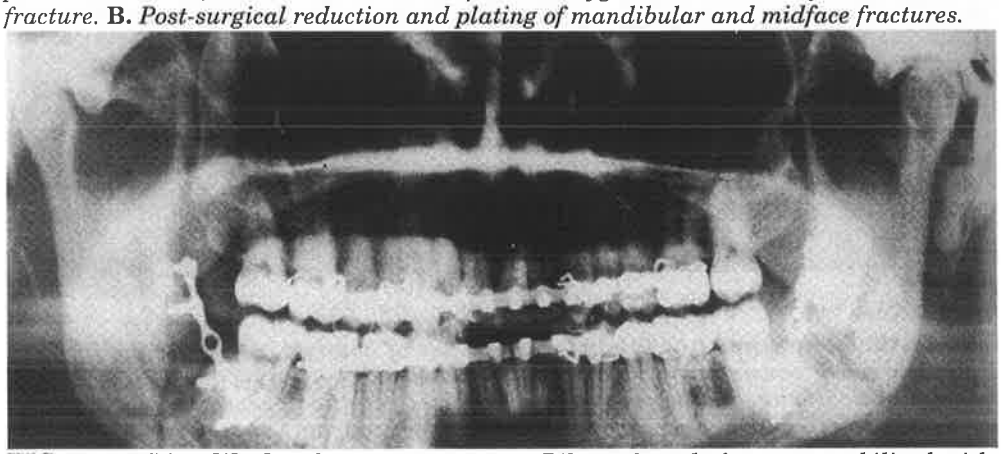


FIG. 11.49. ***Mandibular fracture patterns.*** *Bilateral angle fractures stabilised with miniplates. This method allows immediate movement of the mandible and rapid return to function.*

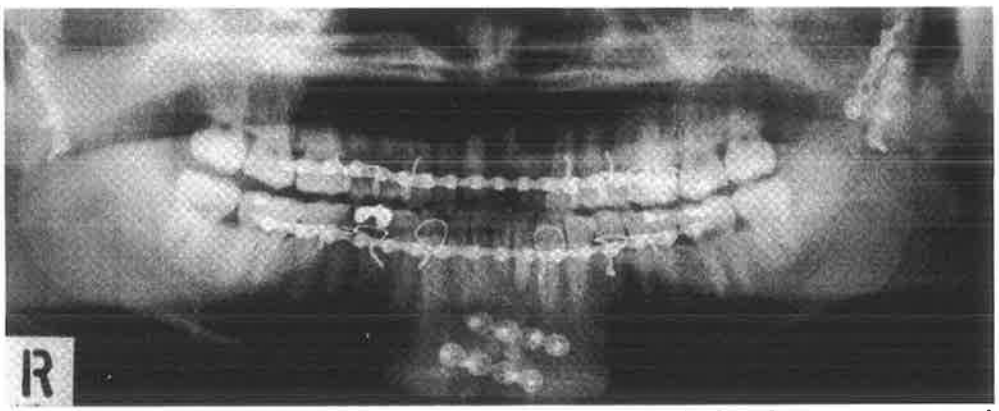


FIG. 11.50. ***Mandibular fracture patterns.*** *Post-surgical orthopantomogram of bilateral condylar fractures associated with symphyseal fracture. Miniplate stabilisation of these fractures provides rapid return to function.*

TABLE 11.1

Classification of mandibular fractures, with percentage incidences in series from South Australia (Australia Craniomaxillofacial Unit: ACFU), Western Scotland (Ellis et al 1985) and Iowa, USA (Olson et al 1982)

	ACFU(%)	Ellis et al 1985 (%)	Olson et al 1982 (%)
Condyle	20.2	29.3	29.1
Coronoid	0.3	2.2	1.3
Ramus	3.05	2.6	1.7
Angle	36.46	23.1	24.5
Body	16.50	33.0	16.0
Symphyseal	23.82	8.4	22.0

Maxillary Fractures

Surgical pathology

An understanding of the blood and nerve supply to the maxilla and upper dental arch is essential both in planning safe incisions for exposure of an already damaged maxilla and in understanding the patterns of numbness associated with maxillary fractures (pp. 48 and 61). On each side, the greater palatine canal transmits the greater palatine artery and nerve which supply all of the bone and mucosa of the hard palate (Fig. 11.52). Branches of the maxillary artery and maxillary nerve enter the posterior maxilla through small foramina to become the posterior superior alveolar (or dental) artery and nerve; the nerve supplies the molar teeth through the dental plexus. Branches from the infraorbital artery and nerve enter the anterior maxillary bone from the orbital floor and supply the anterior teeth (Fig. 11.53A,B) It can be seen that fractures of the orbital floor and/or the anterior maxilla may result in numbness of anterior teeth while fractures extending low in the maxilla posterior to the first molar region may result in numbness of the posterior teeth. Apart from the above-named arteries the maxilla gains blood supply from gingival attachments to the alveolar bone and through its attachments to the soft palate from the pharyngeal and palatine branches of the facial artery and the ascending pharyngeal branches of the external carotid artery. Where this profuse blood supply is interfered with by a significant degloving of soft tissue, and particularly if there is associated detachment of the soft palate from the posterior hard palate, then care must be taken in surgical exposure of the maxilla lest dissection detaches the remaining blood supply from the maxillary bone and the associated teeth.

The skeleton of the midfacial region appears designed to transmit the powerful forces of mastication through to the base of skull. The strong horizontal elements in the maxillae, alveoli and hard palate make up a foundation to support the three paired vertical bony condensations or buttresses (Fig. 2.15). The most anterior of these extends from the dento-alveolar arch in the lateral incisor/canine region superiorly along the piriform margin to the medial orbital rim and frontomaxillary suture. The middle buttress extends from the region of the first molar tooth to the body of the zygoma and through this bone as the lateral orbital wall to the frontozygomatic suture. The posterior buttress is represented by the attachment of the maxillary tuberosity to the pterygoid plates and hence to the spheroid (Fig. 11.54) (Manson et al 1980).

The biomechanical stresses in the midface have been harder to assess than those in the mandible. However, the load paths for distribution of force appear to

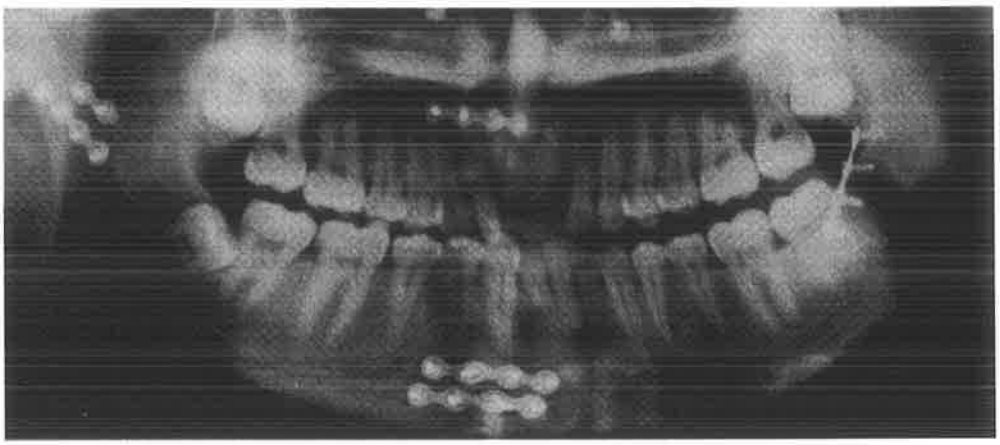


FIG. 11.51. ***Mandibular fracture patterns.*** *Post-surgical orthopantomogram of condyle, body and angle fracture pattern associated with upper jaw fracture. Miniplate stabilisation leads to rapid return of function.*

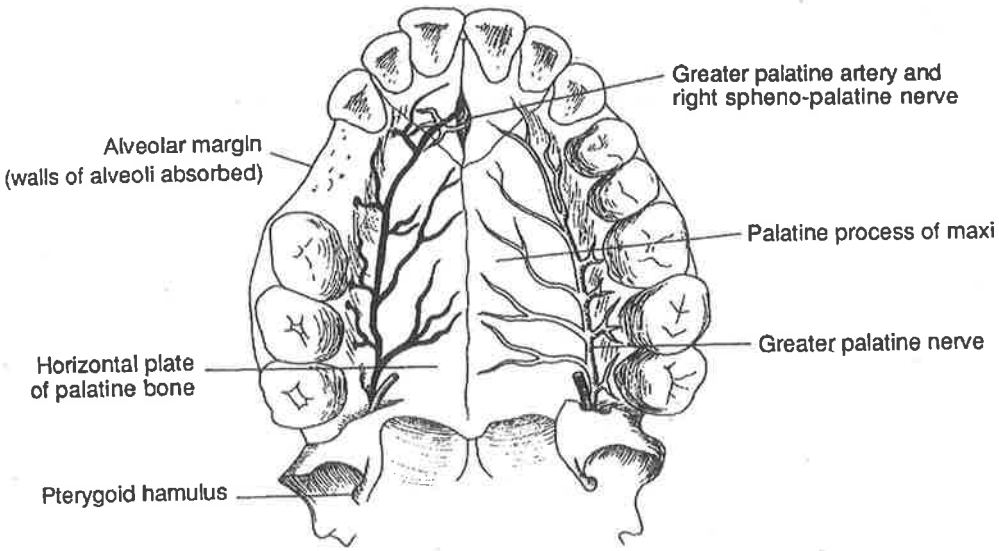


FIG. 11.52. ***Maxillary anatomy.*** *Nerve and artery supply of the hard palate, alveolar bone and mucosa.*

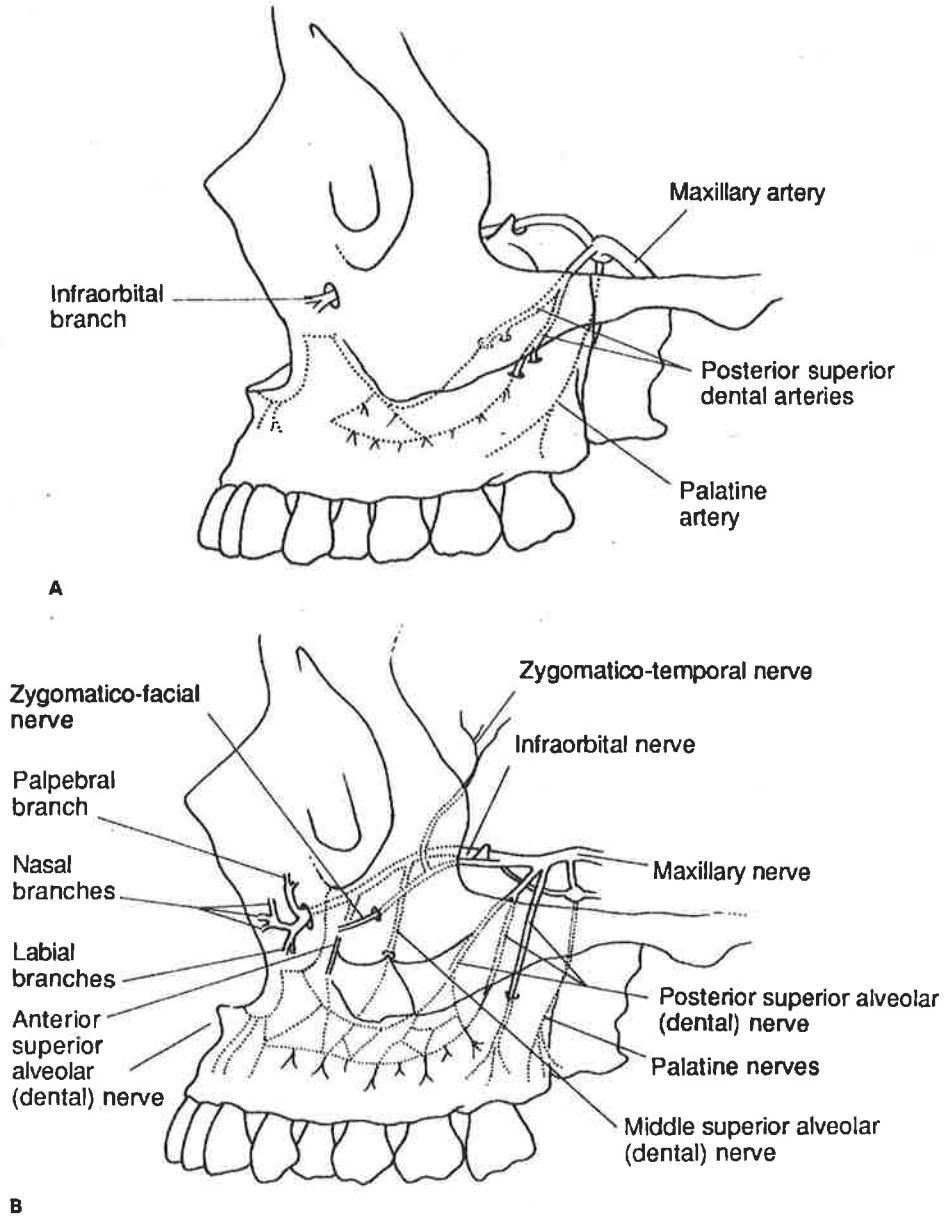


FIG. 11.53. ***Maxillary anatomy.*** **A.** *Arterial supply to the anterolateral maxilla.* **B.** *Nerve supply to the anterolateral maxilla.*

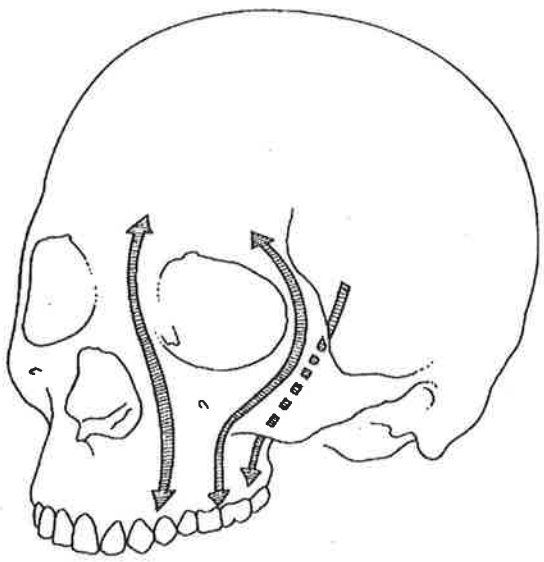


FIG. 11.54. ***Maxillary anatomy.*** *The load-bearing vertical bony buttresses of the midface.*

be the buttresses described above and analysed in more detail in Chapter 2 (p. 44). Ideally, surgical plates screwed in place along the buttresses should stabilize and reconstruct the load paths, and the more screws that are used on each side of a fracture line, the more evenly the distribution of force will be applied along these paths (Fig. 11.55) (Rudderman & Mullen 1992).

Classification

In classifying occlusal fractures of the maxilla, little can be added to Le Fort's original description (p. 18). The common denominator in each of the Le Fort types of maxillary fracture is the status of the upper jaw and the occlusion. In the Le Fort I pattern the upper jaw is detached from the cranial skeleton at a horizontal fracture through the piriform aperture, the zygomatic buttresses and the pterygoid plates. In the Le Fort II fracture pattern, the upper jaw is detached at a fracture line running across the nasal bones through the inferior orbital rims and through the zygomatic buttresses and pterygoid plates. In the Le Fort III pattern the upper jaw is detached from the base of skull at a fracture line running through the nasofrontal suture, the ethmoid bone, the frontozygomatic sutures, the arches of the zygomas and the pterygoid plates (Fig. 11.56). However, this time-honoured classification does not provide a full description of the degrees of comminution and displacement, nor does it mention two common associated and very important lesions—the parasagittal fracture of the hard palate and the smaller dento-alveolar segment fractures of the upper jaw. Furthermore the Le Fort classification does not relate the severity of the maxillary fracture to other areas of the craniofacial skeleton. These deficiencies are redressed in the alphanumeric classification system proposed by Cooter & David (p. 35).

Clinical assessment

The signs depend on the degree of displacement and comminution, and on whether the fracture extends to the Orbits and skull base. Intraoral inspection may show the upper alveolar arch to be intact, or split into two fragments by a longitudinal (parasagittal) fracture, or mobile dental alveolar segments may be seen. Gross loss of bone and oral mucosa can occur in high velocity missile injuries (p. 445). Malocclusion may be evident: it may be complained of by the patient, or be obvious on inspection. However, the examiner must keep in mind the common occurrence of pretraumatic occlusal disharmony, especially anterior open bite and crossbites. In the typical fracture of the upper jaw with an intact alveolar arch, posterior and inferior displacement of the occlusal surface will result in an anterior open bite (Fig. 11.57). This backward and downward movement is in part due to the inclination of the base of the skull, and in part to the vectors of pull by the medial pterygoid muscles. The movement forces the mandible open producing the appearance of elongation of the midface.

On external inspection the cheeks are typically swollen by post-traumatic oedema, bleeding and bruising, or by emphysema from air forced out of the paranasal sinuses. If the fracture of the upper jaw extends into the orbit, then swelling and bruising of the eyelids may be apparent (Fig. 11.58). The clinical methods of examination described in Chapter 6 may be diagnostic. A gloved finger on the hard palate exerting an upward and rocking force may elicit pain, crepitus and abnormal mobility (Fig. 6.5). It may be possible to locate the site or sites of abnormal mobility by performing bimanual palpation at the piriform base and the nasofrontal and frontozygomatic suture areas in turn while applying the upward and rocking force to the hard palate. In some cases gross midface instability can be demonstrated by asking the patient to bite upward with his mandible, resulting in upward movement of the upper jaw. Careful examination along these lines will often enable the examiner to deduce the most likely pattern and level of a fracture of the maxilla.

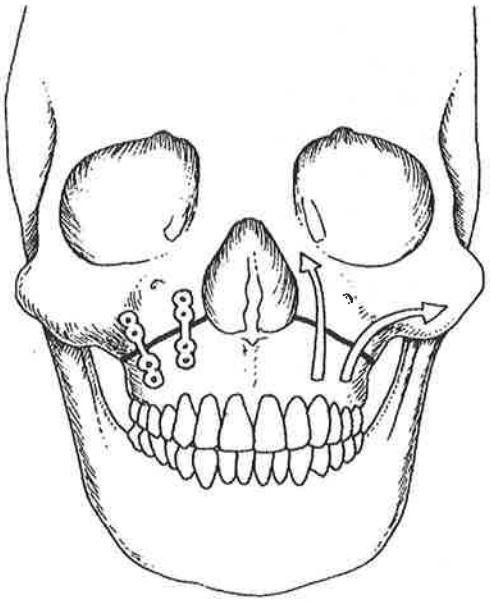


FIG. 11.55. ***Maxillary anatomy.*** *If possible, plates should be placed longitudinally along the line of the load-bearing buttresses.*

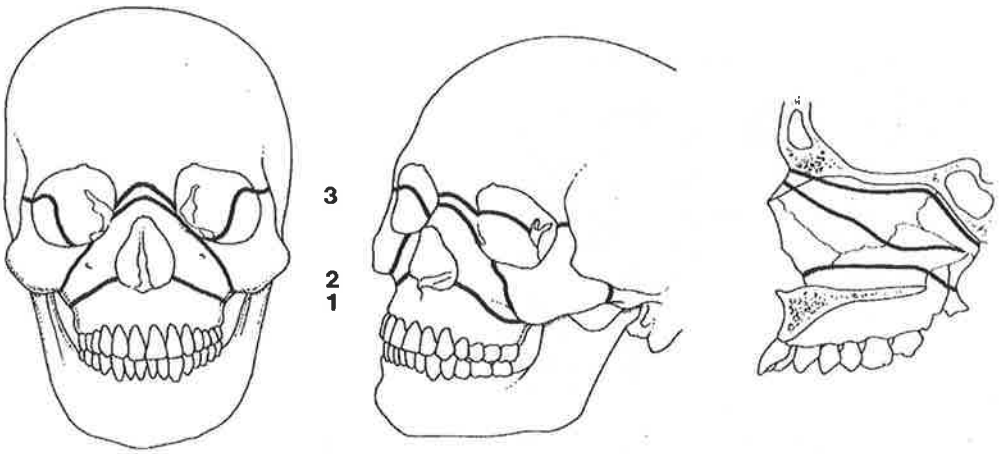


FIG. 11.56. ***Maxillary fracture classification.*** *The classical fracture lines of Le Fort.*

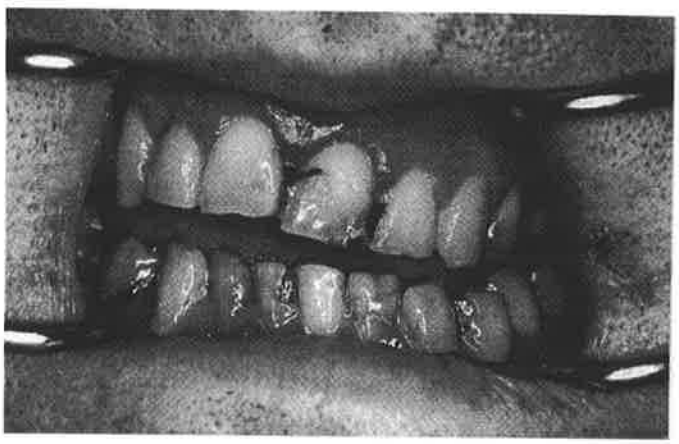


FIG. 11.57. ***Clinical signs of maxillary fractures.*** *Anterior open bite associated with posterior displacement of an upper jaw fracture and gagging of the posterior dentition.*

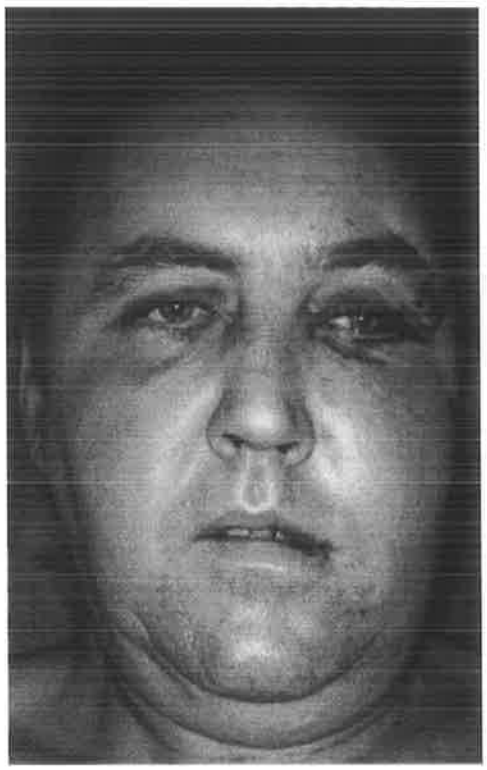


FIG. 11.58. ***Clinical signs of maxillary fractures.*** *Elongation of the midface. bruising and swelling of the cheeks and eyelids accompany a midface fracture involving upper jaw and orbits.*

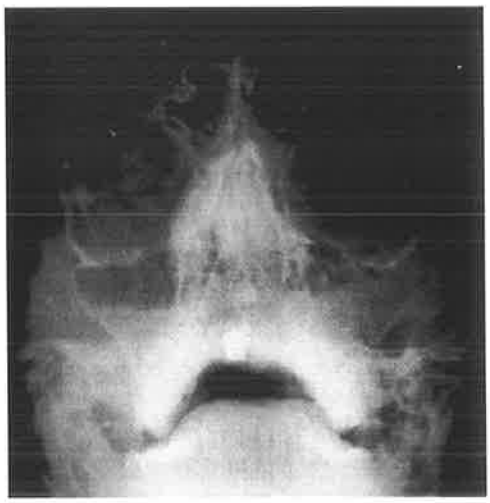


Fig. 11.59 Radiology of maxillary fractures. *Occipitomental view demonstrating fluid levels in both maxillary antra and fractured nose, but little else.*

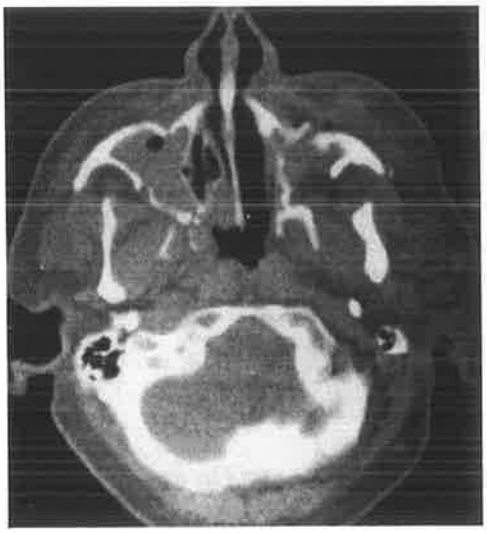


Fig. 11.60 Radiology of maxillary fractures. *Axial CT scan of midmaxilla, demonstrating fractures of the anterior, posterior and medial walls of both maxillae and the right side pterygoid plates.*

Radiological assessment

The radiological findings provide back-up evidence for the clinical diagnosis. Plain anteroposterior and occipitomental X-ray pictures may demonstrate fluid in the maxillary sinuses (Fig. 11.59), or displaced fracture lines in the zygomatic buttress, inferior orbital rim or piriform margin. Parasagittal fractures of the hard palate can sometimes be visualized in plain X-ray pictures. However, the most useful radiological investigation is the CT scan with axial, coronal and three-dimensional (3D) reformatting. This is our standard radiological examination for fractures of the upper jaw (p. 190). Axial cuts show fractures of the posterior wall of the maxillary antrum and of the pterygoid plates (Fig. 11.60). Splits of the hard palate and alveolus, as well as dento-alveolar segmental fractures, are readily seen (Fig. 11.61A,B). Coronal CT scans of the midface give detailed information on fractures of the anterior maxilla, especially those in which the fractures transgress the piriform buttress and/or the inferior orbital rim (Fig. 11.62). The coronal scan also visualises parasagittal fractures of the palate (Fig. 11.63). Axial CT scans must be studied to detect fractures in the region of the pterygoid plates, a confirmatory sign of a fracture of the upper jaw; in the absence of this, the anterior maxillary fracture may be part of an upper midface/naso-orbital fracture with an intact upper jaw (Fig. 11.64). 3D reformatting may add little when displacements are slight, but when displacements and comminution are extensive, the graphic visualisation given by the 3D scan is valuable and may indicate a need for bone grafting.

Principles of management

Management of these fractures depends in the first place on whether the fracture is thought to be in need of surgical reduction and stabilisation. There are several clinical situations where surgical reduction would be contraindicated:

1. Where clinical and radiological examination confirms an undisplaced fracture. In both edentulous and immature patients, an undisplaced fracture is invariably stable to masticatory forces.
2. In the edentulous patient where there is only minor radiological evidence of displacement, and where the fracture appears to be stable to masticatory forces. Bony union of these fractures can be confidently expected (Thaller & Kawamoto 1990). In these cases, prosthodontic adjustment will be sufficient. Patients are given a soft non-chew diet for 6 weeks and then allowed a graduated return to normal eating.
3. Patients with associated severe brain injuries who are not expected to survive.

When none of the above contraindications exist and where there is displacement and instability, then consideration must be given to anatomical reduction and stabilization of upper jaw fractures.

Preoperative dental assessment

In addition to full clinical and radiological examination, patients will require assessment by a dental specialist (p. 355). Impressions of the dental arch and teeth will be used to cast a plaster model. An expert in dental occlusion will study the model, cutting it at sites of vertical fractures through the occlusion and then mounting the fragments on an articulator in the exact position of pretraumatic occlusion. An acrylic bite wafer will then be constructed which will be used peroperatively and often postoperatively to maintain correct occlusal relationship. Close dental collaboration is even more important with upper jaw fractures than with lower jaw fractures. In the latter, the strength and solid form of the mandible ensure that anatomical reduction of the fracture site is relatively easy and the occlusion is used as a secondary check on the correctness of the reduction. In fractures of the upper jaw the complex structure of the maxillary bone makes certainty about anatomical reduction of a fracture more

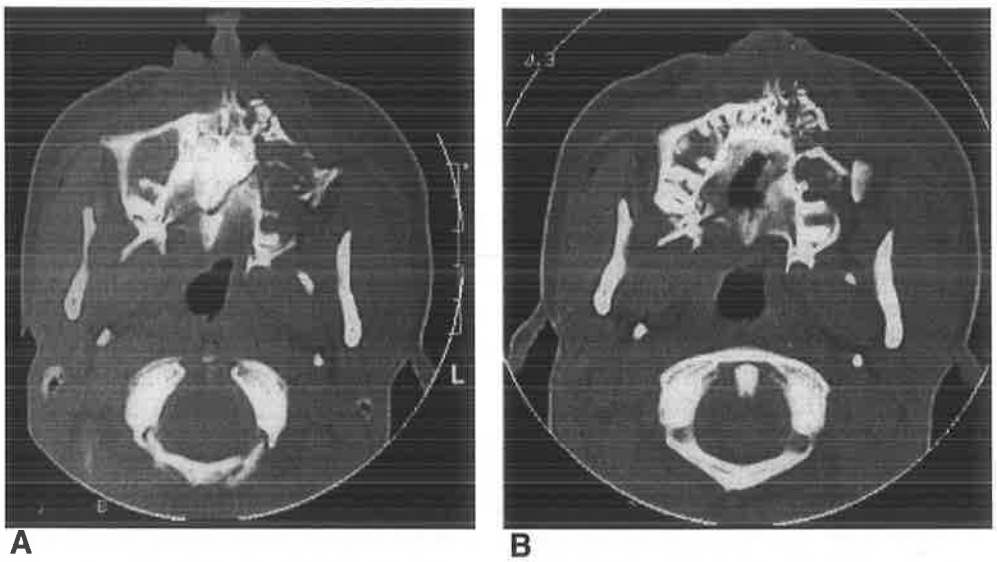


FIG 11.61. ***Radiology of maxillary fractures.*** **A.** *A diagonal fracture of the hard palate and associated dento-alveolar area.* **B.** *A higher cut through the same fracture.*

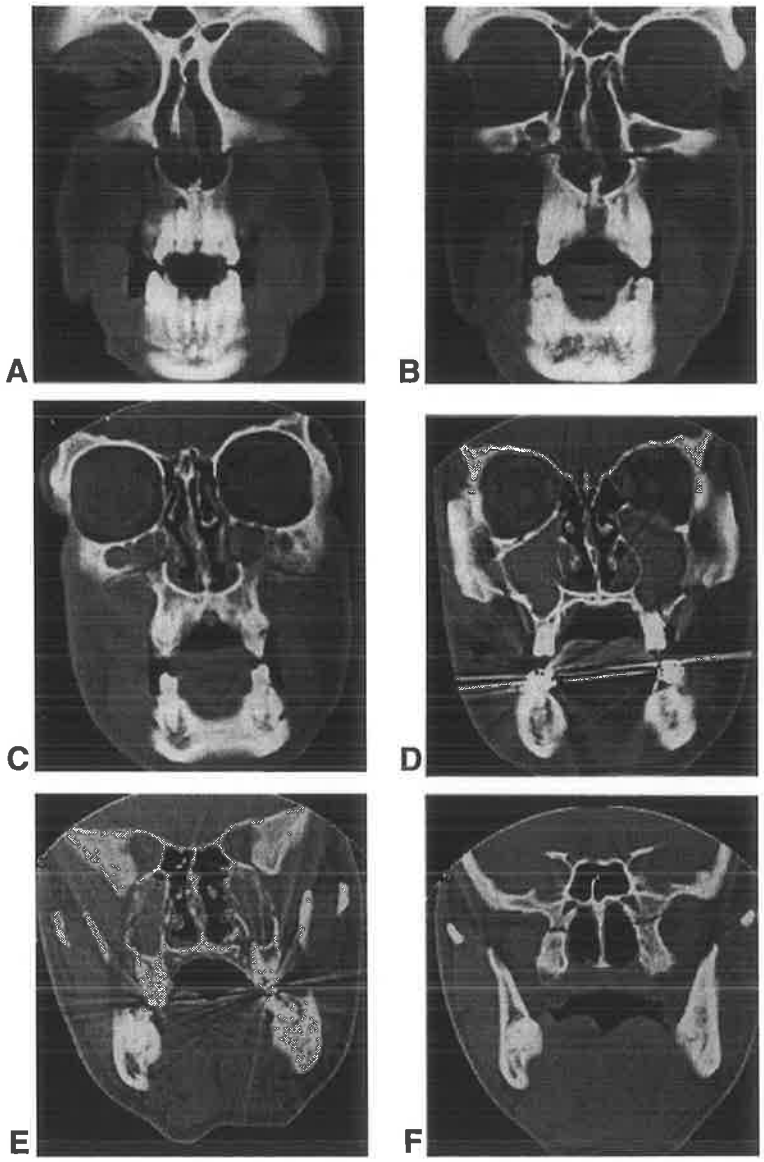


FIG. 11.62. ***Radiology of maxillary fractures.*** *Series of coronal scans anterior to posterior maxilla demonstrating the progression of horizontal maxillary fracture lines from the piriform margins* **A. and B.** *through the mid anterior maxilla* **C.** *to the zygomatic buttress areas* **D.** *and thence to the posterior maxillae* **E.** *and finally the pterygoid plates* **F.**

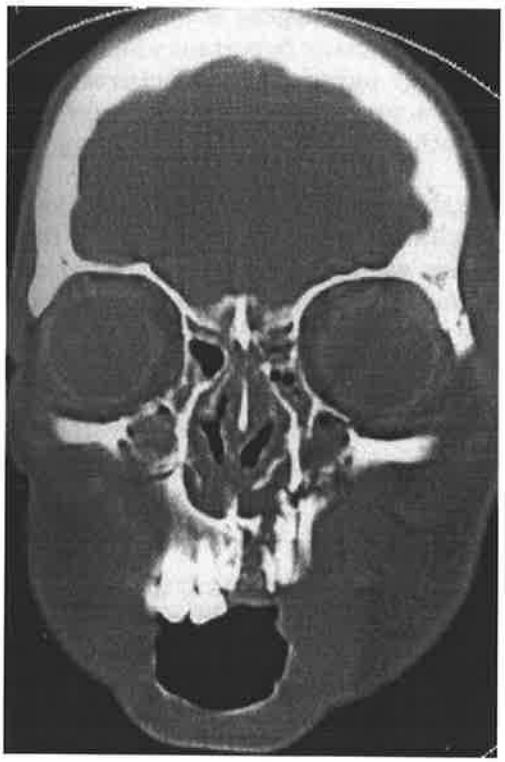
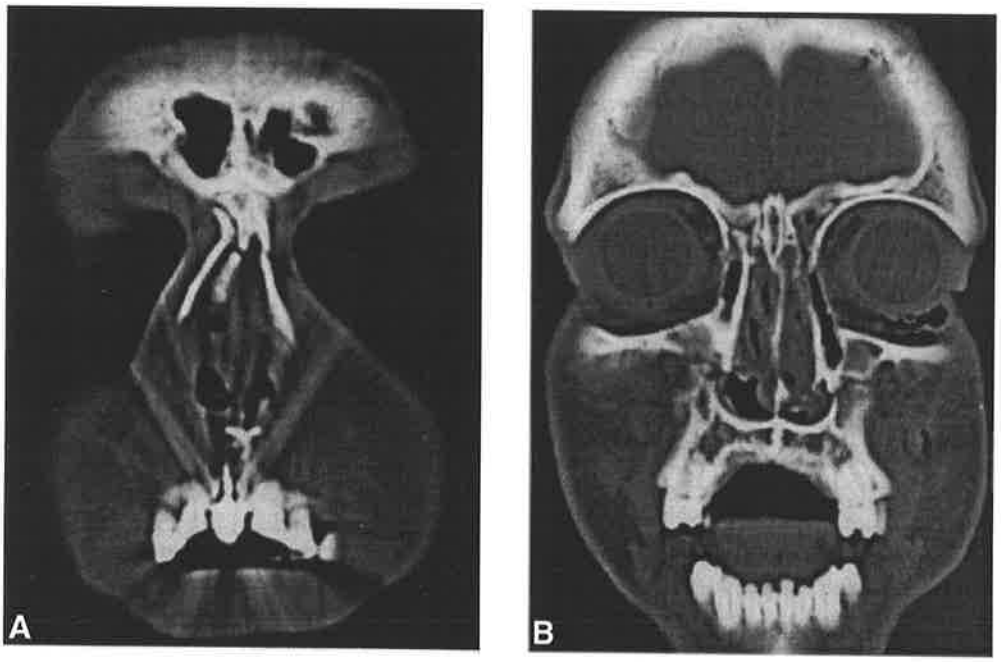


FIG. 11.63. ***Radiology of maxillary fractures.*** *Coronal views of fractures through both piriform margins, the left side of the palate and factures into both orbital floors.*



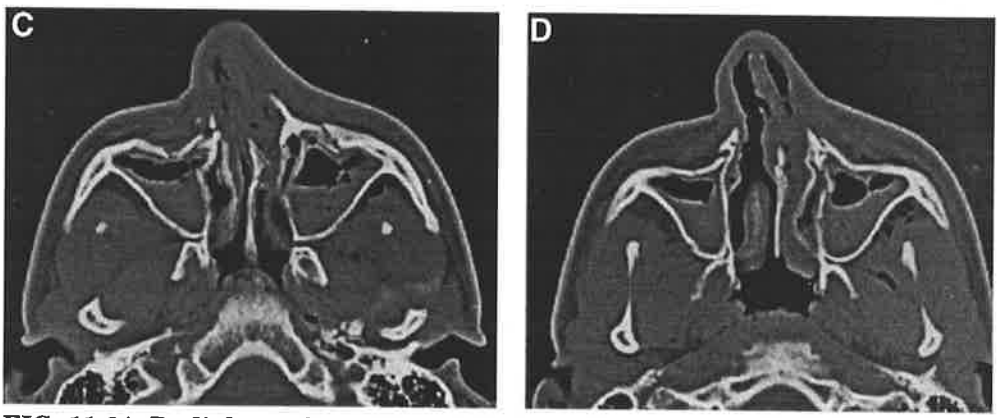


FIG. 11.64. ***Radiology of maxillary fractures.*** *Coronal views of anterior maxillary fracture associated with naso-orbital fracture* **A. and B.** *The upper jaw was intact as evidenced by the intact pterygoid plates on axial cuts* **C. and D.**

difficult and the dental relationships assume a high degree of importance in establishing that reduction is achieved. This is especially important where there are vertical fractures of the upper jaw, e.g. parasagittal fractures and dento-alveolar segmental fractures. In these fractures, the acrylic bite wafer can be used to control rotational forces on the teeth during the period of healing.

Where open reduction and miniplate fixation are indicated in the edentulous patient then Gunning or modified denture splints may be used temporarily. But where intermaxillary fixation and suspension wiring or interfragment wiring are the only option for fracture stabilisation, then appropriate dental splints must be maintained for 6-8 weeks. Similarly, where external frames are the only stabilisation method available for an upper jaw fracture, cast metal cap splints must be bonded to the teeth before surgery: a rod is then attached to the cap splints, and in turn fixed to the external frame. The dental specialist will also manage any acute dental injuries along the lines described in Chapter 12.

Timing of definitive treatment

Much has been written on the desirability of performing surgical correction of the midface fracture as early as possible after injury (Gruss & McKinnon 1986; Manson et al 1985b). The chief argument is the belief that early accurate bony reconstruction will prevent the development of soft-tissue contractures which in turn will lead to post-traumatic deformity. Gruss (1990) has stated the he believes such an outcome will be unavoidable if repeair of midface fractures is not accomplished within 10–14 days after the injury. However, there are other factors which must be considered when deciding on the timing of surgery for upper jaw fracture reduction. Perhaps the foremost of these is the consideration of priorities of management of other injuries. Brain injury is often associated with facial fractures and disturbances of cerebral physiology may take some time to stabilize. Derdyn et al (1990) studied patients with combined facial fractures and cerebral injuries and identified factors which they felt were contraindications to early surgical reduction of facial fractures. These included a Glasgow Coma Score Of five or less, CT evidence of intracranial haemorrhage with midline cranial shift and/or basal cistern effacement, and intracranial pressure > 15 mmHg (p. 369). Brandt et al (1991) in a similar study were unable to identify any neurological complications directly attributable to craniofacial repair in the first 24 h after injury. However, they stated that craniofacial repairs should be delayed in patients with documented intracranial pressure > 25 mmHg. Other systemic injuries that may demand delay in facial fracture treatment include unstable cervical spine injuries, cardiovascular instability and severe intrathoracic injuries; ocular injuries that represent an immediate threat to vision (p. 245) may also impose delay if a combined operation is undesirable. Moreover, a certain amount of time will be required to perform a radiological and dental work-up necessary to allow adequate fracture management. In our unit, this usually takes 2 or 3 days. Finally, consideration will need to be given to the practical requirements of coordinating surgery with other specialties. This may involve collaboration with a neurosurgeon in management of intracranial injuries or with an orthopaedic surgeon in management of distal skeletal injuries. David (1984) has shown the cost-effectiveness of a combined approach in craniofacial injuries. Where there are no contraindications, Our unit policy is to repair midfacial fractures as soon as possible, which is usually 2 or 3 days after injury, but may be any time up to 10 days.

It used to be argued that early reduction and immobilization of Le Fort III fractures will reduce the risk of meningitis. Experience and study of the pathology of anterior fossa fractures give little evidence to support this view. Nevertheless, this consideration suggests that if there are no contraindications, unstable fractures of this type should be fixed as soon as the appropriate preparations have been completed.

Airway management

Upper jaw fractures may compromise both the oral cavity and the nasal cavity, and placement of an endotracheal tube requires careful consideration and discussion with the anaesthetist. When the nasal bones are not significantly displaced or comminuted a nasotracheal tube will allow the necessary surgical procedures to be carried out without special difficulty (Fig. 10.1). If a nasal fracture requires reduction then there are only two alternatives: oral tracheal intubation or tracheostomy. It is often possible to place an armoured tube behind the mandibular molars. This tube is fixed with a wire to the posterior dentition, allowing intermaxillary fixation to be applied during the operation (Fig. 10.2). This avoids the need for tracheostomy. But should the eruption of the posterior molars be such that there is not enough room for even a small tube, then there will be no choice but tracheostomy. Zachariades et al (1983) have listed several other indications for tracheostomy. These include severe gunshot injuries, severe crush injuries, bilateral mandibular and maxillary fractures, gross maxillary fracture with profuse bleeding and midface fractures associated with cranial, thoracic or spinal injuries. We agree that combined cerebral, facial and chest injuries are best managed by early tracheostomy to lessen the risk of cerebral oedema due to hypoxia or increased venous pressure (p. 381) in such cases, tracheostomy should be done as soon as possible. Also we accept tracheostomy as part of the management of profuse facial bleeding from midface fractures, to secure the airway while the facial cavities are packed to control bleeding. However, since the advent of miniplate fixation we have not routinely used tracheostomy in bilateral maxillary and mandibular fractures, preferring nasotracheal or orotracheal incubation as described above.

Disimpaction

Where closed methods of fixation are used, reduction may be carried out using the specially designed forceps described by Rowe & Killey (1955). One blade of each forceps is inserted along each nostril floor while the other blade is placed via the mouth over the hard palate mucosa. When both forceps are in position the surgeon is able to exert powerful leverage on the upper jaw via the hard palate which is securely gripped between the blades of the forceps (Fig. 11.65). It is important to exercise care to avoid tearing the soft palate attachment to the hard palate, which may result in loss of vascularity to the jaw and teeth (see above). While these instruments may also be used in methods employing open exposure of maxillary fractures, disimpaction and mobilization of the upper jaw may be obtained by tapping an osteotome into some part of the fracture and then exerting digital pressure over the alveolus anteriorly in a downward direction. This method avoids the risk of damage to the junction of soft and hard palate with forceps. Minimally displaced non-comminuted fractures of the upper jaw may be difficult to disimpact but this can be achieved in all cases by using one or other or a combination of the above methods.

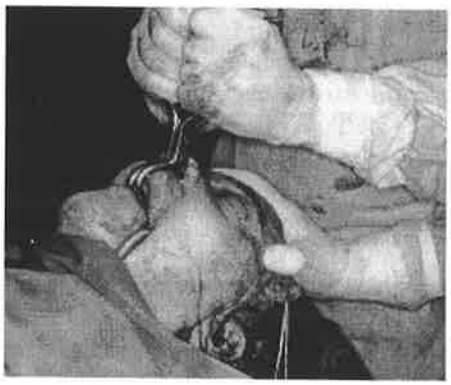


FIG. 11.65. ***Mobilizing upper jaw fractures.*** *Two forceps have been applied to grasp the hard palate and mobilize the upper jaw fracture. This manoeuvre must be done with the head stabilised to avoid injury to the neck and will usually be accompanied by brisk bleeding.*

Fracture stabilisation

Until the last decade, stabilisation of upper jaw fractures was carried out either by external frame fixation or by internal suspension wiring depending on the clinical indications. In both techniques the upper jaw was fixed to the lower jaw in the position of pretraumatic occlusion by intermaxillary wiring or cap splints, or in the case of edentulous patients by Gunning splints; the conjoined upper and lower jaw complex was then secured to the intact upper midface or to the skull base. With fractures having minimal comminution and good vertical stability, stabilisation was achieved by internal suspension wires passed to the intact zygomatic arches. Where there was gross comminution and vertical instability, the jaws were stabilised with an external frame. This frame was removed 6-8 weeks after surgery, as were the suspension wires. As recently as 1985, Wallace described a new frame for craniomaxillary fixation (p. 24). In the past, we have used external frames styled after Le Vant, fixed to the frontal skull by pins in the outer table (Le Vant et al 1969). Halo frames have also been used when frontal bone fractures have dictated the need to use pins inserted more laterally; this frame provides a very rigid platform for stabilising bars. However, no matter how mechanically refined the external frame may be, there are practical problems. For the patient, the halo frame has a major disadvantage: Lying with the framed head on a pillow is uncomfortable. For the surgeon, judgment as to the exact vertical position of the jaw complex in relation to the skull base is more an educated guess than an exact judgment based on anatomical bony landmarks. Finally, the cumbersome linkage of rods and universal joints cannot provide rigid stability. In 1942 Adams (Fig. 1.22) described a method of internal stabilisation of upper jaw fractures.

After reduction, as described above, the upper and low jaws were stabilized in the pretraumatic occlusion using arch bars and intermaxillary wiring. By this means the intact lower jaw provided anteroposterior stability to the upper jaw. Where the zygomas were also fractured, these were first reduced and stabilized with interosseous wires inserted through incisions over the frontozygomatic and inferior orbital rim fractures sites. The central jaw complex was then stabilized against the now stable lateral midface complex using a heavy wire loop inserted through a drill hole in the frontozygomatic area and passed immediately behind the body of the zygoma through an upper buccal incision to the intermaxillary wiring. The wire was secured to the arch bar in the region of the mandibular first molar on each side and tightened to provide an upward and backward compressive force and hence stabilisation. When the lateral midface did not require reduction and stabilisation, the suspension wire was passed around the most anterior part of the zygomatic arch via a small cutaneous stab incision; the wire was then secured to the mandibular arch bar and tightening was then carried out. This technique has had wide application and modifications have been described as recently as 1986 (Farole 1986). But for suspension wiring to be successful, there must be effective integrity of the upper jaw buttresses. Clinical experience and high resolution CT scan imaging have shown that this integrity is often lacking: it is far more common to see significant comminution in one or more of these buttresses. When this is so, suspension wires and intermaxillary fixation will not prevent upward movement of the jaw complex along an arc centred at the TMJs (Fig. 11.66). While external frames could be used to greater effect in such cases, they do not provide absolutely rigid stabilisation. Moreover the problem of correct judgment of midfacial height remains when the midfacial buttresses are not directly visualized.

In the early 1980s, many surgeons were dissatisfied with these methods of upper jaw reduction stabilisation and began looking for new approaches. This movement was stimulated by the new technology of CT scanning which allowed preoperative imaging of the degree of comminution and displacement in midfacial fractures. Stoll et al (1983) in Germany described a method of stabilisation of the midface by an internal metal frame. Two stainless steel rods were passed subcutaneously through an incision over the glabella, one on each side of the

nose, down to the anterior maxillae. After the jaws were secured in intermaxillary fixation, two screws were placed at each end of the rods providing stability in both the vertical and sagittal planes (Fig. 11.67). These rods were removed under local anaesthesia 6-8 weeks later. The authors recommended this method in cases with associated cerebra injury as fixation could be carried out quickly. On the other side of the Atlantic, Gruss and Manson extolled the virtures of extended open reduction of midface fractures and immediate reconstruction of buttresses with direct fragment wiring and bone grafting if necessary. Only the accessible nasofrontal and zygomaticofrontal buttresses were reconstructed but the combination of interfragmentary wiring and bone grafts produced good stability and avoided the need for suspension wiring or internal frames (Fig. 11.68) (Manson et al 1985b, Gruss et al 1985b). Schilli et al (1981) pointed out the benefits of extended open reduction followed by stabilization by miniplates and screws to give three-dimensional stability and less need for bone grafting. In a recent review of midface stabilisation, Gruss & Phillips (1989) confirmed that miniplate fixation of the midface had almost totally replaced interosseous wires and primary bone grafts in their practice. However, when there were bony buttress defects > 0.5 cm in length they felt that interposition bone grafting was indicated, the bone graft being stabilized to the miniplate. The advent of malleable titanium miniplates has been an important part of the evolution of treatment.

Miniplate technique

Since 1987 open reduction with stabilisation by miniplates has been our treatment of choice for displaced upper jaw fractures. Originally we used the titanium miniplates of the Wurzburg System, but latterly we have moved to locally manufactured titanium plates and screws (see above). We find these more malleable, conforming better to the underlying bone and reducing the risk of plate spring with subsequent movement and malunion.

Operative management

As stated above, operation is done under endotracheal anaesthesia, tracheostomy being rarely required. Arch bars are located to teeth of upper and lower jaws. Exposure of the upper jaw fracture lines is performed through an incision in the mucosa of the upper buccal sulcus, taking care not to injure branches of the inferior orbital nerves. When a midface fracture involves the orbit(s), subciliary lower eyelid incisions (p. 238), with or without a coronal scalp flap, may be needed to complete the exposure. The soft tissues are dissected from the maxillae on each side to expose the nasomaxillary and the zygomaticomaxillary buttresses and the related fractures. Often there is significant comminution and small fragments of bone are removed. Larger fragments will be preserved in a weak antibiotic solution—usually 0.2% flucloxacillin in normal saline. This solution is used copiously to irrigate the maxillary sinuses. Disimpaction of the fracture and mobilization of the upper jaw are carefully carried out as described above. An acrylic occlusal wafer is wired to the upper jaw and the jaw is placed into occlusion and stabilized with intermaxillary wires. We are aware that in some centres, interocclusal acrylic wafers are not considered necessary when there is a good dentition, an accurately determined pretraumatic occlusion, and no demo-alveolar fracturing. However, we find the use of wafers invaluable in fractures with more complicated impairments of occlusion. The relationship of the upper jaw to the midface buttresses is then established; an assistant maintains the seating of the mandibular condyles in the glenoid fossae. Usually at least one of the four anterior vertical midface buttresses will not be severely comminuted, providing a guide to the correct vertical relationships between the upper jaw and the remaining midface. Once the relationship is established an AusSystem miniplate is used to stabilize the reduced fracture, with adherence to the principle of reconstructing the vertical load lines and using at least two screws on either side of the fracture line. Accurate shaping of the plate to the bone contours is also important. Care must be taken to avoid damaging dental roots when fractures extend inferiorly in the zygomaticomaxillary buttress. When all four anterior

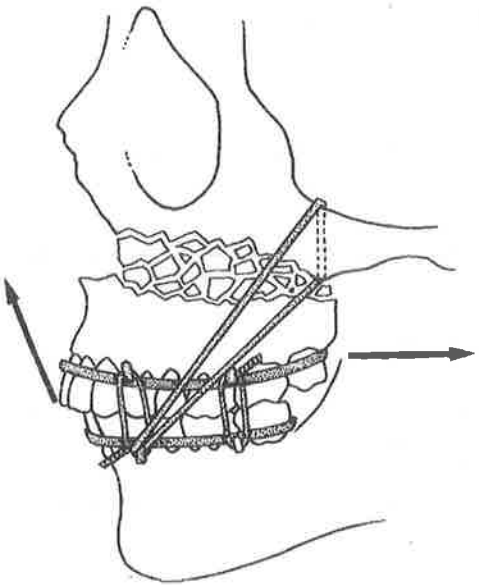


FIG. 11.66. ***Vectors of force following suspension wiring.*** *Adams circumzygomatic compression wiring results in upward movement of the midface when there is comminution of the midface vertical buttresses. Posterior displacement is prevented by the mandibular condyle remaining seated in the glenoid fossa.*

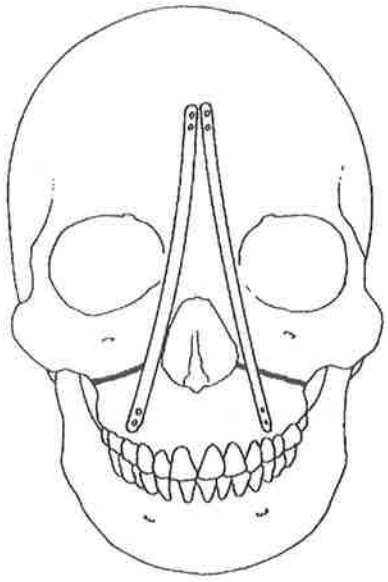


FIG. 11.67. ***Upper jaw stabilisation.*** *Temporary midface stabilising rods as used by Stoll et al.*

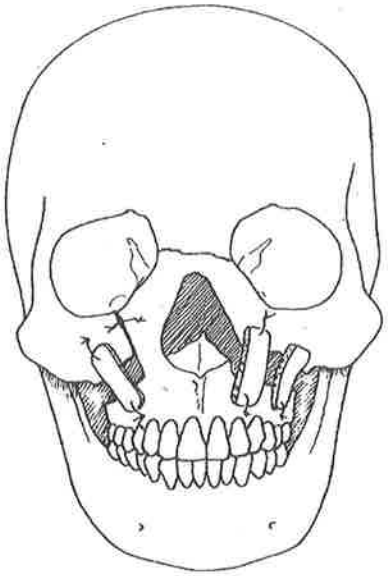


FIG. 11.68. ***Upper jaw stabilisation.*** *Interfragmentary wiring and primary bone grafting as popularized by Gruss and Manson.*

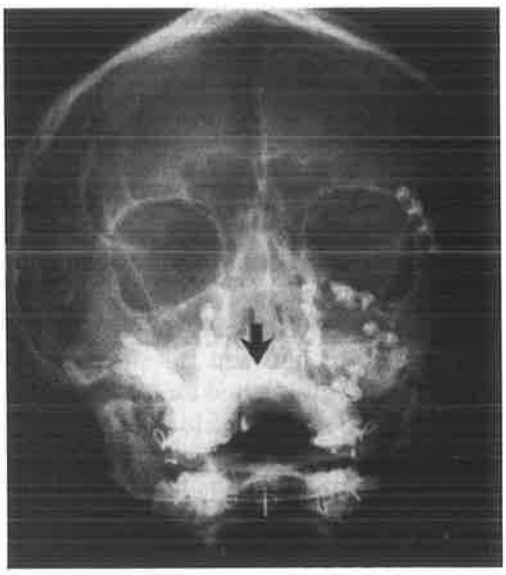


FIG. 11.69. ***Miniplating of upper jaw fractures.*** *Post-surgical reduction antero-posterior radiograph showing stabilization of fractures, seen in Fig. 11.61. The fracture of the alveolus and hard palate has been controlled by locating the teeth in an acrylic bite wafer and interosseous stabilization with a horizontal miniplate and screws (arrow). Four vertical buttress miniplates and screws can be seen stabilizing the occlusal complex to the upper midface.*

vertical midface buttresses are severely comminuted, it will be necessary to judge the correct vertical positioning of the upper jaws by eye, using the line of contour of the buttresses as a guide; in such cases, bone grafts may be required. Depending on the operator's preference, either outer table of calvaria or split rib graft will be suitable: we prefer calvarial bone, because it is more rigid and holds screws very well and because donor site morbidity is less (p. 241). All four buttresses are stabilized with miniplates and screws before intermaxillary fixation is released. The success of the reduction is then finally confirmed. All surgical fields are copiously irrigated with weak antibiotic solution. Oral mucosal wounds are closed with 3/0 chromicized cat gut or 4/0 Vicryl, depending on individual preference. Finally the mandible is manipulated back and forth into the bite facets of the wafer: the surgeon then confirms that the reduction is satisfactory and that the lower teeth engage cleanly into the wafer and all at the same time (Fig. 11.69).

Management of complex midfacial fractures

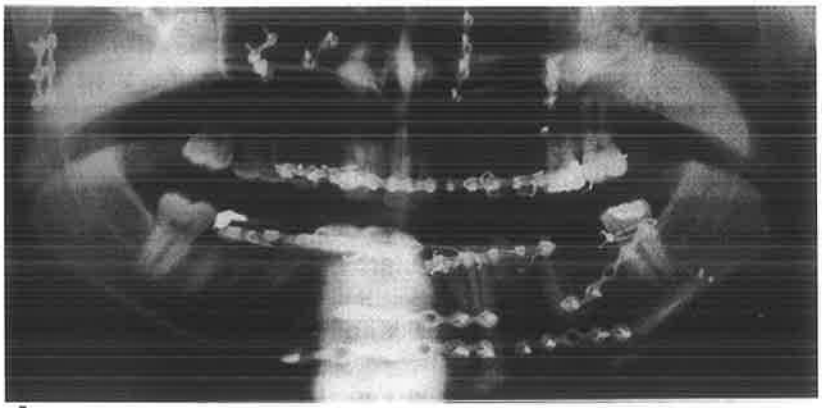
The above discussion relates to fractures of the upper jaw at the Le Fort I level. Different treatment sequences must be considered when other bones of the midface are also fractured, and when there is mandibular fracture. Our policy is based on the principle outlined by Markowitz & Manson (1989).

Vertical fractures through the alveolar process of the upper jaw and associated palate can occur with any type of Le Fort fractures. Antoniades et al (1990) found an incidence of 24% of parasagittal fractures of the alveolus and hard palate associated with Le Fort fractures; in this group, there was a 78% incidence of associated mandibular fractures. Manson et al (1990a) described difficulty in preventing rotation of the minor dento-alveolar segment in parasagittal fractures of the upper jaw and recommended open reduction of the posterior aspects of the hard palate fracture with stabilisation by miniplate and screws under direct vision; miniplate fixation was also used across the fracture in the vicinity of the piriform margin. Acrylic occlusal wafers were needed to stabilize the occlusion in 15% of patients. With this technique, there was a 10% incidence of plate exposure in the roof of the mouth.

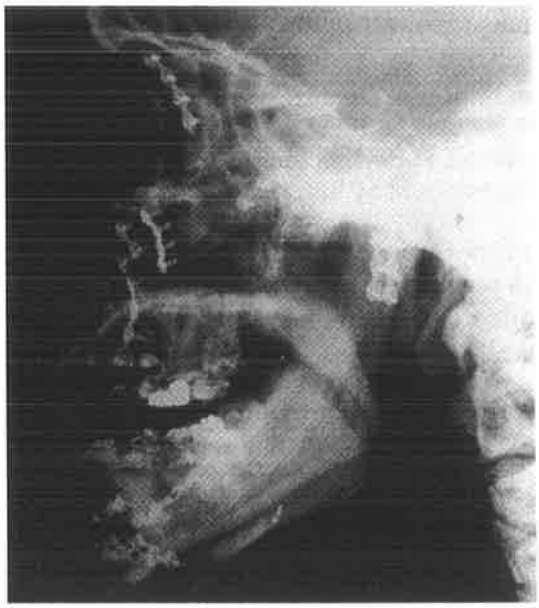
Where the mandible is also fractured, it is stabilized with miniplates (see above). This is particularly important when there is a displaced condylar fracture in association with an upper jaw fracture; only by open reduction and miniplate stabilisation of the condylar fracture can the correct anatomical height of the posterior facial skeleton be established (Fig. 11.70A,B).

More than in any other constellation of CMF fractures, the combined maxillary and mandibular fractures with comminution and detached dento-alveolar segments demand expert presurgical dental model planning. We routinely use an acrylic wafer to locate the teeth on both upper and lower jaws. The mandibular arch is stabilised first and checked against the wafer. The maxillary arch is then reduced by an extended open approach through the upper buccal sulcus incision. Arch bars are ligated to the teeth either segmentally or along the whole upper arch and the teeth are then fixed into the wafer which is stabilized to the arch bars with wires. Miniplate fixation across the alveolar fracture, at the level of the piriform margin or further posteriorly, is carried out and then the lower jaw is again brought into the wafer to complete intermaxillary fixation (Fig. 11.69). The patient must wear the acrylic splint and arch bars on the upper teeth for a minimum of 6 weeks. With this approach we have only infrequently found it to be necessary to expose the hard palate and apply miniplate fixation. But when there has been loss of alveolar bone and teeth, with significant comminution, then exposure and miniplate stabilisation of the hard palate component of the fracture may be the only way of establishing the correct transverse posterior maxillary relationships.

Where the upper jaw fracture is associated with displaced orbital fractures then these are stabilized in the manner described below. The occlusal complex, consisting of the upper and lower jaws united by intermaxillary fixation, is then



A



B

FIG. 11.70. ***Miniplating of upper and lower jaw fractures.*** *Orthopantomogram* **A.** *and lateral plain film* **B.** *demonstrating miniplate stabilisation of a complex mandibular fracture including right condylar fracture. Doing this first allowed accurate localisation and plating of the upper jaw fracture and associated midface complex fracture.*

brought to the stabilised upper midface and final stabilisation (with or without bone grafting) is carried out at the Le Fort I level using miniplates according to the principles already described (Fig. 11.71). Particular care is taken at the close of the reduction to ensure that the occlusion is satisfactory.

Edentulous upper or lower jaws

In all cases, it is necessary to establish the pretraumatic anteroposterior jaw relationships; in the edentulous patient it is also important that the correct vertical posterior facial height be established so that dentures can be fitted. If possible a Gunning-type splint is made from the patient's denture. If this is not available then a splint or splints made from models cast from dental impressions of the edentulous jaw should be used. These splints are wired to the upper and lower jaws and then to each other during the surgery to allow correct reduction of the fractures in both the anteroposterior and vertical planes (Fig. 11.72A,B). The management of fractures of the edentulous jaws is further discussed in Chapter 19 (p. 520).

Postoperative management

This is virtually as for mandibular fractures, and can be divided into the same temporal sequences. We release the intermaxillary fixation during the first 24 h to allow safe recovery of the airway (p. 255). The acrylic occlusal wafer may be left on the upper jaw and the mandible permitted to engage in the dental facets on the inferior aspects of the wafer. Light rubber band traction may be placed

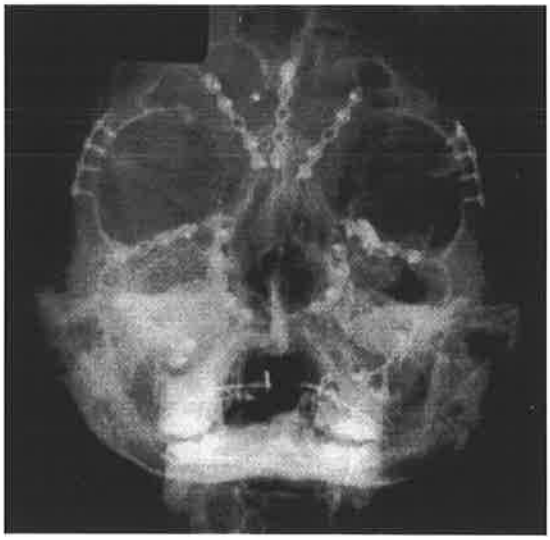
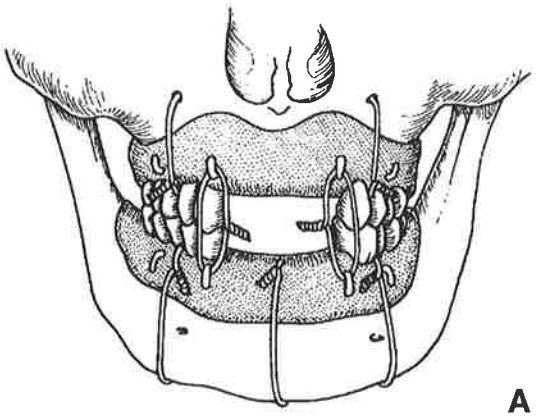


FIG. 11.71. ***Miniplating of combined upper jaw and orbital fractures.*** *Post-surgical reduction X-ray of upper jaw fractures combined with orbital fractures (Le Fort I, II and III). The reduced occlusal complex has been stabilised to the reduced and stabilised orbital complex using vertical buttress miniplates and screws.*



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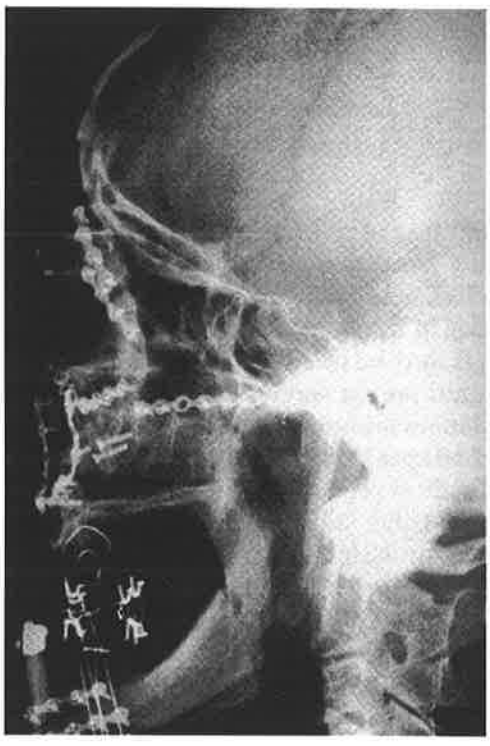


B

FIG. 11.72. ***Denture splintage of upper jaw fractures.*** **A.** *Diagram of denture type splints wired to the upper and lower jaws and then to each other.* **B.** *Lateral radiograph of an edentulous patient with upper and lower jaw fractures where the dentures were modified to enable intermaxillary fixation of the jaws and aid in reduction of the upper jaw fracture.*

between upper and lower jaws in the premolar region after the first 24 h to inhibit contraction of the medial pterygoid muscles during the early stages of mobilization. These muscles may cause posterior and caudal displacement of the upper jaw if used strenuously; by providing a closing force, the rubber band effectively inhibits the muscle action. Depending on the severity of the injury, this elastic traction is maintained for 7–14 days before removal. At that time the acrylic bite wafer will also be removed unless it is needed to stabilize the upper jaw arch form as in parasagittal and dento-alveolar fractures, when the acrylic bite wafer will be left in situ for 6-8 weeks. During the first 24 h the patient is allowed to sip water by mouth and oral hygiene is attended to by nursing staff using suction, dressed probes or more sophisticated water picks or air guns. A small-headed soft toothbrush is useful for cleaning around arch bars and wires while mouthwashes will give the patient a sense of comfort and improved hygiene.

The intraoperative antibiotics are continued intravenously for 48 h; we use flucloxacillin and metronidazole (p. 236). The patient is nursed sitting up as soon as possible to reduce facial swelling. Jaw mobilisation is commenced as soon as possible, unless other injuries cause delay. Pain relief is given as required (p. 257). Over the first week a non-chew diet is instituted with appropriate caloric and nutritional content. Once the patient is managing this diet and maintaining good oral hygiene, and depending on the demands of other injuries, then he/she is discharged from hospital, often as early as 2–5 days after surgery. Before discharge, the position of plates and screws and the relationships of the condyles in the glenoid fossae are checked by OPG of the mandible, and anteroposterior and lateral views of the skull and facial bones as well as appropriately tilted occipitomental views. Between 2 and 6 weeks, outpatient follow-up is maintained on a fortnightly basis. Hygiene and nutritional status are checked as well as mouth opening and occlusion. Dental arch bars and bite wafer are removed between 6 and 8 weeks in cases where these had been judged to be necessary. Patients are then referred for dental assessment of tooth vitality, occlusion and hygiene (p. 360). Further reviews are carried out over 1 year to establish the restoration of oral function as well as recovery of soft-tissue scarring and cutaneous nerve function. The success of dental restorative measures is also confirmed and recorded.

Complications

These include:

- Malunion and malocclusion
- Non-union
- Facial deformity, especially soft-tissue sagging
- Infection
- Nasal obstruction
- Lacrimal duct obstruction

Malunion and malocclusion

Malunion with associated malocclusion and loss of normal facial form are the most significant complications of upper jaw fractures. Severe comminution of midfacial buttresses with or without bone loss may make stabilization of upper jaw fractures difficult. Poor fixation of screws in thin maxillary bone may lead to movement after operation; to prevent this, it may be worth using a piece of bone graft behind the maxillary bone to act as a nut for the screw, which thus acts as a bolt. Failure to seat the condyles in the glenoid fossae during the time that the plates are being applied is another cause of relapse and malocclusion, commonly resulting in anterior open bite. Failure to produce good conformation of plates to the underlying bony shapes may result in additive displacing forces and again malunion is the result; this is less likely to occur with the more malleable plates favoured by us. The finding of malunion will require full dental and radiogical reassessment. If deformity is mild, spot grinding of tooth facets may provide sufficient adjustment to allow normal function. Where malunion is gross, then osteotomy may be required with or without preliminary bone grafting of the anterior maxilla and appropriate buttress stabilization with miniplates.

Non-union
Today this is a rare event in maxillary fractures, though occasionally seen when severely comminuted fractures were treated by closed suspension wiring.

Facial deformity
While the positioning of the posterior maxilla, in any plane, probably has little or no effect on the facial contour, the anterior maxilla is critical for proper balance. Inadvertent impaction of the maxilla, a significant problem in the days of wire fixation, creates the impression of an aged face by eliminating maxillary incisor display and foreshortening the face. Inferior malpositioning, much less common, creates an anterior open bite (due to posterior interference) and displays excessive gingiva. Obviously, improper positioning in the anteroposterior direction creates similar problems. Furthermore a discrepancy in midline position is readily apparent to the casual observer.

Where fracture exposure has required dissection of soft tissues from the zygomatic body, careful attention must be given to adequate suspension of soft tissue in this area. Failure to appreciate this point may lead to loss of pretraumatic cheek prominence through soft-tissue sagging.

Infection
We have seen several cases of chronic maxillary sinusitis some years after a fracture of the upper jaw. Management includes removal of buttress plates and screws, after which the infection will usually subside.

Nasal obstruction

Nasal obstructive symptoms may follow upper jaw fractures through fracture dislocations of nasal septal cartilage and bone. Once the nasal mucosa has soundly healed then appropriate septoplasty may solve this problem.

Lacrimal duct obstruction
Lacrimal obstruction is an uncommon complication of upper jaw fractures. Balle et al (1989) found eight cases of lacrimal obstruction in 104 maxillary fractures of Le Fort types II and III. Obstruction presents as epiphora, with or without recurrent dacryocystitis. Diagnosis of the level of lacrimal obstruction is confirmed on dacryocystography; if confirmed the condition may be treated by dacryocystorhinostomy (p. 429).

Fractures Affecting the Orbit

Naso-Orbito-Ethmoid Fractures

Surgical pathology

Naso-orbito-ethmoid fractures are relatively uncommon injuries. The result of direct trauma to the midface, the fractures may be confined to the naso-orbito-ethmoid region, or more frequently occur in association with other midfacial injuries. While the primary point of injury is the nasal bone complex, a high energy impact often produces radiation posteriorly in the underlying delicate ethmoid framework, superiorly into the frontal sinus, laterally to the orbital floor and inferiorly into the nasal septum and maxillae (Fig. 11.73). Consequently the nomenclature to describe these injuries has varied according to the particular area of interest to individual authors. McCoy (1959) coined the term 'naso-ethmoid-orbital', more frequently phrased as naso-orbito-ethmoid, which best delineates these injuries. Management is challenging because of the intricate and complex three-dimensional skeletal anatomy of the region, the frequent coexisting soft-tissue injuries (especially of the medial canthal ligament and nasolacrimal duct) and the difficulties in achieving and maintaining fracture reduction.

As with most facial fractures there has been a move from techniques of closed reduction and splinting, often with poor outcomes, to a more invasive strategy of open reduction and stable internal fixation. The challenge in the treatment of naso-orbito-ethmoid fractures remains the restoration of bony nasal projection and the restitution of nasal and orbital soft tissues, in particular the medial canthal ligament. Only by understanding the anatomy of this region is optimal management possible.

These fractures are characterised by disruption of the naso-orbito-ethmoid region with isolation of a central unstable bone fragment to which the medial canthal ligament remains attached, exerting a laterally displacing force. The resultant deformity is a depressed nasal root telecanthus and shortening of the palpebral fissure (Fig. 11.74).

The naso-orbito-ethmoid region lies at the confluence of the cranium, orbit, nose and maxilla and the reconstruction of all these regions is required for an acceptable outcome.

The frontal process of the maxilla, the maxillary process of the frontal bone and the proximal segment of the nasal bones constitute the upper portion of the central midfacial buttress or pillar. Protected posteriorly is the thin medial orbital wall composed largely of the lacrimal bone, and the orbital plate of the ethmoid bone (lamina papyracea). Dissipation of the impacting force as it proceeds posteriorly usually prevents the fracture displacement from extending to the optic canal. Above and laterally the medial orbital wall becomes the thin orbital roof, the anterior portion of which is the floor of the frontal sinus. Medial to, and lying behind, the naso-ethmoid buttress, and below the anterior cranial fossa, the interorbital space is composed of the ethmoid sinuses. The posterior boundary is the spheroid bone. With the application of significant force to the naso-orbito-ethmoid region, disruption and buckling of the interorbital space occurs, with potential for displacement posteriorly. Radiation of fracture lines superiorly will involve the anterior cranial fossa floor with attendant risks of a cranionasal fistula (p. 376) and olfactory tract disruption. In our experience, the association of a naso-orbito-ethmoid fracture with extensive disruption of the anterior cranial fossa is not very common (Table 13.2); however, when this association does occur, it may have serious implications.

The frontal sinus lies above the anterior interorbital space, with variable lateral extensions. The anterior wall is thick, providing structural support from side to side. Bony septa, the patterns of which vary, traverse the sinus, attaching the anterior wall to the thin posterior wall. The floor of the frontal sinus is the medial roof of the orbit. Centrally the frontal sinus drains into the nose; the drainage of the sinus into the ethmoid infundibulum or middle meatus (Fig. 2.8) is at risk when the anterior ethmoidal labyrinth is severely comminuted. The nasal bones complete the bony pyramid anteriorly. The medial canthal ligament and lacrimal drainage system comprise the soft tissue elements likely to be injured in this region. The three limbs of the medial canthal ligament — superior, anterior and posterior — envelope the lacrimal sac. The ligament maintains eyelid position in relation to the globe and forms the medial component of the suspensory sling of the eyelids (Fig. 11.75). Disruption by laceration, or through mobilization of the bony attachment, allows an unopposed lateral pull in the suspensory sling, causing telecanthus and shortened palpebral fissure.

Lacrimal drainage occurs via both lacrimal canaliculi which run in a fibrous sheath, the palpebral extensions of the medial canthal ligament. Uniting, they enter the posterolateral aspect of the lacrimal sac located in the lacrimal fossa and overlaid in its upper portion by the medial canthal ligament (Robinson & Stranc 1970). The sac is continuous with the nasolacrimal duct (Fig. 2.38) as it passes inferiorly, posteriorly and slightly laterally through a distance of approximately 12 mm in the bone to enter the nose below the inferior turbinate (Gruss et al 1985a).

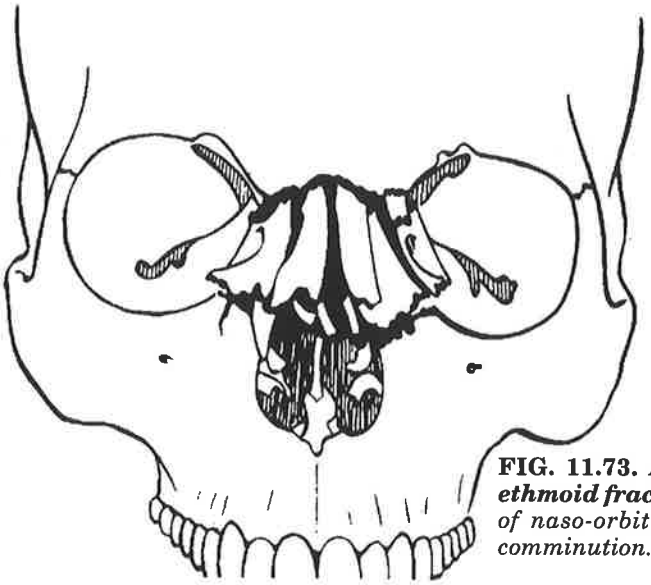


FIG. 11.73. ***Anatomy of naso-orbito-ethmoid fractures.*** *Conventional pattern of naso-orbito-ethmoid fracturing with comminution.*

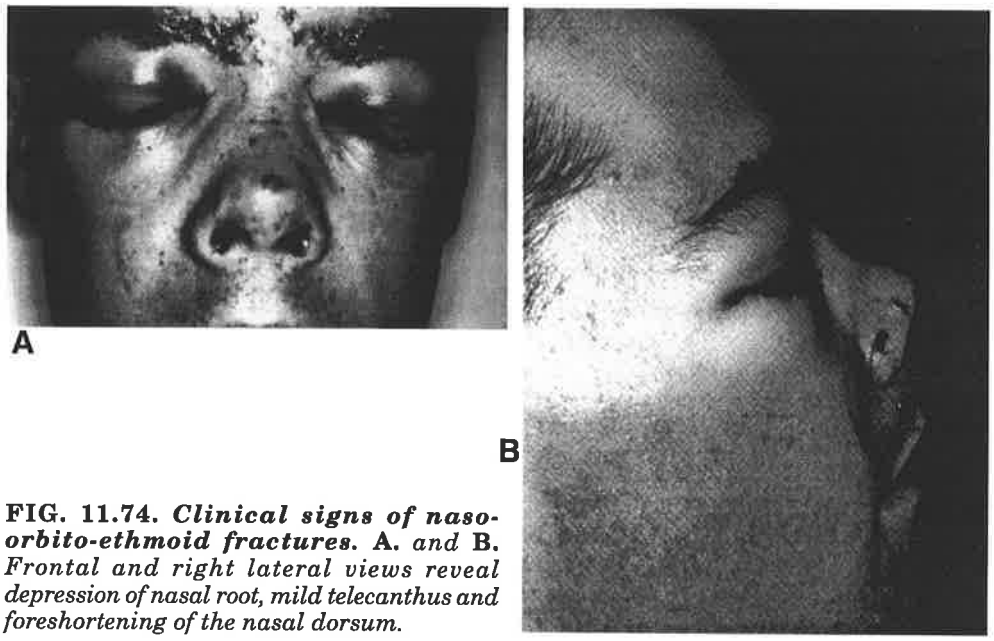


FIG. 11.74. ***Clinical signs of naso-orbito-ethmoid fractures.*** **A.** *and* **B.** *Frontal and right lateral views reveal depression of nasal root, mild telecanthus and foreshortening of the nasal dorsum.*

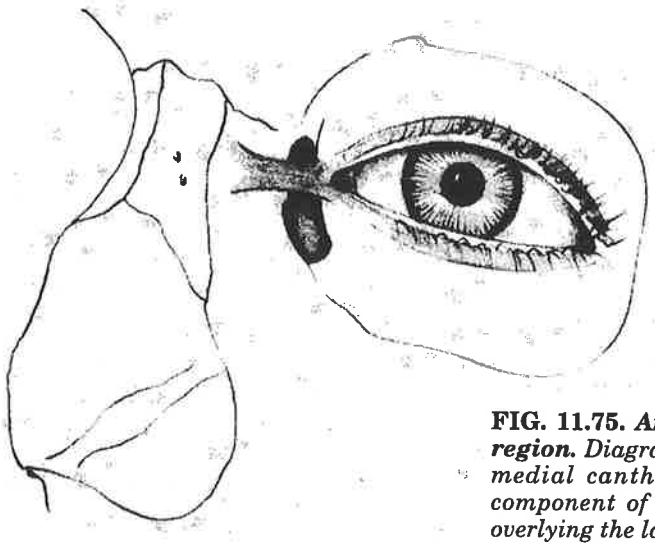


FIG. 11.75. ***Anatomy of medial canthal region.*** *Diagrammatic representation of the medial canthal ligament as the medial component of the eyelid suspensory sling overlying the lacrimal sac.*

Classification

The fractures may be unilateral or bilateral, simple or comminuted, closed or compound; they may occur in isolation or in association with more extensive fracturing of the forehead, orbit or maxilla. Fractures are bilateral in two-thirds of cases; while in > 90% there are other associated facial fractures (Cruse et al 1980). These authors reported 33 cases in 182 patients, an incidence of 18%. With improved imaging techniques and wide exposures, the true incidence of these injuries should be better understood.

Several classifications of naso-orbito-ethmoid fractures have been proposed. Markowitz et al (1991) group them into Types I-III, within each subdivision an ascending scale of severity of comminution. Gruss (1982) extends his classification in accord with the almost uniform extension of these fractures into the adjacent bony skeleton (Table 11.2).

Our system of scoring by the craniofacial disruption index quantifies the extent and pattern of bony damage in a global fashion.

TABLE 11.2

Classification of naso-orbito-ethmoid fractures

A. Markowitz et al (1991)	
Type I	Single central fragment bearing medial canthus
	— unilateral
	— bilateral
Type II	Comminution of bony central fragment without extension beneath area of canthal insertion
	— unilateral
	— bilateral
Type III	Comminution of bony central segment with extension beneath area of canthal insertion
	— unilateral
	— bilateral
B. Gruss (1982)	
1.	Naso-orbital alone
2.	Naso-orbital and central maxillary
3.	Naso-orbital and Le Fort II and III
4.	Naso-orbital and orbital dystopia
5.	Naso-orbital and loss of bone

Clinical assessment

A history of significant blunt trauma to the central midface, especially the glabella and nasal root region, should evoke suspicion of a naso-orbito-ethmoid fracture.

In the past, extreme soft-tissue swelling and the absence of significant early localising functional deficits often led to late diagnosis or misdiagnosis. The resulting cosmetic and functional deformities were significant and almost impossible to correct by secondary means.

The advent of axial and coronal CT scan delineation of the central midface has allowed detailed display of the anatomy of the fracture patterns. Specific clinical examination confirms or refutes the CT diagnosis and establishes whether there is a need for intervention, while the radiological assessment determines the operative approach and the patterns of fixation.

Inspection reveals localized bruising and ecchymoses, sometimes burst open lacerations over the nasal root, and periorbital swelling. Where the fractures spread more widely, diffuse facial oedema is usually seen and obscures the clinical

findings. Medially located subconjunctival and periorbital haemorrhages are common. The presence of a laceration in the region of the medial canthal ligament raises the possibility of injury to the nasolacrimal drainage (p. 428).

The characteristic skeletal disruption is best seen in the lateral profile (Figs 11.74 and 11.76). Collapse of the entire bony nasal pyramid results in gross posterior displacement of the nasal dorsum, with tilting superiorly of the nasal tip and creation of an abnormally obtuse nasolabial angle. In frontal view, there is an increase in nasal width superiorly, with obvious upward rotation of the nasal tip. The caudal end of the nasal septum is usually buckled laterally beneath the collapsed nasal dorsum.

Palpation of the naso-orbito-ethmoid region confirms the loss of nasal bony support. Backward pressure on the nasal dorsum accentuates the collapse of nasal contour; there may be bony crepitus. Transverse compression of this region between thumb and index finger over the region of the medial canthal ligaments will elicit painful bony movement. Under anaesthesia, bimanual examination is performed by placing a blunt-tipped clamp intra-nasally against the portion of the medial orbital wall to which the medial canthal ligament attaches; this produces movement in the unstable fracture. Paskert & Manson (1989) advocate this as a test of need for surgical repair. A complete ophthalmological assessment is mandatory to exclude associated injuries and as a baseline for follow-up after surgical repair. Neurosurgical evaluation is also needed because of the proximity to the anterior cranial fossa and the potential for fracture extension into this area often with a dural tear, pneumocephalus, CSF rhinorrhoea and olfactory tract injury. Olfaction should be tested if possible (p. 166), though nasal obstruction often prevents this.

Radiological assessment

Plain radiographs have usually been performed in the emergency room as part of the screening assessment. These seldom contribute significantly to the diagnosis of the fracture pattern and planning management, except when there is pneumocephalus or other sign of anterior fossa involvement.

Direct axial and coronal CT scan evaluation of the naso-orbito-ethmoid region is essential to document the pattern and extent of the bony injury (Fig. 11.76). Extensions of the fracture into the adjacent frontal sinus above, and midface below, are identified; these may modify treatment

Principles of treatment

In the past these injuries were treated by closed reduction with fixation by external splintage. A number of authors including Adams (1942) and Converse & Smith (1966) employed external lead plates or buttons held by transnasal wires to restore nasal form. The inevitable result in anything but a minimally displaced fracture, was characteristic nasal root flattening and collapse. Where comminution was marked the compression plates indeed accentuated the posterior recession of the nasal dorsum. The associated shrinkage and contracture of the overlying soft-tissue envelope produced a state almost insoluble by secondary reconstructive means. Mustardé (1963), Dingman & Natvig (1964) and Stranc (1970) identified and advocated the importance of direct open reduction, direct fixation with wire and reattachment of the medial canthal ligament (Fig. 11.77). When performed through restricted incisions, and where the extent and severity of comminution of the fractures were not accurately defined, the results remained suboptimal. Modern management identifies in detail the pattern of skeletal injury, to permit planned early wide exposure, accurate anatomical reduction with stable internal fixation and primary bone grafting where bone loss or comminution is extreme (Leipziger & Manson 1992). Just as the complex facial fracture demands accurate reconstruction of facial skeletal width and projection, with precise redraping of the soft-tissue cover, so injury to the naso-orbito-ethmoid region requires

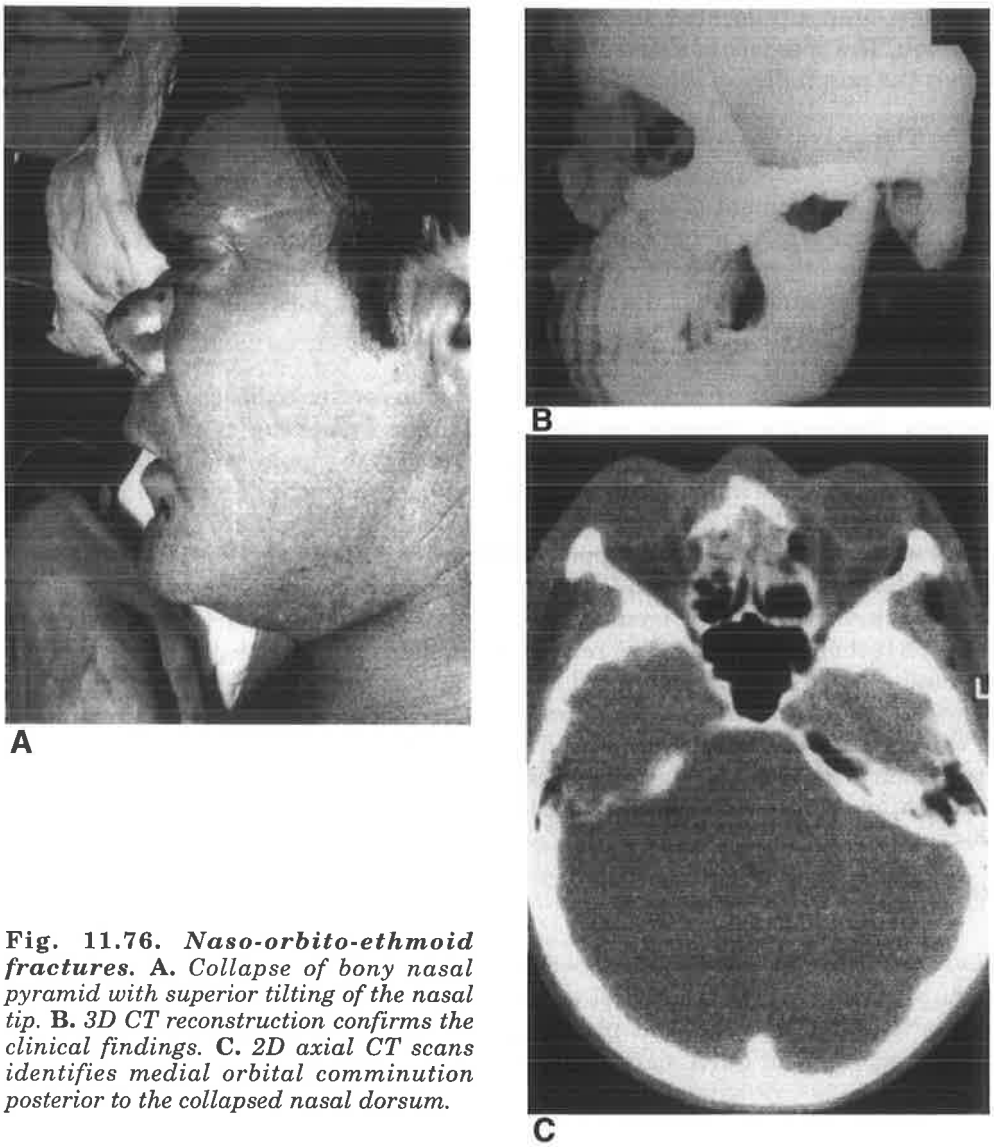


Fig. 11.76. ***Naso-orbito-ethmoid fractures.*** **A.** *Collapse of bony nasal pyramid with superior tilting of the nasal tip.* **B.** *3D CT reconstruction confirms the clinical findings.* **C.** *2D axial CT scans identifies medial orbital comminution posterior to the collapsed nasal dorsum.*

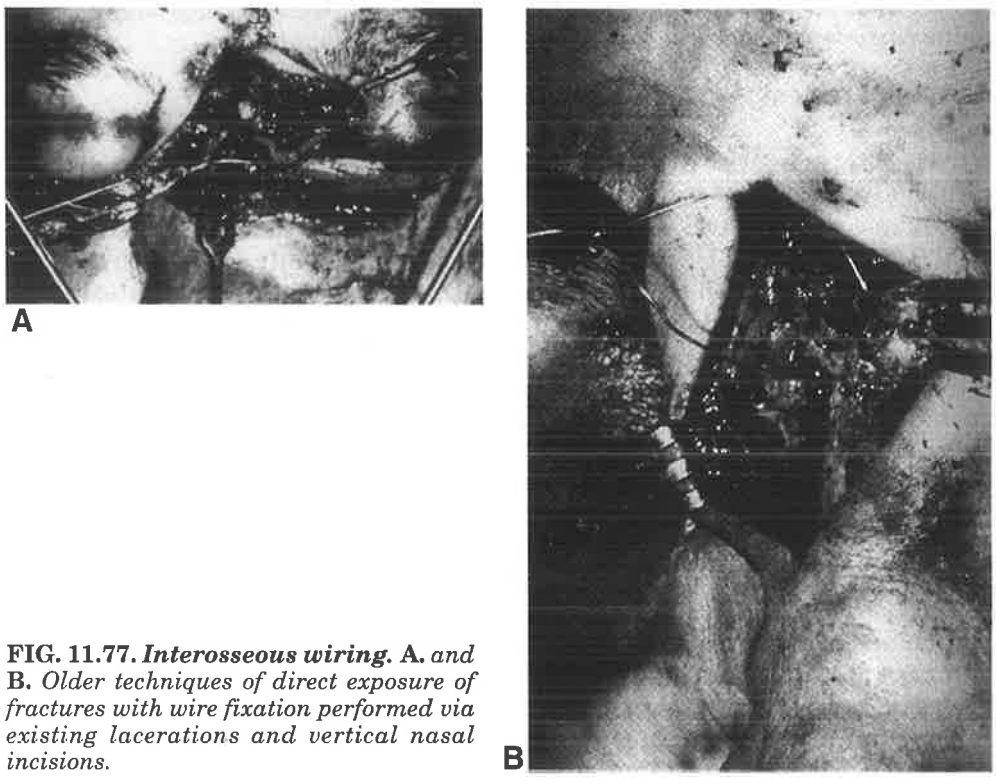


FIG. 11.77. ***Interosseous wiring.*** **A.** *and* **B.** *Older techniques of direct exposure of fractures with wire fixation performed via existing lacerations and vertical nasal incisions.*

restoration in three dimensions in a discrete fashion, correction of the intercanthal distance and anteroposterior positioning being of critical importance. Maintenance of the close bond of the soft tissues to the underlying skeleton, particularly in the medial canthal region, will permit ideal reconstruction in both transverse and anteroposterior directions. Finally the functional and aesthetic reconstruction is completed by appropriate redraping of the soft-tissue envelope, with repositioning of the medial canthal ligament and repair of the nasolacrimal drainage system where necessary.

Operative management

A coronal scalp incision gives the best exposure. Subperiosteal dissection of the entire lateral orbital wall and orbital roof allows wide, tension-free exposure of the medial orbital walls, nasal bones and frontal process of the maxilla. The trochlea is elevated, but care is taken to preserve the bony insertion of the medial canthal ligament.

A conjunctival incision with lateral canthotomy or lower eyelid subciliary incision exposes the inferior medial orbital wall and orbital floor (Fig. 9.5).

An upper vestibular (buccal sulcus) incision will identify and enable fixation of fracture extension to the nasomaxillary buttress and margin of the piriform aperture.

Local nasofrontal lacerations may provide midline exposure, but open incisions in this region have a place only in the elderly or bald patient where there is a localized fracture and where the coronal approach is aesthetically undesirable; except in such circumstances, the 'open-sky' incision of Converse & Hogan (1970) is now largely of historical interest.

Previously, multiple interosseous wires were employed to reassemble the bony jigsaw, but these suffered from the same inability to maintain nasal projection and width that had been experienced with external frames and splints. The demand for precise stable anatomic reduction in this area has seen the rapid implementation of miniplate, microplate and screw fixation.

Where the naso-orbito-ethmoid region is fractured as a single segment, either on one side or bilaterally, the deformity can be controlled by miniplate or microplate and screw fixation at two points: in some cases, a single plate on the piriform aperture or medial inferior orbital rim may be sufficient. In these cases the canthal attachment is in continuity with the major bony fragment and hence canthal position is restored by the skeletal reduction.

In injuries where bony comminution is more severe, adequate exposure is necessary to identify the nature of the bony attachment of the medial canthal ligament. Where this is intact, and the segment of bone of sufficient size, the segment itself may be used to maintain the correct intercanthal distance. Either by 28 gauge transnasal wires placed posterior and superior to the lacrimal fossa and tightened, or by an appropriately contoured microplate or miniplate, the central segment is reduced to minimize the bony distance between bony medial orbital margins. This is most frequently undercorrected, with inappropriately anterior placement of the medial canthus so that over correction should be attempted. The central segment is then fixed above and below to the stable bony elements by plates and screws.

Reconstruction of the medial orbital wall with rib or calvarial graft is usually necessary. In extreme comminution with avulsion of the medial canthal ligament, trans nasal canthopexy with wires is required; this is done through a hole in the bone graft of sufficient size (3 mm) to allow anchoring of the soft tissue. Use of a primary bony graft as a cantilever restores the nasal bridge projection, and maintains the soft tissues in correct length, by tissue expansion, which both

minimizes and aids later Secondary reconstruction. The nasal dorsum is reconstructed by an appropriately contoured costochondral graft or calvarial bone graft fixed superiorly at the nasofrontal region by miniplate and screws. The distal portion of the graft is inserted under the lower lateral cartilages to preserve normal tip anatomy. A columella strut is seldom required at the time of primary reconstruction if cantilever nasal support is adequate (Figs 11.78 and 11.79).

The nasal septum, which is invariably grossly buckled and oedematous, may be centred by a closed manipulation. A later definitive correction may be necessary. Primary septal reconstruction has so far not been widely attempted; the risks and difficulties are discussed below (p. 335).

Associated frontal sinus and cranial fractures are repaired at the same time with stabilising miniplates or microplates and screws. The presence of such fractures demands care in ensuring that the support for cantilevering the nasal dorsum is stable; this may require extension of plates further superiorly on the forehead. Compromise of the frontonasal drainage is only seen where both anterior and posterior walls of the lower portion of the frontal sinus are grossly fractured, and only then is exenteration and obliteration of the sinus necessary.

Management of nasolacrimal injuries

Despite its proximity, damage to the nasolacrimal system in naso-orbito-ethmoid fractures is not very frequent even in severe injuries; Cruse et al (1980) recorded laceration of the nasolacrimal duct system in 7 (21%) of 33 cases. Early exploration is not indicated except where there is an obvious lacrimal system transection in an external laceration, when standard repair over a silicone tube or nylon suture produces satisfactory results. Late obstructive symptoms requiring dacryocystorhinostomy are seen in only 5-10% of patients treated by open acute fracture reduction. When obstruction is noted it is usually in the bony nasolacrimal canal; this can be confirmed by dacryocystography (p. 178). Definitive dacryocystorhinostomy s best performed at least 3 months after the primary fracture repair. By contrast, closed reduction and external splinting of naso-orbito-ethmoid fractures was associated with a higher rate of obstruction by malpositioned bones and more frequent need for later dacryocystorhinostomy (Gruss et al 1985a).

Complications

These include:

- Uncorrected deformity
- Nasal obstruction
- Enophthalmos
- Soft-tissue thickening and contractures
- Prominent plates and screws.

Uncorrected deformity
This is the chief complication. Diagnosis of the naso-orbito-ethmoid fracture in the severely comminuted case is seldom difficult, even when diffuse soft-tissue swelling is evident. But where displacement is not so marked, oedema and swelling may result in misdiagnosis and a later presentation with deformity. Loss of nasal projection and increased nasal width produce a contracted, distorted soft-tissue envelope in which osteotomy and bone grafting is likely to produce a result inferior to what can be achieved by early correction.

Following early open reduction and fixation of naso-orbito-ethmoid fractures, telecanthus and inappropriately anterior placement of the medial canthus remain a common problem. This reinforces the importance of maintaining

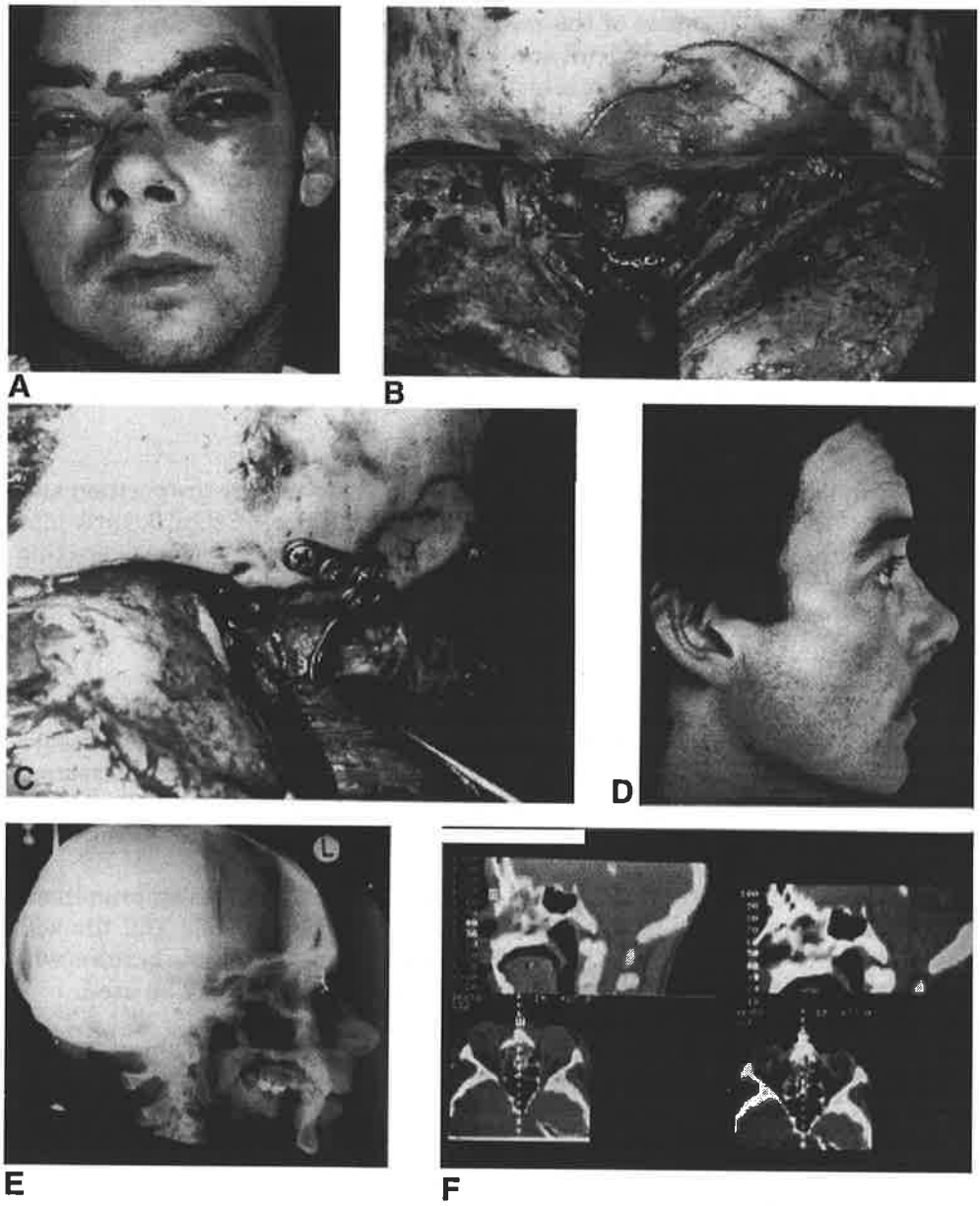


FIG. 11.78. ***Management of naso-orbito-ethmoid fracture with a nasal graft.*** **A.** *Significantly depressed naso-orbito-ethmoid fracture with frontal extension following assault with baseball bat.* **B.** *Operative exposure via bicoronal flap confirms extension into anterior wall of the frontal sinus.* **C.** *Operative view following reduction and internal fixation with miniplates.* **D.** *Postoperative clinical appearance with restoration of nasal dorsum including primary bone graft.* **E.** *Postoperative plain lateral radiograph.* **F.** *Pre- and postoperative midsagittal 2D CT reformats detail restoration of profile.*

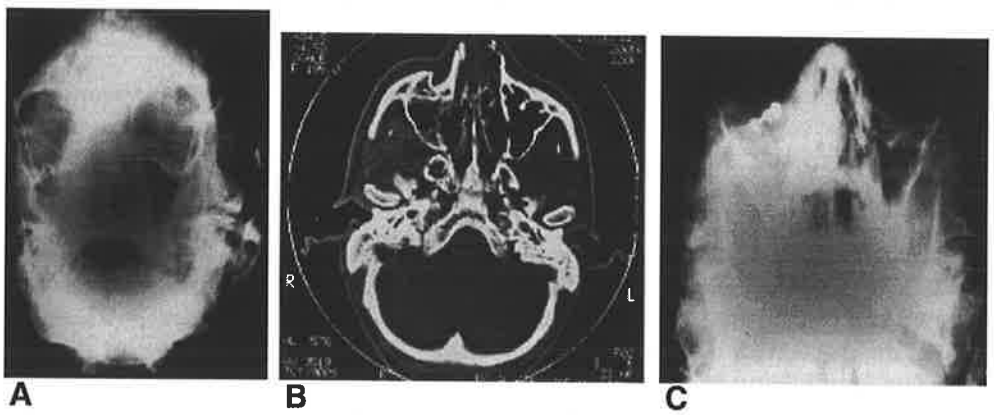


FIG. 11.79. ***Management of naso-orbito-ethmoid fracture.*** **A.** *Plain radiograph of isolated right naso-orbito-ethmoid fracture.* **B.** *Axial 2D CT scan details posterior displacement at right nasomaxillary region.* **C.** *Postoperative plain radiograph confirms reduction and fixation at the medial end of infraorbital rim and the piriform margin.*

the central bony attachment of the medial canthus, as exposing and reducing this structure is technically difficult and seldom recreates the pretraumatic state. Late reconstruction then demands osteotomy to reduce bony intercanthal distance together with a repeated medial canthopexy.

Collapse of the bony nasal dorsum is usually satisfactorily reconstructed by augmentation with a bone graft. Resorption and late irregularity may be corrected by further grafting, using standard rhinoplasty incisions and approaches (p. 577).

Nasal obstruction
Nasal airway obstruction is not infrequent following these injuries. Appropriate evaluation and septoplasty are commonly required.

Enophthalmos
Post-traumatic enophthalmos may result from failure to restore the position and continuity of the medial orbital walls where the injuries extend significantly into the orbit. Careful stable orbital wall reconstruction will prevent or mitigate this deformity; the late treatment of enophthalmos is difficult and often unsatisfactory (p. 559).

Soft-tissue thickening and contractures
Soft-tissue thickening and scar contracture with webbing may follow open lacerations and the surgical correction of the underlying fractures. The soft tissues in the medial canthal region are normally thin and pliable; trauma in dissection of this area must be minimal to avoid intractable problems with the soft tissues.

Prominent plates and screws
Miniplate fixation has greatly improved fragment stability. However, prominent plates and screws in this region may become visible or palpable and the soft tissues around them may be thickened. Microplates and small screws will minimize this problem; alternatively, fine interosseous wires may be used.

Fractures of the Zygoma

Surgical pathology

The description and management of fractures of the orbit and zygoma have often suffered from imprecision and inaccuracy. A superficial consideration of these injuries frequently fails to elucidate the complexities of the extent and pattern of the bony disruption and of the soft-tissue elements which act to displace the skeleton or are themselves disrupted by the fracture. Inadequate understanding leads to inadequate treatment, and the resulting orbitozygomatic depression, enophthalmos and ocular dystopia are among the more difficult facial deformities to treat.

Advanced imaging techniques and wide exposure of the orbitozygomatic region have revealed the true extent of these injuries and provided an impetus to prevention of the frequent complications which used to attend operative b treatment in this region.

Zygomatic fractures are among the most frequent facial injuries. They have frequently been described as *'tripod'* fractures: this term focuses attention on the resilient bony extensions of the zygoma that locally relate to adjacent bones, without due recognition of the almost inevitable involvement of the orbital wall and of the fourth bony process — the zygomatic arch. In fact, the zygomatic bone is truly a *'quadripod'* which borders the inferolateral margin of the orbit. Displaced fractures of the zygoma therefore involve the boundaries and walls of the orbit, though to a variable degree (Fig. 11.80).

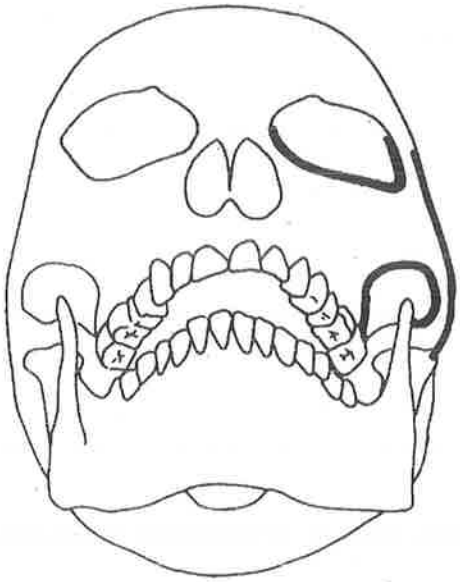
For many years, there was a belief that restoration of the solid bony contours of the cheek was sufficient, and this favoured the use of simple techniques of fracture reduction. But when older techniques of reduction were seen to produce a high incidence of residual displacement, and with the increasing frequency of high velocity severely comminuted fractures, there arose a demand for techniques of stable fixation in three dimensions and accurate restoration of all bony orbital contours both external and internal.

The zygomatic bone makes a key contribution to orbital contour, facial width and projection (Figs 11.80 and 11.81). Comprising the inferolateral orbital rim, the zygoma articulates with the frontal bone superiorly, the maxilla inferiorly and medially, and with both the spheroid and temporal bones posteriorly and laterally. Four bony processes radiate from the zygomatic body providing strong articulations with the frontal, maxillary and temporal bones. The bony junction between the temporal process of the zygoma and the zygomatic process of the temporal bone constitutes the zygomatic arch. Attached to the arch are the temporalis fascia and masseter muscle, this muscle being usually considered to be the major deforming force in maintaining displacement of a depressed zygomatic fracture, though this is in debate (Dal Santo et al 1992). Immediately superficial to the arch are the superficial temporal vessels and frontal branch of the facial nerve, while deeply pass the internal maxillary vessels, temporalis muscle and coronoid process.

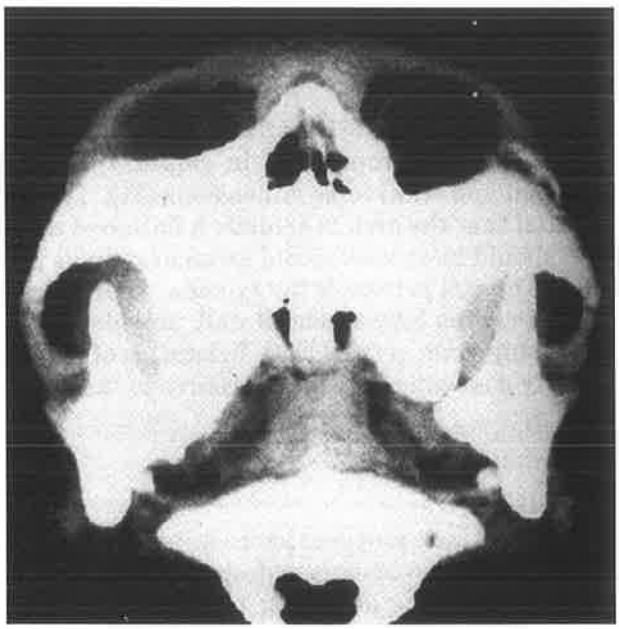
Gruss et al (1990) emphasize the importance of the position of the zygomatic arch to locate the zygoma appropriately in relation to the skull base in anteroposterior, vertical and transverse dimensions (Fig. 11.81); more recently Gruss has also stressed that the arch is actually a flattened arciform structure, and that this shape should be reconstructed as accurately as possible. Perhaps equally important, the orbital portion of the zygoma, where it meets the greater wing of the spheroid along the lateral orbital wall, provides similar information regarding the relationship to the cranial base. Relocation of the displaced zygoma cannot be anatomically guaranteed without recourse to exposure of a number of these articulations.

Classification

Various classifications have been proposed for fractures of the zygoma. The system of Knight and North (1961) achieved early widespread use. These authors detailed patterns of zygomatic fracturing according to direction of displacement and rotation of the zygoma as viewed on plain radiographs. This was a simplification of the pattern of injury, implying dislocation in two dimensions about a single axis or point of rotation; the classification was consistent with the imaging techniques and level of open surgical exposure available at that time. With the advent of CT scanning, and with improved methods of operative exposure and fixation of the orbitozygomatic region, the extent rather than the pattern of fracturing was seen to be of greater significance. Where low velocity impacts produce linear fractures with lesser degrees of displacement, high velocity impacts result in comminution, displacement and extension into the adjacent facial skeleton. Jackson (1989) evolved a simplified general classification of zygomatic fractures relating pattern and displacement to treatment (Table 11.3). Gruss et al (1990) recognized the role of the zygomatic arch injury in accurate reduction and classified these injuries according to the level of bony damage to the zygomatic body and the zygomatic arch. We (Cooter & David 1989) quantify the degree of bony comminution in each element of the zygoma as well as the level of displacement of the skeletal articulations. This system, although complex, permits more accurate comparison of treatment results between different series (p. 35).



A



B

FIG. 11.80. ***Anatomy of zygomatic fractures.*** **A.** *and* **B.** *Diagram and 3DCT reconstruction identify the contribution of the zygoma to the orbital contour and facial width and projection.*

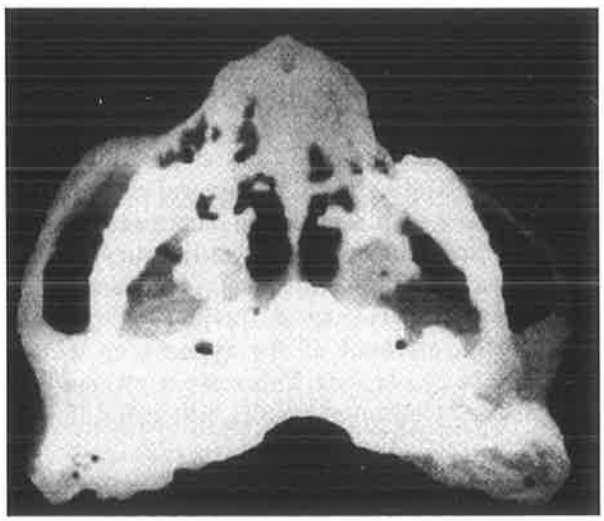


FIG. 11.81. ***Radiology of zygomatic fractures.*** *Basal 3D CT reconstruction of untreated displaced left zygomatic fracture. Lateral bowing of the zygomatic arch accompanies posterior displacement of the zygomatic body.*

TABLE 11.3

*Classifications of zygomatic fractures**

A. Jackson (1989)	
Group I	Undisplaced fractures—no treatment
Group II	Localized segmental fractures—exposure and fixation
Group III	Displaced 'tripod' fractures (low velocity)—simple elevations, or elevations, exposure and fixation
Group IV	Displaced comminuted fractures (high velocity) wide exposure and multiple point fixation

B. Gruss et al (1990)	
1. Zygomatic body	2. Zygomatic arch
(a) Intact	(a) Intact
(b) Undisplaced	(b) Undisplaced
(c) Segmental	(c) Segmental
(d) Displaced	(d) Displaced
	— inferiorly with depression
	— laterally
(e) Comminuted	(e) Comminuted

* These simple systems of classification provide a grading of severity which can be related to management protocols.

Clinical assessment

A history of local trauma to the cheek should arouse suspicion of orbitozygomatic fracture. Recording the mechanism, direction and particularly the velocity of impact is essential; and the observer should maintain a high index of suspicion for associated ocular, midfacial, craniocerebral and cervical spine injuries. The association of zygomatic fractures with other midfacial injuries and with fractures of the adjacent segments of the mandible (condylar process, ramus, coronoid and angle) has to be kept in mind; mandibular injury is especially likely where a localized high velocity impact is sustained.

The symptomatology typically relates to the effect of the displaced zygomatic bone on the surrounding soft tissues. The orbital soft tissues manifest swelling, ecchymoses and subconjunctival haemorrhages, particularly laterally (Figs 11.82-85). Diplopia or blurred vision may be a complaint due to orbital soft-tissue swelling, ocular dystopia or primary ocular injury. Numbness or altered sensation in the distribution of the infraorbital nerve (cheek, lateral portion of nose and upper lip) indicates infraorbital rim fracturing. Trismus and complaints of malocclusion are recorded where arch fractures impinge on the underlying temporalis muscle. A sense of an altered occlusion may also follow injury to the infraorbital and superior alveolar nerves. In both situations a true occlusal disturbance by extension of the zygomatic fracture into the maxillary alveolar process must be excluded by careful intraoral examination (Fig. 11.83).

As the zygoma is largely superficial, physical examination and especially palpation will readily identify displacement. Observation from above (bird's eye view) provides an impression of loss of the zygomatic prominence (Fig. 11.84). This is confirmed by placing examining fingers on the most prominent part of the zygomatic body and comparing with the non-injured side. When there is displacement in the inferior orbital rim, there may be a palpable step-down as one feels from medial to lateral at the infraorbital rim, with associated tenderness. More laterally a tender palpable depression in the zygomatic arch is present, often the only abnormality in isolated arch fractures. In contrast, where the zygomatic fracture accompanies extensive midfacial fracturing the zygomatic arch is bowed outward and the anteroposterior projection of the zygomatic prominence is diminished.

Intraoral examination will identify bruising in the upper vestibule where fractures of the maxillary buttress may also be palpable. Alveolar extensions of the fracture or malocclusions caused by trismus or associated midfacial and mandibular fractures may also be noted.

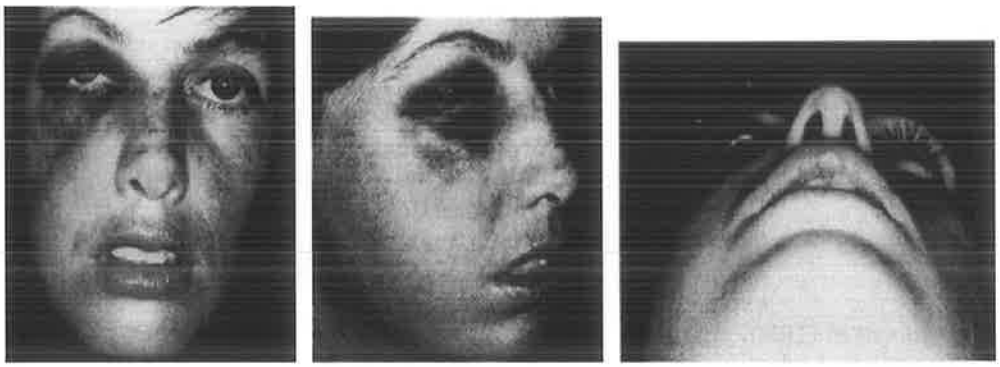


FIG.11.82. ***Clinical signs of zygomatic fractures.*** **A.** *Frontal,* **B.** *right oblique and* **C.** *worm's-eye views of women with depressed right zygoma after a horse riding accident. Marked periorbital bruising does not disguise the flattening of the right cheek. Enophthalmos on the right produces a pseudoptosis.*

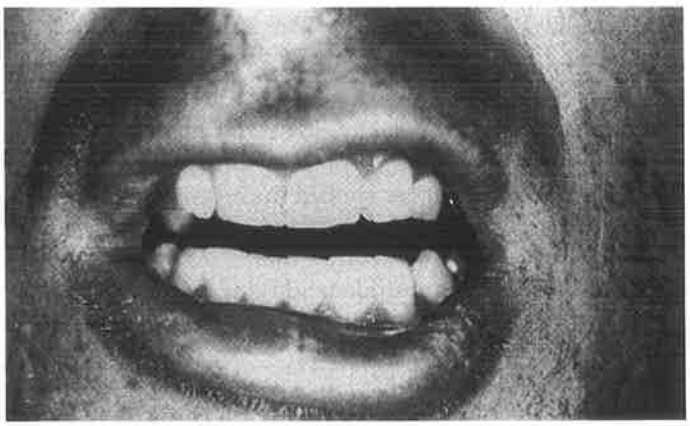


FIG. 11.83. ***Clinical signs of zygomatic fractures with maxillary extension.*** *Open-bite occlusal disturbance in patient with a depressed zygomatic fracture with extension into the maxillary alveolus.*

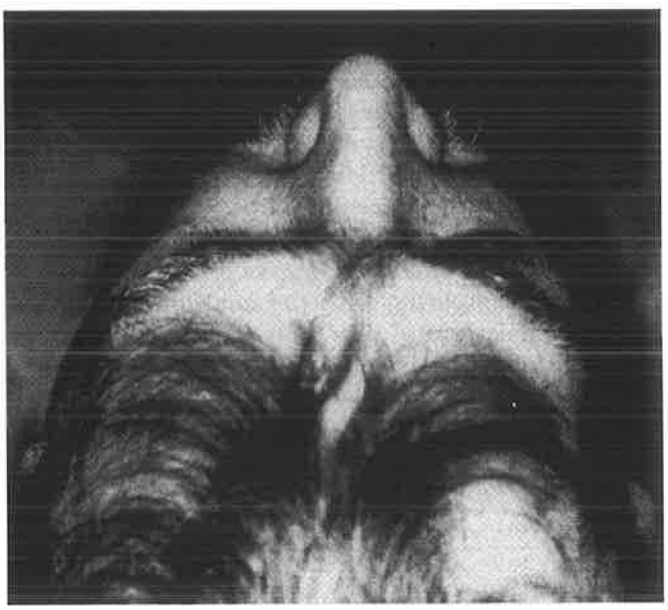


FIG. 11.84. ***Clinical signs of zygomatic fractures.*** *Birds-eye view identifies flattening of the left zygoma.*

Detailed ophthalmological examination (p. 167) includes assessment of visual acuity, extraocular muscle function and degree of enophthalmos. Enophthalmos may be masked by early severe facial and periorbital swelling and indeed exophthalmos is not uncommonly the early finding. Unusual zygomatic fractures with medial impaction may be associated with a reduction in the bony orbital volume and hence a tendency to exophthalmos. Ophthalmoscopic examination will disclose any retinal injury or hyphema, the presence of which sometimes requires further investigation and perhaps delay in the operative repair of the skeletal injuries.

Radiological assessment

Plain radiological assessment of the zygoma consists of the Waters, reverse Waters, Caldwell, submentovertical and lateral views (p. 199). The Waters view records displacement at the inferior orbital rim, maxillary buttress and zygomatic body; the Caldwell view shows the frontozygomatic region and zygomatic arch; the submentovertical view shows the zygomatic arch. For isolated zygomatic arch fractures, no further radiological assessment is really necessary, though we commonly perform CT scanning to amplify the anatomical diagnosis and as a baseline for postoperative verification of reduction.

For more complex patterns of fracturing requiring accurate assessment of orbital wall involvement, degree of zygomatic body and arch displacement, and degree of comminution, axial and coronal two-dimensional (2D CT scanning of the orbitozygomatic region is certainly indicated (Fig. 11.86). Coronal scans visualize the orbital walls and the orbital floor; the axial views show accurately any deformation of the zygomatic body, arch and lateral and medial orbital walls. Both views, by identifying the appropriate stable skeletal elements, assist in planning the surgical exposures necessary for reduction and stable fixation (Fig. 11.87). 3D CT scan reconstructions provided graphic pictorial assistance but add nothing to surgical decision-making which relies much more on the baseline 2D CT scan data.

Principles of management

The evolution of treatment of zygomatic fractures has seen continuing debate with regard to the indications for reduction and the techniques and patterns of fixation of zygomatic fractures.

The mainstay of reduction alone has been Gillies' technique of percutaneous elevation by leverage from an incision in the temporal hair-bearing scalp (Gillies et al 1927), or Dingman's variation (Dingman & Natvig 1964) of elevation from the eyebrow, or even from an intraoral approach. Few studies have detailed objective results regarding outcome after simple Gillies reduction. Melmed (1972) studied a group of low velocity, minimally displaced body and depressed arch fractures and reported a 30% rate of results considered to be less than acceptable. This report also detailed an improved aesthetic outcome and fewer poor results when fixation was added, with further increments of benefit when reduction was performed under direct vision. An opposing view has been presented by Schubert (1991), with excellent anatomical restoration in 73% of cases treated by percutaneous reduction alone, but in this study the comminuted or significantly displaced fractures were treated by some form of fixation while the more stable, minimally displaced, low velocity fractures were treated by reduction alone.

The progression through techniques of antral packing, interosseous wiring and transfixion with Kirschner wire to the use of miniplate fixation has seen more consistent restoration of symmetry. Rohrich et al (1992) reported fewer problems with infraorbital nerve complications, malpositions of the globe and inaccurate cheek positioning when miniplate fixation was compared with fixation by interosseous wires. It is now standard practice to employ miniplate fixation and wide operative exposure of the orbitozygomatic skeleton, thus allowing

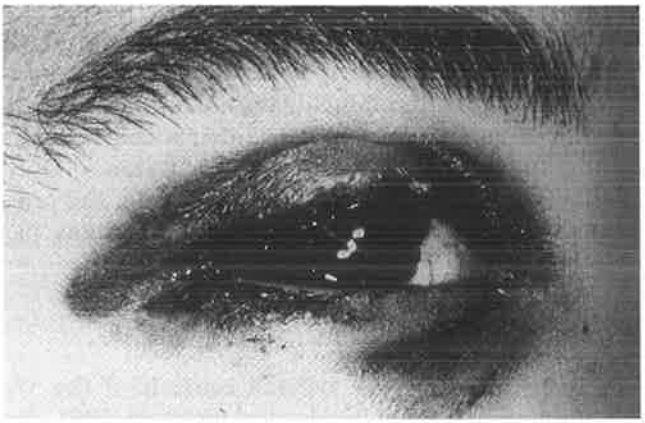


FIG. 11.85. ***Clinical signs of zygomatic fractures.*** *Laterally based subconjunctival haemorrhage in a fracture of the right zygoma.*

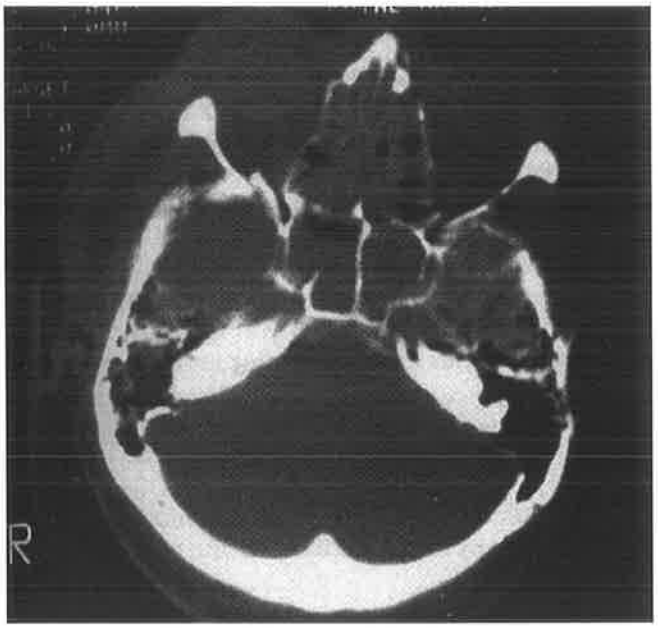


FIG. 11.86. ***Radiology of zygomatic fractures.*** *Axial 2D CT scan shows very posterior fracturing of the lateral orbital wall, medial impaction of the orbital rim and marked exophthalmos.*

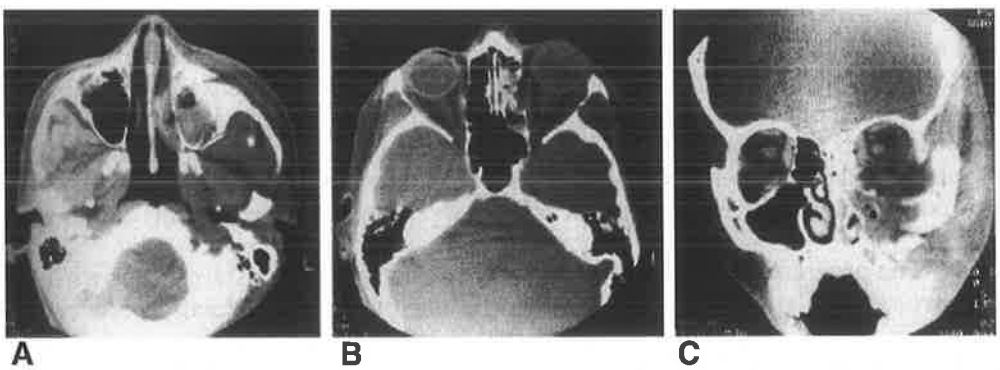


FIG 11.87. ***Radiology of zygomatic fractures.*** **A.** *Axial 2D CT scan identifies posterior displacement of the zygomatic body, fracturing through the infraorbital rim and lateral bowing of the zygomatic arch.* **B.** *Medial orbital wall blow-out in association with displaced zygomatic fracture.* **C.** *Coronal 2D CT scan displays zygomatic body displacement, medial and lateral wall, and orbital floor damage in a high-velocity impact.*

anatomical restoration of the zygomatic prominence with fewer positional disturbances of the globe, such as enophthalmos and vertical dystopia.

Locating the required points of fixation demands an understanding of which articulations are most easily displaced. Fracturing and comminution begin in the zygomaticomaxillary buttress and inferior orbital rim, with extension to and involvement of the greater spheroid wing (lateral orbital wall); disruptions of the zygomatic arch and zygomaticofrontal suture are seen in the more severe injuries. Since the masseter muscle is the major displacing force acting on the zygoma, miniplate fixation of the zygomaticomaxillary buttress and the zygomaticofrontal articulation should orientate the vectors of fixation in the optimal axis. The techniques of exposure, reduction and miniplate fixation are now well described, but surgeons differ on how these principles are applied in each pattern of fracture.

In discussing treatment approaches, it is logical to subdivide fractures of the zygoma into those where there is no orbital wall involvement (isolated zygomatic arch fracture) and the more common orbitozygomatic fracture, where the fracture lines involve at least one wall of the orbital cavity.

Isolated zygomatic arch fracture

The isolated zygomatic arch fracture is usually a low velocity injury, with characteristic symptoms of trismus and clinical signs of localized arch contour depression. Treatment is by the closed reduction technique of Gillies et al (1927). A short oblique incision in the hair-bearing temple allows passage of an elevator deep to the temporalis fascia behind the depressed arch. This achieves stable reduction in the majority of cases (Fig. 11.88). In rare cases, isolated arch fractures may be unstable after reduction. While it is possible to fix arch fractures internally via a bicoronal approach, the degree of contour abnormality seldom justifies an open operation of such magnitude. The less invasive technique of stenting of the arch with a percutaneous needle or tubing sutured over the arch may have a role. Alternatively, the swelling and oedema in the temporalis muscle after the performance of the Gillies manoeuvre may provide a degree of internal splintage sufficient for contour maintenance in these cases.

When untreated or neglected, zygomatic fractures manifest a cosmetic deformity — the localized depression of the lateral cheek. Only in extreme cases where depression is marked is there a long-term disturbance of jaw opening and closing. There are no significant complications of the percutaneous surgical correction.

Orbitozygomatic fractures

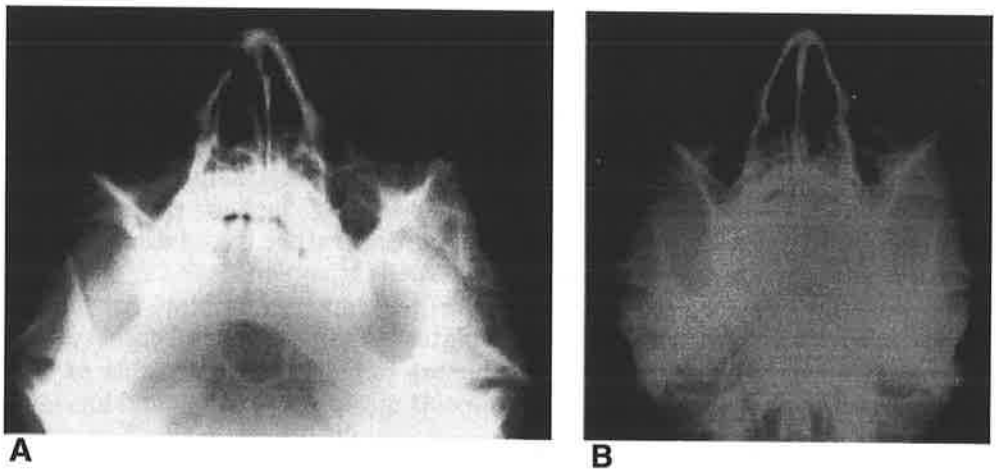


FIG. 11.88. ***Radiology of zygomatic arch fractures.*** **A.** *and* **B.** *Plain radiographs confirm reduction of an isolated left zygomatic arch fracture following a Gillies elevation.*

Undisplaced fractures require no operative treatment. Advice is provided to avoid local pressure on the zygomatic prominence during the initial weeks, with follow-up maintained until healing at about 6 weeks. The incidence of late displacement of initially conservatively managed undisplaced zygomatic fractures is unknown, but appears to be very low. Early recognition will permit appropriate operative intervention.

Displaced orbitozygomatic fractures demand careful open reduction and internal fixation on an individualized basis. Most fractures fall within the category of mild to moderate displacement, in association with a variable degree of comminution. Fracture separation in these cases is invariably more marked at the zygomaticomaxillary buttress and the inferior orbital rim, with lesser injuries at the superior articulations. Exposure via a lower eyelid subciliary skin-muscle flap or transconjunctival incision with lateral canthotomy reveals the zygomaticofrontal suture, lateral orbital wall, inferior orbital rim, orbital floor and anterior zygomatic arch (Fig. 11.89). An upper buccal vestibular incision permits exposure of the zygomaticomaxillary articulation and anterior attachment of the masseter muscle to the zygoma (Fig. 11.90). Following fracture mobilization and elevation either by the intraoral approach or by Gillies' procedure, anatomical restoration is achieved by visualizing the anterior zygomatic articulations, and comparing the anteroposterior projection of the zygoma and inferior orbital rim with the contralateral side. The addition of lateral orbital wall exposure reveals the alignment of the zygoma with the greater wing of spheroid and confirms accurate reduction.

Miniplate fixation in the zygomaticomaxillary buttress has a vector of action which opposes the displacing influence of the masseter, albeit in bone which is thin and has little strength. The zygomaticofrontal suture, while a poor single indicator of reduction, provides the strongest bone for miniplate fixation. This two-site fixation will provide sufficient stability in the majority of fractures of this type. An additional third point of fixation at the inferior orbital rim with miniplates, or preferably a micro-plate or fine wire, will counteract the remote possibility of lateral rotation. Where significant comminution is present in the zygomaticomaxillary buttress, inferior orbital rim fixation will contribute significantly to stability in this level of injury; this may be unnecessary if bone grafting is employed (see below). Operative treatment is completed by careful reattachment of the midfacial and periorbital soft tissues to the zygomatic skeleton to minimise subsequent soft-tissue sagging under gravity, causing scleral show, lower lid ectropion, lateral canthal distortion and depression of the zygomatic soft-tissue prominence.

In many zygomatic fractures where displacement is significant, but where comminution is not marked, single miniplate fixation at the zygomaticofrontal suture is adequate (Figs 11.91 and 11.92). Visualization of the orbital rim, lateral orbital wall and zygomaticomaxillary region is also required to confirm the accuracy of the reduction.

In thinly built individuals and females the zygomaticofrontal miniplate is often palpable and sometimes visible, requiring later removal. This is accentuated where there is dissection, displacement or atrophy of the temporalis muscle. Countersinking the miniplate by burring or contouring the bone prior to plate placement, or by locating the plate on the posterior, rather than lateral aspect off suture may obviate this problem. Microplate fixation causes fewer cosmetic problems (Fig. 11.93). Yaremchuk et al (1993) recommend miniplate fixation of the zygomaticomaxillary suture, with microplates at the zygomatico frontal suture and the infraorbital rim. This fixation appears to be at least as stable as the more conventional pattern of two- or three-point miniplate fixation, and has less likelihood of aesthetic complications.

Comminuted and grossly displaced orbitozygomatic fractures seldom occur in isolation. Almost always these fractures occur in high velocity impacts

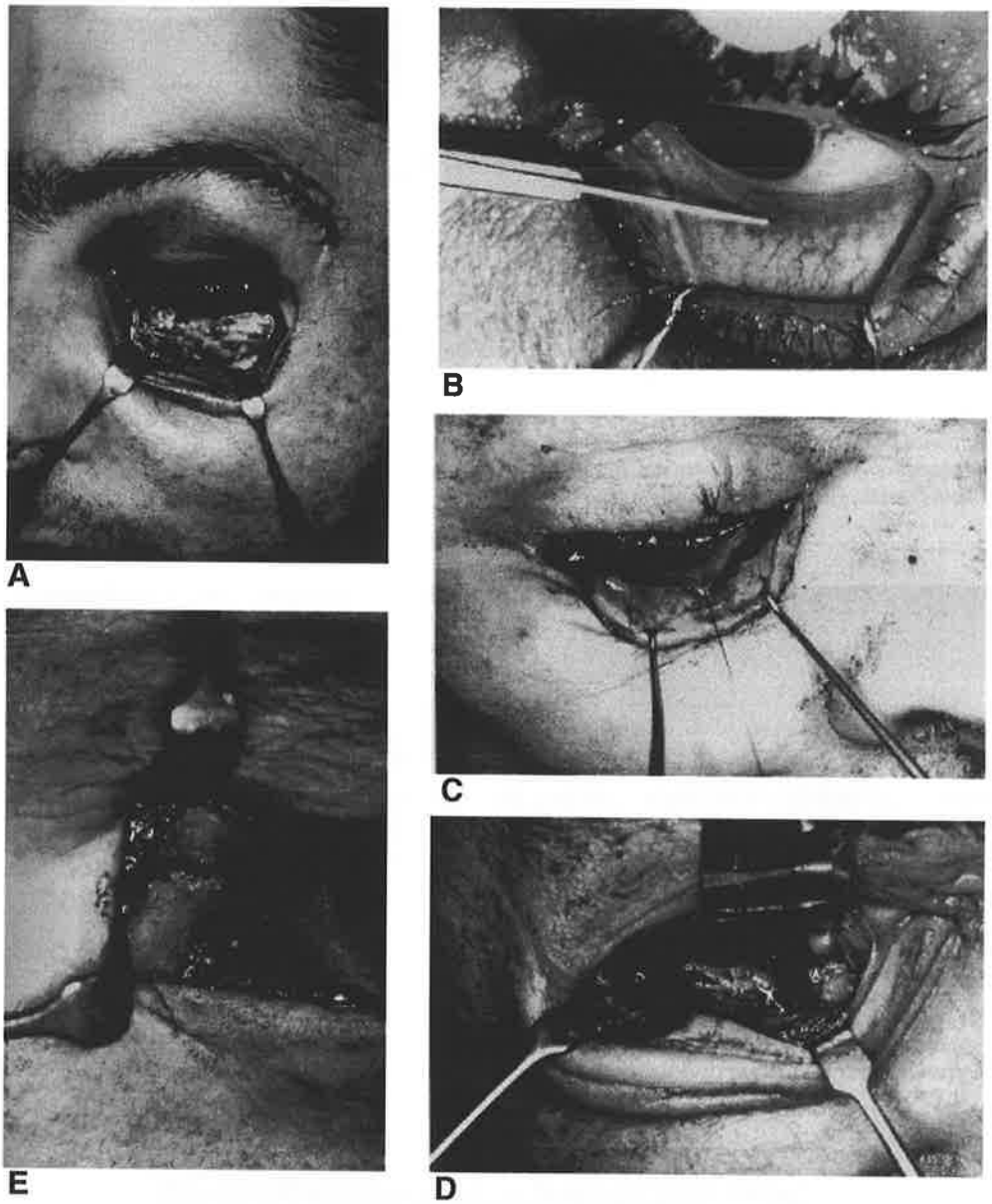


FIG. 11.89. ***Surgical exposure of orbital rim.*** **A.** *Subciliary skin–muscle flap approach to the inferior orbital rim and floor.* **B.** *Transconjunctival approach begins inferior to the tarsal plate of the lower eyelid.* **C.** *Extension with lateral canthotomy improves width of exposure.* **D.** *Displaced fracture of the inferior orbital rim easily exposed.* **E.** *Lateral and superior dissection allows exposure and plating of the frontozygomatic fracture via conjunctival incision and lateral canthotomy.*

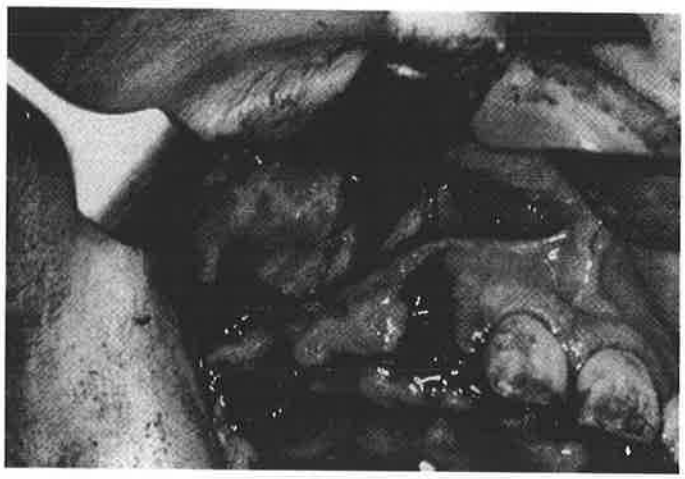


FIG. 11.90. ***Surgical exposure of zygomaticomaxillary region.*** *Upper vestibular incision exposes the anterior maxilla, piriform margin and zygomaticomaxillary fracture.*

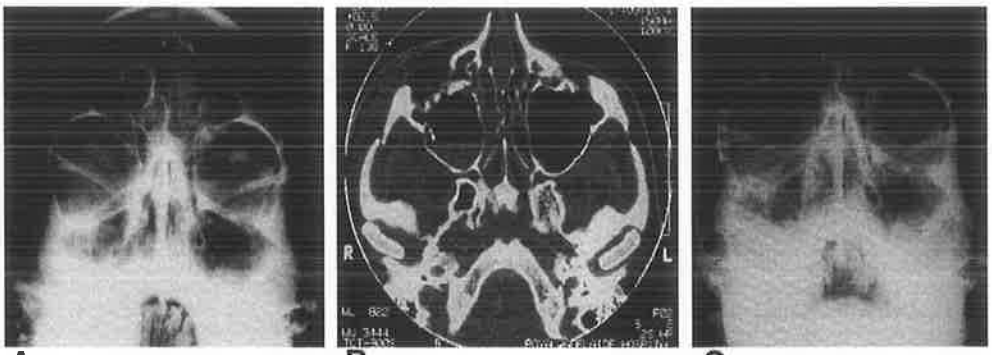


FIG. 11.91. ***Radiology of zygomatic fracture management.*** **A.** *Displaced but minimally comminuted fracture of the right zygoma* **B.** *Axial 2D CT scan confirms displacement.* **C.** *Postoperative plain radiograph displays satisfactory reduction with single frontozygomatic mimplate fixation.*

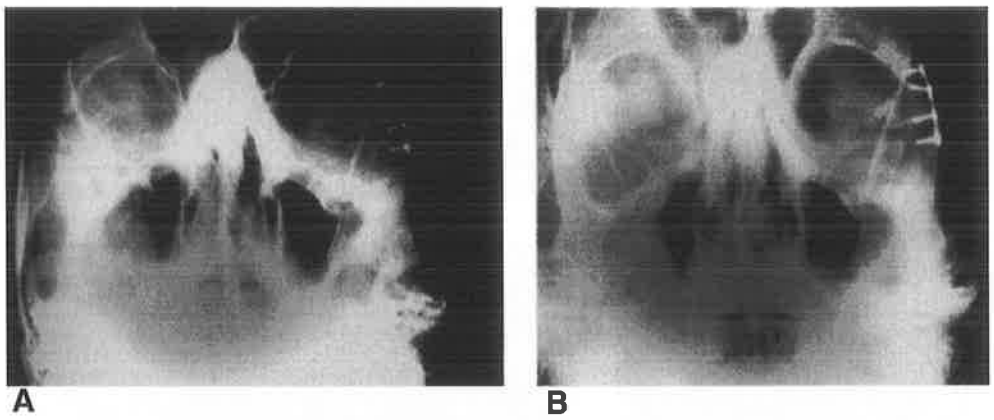


FIG. 11.92. ***Radiology of zygomatic fracture management.*** **A.** *Displaced right zygomatic fracture in patient with previously treated old fracture of the left zygoma* **B.** *Postoperative radiograph with single frontozygomatic plate fixation.*

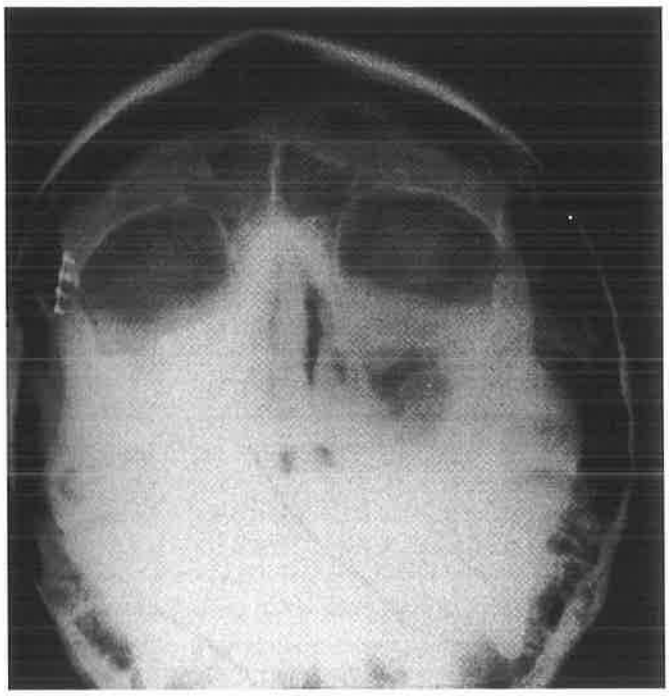


FIG. 11.93. ***Microplate fixation of zygomatic fractures.*** *Significantly displaced right orbitozygomatic fracture with microplate fixation at frontozygomatic fracture and micromesh at inferior rim to restore orbital floor position*

producing major midfacial or panfacial fractures, of which the zygomatic injury forms one portion. Superimposed on the pattern of the moderately displaced fracture group, and reflecting more violent distortion, are comminution of the lateral orbital wall, including the greater wing of the spheroid, and disruption of the zygomatic arch. Recognizing the disruption and restoring the arch, as advised by Gruss et al (1990), is the key to restoration of the zygomatic projection, the midfacial width and the anteroposterior projection (Fig. 11.94). Axial CT scan images identify the arch fractures, usually anteriorly at the junction with the zygomatic body and posteriorly passing obliquely close to the glenoid fossa, with intervening lateral bowing of the arch. A bicoronal approach facilitates exposure, mobilization and reduction of the arch fractures. Disinsertion of the masseter muscle may be required for reduction; correct anatomical positioning is achieved by comparison with the contralateral side.

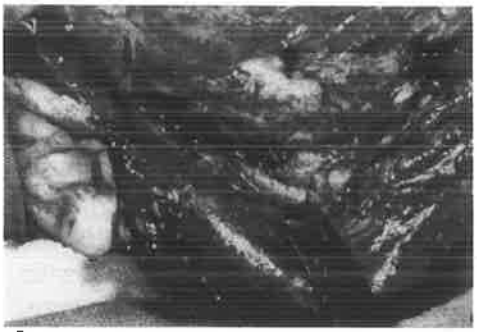
Fixation in these cases follows the 'best fit' principle, comparison with the normal contralateral side being readily available. Temporary stabilisation at the zygomaticofrontal suture with an interosseous wire or miniplate allows the surgeon to verify that there is correct rotational and projectional alignment at the zygomaticomaxillary buttress, inferior orbital rim and lateral orbital wall, before proceeding to definitive miniplate and screw fixation of the zygomatic arch. Formal miniplate fixation of the remaining sites is then performed to position the zygoma rigidly between the fronto-orbital borders above and the midface below.

The role of microplate fixation in these high energy injuries has not been clearly defined. Yaremchuk et al (1993) believe these to be inadequate on their own in situations where extreme soft-tissue swelling may exert considerable deforming forces. We have had no experiences to support or disprove this view.

Comminution and loss of bone at the major articulations, the anterior maxillary wall, the orbital floor and the lateral orbital wall demand careful operative display, reduction and reconstruction by primary bone grafting if the major complications of enophthalmos and orbito-ocular dystopia are to be avoided (Fig. 11.95). High velocity injuries with their characteristic comminution all require exploration of at least the orbital floor and lateral orbital wall. The lateral orbital wall is situated entirely posterior to the axis of the globe, and nonanatomical reduction produces an increase in bony orbital volume with resulting enophthalmos (Fig. 11.96). Comminution in this region invariably requires the addition of bone grafts from calvarial, rib or iliac donor sites. Grafts are easily placed into any wall of the orbit, particularly where the posterior 'ledges' are used as guides to alignment and stability, but bone graft displacement is too often seen. Stabilizing these grafts by miniscrew, miniplate, microplate or titanium mesh should improve predictability of outcome and reduce the incidence of resistant enophthalmos.

The orbital floor characteristically bulges upward in its most posterior extent, 35-38 mm behind the inferior orbital rim where the intact bony ledges are located (Fig. 11.97). Failure to expose this area and to employ it for bone grafting may lead to inadequate initial bony orbital wall reconstruction and also to subsequent graft displacement and orbital volume expansion.

The case for routine wide orbital exploration in medium velocity orbitozygomatic fractures is not so well defined. Orbital wall components of the zygomatic fracture do not require exploration if CT scanning shows no displacement: in such cases the periorbita is intact and maintains the soft-tissue orbital volume. Unnecessary exploration risks disruption of the periorbita and may cause orbital volume changes secondary to the surgery.



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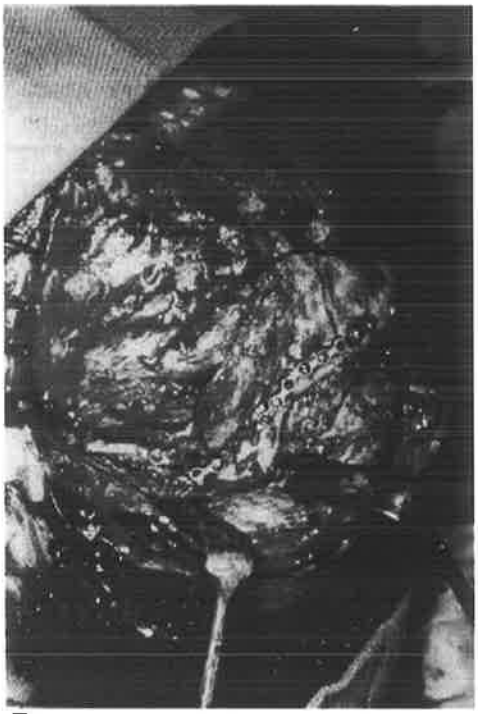
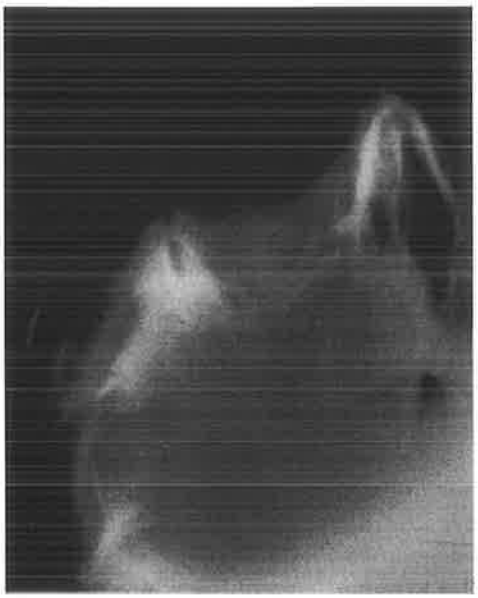


B

FIG. 11.94. ***Management of zygomatic arch fractures.*** **A.** *Zygomatic fracture occurring in association with midfacial fractures. Displaced arch fragments at tip of retractor.* **B.** *Stabilization at the frontozygomatic region and zygomatic arch restores facial projection and width.*



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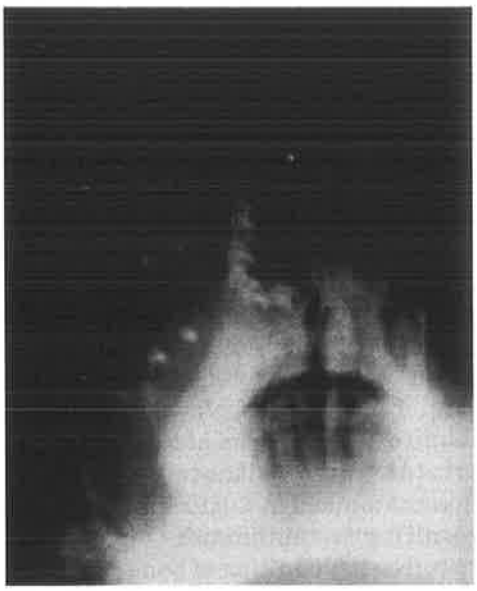


B

FIG. 11.95. ***Management of comminuted zygomatic fractures.*** **A.** *Severely comminuted right zygomatic fracture.* **B.** *Plain radiograph after wide exposure, reduction, internal fixation and primary bone grafting.*

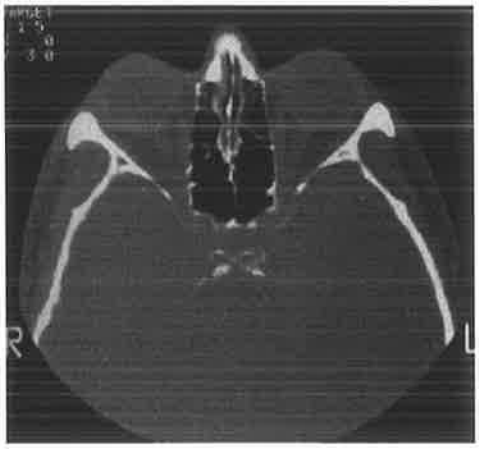


FIG. 11.96. ***Radiology of orbital margins.*** *Axial 2D CT scan of uninjured orbits. The lateral wall lies entirely behind the equator of the globe.*

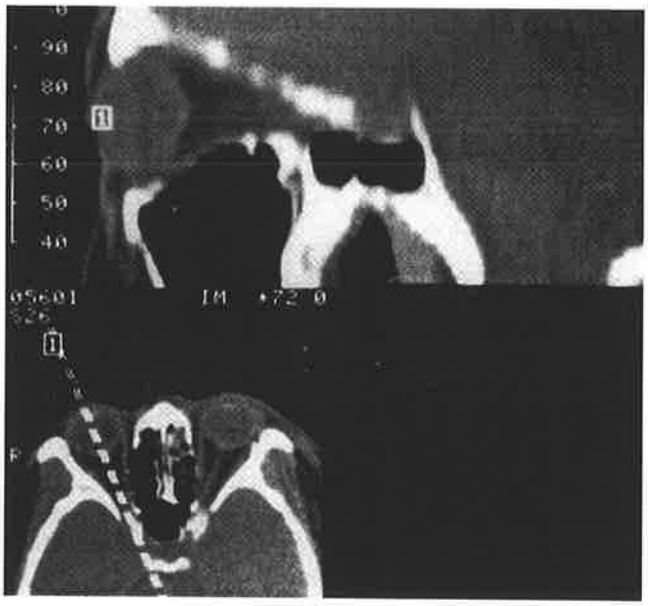
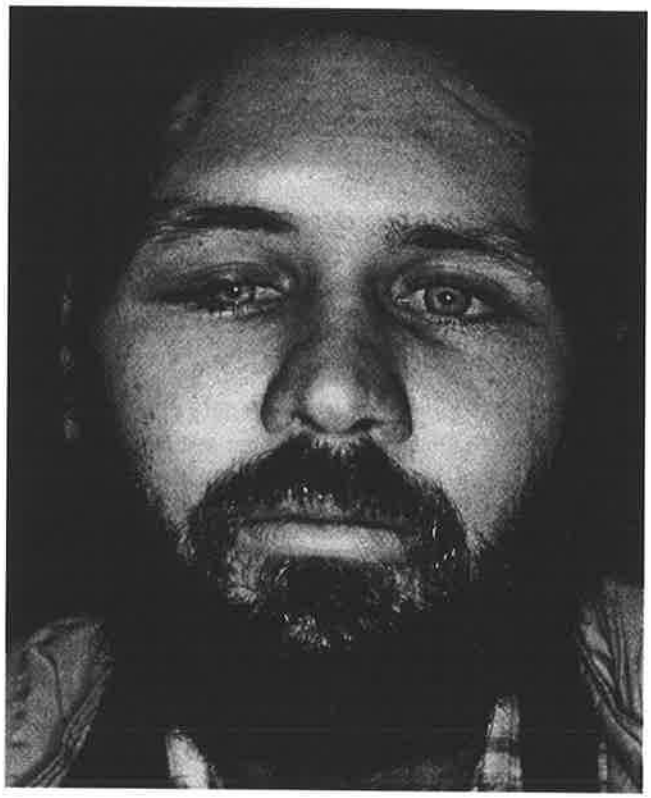


FIG. 11.97. *Radiology of orbital floor.* *Oblique sagittal 2D CT reformat identifies the upward bulge of the orbital floor in its posterior portion.*



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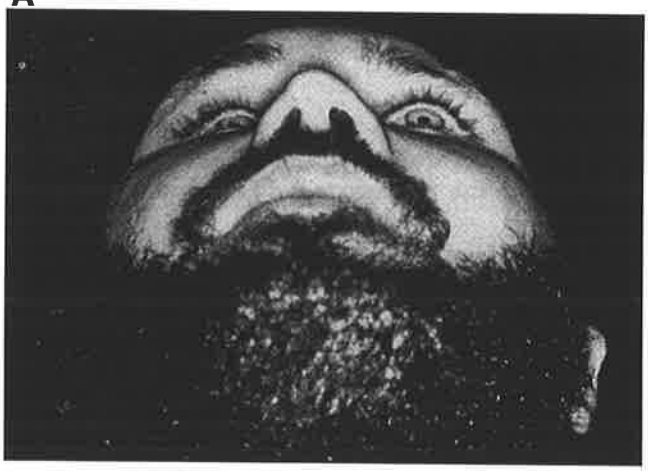

B

FIG. 11.98. *Post-traumatic enophthalmos.* **A.** *Frontal and* **B.** *worm's-eye view of marked enophthalmos on right side following severe right frontozygomatic fracture. Note the supratarsal hollowing and pseudoptosis.*

Complications

These include:

- Deformity
- Prominent plates
- Disturbance of globe position
- Ocular injury
- Nerve injury.

Deformity
The chief complication of the older techniques of management was inadequately or uncorrected deformity. Osseous deformity has been much reduced by wide exposure, open reduction, internal fixation and primary bone grafting; these techniques carry with them certain side effects which demand finesse in surgical technique. Soft tissue deformities include sagging of eyelid and cheek after zygomatic dissections (Phillips et al 1991); meticulous redraping of the dissected soft tissues should lessen the risk of this deformity.

Prominent plates
Plate prominence is common: plate placement must minimize palpability and visibility.

Disturbance of globe position
Disturbance of globe position in both the anteroposterior and vertical dimension remains the major long-term complication. Enophthalmos and vertical ocular dystopia as late post-traumatic manifestations are cosmetically and often functionally disturbing, and almost impossible to correct perfectly when well established. These deformities constitute the major complication in high velocity, severely comminuted zygomatic fractures (Fig. 11.98).

Ocular injury
Direct globe injuries are common in association with these fractures, but most are not serious and without long-term sequelae. These injuries are discussed in detail in Chapter 14.

Nerve injury
Infraorbital nerve injuries causing persistent numbness, paraesthesia or hyperaesthesia in the distribution of the nerve may occur; Dielert & Jais (1992) reviewed 176 case of zygomatic fractures, and found disturbed sensation in the face in as many as 28.5%. Most patients experience significant recovery of function early, but occasional patients experience progressive improvement as late as 18 months after injury.

Orbital Fractures

Surgical pathology

The position of the orbit at the interface between the cranium above and the midface below, in intimate relation to the thin-walled paranasal sinuses, demands that this region be carefully evaluated in the assessment of facial trauma.

Advances in the management of orbital injuries have resulted from better understanding of the bony and soft tissue anatomy (Pearl 1987), and have improved awareness of how each component relates to the other to produce the final position of the globe in three dimensions. Advances in the imaging of these injuries now permit preoperative planning, appropriate operative exposure, and reconstruction of both skeletal and soft tissues to minimize the major long-term sequelae of enophthalmos and attendant soft-tissue contractures. A high and predictable level of anatomical skeletal restoration by rigidly fixed bone grafts

now appears possible, and the only remaining unpredictable variable for outcome is the periorbital soft tissue and how this changes after the initial injury and the subsequent surgery. Good primary correction is essential; late correction will not reverse the soft tissue disorganisation and muscle shortening resulting from prolonged posterior displacement of the globe.

Simplistically a quadrilateral pyramid composed a seven bones, the orbit has a strong anterior rim or base and four thin walls which converge on a strong bony apex. The thick protective outer rim is formed by the frontal zygomatic and maxillary bones; at the rim, the orbit is ovoid in coronal section, becoming more rectangular as one proceeds posteriorly to the apex, where the cavity is almost triangular (Fig. 2.7). The orbit has four walls— superior (roof), medial, inferior (floor) and lateral—all thin and delicate and each unique in the manner in which it responds to trauma. Lang (1983) quoted measurements of the orbital walls in adults; these are given on p. 37. The portion of the orbit in front of the transverse axis of the globe—the anterior segment—has little effect on bony orbital volume and hence on the projection of the globe of the eye, and relatively more influence on its vertical and transverse ocular position (Fig. 11.97). Alterations to the size and shape of the posterior segment, the portion of the orbital cavity behind the axis of the globe, determine the orbital skeletal volume and therefore the ocular projection.

Disruption of the orbit may result from fracture of the orbital rim and walls by extension of *extrinsic* (= external) fractures, which are classified according to their major anatomical component, or from indirect forces transmitted via the globe and periorbital soft tissues to produce an *intrinsic* blow-out fracture of the floor or medial wall (Table 11.4).

TABLE 11.4

Classification of orbital Fractures

1.	Intrinsic—orbital rim intact	
	(a)	Blow-out
	(b)	Blow-in
2.	Extrinsic—orbital rim fractured	
	(a)	Blow-out
	(b)	Blow-in

The superior orbital rim, formed by the frontal bone, is thick. The orbital roof is arched in both anteroposterior and transverse directions, and relatively resistant to fracturing. 'Blow-in' fractures (inferior displacement) of the roof occur as frequently as blow-out fractures in this area and in most cases are associated with orbital rim disruption. Orbital roof fractures, usually occurring in high energy impact injuries with extension into the face, are more frequently seen in children (p. 511); such fractures often result from direct impacts on the frontal bone (p. 102).

The orbital floor is the classic site of 'blow-out' fractures. The anterior orbital floor medial to the infraorbital canal is concave in an anteroposterior direction. Posterior to the axis of the globe it becomes convex upwards, the underlying maxillary antrum bulging superiorly and obliterating the angle between the floor and the medial wall (Fig. 11.97). Restoration of this orbital floor convexity after injury significantly influences forward positioning of the globe.

The medial walls are approximately vertical and run parallel with each other. The most inferior portion diverges laterally and runs into the floor. The lacrimal bone and the lamina papyracea (orbital plate) of the ethmoid comprise the thin, vulnerable portion of the medial wall. Supported by the bony septae of the ethmoidal air sinuses, the medial wall, although often thinner than the floor, does not fracture as frequently as the orbital floor in pure 'blow-out' injuries. Isolated medial wall defects in the middle third of the orbit produce increases in orbital volume posterior to the ocular axis and may cause enophthalmos. Medial rectus entrapment in such injuries is possible but infrequent. At the anterior extent of the medial wall lie the lacrimal drainage system and medial canthal ligament; injuries of these structures are most frequently seen in association with naso-orbito-ethmoid and complex midfacial injuries.

The lateral orbital wall (greater wing of the spheroid and orbital process of the zygoma) diverges from the medial wall at 45° (Fig. 11.96), and lies entirely posterior to the transverse ocular axis. Infrequently fractured in isolation, a fracture in this site is an almost invariable accompaniment of zygomatic fractures. Any disturbance in orientation or positioning of the lateral wall, lying as it does posterior to the globe axis, produces orbital volume change and alteration in globe projection (Fig. 11.99). Muscle incarceration in these injuries is rare, but muscle contusion may occur.

The posterior third of the orbit and orbital apex is composed of thicker, more resilient bone, surrounding the inferior and superior orbital fissure and optic foramen. Comminution of the thinner, more anteriorly placed orbital bones minimizes transmission of displacement forces to the posterior orbit.

Extraocular muscles in the anterior half of the orbit are separated from the bony walls by small fatty cushions, in contrast to the posterior orbit where they are close to the walls and more vulnerable to injury from the fracture or from the trauma of surgical dissection. The orbital fat of the anterior orbit is extraconal. Of special interest is a sausage-shaped orbital fat compartment which separates the levator palpebrae superioris, superior rectus and superior oblique muscles from the orbital roof. Where dystopia of the globe occurs with orbital fracture this superior fat pad prolapses away from the upper eyelid producing 'supratarsal hollowing'—corrected by repositioning and elevation of the globe behind the equator. In the posterior orbit most fat is intraconal and it is the volume of this fat that is the most important determinant of globe position. Most authors believe that loss of extraconal fat produces no significant change in globe position, but fat displacement from the intraconal to the extraconal compartment in the posterior orbit reduces globe support, with possible resultant enophthalmos (Manson et al 1985a).

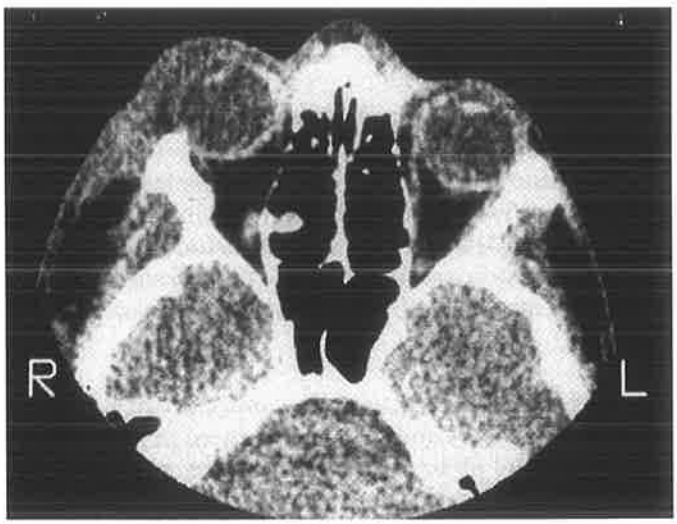

FIG. 11.99. ***Radiology of blow-in fractures.*** *Axial 2D CT scan of lateral orbital wall 'blow-in' fracture (impure) with resultant exophthalmos.*

Classification and mechanisms

Fractures of the orbit may be simply grouped as those which are intrinsic to the orbital walls and those fractures of the orbital walls which occur in association with extrinsic (extraorbital) fractures involving the frontal, zygomatic, midfacial or naso-ethmoid regions (Table 11.4). Intrinsic fractures of the orbital walls are categorised as blowout and blow-in fractures. The pure blow-out or blow-in fracture is distinguished by an intact orbital rim, while what are often inaccurately referred to as 'impure' blowout or blow-in fractures are characterised by extension of an orbital wall fracture to involve the orbital rim.

The application of sudden blunt trauma to the periorbital soft tissues is transmitted as a hydraulic pressure wave to the orbital walls, resulting in a characteristic pattern of fracturing of these walls. Because of its thin, fragile and comparatively unsupported position the orbital floor gives way medial to the infraorbital nerve and medial orbital wall. The sequelae of this disturbance include enophthalmos, muscle entrapment and diplopia: these are direct consequences of the suddenly increased orbital volume and the displacement of the globe and its related muscles and periorbital fibrous septae. Pure blow-out fractures usually result from a low energy impact: the typical cause is a ball striking the orbit from in front (Fig. 4.5). Controversies on the mechanisms producing blow-out fractures are discussed in Chapter 4 (p. 104). The blow-in fracture, described more recently (Antonyshyn et al 1989), and much rarer, is characterised by inward displacement of bony fragments of the walls and/or rim (pure and impure) with resultant decreased orbital volume and increased globe projection (Fig. 11.99).

Low velocity impacts from assaults or falls most commonly produce simple, isolated linear, or 'blow-out' type fractures, with minimal comminution involving one or two orbital walls. Medium velocity impacts produce similar injuries, but usually with associated orbital rim disturbance and involvement of at least two orbital walls. The extrinsic fractures which extend to involve the orbital walls invariably result from medium and high energy impacts.

High velocity impacts result in complex orbital fractures, involving three or four walls, with associated panfacial fractures and increased incidence of orbital soft tissue and globe injuries; such fractures exhibit much les well defined patterns of fracturing, and more comminution and bone loss.

Clinical assessment

Clinical examination of the orbit in trauma is often of limited value, but a history of the velocity of impact, an the presence or absence of associated craniofacial and. multisystem injuries will alert the observer to the likelihood of simple or complex orbital injuries. Periorbital; bruising and swelling is inevitable, and in major injuries is at first so severe as to obscure the globe (Fig. 11.100). Eversion of the eyelids with Desmarre retractors in these severe cases is mandatory and should be done early to avoid missing primary globe injuries, which occur with increased frequency as the impact energy rises.

Visual disturbance, blurring or diplopia may be complained of, and these symptoms require full ophthalmic logical assessment. Holt & Holt (1983) reported on a large series of patients with facial trauma and identified ocular and/or periorbital soft tissue injury in 65%; 18% had severe injuries and 3% blinding injuries. Our experience suggests a much lower incidence (4.9%) of significant ocular injuries, predominantly in association with pure orbital, zygomatic or mid- or pan facial fractures (p. 397). Altered sensation and numbness in the distribution of the infraorbital nerve and the anterior and middle superior alveolar nerves are frequent accompaniments of orbital floor fractures (Fig. 2.29).

Clinical examination follows the sequence detailed for zygomatic fractures. Assessment of alterations in the anteroposterior projection of the globe is

frequently difficult during the acute phase when swelling is marked. The enophthalmos associated with a significant orbital wall injury may only become overt after the oedema has subsided (Fig. 11.101). Early marked exophthalmos suggests a significant reduction in bony orbital volume and requires urgent and complete ophthalmological and radiological examination to exclude an orbital injury which may be directly injurious to the globe or contents of the orbital apex. The ophthalmological assessment of the globe and the ocular motility is especially important; Antonyshyn et al (19891 resorted a 12% incidence of globe rupture and 10% incidence of superior orbital fissure syndrome in patients with blow-in fractures.

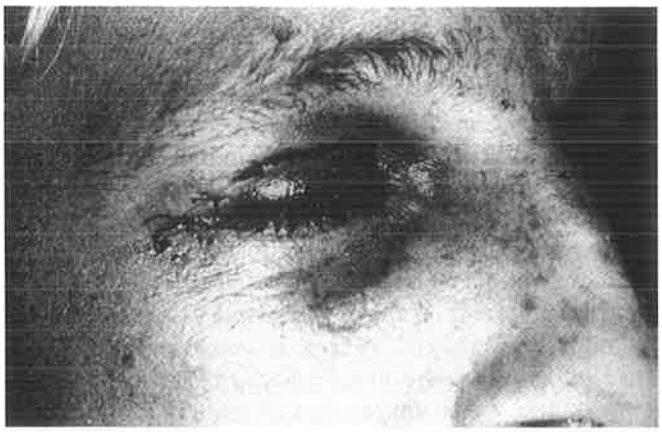

FIG. 11.100. ***Clinical signs of orbital blow-out fractures.*** *Right orbital blow-out fracture following blunt trauma. Moderate periorbital bruising closes the eye acutely.*

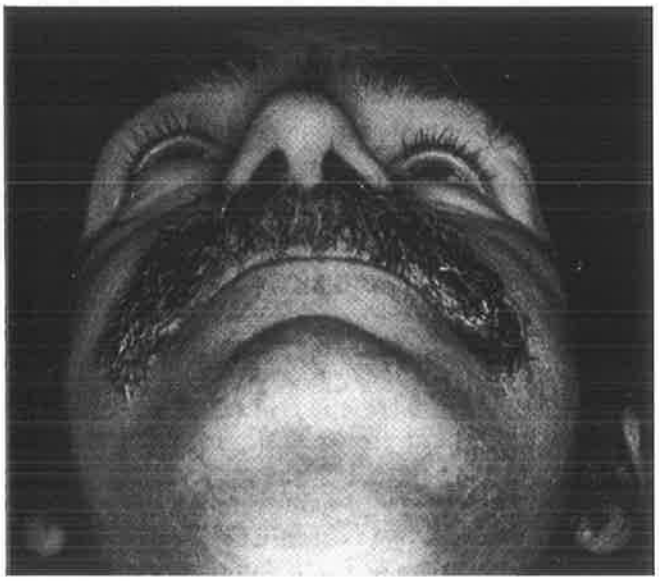

FIG. 11.101. ***Clinical signs of orbital blow-out fractures.*** *Worm's-eye view identifies significant enophthalmos in blow-out fracture after resolution of oedema.*

Radiological assessment

CT examination is now the diagnostic investigation of choice in orbital trauma. Plain radiographic studies are often employed as screening tests, but CT scan assessment is mandatory for accurate diagnosis and in planning appropriate reconstruction (Fig. 11.102).

High resolution axial CT scan examination with films of both bone and soft-tissue windows show damage to the medial and lateral walls and extension of fractures into the cranial base, zygoma and midface. Direct coronal views, or those produced by reformatting the data obtained from the axial scans, visualize the status of the orbital roof, medial wall and orbital floor (Fig. 11.103). When a

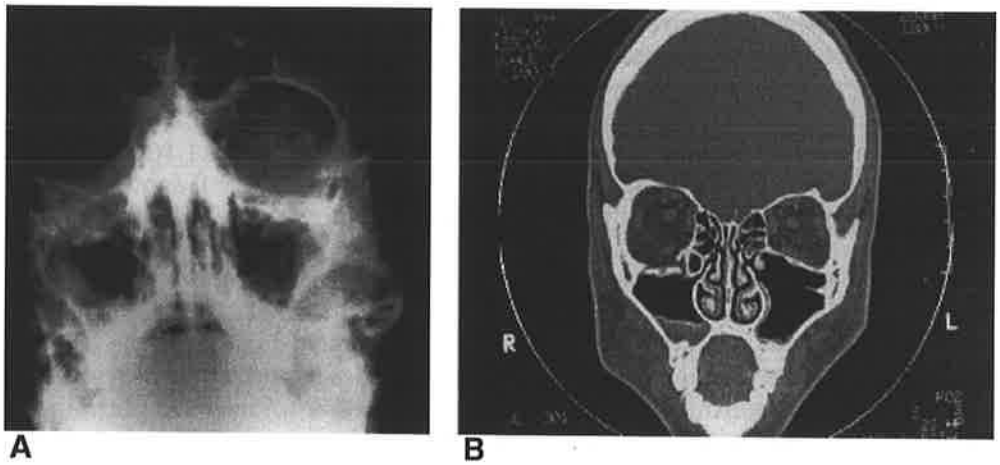

FIG. 11.102. ***Radiology of orbital blow-out fractures.*** **A.** *Plain radiograph shows 'tear-drop' deformity of right orbital floor, with inferiorly displaced orbital floor bone fragments.* **B.** *Coronal 2D CT scan details the isolated right orbital floor fracture with no involvement of the medial orbital wall.*

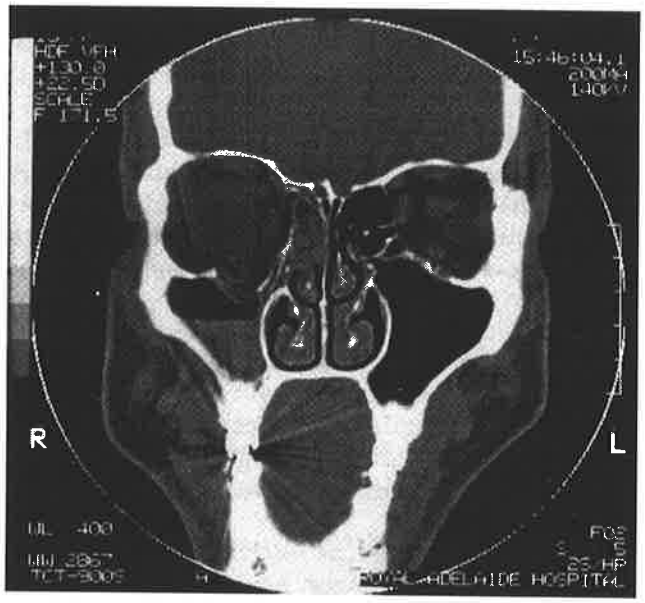

FIG. 11.103. ***Radiology of orbital blow-out fractures.*** *Coronal 2D CT scan pictures the trap-door like depression of the right orbital floor with concomitant blow-out of the medial orbital wall. Note the transverse narrowing of the ethmoid air cells on the right.*

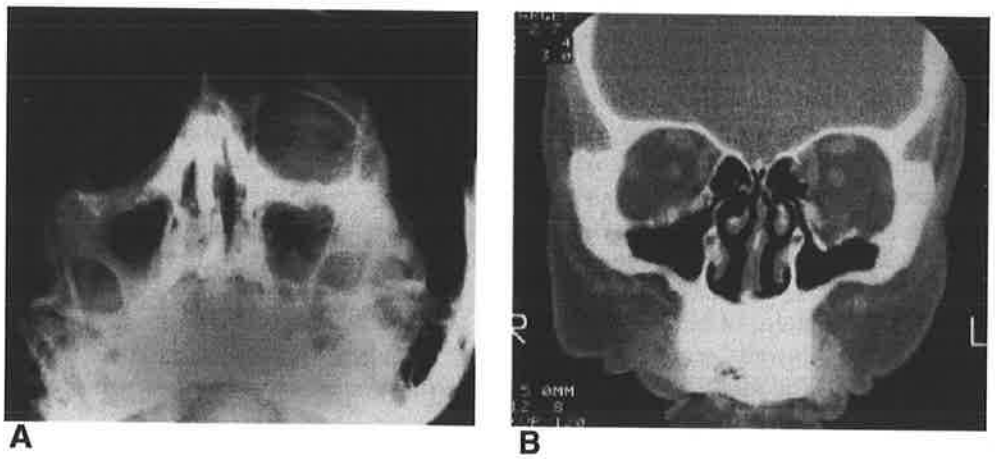

FIG. 11.104. ***Radiology of orbital blow-out fractures.*** **A.** *Plain radiograph identifies relatively minor irregularity and depression of left orbital floor bony fragments.* **B.** *Coronal 2D CT scan denotes irregularity of the left orbital floor and apparent inferior displacement of the inferior rectus muscle into the region of the fracture.*

pure blow-out fracture is suspected, direct coronal scans of the orbit will accurately identify both the extent of the floor and medial wall involvement and the nature of the soft tissue abnormality (p. 193). Fatty prolapse into the area of the blowout fracture without muscle entrapment is identified by the presence of the muscles in the orbit above the orbital wall fracture (Fig. 11.104). Muscle displacement into the sinus or muscle impinging on the bony fragments may mean muscle entrapment. Submucosal haematomas or mucosal polyps of the maxillary sinus roof, which on conventional radiology may resemble herniated soft tissues, can be differentiated on coronal CT scan. Sagittal oblique reformats of CT image data in the line of the inferior rectus muscle will similarly relate this muscle and the adjacent orbital soft tissues to bony damage to the orbital floor. Comparison with the contralateral orbit provides a qualitative assessment of the degree of enophthalmos; quantification of post-traumatic enophthalmos and the changes produced by surgical correction is also possible (Marsh & Gado 1983).

Management

Closed methods of managing fractures which involve the orbital walls risk non-anatomical reconstruction of the bony orbit, post-traumatic enophthalmos, ocular dystopia and motility disturbances. Some authors have advocated conservative management of blow-out fractures on the basis that the soft-tissue ligamentous supports of the globe remain undamaged and able to maintain eye position (Korneef 1982). These views have been largely rejected because of the incidence of unacceptable late post-traumatic enophthalmos, which is very difficult to treat. Non-operative management is considered only in those cases where bony displacement is negligible and there are no other clinical signs of note: there is no evidence to support infraorbital nerve numbness alone as an indicator for operative intervention.

Management now aims at accurate restoration of the orbital rim and walls, thus minimising the troublesome secondary soft tissue disturbances. The orbital rim is reduced and fixed internally with a combination of miniplates and microplates, while the orbital walls are reconstructed with primary bone autografts if comminution or bone loss is extensive (Figs 11.93 and 11.95).

Operative repair is performed as early as possible to minimize the development of soft tissue fibrotic changes. Acute periorbital swelling prevents exploration and repair within the first 24–48 h except in such exceptional circumstances as the presence of a bony fragment impinging on the globe or optic nerve in a 'blow-in' fracture. This demands immediate exploration.

The exposure, exploration, reduction and fixation of the orbital rim are described in the section on orbitozgomatic fractures (p. 320).

Reconstruction of the orbital cavity, together with orbital rim restoration, returns the globe to its pre-injury position with full maintenance of ocular motility. Subperiosteal dissection of the orbital walls delineates the site and pattern of the fracture, identifying the areas of bony deficiency and the surrounding stable, intact bony margins. All the herniated soft tissues are retrieved from the bony defect, minimising fibrosis or loss of soft tissues.

In small isolated blow-out fractures, the displaced bony fragment may be elevated and allowed to override, producing a stable restoration of the orbital wall anatomy. For most defects this is inadequate, and correction requires the introduction of an orbital wall substitute.

The choice of materials for orbital reconstruction lies between autogenous bone or cartilage grafts and alloplastic implants, either resorbable or non-resorbable. Supposedly resorbable alloplastic materials such as Gelfilm® and Vicryl® mesh have little strength and are of use only where orbital wall

defects are minimal, or where the surgeon feels that a small smooth space filler will improve a localized contour irregularity in the restored orbital cavity. Being resorbable, they are well tolerated and are not associated with late infective or extrusion problems. Various non-resorbable alloplastic implants have been employed for small and large orbital defects (Maas et I 1990). The easy availability of compounds such as Silastic® sheeting, Marlex® mesh, Prolene®, Teflon® and titanium or Vitallium® mesh, and the wish to avoid secondary donor site problems, have continued to promote the widespread use of these compounds in the orbit despite significant graft extrusion rates. As with similar grafts utilised elsewhere in the body the orbital tissues respond to the foreign body with the production of a fibrous avascular capsule. Situated in close proximity to the paranasal sinuses and often immediately subcutaneous, alloplastic implants remain at long-term risk of infection and extrusion. The biocompatibility of titanium has encouraged its use as a mesh to restore orbital cavity contours, especially in complex orbital and extensive panfacial fractures where all normal orbital supporting structures are lost. Able to be bent and shaped very accurately, titanium mesh is best utilized as a framework on which autogenous bone may be placed. Some of the bone will resorb, but experimental studies by Sullivan et al (1993) suggest that enough remains to constitute a smooth orbital floor. The use of mesh in isolation allows extensive soft-tissue ingrowth, making removal of the mesh at a later date for infective or other complications a possibly insurmountable technical challenge. In addition the porosity of the mesh does not prevent soft-tissue loss from the orbit and hence the development of enophthalmos. We believe that autogenous bone is usually the best material for orbital repair, provided that the grafts are properly shaped, placed anatomically, rigidly fixed, and with substantial volumetric overcorrection to allow for future resorption. Autogenous grafts have indeed been utilized in this way for many years, being favoured because of the lack of infective and extrusion problems. Nevertheless, autogenous grafts are attended by variable degrees of disadvantage relating to donor site morbidity and graft resorption. These relate to the choice of bone or cartilage, and to the site from which the graft is obtained.

Calvarial bone enjoys widespread popularity for use as onlay and inlay grafts in the craniofacial region. The need for a further incision may be avoided if bone is harvested from the inner table when a formal craniotomy has been performed, or split from the outer table of the temporoparietal skull when a coronal flap is used; with a sharp osteotome, calvarial bone can be cut with preservation of the overlying pericranium. The brittle nature of calvarial bone makes contouring of the graft to the appropriate orbital wall shape rather difficult. Calvarial bone appears most useful where it can accurately reconstruct a single orbital wall, or as an autogenous onlay overlying titanium mesh restoring two or more orbital walls.

Split-rib graft is available in large quantities and is most readily contoured to the correct curvature of the orbital walls. It is the graft material of choice in those cases where several orbital walls require reconstruction. Resorption of onlay rib or iliac grafts is said to be higher than when calvarial bone is employed (Zins & Whitaker 1983). Stacking bone graft from anterior to posterior, making use of the stable posterior ledge, gives the correct convex upslope of the orbital floor and minimizes the risk of displacement of the graft. Rigid internal fixation by lag screws or contoured microplates or miniplates improves graft retention and gives ideal orbital wall reconstruction. These additional techniques are seldom necessary in the isolated blow-out fracture, but increase the predictability of a good outcome in complex injuries.

Autogenous cartilage graft, either costal or auricular, can be valuable in correcting small orbital wall defects. While not prone to significant resorption, cartilage grafts have a tendency to warp; if placed in the orbit, difficulties with fixation may lead to displacement of the cartilage into the adjacent maxillary sinus. Few studies have detailed significant experience with the use of cartilage graft in the orbit.

In uncomplicated blow-out fractures, after anatomical correction of the orbital bony anatomy with or without a graft, the orbital soft tissues are simply allowed to redrape. A forced duction test is performed by grasping the rectus muscle insertion with forceps to ensure that all muscle entrapment has been relieved. The lower lid incision or conjunctival incision is then closed in a single layer. In complex orbital injuries where the exposure requires extensive subperiosteal dissection of the facial soft tissues, the final act in operative treatment is careful fixation of the periorbital soft tissues.

Complications

Complications of orbital fractures are still seen despite open reduction and primary bone grafting. Residual problems may result from the initial injurious process, or from failure to correct the disordered anatomy, or from changes induced by the operative exposure (Wolfe 1988). Common complications include:

- Ocular problems
- Enophthalmos
- Ocular dystopia
- Canthal dystopia.

Ocular problems
These relate primarily to the initial injury and are not affected by the bony reconstruction. Persistent diplopia in the initial postoperative period is not infrequent but is seldom noted on long-term follow-up. Some patients do continue to have subjective complaints of double vision on extremes of gaze. It is sometimes suggested that these complaints are not supported by objective evidence of ocular disturbance; nevertheless a careful test of ocular motility will usually show muscle weakness or restriction of gaze and some of these patients need ocular muscle surgery (p. 420).

Where significant bony displacement occurs in the region of the orbital apex and superior fissure with associated optic nerve injury and/or ophthalmoplegia, the outcome after bony reduction is often unhappy. In most cases of blindness following high energy impacts the optic nerve is irreversibly damaged in the optic canal (p. 422). By contrast Antonyshyn et al (1989) were able to show a significant improvement after immediate orbital decompression in orbital blow-in fractures complicated by ophthalmoplegia.

Enophthalmos
The commonest complication remains persistent enophthalmos. This relates principally to inadequate exposure of the extent of the bony defect, and particularly the medial orbital wall extension. Inadequate or inaccurate orbital wall reconstructions by graft, or graft displacement and resorption, contribute significantly to this problem. In complex orbital injuries a degree of bony overcorrection is needed to achieve an acceptable long-term outcome with respect to globe position; the unpredictable factors of oedema, fibrosis and fat loss make it impossible to get a perfect result.

Ocular dystopia
Transverse and vertical dystopia of the globe may follow, inaccurate reduction of the zygoma, and hence the portion of the orbital walls anterior to the axis of the globe, or from incorrect graft placement in the same region.

Canthal dystopia
The main complications relating to the surgical exposure and subperiosteal dissection of the orbit are degrees of medial and lateral canthal dystopia. Careful maintenance of the bony attachment of the medial canthus in isolated orbital injuries should prevent the development of telecanthus or medial canthal dystopia. Medial canthal abnormalities are more frequent and are difficult to correct where

bony orbital injury is accompanied by local soft-tissue lacerations in this region. In addition to telecanthus an inappropriately anterior positioning of the medial canthal ligament is a frequent complication. Lateral canthal dystopia, with poor apposition of the lower lid to the globe, is a common finding soon after a lateral canthotomy performed to expose the orbit but settles with time. Careful attention to wound closure and reattachment of the lateral canthus will prevent the development of this eyelid abnormality.

Fractures Primarily Affecting the Nasal Airway

Nasal Fractures

Surgical pathology

Whether alone or in combination with more complex injuries, fractures of the nose are the commonest facial fracture in most published series. The prominent position of the nose makes it often injured in isolation when struck by a low energy impact (Fig. 11.105); the nasal bones are the most fragile element in the facial skeleton, and break under forces well below those needed to fracture the other components of the middle third of the face (p. 103). With high energy impacts, nasal fractures are often seen as one element of a more complex facial injury. The exposed position of the nose makes it liable to injury in assaults, accidents and falls. An understanding of the mechanisms and patterns of injury and their variation according to the force and direction of the blow is of importance both for individual patient management and prognosis and also in the prediction of outcome and resolution of litigation arising from assault.

While the visible injury of the nose demands immediate attention, most nasal fractures have posterior extensions which produce bending or displacement of the bony and cartilaginous nasal septum. The management of these its a great long-term challenge.

The patterns of nasal injury relate to the complex anatomy of the nose (p. 37) and to the velocity, mass, direction and site of impact. High energy impacts, by objects with high velocity and/or large mass, cause increased comminution, loss of nasal dorsal support and likely extension into adjacent facial regions; the nasal bones and anterior nasal spine are likely to collapse under an impact force able to create a Le Fort type of fracture (p. 103). The spread of bony-cartilaginous disruption widens as the effective force of impact increases (Pollock 1992). Direct frontal impact usually displaces and comminutes both the nasal skeleton and septum. Laterally directed forces produce deformation ranging from minor unilateral medial displacement of a single nasal bone through to significant lateral displacement of the entire nasal complex; with the latter, septal involvement may be complex in several dimensions. Inferior impacts damage initially the anterior nasal spine with extension more posteriorly to involve the septum and lateral nasal structures, injuring both soft tissues and bony elements.

Stranc and Robertson (1979) analysed nasal fractures according to lateral and posterior displacement and by the unilateral or bilateral extent of the injury. They noted that lateral force fractures were most common, and because there is seldom serious disruption between the bony and cartilaginous elements, the aesthetic and functional result after reduction is usually good. By contrast injury from a frontal impact is less common and less well defined and the results are less often acceptable. Generally the degree of posterior displacement correlates closely with the amount of comminution of the nasal bony-cartilaginous complex.

As with other regions of the facial skeleton, identical fracture patterns are seldom seen in the nose, yet a degree of predictability is evident. The cartilaginous elements are the most forward-projecting portions of the nose and are thus the first components to deform on impact. The dissipation of energy then proceeds

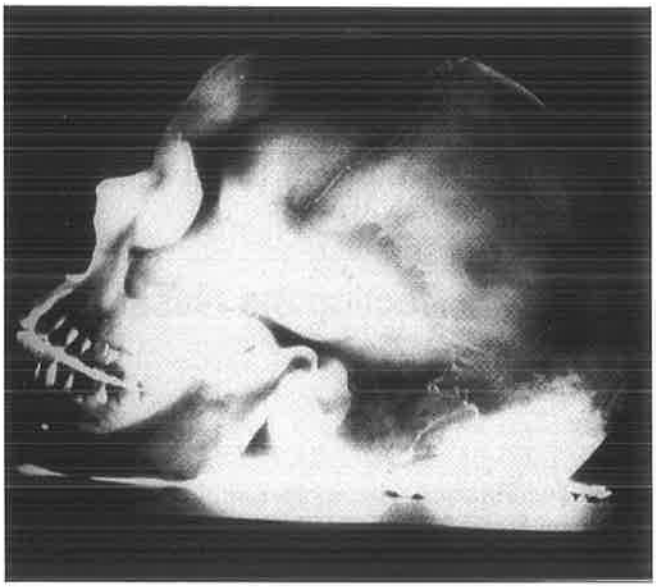

FIG. 11.105. ***Nasal skeleton.*** *Lateral profile of adult skull confirms the prominent position of the nose, exposed to frontal and oblique lateral impacts.*

through the next most vulnerable structures, the nasal bones and septum, and so on into the skeletal base until dissipated. The fractures due to high energy impacts are invariably bilateral with extensions proximally toward the glabella, inferiorly to the piriform margin, with involvement of the supporting facial skeleton.

Clinical assessment

The history of injury usually identifies the probable energy of impact and the likely extent of injury. Details of the nature of the traumatic force, and its direction, will aid in predicting fracture patterns. A record of pre-existing nasal deformity and previous operations on the nose should be sought.

Examination early after injury is ideal, but is seldom possible before the onset of oedema obscures the definition of the nasal bones and cartilages. Local injury to the region of the anterior nasal spine produces swelling and pain in the upper lip with marked tenderness on palpation of the columella. Where the injury is localized to the nasal bones and frontal processes of the maxilla periorbital bruising and paranasal oedema are evident (Fig. 11.106). With further extension of the injury into the naso-ethmoid and midfacial regions the deformity associated

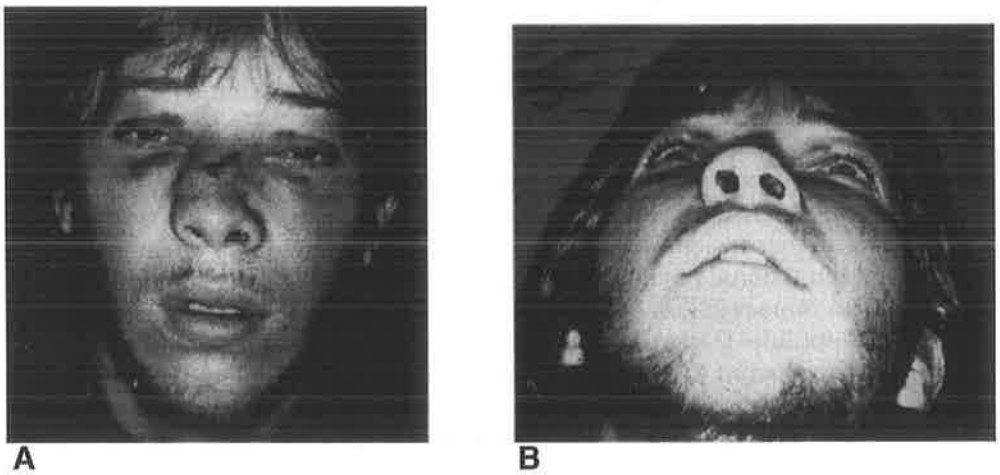

FIG. 11.106. ***Clinical signs of nasal fractures.*** **A.** *and* **B.** *Clinical appearance where gross deviation of the nasal bony skeleton to the right with attendant tilting of the cartilaginous septum.*

with these injuries predominates and the clinical indications of displacement of the nasal bones are correspondingly less evident. In general, the more extensive the injuries, the greater the oedema and bruising.

A systematic examination and palpation of the nose, with attention both to intra- and extranasal structures, is done along routine lines (Fig. 6.5). This must be gentle, and may rarely require local or general anaesthesia, especially in children. Palpation of the nasal bones and frontal processes of the maxilla is performed to elicit mobility, bony deformity and crepitus. Oedema often limits the detailed assessment of the displacement of the thin nasal bones.

Intranasal examination allows assessment of nasal obstruction from blood clot, or bleeding from mucosal lacerations which may require control to allow complete evaluation. Displacement of the septal cartilage, bony deviation, mucosal tears and septal haematoma are all assessable by intranasal examination (Fig. 11.107).

Reassessment may be indicated when the initial examination occurs at a time of maximal swelling, in order to avoid missing a fracture. The final and often most effective clinical evaluation for surgical planning may be performed intraoperatively. A periosteal elevator placed high in the nasal vault will help in bimanual palpation of individual nasal bones and the nasal bone complex as a whole. Intraoperative pressure over the nasal dorsum is helpful in assessing collapse of the underlying bony-cartilaginous structure (Gruss 1982). Although this procedure was originally used in the assessment of naso-orbito-ethmoid fractures, Pollock (1992) has utilized it selectively at different levels of the nose to identify collapse of the upper, middle or lower vault. When applied to the upper nose in complex injuries, the nasal bridge is found to disappear back between the eyes.

Radiological assessment

Routine radiographical examination of the facial bones may not always reveal the nasal fracture. A lateral view coned on the nose and occipitomental views with additional backward projection may improve the visualisation of bony injury. These views are frequently requested in the casualty department, particularly if the mechanism is an assault and litigation is likely; however, the radiographs seldom influence the decision on operative repair. Coronal or axial CT scans, with the addition of 3D CT reconstruction, will elegantly visualise a nasal fracture superimposed on a major midfacial or naso-orbito-ethmoid fracture (p. 198), but CT investigation is certainly not indicated for isolated simple nasal fractures.

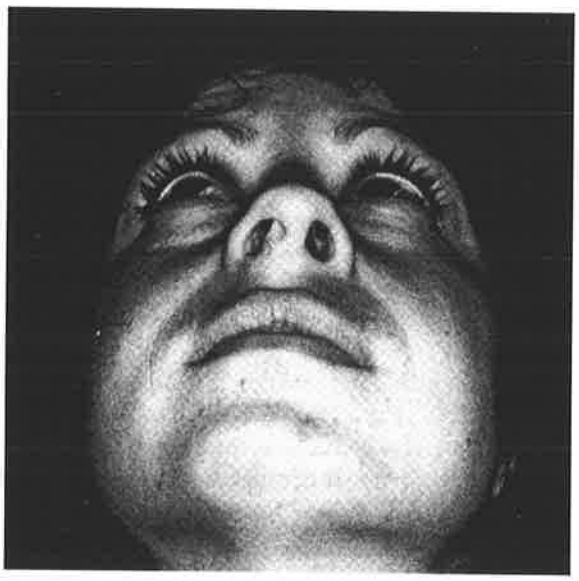

FIG. 11.107. ***Nasal septal injury.*** *Worm's-eye view identifies characteristic caudal septal dislocation.*

Management

Treatment aims at restoration of appearance and restablishment of nasal airway function bilaterally. As a rule, the nasal fracture must be reduced. Convention has it that reduction is ideally undertaken either immediately following injury, prior to the development of oedema, or up to 14 days later when the oedema has subsided. Most cases fall within the latter category, whether simple, or complex. Where there are other extensive facial fractures reduction may be further delayed while the patient is resuscitated and appropriately investigated. In fact, the timing of operative treatment must be individualised. While immediate centralisation of the nasal pyramid in a laterally deviated nose is possible, an inadequately treated nasal septum or inaccurately reduced nasal bones will necessitate more complex secondary surgery. Most nasal fractures are reduced within the first 7–10 days although it is possible to accomplish an appropriate reduction after a delay of 3–4 weeks

Because simple techniques of reduction will not suffice in all cases, nasal fractures should as a rule be treated in an operating theatre, so that more complex forms of treatment can be undertaken at once if necessary.

Simple closed reduction is the standard treatment for nasal injuries due to low and middle levels of impact energy. In the acute period, the application of local thumb pressure may successfully reduce the displaced nasal complex to its normal position, sometimes without the need for an anaesthetic. Delayed reduction may be possible in the same fashion, using local anaesthetic and sedation or general anaesthesia. General anaesthesia permits detailed examination, and is particularly useful in the younger patient where cooperation might otherwise be difficult. Intranasal packing and external tapes and splints minimize subsequent swelling and bleeding, and may aid in positioning unstable fragments.

In middle level energy impacts where displacement of the nasal complex may be both posterior and lateral, closed reduction is similarly employed, but requires the use of elevating instruments. Upward and outward manipulation of the nasal bone fragments with a Howarth elevator or similar blunt edged tool permits realignment whilst minimizing damage to the nasal lining. Nasal compression forceps (e.g. Walsham's forceps) are widely employed but may cause additional soft-tissue crush injury. Sharper instruments such as a large curved haemostat have been used, but risk tearing the nasal mucosa.

Treatment of low and middle energy nasal injuries by simple closed reduction has been generally accepted, despite studies which indicate that this method has potential for a poor outcome. Mayell (1973) in a retrospective report showed that only 30% of cases had restoration of normal appearance, though 50% had normal nasal airway function. Similarly Harrison (1979) identified only 33% of his patients as having both adequate airway and ideal cosmesis.

Closed reduction is ideal for the unilateral, depressed nasal bone fracture where septal injury is absent; inadequately treated septal displacement is the chief cause of the poor results of closed reduction. In the middle energy impacts the nasal complex displacement is both posterior and lateral with resultant increased septal injury, and more elaborate procedures may be needed.

Nevertheless, nasal septal dislocation is routinely managed in the first instance by closed reduction (Fig. 11.107). As the septal position will often influence nasal bone position, the reduction must be rechecked and occasionally repositioned. Asch septal forceps will correct both superior and inferior septal deviations. The superior portion of the cartilaginous septum, being contiguous with the bony septum, is reduced first before dealing with the inferior septum. Adequate reduction of the superior portion can usually be achieved; inferior septal deviations are not so often successfully reduced with closed techniques.

Where closed reduction has failed, the choice lies between acute open reduction or a late secondary procedure. Early open reduction is difficult because of the soft-tissue swelling and the increased risk of lacerations when elevating the mucoperichondrium of the septum. Harrison (1979) suggested early limited submucosal resection of the bony-cartilaginous junction for those cases where septal deviation is marked: after lower horizontal and posterior vertical submucous septal resection, the cosmetic and functional results were improved. There has been only limited acceptance of this management, because it could result in mucosal injury and septal perforation. Pollock (1992) has advocated early cartilage repositioning and fixation where significant septal deviation adversely affects nasal bone positioning, using techniques of cartilage preservation. This has not been our policy: rather, we have advocated secondary correction.

Lacerations of the overlying skin are frequent in cases of nasal fracture, and may permit open reduction and fixation with interosseous wires, miniplates or microplates. When bicoronal and upper vestibular incisions are used for exposure of associated complex fractures, ideal exposure of the nasal region is given.

Complex nasal fractures demand careful reconstruction proceeding from a stable proximal base. The nasal bony anatomy must be restored and the bones must be reattached at the frontal articulation to recreate the nasofrontal angle. Interosseous wires, micro- and miniplates and screws will provide anatomical reconstruction where the bony fragments are sufficiently large. The middle and lower thirds of the nose are then reconstituted using the techniques of closed reduction and limited open septal reduction described above. In all cases nasal packing is employed for 1-2 days in concert with a moulded nasal splint or plaster for the first 5-7 days. Where bony comminution is extreme and proximal skeletal support inadequate this approach is insufficient to expand the nasal soft-tissue envelope. If uncorrected, severe comminution may result in a variable combination of saddle deformity, loss of nasal tip projection and nasal foreshortening in the vertical dimension. There is a need for bone or cartilage grafts to augment the skeleton and to splint the nasal soft tissue.

Cantilever bone grafts overlying the existing nasal bone and interposed into the nasal tip will satisfy this reconstructive need. Introduced via a bicoronal approach if this is already available, or through a local intranasal incision or an external rhinoplasty approach, the graft is fixed to the stable proximal elements of the nasal bones with miniscrews or by a contoured miniplate anchored to the central forehead. In the less complex fractures where no bicoronal incision has been used, the anchoring screws can be inserted through a small skin incision in the glabella. Attention to nasal tip aesthetics is essential when using these grafts to ensure accurate positioning and attachment of the alar cartilages, and to minimize the need for secondary revision. Underlay grafts have been described as a variation with miniplate fixation of the graft below at the anterior nasal spine. No significant advantage over onlay cantilever grafting has been noted, especially in operations performed soon after injury. Cartilage grafts may be of value in the middle section of the nasal dorsum overlying the upper lateral cartilages where isolated collapse in this region is noted; they are usually inadequate to maintain forward nasal projection in severe complex injuries.

Complications

These may be broadly classified as either aesthetic or functional, interplay between both being common.

Facial aesthetics

In terms of facial aesthetics, persistent lateral deviation of the nose, and loss of nasal projection and saddle deformity, are the most significant common residual problems.

In the common low and middle energy nasal fractures persistent lateral deviation and irregularities of the nasal bridgeline, particularly a nasal hump, are the commonest aesthetic problems requiring secondary surgery. Corrective rhinoplasty with hump reduction and infracture together with septoplasty may be required (p. 575), the timing being variable; usually the rhinoplasty is performed some months following the initial injury.

Loss of nasal dorsum projection is managed in the same fashion, with an autograft for augmentation. Contracture of the soft-tissue envelope is the major limitation on what can be achieved to restore the nasal dorsum; as a rule, less projection can be achieved than with primary surgery and attempts at over-projection risk graft exposure at the nasal tip.

Function
Nasal obstructive symptoms from inadequate correction of the nasal septal cartilage represent the chief functional disturbance. Obstructive symptoms are managed by definitive septoplasty in conjunction with any external nasal surgery that may be required; this is done when the natal mucosa is soundly healed from the initial injury.

Multiple and Panfacial Fractures

Clinical significance

Modern retrieval and resuscitation services result in the survival of many very severely injured patients, some of whom present for management of panfacial and craniofacial fractures. These victims of high velocity impact with multi-system injuries which include craniofacial fractures need initial intensive care and treatment of life threatening injuries; they then require acute stable fixation of their cranial and facial fractures to maximize the restoration of form and function.

Each injury is unique, demanding a flexibility of management that is not conveyed in any cookbook recipe approach. The confusion and poor outcome that follow rigid application of such approaches can be overcome by correct understanding of the aims and principles of reconstruction and proper sequencing of repair. In essence repair proceeds from knowledge of the stable, uninjured; bony buttresses, determined preoperatively by detailed radiology and intraoperatively by wide exposure. The aim of treatment is correct restoration of midfacial projection and width in relation to the cranial base above and the mandible below. The twin requirements of form and function can then be realised. Management of these injuries involves the application of all the principles and approaches to individual subregions with a specific focus on timing and sequence of repair.

Surgical pathology

The patterns of fracture in these injuries are not defined by any conventional classification system applicable to individual facial subregions. However, in the majority of cases there are three important anatomical sites of injury, and an understanding of the fracture patterns and exposure of each of these is necessary for effective repair.

1. Cranio-orbital interface. This is the junction between the anterior cranial fossa and frontal sinus above and the orbits and naso-ethmoid region below. Anteriorly the sinus and its nasal drainage conduit (p. 37) demand consideration in the repair process, to minimize the risk of secondary sinusitis, mucocele and other infective problems. More posteriorly, maintenance or restoration of the normal interface between the contents of the anterior cranial fossa and the nasal cavity is essential in avoiding cerebral infection ascending from the nasal air

passages; a dural tear makes cerebral infection a threatening possibility in both the short and the long term (p. 147). At the posterior limits of the craniofacial junction, extension of cranial base fractures through the spheroid bone risks injury to the neurovascular structures at the orbital apex, superior orbital fissure and cavernous sinus. Away from the midline, damage to the orbital roof and lateral orbital walls may affect the projection and/or motility of the eye, or may cause orbital pulsation. Injuries in this area are often complicated by frontal lobe damage.

2. Zygomaticomaxillary junction. The zygomatic arch and the lateral orbital wall most accurately relate the orbit and midface to the cranial base above. Correct stable arch position determines lateral midface projection and midfacial width. Reconstruction of the maxilla in its correct place between the zygomas will complete the midfacial arch and central midfacial projection, with a proper relation to the mandible.

3. Mandibular condoles. These demarcate the posterior lateral limit of the craniofacial interface. The mandibular condyles and the rami establish posterior facial height The condyles are frequently injured in panfacial fractures and demand careful restoration to re-establish the vertical dimension of the face. It is the intact mandibular arch to which the midface must be related at the occlusal level, and the mandibular arch determines facial height and to a lesser extent central facial projection.

Classification

The extent and range of bony injury in high velocity impacts frustrate any attempt to produce a detailed and precise system of classification. However, groupings have been formulated in relation to the direction and point of impact on the craniofacial region, and related to areas of skeletal strength, the facial buttresses, and adjacent zones of relative bony weakness—the sinuses and the nasal, orbital and oral cavities.

Gruss et al (1989) have grouped these injuries into three broad anatomical divisions:

- Central craniofacial fractures
- Lateral craniofacial fractures
- Combined central and lateral fractures

Centrally directed impacts over the frontonasal region cleave the central facial buttresses from the anterior cranial base. Variable extensions into the frontal bone and orbital roof occur around and through the impact-absorbing frontal sinus. In the paediatric group where frontal sinus development is not complete (Fig. 2.10), Impacted oblique linear and segmental fractures of the frontal bone and orbital roof occur more frequently. More extensive and/or high velocity impacts produce vertical extension of the fracture pattern through the midface and central mandible.

Laterally situated impacts damage the frontozygomaticomaxillary (lateral facial) buttress with posterior extension to the greater wing of the spheroid, temporal and parietal bones. Intracranial complications are less common, but extension into the lower face is often seen.

Extremely violent impacts produce gross comminution of both the central and lateral facial elements with frequent sagittal fracturing of both the midface and mandible, mandibular condylar fractures and an increased incidence of intracranial injury. Although collapse of the facial skeleton has protective value (p. 106), the brain may suffer both local and diffuse damage, and the floor of the anterior cranial fossa is often disrupted.

Clinical assessment

This begins with evaluation of airway, breathing, circulation and conscious level. The immediate concern in the management of these severely injured cases is the institution of appropriate life-support measures and the assessment of life-threatening intracranial and extracranial complications.

The hallmark clinical features are deformation of extreme aesthetic and functional significance, and marked skeletal instability. Clinical observation reveals extensive diffuse facial swelling and bruising, often with lacerations over the bony prominences — the forehead, the supraorbital ridges and the root of the nose. Visible bony deformation is often evident with loss of nasal projection and disturbances in midfacial projection, symmetry and height.

Palpation of the facial skeleton may reveal contour irregularities of the orbital margins and widespread bony instability involving both orbital and occlusal skeletal elements (Fig. 6.5). Irregularities of the supraorbital rim may identify cranial extensions of the fracture patterns whilst flattening of the zygomatic region may be accompanied by marked proptosis, raising suspicion of a blow-in displacement of the lateral orbital wall, including the greater wing of the spheroid with involvement of the orbital apex. Compressive force applied to the nasal root in both anteroposterior and transverse dimensions may demonstrate massive comminution and mobility of the naso-ethmoid region on the anterior cranial base.

Intraoral examination identifies bruising, soft-tissue lacerations or even tissue loss, and may show segmental and sagittal maxillary and mandibular fractures often involving dento-alveolar segments. The extreme mobility of a flail midfacial segment is often easily demonstrable. Detection of condylar fractures is especially easy in patients who are already intubated and ventilated when the first detailed clinical examination is carried out. By grasping and moving the mandible in the region of the mandibular angle with a gloved hand while the index finger of the opposite hand palpates the anterior wall of the external auditory canal, it is possible to appreciate crepitus, abnormal mobility and/or displacement of the mandibular condylar process.

In most though not all cases of panfacial fractures, there are impairments of neurological function, and a neurological assessment should be carried out as soon as possible (p. 159), though often this is limited by the need to institute endotracheal intubation and ventilation. It is also desirable to assess visual function as soon as possible and especially to exclude a penetrating injury of the globe; this may be impeded by orbital swelling.

Radiological assessment

Definitive description of the fracture pattern in these cases is possible only with high resolution CT scans. Ideally axial scan data from the vertex to chin point should be provided on bony settings. Soft-tissue window hard copy images should be provided in the cranial slices to identify the nature of any intracranial involvement and to visualize the globes and the optic nerves. The creation of 3D CT images from the axial data provides a qualitative ready reference image pre- and intraoperatively to assist in planning treatment (Broumand et al 1993). However, these images do not provide an accurate guide to fracture patterns and displacement in all areas of the facial skeleton.

Management

The treatment of these complex injuries is the sum of the repair of all the fractures of individual facial and cranial structures. It is desirable to minimize reoperation and prolonged hospitalisation. In the past, difficulties often arose in the timing of facial repair in relation to neurosurgical or less often ophthalmological interventions; the modern craniofacial unit allows interdisciplinary coordination

of effort and the development off coherent, logical sequence of repair which is applicable to all eventualities. In particular, the combined craniofacial and neurosurgical operation permits repair of multiple facial fractures and closure of cranionasal fistulas in a single procedure (David 1984).

Preoperative care

The patient's cardiorespiratory condition must first be fully stabilised. Tracheostomy is no longer required solely for airway management of complex facial fractures, but where prolonged incubation is necessary for coma or other injuries then tracheostomy may be employed (p. 382).

It may be necessary to monitor the intracranial pressure (ICP), since the clinical signs of a fall in the conscious level may be masked by orbital swelling or by the use of artificial ventilation (p. 369). The initial CT scan gives warning of the likelihood of delayed cerebral swelling and other complications, and it is our policy to institute ICP monitoring if there is CT evidence of intracerebral haemorrhage, obliteration of basal cisterns or midline shift. Even if the CT scan is within normal limits, it maybe wise to monitor the ICP, at least during the first 48 h.

Timing of repair

The severely injured patient with complex craniofacial/ panfacial fractures ideally requires definitive correction of the bony injuries within the first 5-7 days. Some have suggested primary fixation within 12-48 h of injury (Gruss 1990), but such a time frame may be incompatible with adequate preoperative radiological, ophthalmological and dental assessment—especially when the patient is comatose or poorly cooperative. This is also the period when soft-tissue swelling peaks and operative exposure and assessments of facial projection and symmetry are at their most difficult. Cerebrospinal rhinorrhoea or intracranial aerocele may provide an additional reason for delay: for reasons set out in Chapter 13, we believe that these signs of a cranionasal fistula do not invariably demand an anterior cranial fossa dural repair, and delay of a few days will often clarify the need for this procedure, besides allowing raised ICP to subside.

Where early acute neurosurgical intervention is necessary for intracranial bleeding or compound depressed calvarial fractures, the collaboration of the craniofacial surgeon is mandatory. If scalp incisions are made and cranial bone flaps elevated, it is important that they be designed so as not to complicate the exposure and stabilisation of cranio-orbital fractures. For example, where fixation of the mobile face below demands an intact fronto-orbital bar above, any frontal bone flap elevated for exposure of the anterior cranial fossa must be positioned so as to maintain these stable superior points. Loss of the fronto-orbital bar complicates superior localisation and predisposes to inaccurate fracture reduction. Where depressed fractures of the temporal region have extensions to involve the lateral orbital wall and greater wing of the spheroid, the opportunity exists to correct and stabilize the orbital component as well as elevating the cranial element of the fracture. Leaving the orbital fracture element till a later definitive facial repair risks displacing the already corrected cranial component and may cause intracranial bleeding or dural injury.

Occasionally, patients with panfacial or complex facial fractures present late, more than 3 weeks after injury, having undergone extensive life-saving procedures for extracranial injuries. In such cases bone union is often well advanced, and soft-tissue contracture may already be evident. Soft-tissue dissection for exposure must then be extensive and the fractures must be disimpacted to permit stable reduction and anatomical fixation; fracture mobilisation will often require formal osteotomy of the fracture lines.

Sequence of repair

Here are several possible approaches to the complex panfacial fracture, relating to the status of the stable boundaries of the injured face—the calvarial vault above and the mandible below.

Where the frontal cranium is intact, or where there is fracture but no loss of bone in the anterior cranial fossa or fronto-orbital bar, or where these structures have already been rigidly reconstituted, the orbito-zygomatic land midfacial regions can be built *from above downward.* Projection and symmetry of the orbitozygomatic facade sets the facial width and lateral facial projection, between which the midfacial arch can be positioned to recreate midline facial projection and pretraumatic occlusion.

There are situations where repair proceeds *from below upward.* When the mandibular arch is disrupted, it should be repaired as the first part of the procedure. If the mandibular condyles are fractured either unilaterally or bilaterally, they require early reduction and internal fixation by miniplate, to re-establish posterior facial height. In the presence of comminuted intracapsular condylar fractures this may not be possible and intermaxillary fixation must be maintained; primary costochondral graft reconstruction of the mandibular condyle can be contemplated (p. 604). Once the mandible is rigidly restored the midface may be disimpacted and placed into the predicted occlusion; intermaxillary fixation is then instituted. Repair then proceeds *from above down* to meet the already fixed maxillomandibular segment.

Sagittal fractures of the midface introduce another dimension in reconstruction. Where the mandibular arch is intact an occlusal reference point exists below to restore lower midfacial width and maxillary dental arch form. The upper midfacial width is set by correct positioning of the zygomas and this determines the transverse dimension of the split midface at its upper border. Fixation of the sagittal fracture may be possible with transversely placed miniplates anteriorly on the maxilla, or may rarely require posterior microplate fixation of the hard palate. Where sagittal midfacial and complex mandibular fractures occur concomitantly, both upper and lower dental arches appear splayed open and there are no immediately available reference points. Gruss et al (1990) stress the importance in such cases of using the zygomatic arches to set the facial width. The maxillary arch width is then set between these boundaries with miniplate fixation of the midface to produce stability both transversely and vertically in relation to the orbitozygomatic region above. The mandibular arch can then be rebuilt on the above structure, producing ideally a stable functional result and (most-importantly) a symmetrical aesthetic result. In these cases, the danger in the reverse approach, proceeding from below upwards, is that reconstruction commences at a distance from the only stable symmetrical element—the cranial base. The inaccuracies inherent in condylar fracture repair and repositioning are great and introduce variables of asymmetry much beyond what can be achieved by starting at the zygomas and zygomatic arches, which are a direct extension of the cranial base.

The only other situation of note where a repair involves rebuilding from *below upward* is in the presence of cranial bone loss and disruption of the floor of the anterior cranial fossa. There the reference points are lost or distorted. To repair the frontal cranial base and then secure on it the midface may risk damaging or displacing the already repaired anterior cranial fossa. The mandible and midface are therefore first repaired and this reconstituted complex is reattached to the cranium above, once its form has been restored and the appropriate barriers between nose and intradural space have been repaired, if this is considered necessary. The indications and technique of transcranial repair of the anterior cranial fossa are detailed in Chapter 13 (p. 376); when this is done as part of a combined procedure, great care is needed to maintain appropriate asepsis. We have not employed subcranial extradural repair of the shattered

anterior fossa when there is likelihood of a cranionasal fistula. If extradural repair can be shown to be reliable, the procedure has attractions both with respect to simplicity and because it need not be contraindicated by the presence of brain swelling (p. 381). At present, we are sceptical of the reliability of this method of closing a cranionasal dural fistula except under very favourable conditions.

At all levels, an acceptable outcome demands repositioning of existing bone, with primary autogenous bone grafting where there is severe bony comminution or bone loss and replacement or expansion of the craniofacial soft tissues to the pretraumatic state.

A predictably good outcome for the skeletal elements in these complex injuries is now achievable. But further attention to the soft tissues is required. The older semi-closed management of these injuries by suspension wiring and external frames was followed by contracture of the soft tissues because of failure to restore normal bony contours, whether orbital with post-traumatic enophthalmos, or paranasal with loss of nasal projection. Modern subperiosteal dissection of the facial soft tissues permits anatomical skeletal repositioning, primary bone graft augmentation and anterior re-expansion of the facial soft tissues to approximately the pre-injury situation. Such exposures, however, risk sagging of the facial soft tissues especially the cheeks, under the influence of gravity, if resuspension of the soft-tissues to the underlying bone has been inadequate. The resulting soft-tissue alterations may produce a perception of asymmetry and a disappointing aesthetic outcome, attributable to unsatisfactory soft-tissue cover and not to skeletal deformity. Reattachment of the periosteum to the zygoma and zygomatic arch has been advocated (Gruss et al 1990) but no long-term results have been reported on maintenance of the soft-tissue position. Atrophy of the temporal soft tissues (the temporalis muscle and the temporal fat pad) may also follow these wide exposures. Secondary correction with bone grafts placed deep to temporalis is then necessary to augment the contour of the region and to restore symmetry.

Complications

The complications attending these injuries are those seen in each individual craniofacial subregion. The potential for asymmetry of appearance and disturbance of function is extreme. The continued evolution of exposure, rigid fixation and primary bone grafting should ensure increasing accuracy of reconstruction and reduction in rates of post-traumatic deformities and other complications in the facial region.

The complications of concurrent transcranial anterior fossa repair include frontal lobe oedema and early or late cerebral infection (p. 380); we believe that the risks of these complications are acceptably low if our criteria for transdural exploration are respected and if operation withheld until cerebral swelling has subsided.

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Massive tissue loss

D. J. David, E. Tan

Introduction

In Chapters 11–15 we presented a plan of multidisciplinary management of the craniomaxillofacial (CMF) injuries commonly seen in peacetime practice, in which tissue loss is not usually a major factor; this chapter considers the early management of injuries in which there is substantial tissue destruction or ablation. In Chapter 21, we consider the correction of the long-term deformities that may result from such injuries.

Even in peacetime, accidents inflicting massive avulsive tissue loss do occur. Such wounds are seen as the result of gunshots fired at close range, or from industrial explosions. Freak road accidents also sometimes result in avulsive injury and, in some parts of the world, bites by large animals may ablate portions of the face (p. 495). In war, avulsive facial injuries are very common and this has been so since firearms first began to dominate the world's battlefields (p. 2). Military missiles frequently avulse large segments of the face or extensive portions of the frontal scalp and calvarial bone. These wounds, if not immediately lethal, cause frightful aesthetic deformities and often severe impairments of speech, vision and other functions; Fig. 1.15 records some typical cases from World War I. In war, the management of such wounds is likely to be complicated by problems in evacuation, assessment and resuscitation, and definitive management is soften delayed.

Our experience of major avulsive CMF trauma (Table 16.1) is based chiefly on peacetime practice, supplemented by study of wounded soldiers referred to us from other centres for secondary repair; in preparing an account of the management of these wounds, we have drawn heavily on the published and unpublished experience of other surgeons working in recent wars and civil conflicts.

TABLE 16.1

Massive avulsive injuries in the CMF area

Cause of avulsive injury	No. of cases
Gunshot wounds	16 (7 wartime)
Vehicular accidents	11
Other	4
Total	31

The figures give the experience of the Australian Craniofacial Unit during the period 1973–1993. Excluded are cases requiring only neurosurgical management and gunshot wounds not causing significant loss of tissue.

Surgical Pathology

The pathology of missile injury expresses the nature and velocity of the missile; the anatomy of the impact is also relevant, impacts on bone being especially injurious. Wounds resulting from gunshot are classed as penetrating, perforating and avulsive or ablative. A penetrating wound is typically inflicted by a low velocity missile which remains embedded in the tissues: little damage is done unless a vital structure is hit. Perforating wounds may be caused by higher velocity missiles; in these, there is often a small entry wound, a larger exit wound, and a core of tissue damage between the wounds. Massive avulsive wounds show loss of anatomical structures, with great variety of tissue damage; the damage may be more widespread than the apparent tissue defect, because seemingly intact structures may have been devitalized by the missile impact or may have suffered traction injury from the avulsion.

Avulsive wounds result from very high velocity impacts by missiles of any size, or from large missiles or groups of missiles at lower velocities, or from tearing forces such as the bite of a large carnivorous animal. In war, fragments from mines, bombs and shells and high velocity rifle bullets are likely to cause avulsive injury; in peacetime, such injuries may result from short range (<6 m) blasts from shotguns or large calibre pistols, or from a variety of rifles used in hunting, civil homicide, or attempted suicide. The ballistics of missile injuries are considered in Chapter 4. Avulsive injuries can also occur from industrial explosions, such as the explosion of a tractor tyre, and from a variety of accidents with machinery. On occasion massive craniofacial injury results from a vehicle accident. Ablative injury may result from tissue destruction by post-traumatic infection; the outcome is often a tissue defect that requires correction by procedures similar to those used in repairing acute avulsive trauma. A severe burn may also constitute an ablative injury.

The permutations of bone and soft-tissue avulsion are infinite, and any part of the CMF area may be damaged as a primary effect of the impact, or as a secondary complication. Soft-tissue injuries caused by missiles are often complicated by the presence of indriven bone and tooth fragments which must be carefully removed; if this is not done, they may be the source of chronic infection and sinus formation.

The eyes and the brain may be injured, either directly or by vascular interference. Extensive damage in the facial region can inflict severe tissue disruption without immediate effect on the central nervous system, yet with delayed cerebral or visual complications of ischaemic type resulting in increased disability (Fig. 16.1).

Wounds of the lower face and mandible

Injuries range from gross destruction of the lower jaw (Fig. 1.15d) to a simple mandibular fracture with soft tissue ablation. In the rifle bullet wound shown in Fig. 16.1, there was gross separation and destruction of the mandibular symphysis, loss of the floor of the mouth and tongue and part of the lower lip; one condylar process was missing, as was the central part of the maxilla. Such injuries are very dangerous: tongue control is lost, and with fading consciousness there may be a lethal airway obstruction. Injuries of the base of the tongue may go on to gross swelling and bleeding and are then an even greater threat to the airway. Loss of the lower lip is a challenging problem because effective reconstruction of the orbicularis oris muscle is a very difficult procedure and repair with distant flaps always gives a second rate functional result.

Missile injuries in the lower face can result in virtually no damage at all, with only a very small entry wound and minimal destruction of the missile tract, with fragments of metal and/or bone dispersed in the tissues or in the palate or

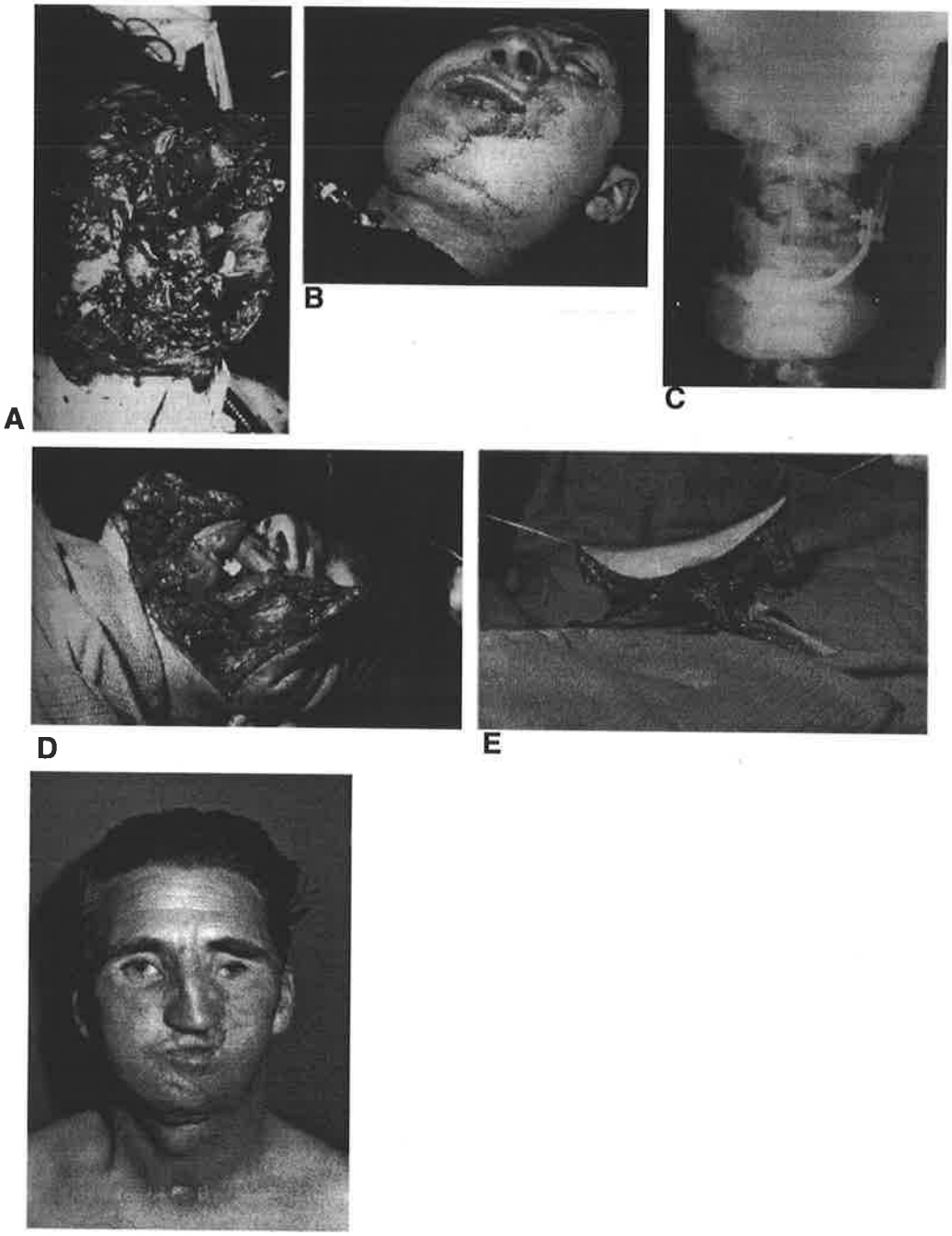

FIG. 16.1. ***Avulsive facial gunshot wound.*** *A massive soft-tissue and bony injury was inflicted in the face by a Lee Enfield .303 in rifle at close range. The patient drove his car to seek help. Some 8 hrs later he became blind, presumably from retinal arterial spasm. He was never unconscious. He underwent intubation, tracheostomy, debridement and primary closure of the wound. Subsequently the bone fragments were stabilized with a metal spacer; later, microvascular reconstruction of the mandible was done with other corrective procedures.* **A.** *Initial presentation, with massive tissue loss and bony destruction. Despite the forbidding appearance, he was easily intubated because the tissues could be peeled apart and the laryngeal inlet observed.* **B.** *Semi-elective tracheostomy and repair of the soft tissues were performed. More debridement was necessary to remove damaged muscle and pieces of bone and tooth which had been driven into the wound.* **C.** *The lower jaw fragments were separated by a Bowerman-type prosthesis. At the time of the stabilisation of his jaw, the zygomas were reduced and wired into position, establishing the width of the upper pan of the middle third of his face.* **D.** *2 months later, the definitive repair was performed. A composite osteocutaneous free groin flap was raised on the deep circumflex iliac vessels with a skin paddle which was placed in the floor of the mouth coming through the lip and onto the chin. The defect is shown recreated with the bone ends separated and the metal prosthesis removed in readiness for insertion of the graft.* **E.** *The osteocutaneous composite graft on its vascular pedicle.* **F.** *The patient remained blind and had a continuing problem related to the soft-tissue deficit in the orbicularis oris. Since that time he has had a number of operations to sculpture the bone and redrape the soft tissue.*

adjacent to the base of skull. More frequently, however, we are confronted with some damage to the mandible and lower teeth (Figs 16.1 and 21.73) Banks et al (1985) note that bullets striking the mandible may cause fractures of teeth below the gingival margin at a distance from the impact site, presumably by a shock wave transmitted through the dense bone of the mandible.

Wounds of the midface: maxillae, zygomas and nose

In our practice, the acute midface injuries most commonly seen have been the results of attempted suicide or homicide; we have also had to treat the late problems resulting from war injuries. An avulsive impact from the front, such as a shotgun blast, will frequently produce cone-shaped destruction of the middle third of the face, leaving lateral elements intact. A missile impact from the side may ablate the whole of the upper jaw and nose (Fig. 1.15C), or may tear off half of the upper jaw leaving the other half relatively intact. It is unusual to find a complete loss of the middle third of the face; more often a segment of palate is missing and the residual fragments are blown aside into abnormal positions. Often the nasal pyramid is completely disrupted. The zygomas are often splayed apart and there may be associated fractures of the coronoid processes of the mandible, or disruption of the temporomandibular (TMJ) joints. If not treated effectively, ankylosis may develop. There are likely to be wide oro-antral and oronasal fistulae, with exposure of the antrum to the outside. According to the direction of the missile(s) there are injuries of adjacent vital structures such as the eyes or the brain; missiles traversing the middle third region may disrupt the middle cranial fossa as well as the anterior fossa, and both temporal and frontal lobe damage have been seen.

In surviving cases of midface regional injury, the structures likely to give long-term surgical problems are the lacrimal apparatus, the facial nerve and the parotid gland. Residual oro-antral and oronasal fistulae, almost inevitable when the hard palate has been widely destroyed, are also important surgical problems. Comprehensive destruction of the nasal structures is extremely difficult to repair in the first instance, and this leads to the necessity for complex secondary surgery. Where there is extensive loss of tissue over the anterior midface with exposure of the maxillary antrum then it is necessary to provide soft tissue and skin cover to this area.

Wounds of the upper face and frontal convexity

A missile wound in the frontal area may show more or less extensive loss of skin and frontal bone. The paranasal air sinuses are often injured; there may be dural penetration and cerebral damage. Such dural injuries may result in a permanent cranionasal communication, with risk of early or late meningitis and/or brain abscess. In our view, frontal wounds with proven or probable dural penetration require transcranial exploration and full dural repair; the arguments for and against this policy are discussed on p. 376. This is less necessary when the middle fossa dura is torn, though here too the possibility of a cranionasal or cranioaural fistula must be kept in mind. We have seen delayed meningitis from a missile wound of the middle fossa.

Missile wounds of the upper face are likely to involve the orbit and especially the eye; the inherent anatomical resistance of the mobile globe of the eye to blunt trauma of the facial skeleton, so often seen after in road crashes, does not give protection from a missile (Fig. 1.15B). Injuries of the naso-ethmoidal region are frequently associated with damage to the globe. Reconstruction of the root of the nose and cranial base is often needed, with repair of the nasal pyramid (Fig. 21.48).

Missiles striking the frontal convexity above the orbital level often cause lethal brain damage. However, when the missile path is tangential to the frontal convexity (p. 141), the patient may survive with a variable loss of skin, frontal bone and dura mater; such injuries often entail severe and disabling frontal lobe damage (p. 53).

Frontal ablative defects secondary to bone infection are less serious. Post-traumatic infection may destroy much of the frontal bone, leaving gross deformity; as a rule, there is no associated scalp loss, though sometimes sinus formation may have caused extensive scarring (Figs 21.1 and 21.7).

Scalp avulsion

Frontal scalp avulsion as an isolated injury has been discussed on p. 437. In scalping injuries due to traction on the hair, the plane of separation leaves the pericranium intact, and viable unless it becomes desiccated. In missile injuries, substantial areas of scalp may be avulsed or destroyed, sometimes in conjunction with loss of bone and exposure of dura or brain.

Management

General principles

Vital functions and vital structures must be protected. The vital functions chiefly under threat are the airway and the circulation. Airway obstruction and haemorrhage from large cervical and facial vessels have been the causes of many battlefield deaths, and despite advances in emergency trauma care, preventable deaths still result from peacetime injuries of this type.

After the essential preliminaries of evacuation, together with efforts to save life and to protect function, the primary management of the wound must be undertaken. The vital structures that need to be covered urgently are the brain and the dura, the eyes and the large vessels in the neck and face. This should be done with the aim of minimising long-term deformity and disability. The maxillofacial region is different in many ways from other parts of the body in that blood supply of the area is excellent. With the advent of antibiotics, the risk of secondary haemorrhage has been minimized, and one can contemplate a programme of minimum debridement and early closure with a considerable amount of reconstruction being performed in the early phase, if not at the first operation then certainly within the first 10 days to10 weeks.

Gruss et al (1991) recently reviewed their experience in 37 cases of massive facial injury from gunshots, chiefly suicidal attempts with shotguns or sporting rifles. They, argued against traditional programmes entailing early soft-tissue closure and delayed multistaged bone replacement, and advocated early reconstruction with model craniofacial techniques, notably rigid internal fixation with plates and bone grafts. For severe avulsive injury with loss of bone and soft tissue, Gruss and his colleagues proposed:

1. Immediate conservative debridement, with cover of exposed bone.
2. Delayed (7–10 days) primary definitive repair. Missing midfacial bone should be replaced with split calvarial and rib grafts, with stabilisation by miniplates and screws; the correct facial width is restored. The mandible should be reconstructed, fragments being reduced and fixed with miniplates and lag screws. Gaps are bridged with long reconstruction plates. Soft tissue may be replaced with a free vascularized omental graft.
3. Late repair procedures may be necessary: in their practice, these included scar revision, reconstruction of the oral commissure, rhinoplasty and mandibular reconstruction by vascularized or conventional bone grafts.

Two strategies can be considered when one is confronted with the eventual need to reconstruct the mutilations due to ablative midfacial injuries, and they are not mutually exclusive. The first is prosthetic: the defect is replaced as far as possible by an external prosthesis or by an implant of some biocompatible material. The second is truly restorative: the defect is repaired by transplanted autogenous tissue. The advent of microvascular anastomosis (see Ch. 21) has greatly enlarged the utility of restorative transplantation. Indeed, modern reconstructive

techniques involving flaps of skin, muscle and bone and free tissue transfer, together with the development of osseo-integrated implantology, have brought the two strategies into a close partnership. By aiming at the best possible early definitive repair, we hope to minimise the need for late correction or prosthetic replacement—and to maximize the success of these forms of management.

Wartime conditions may make compromises unavoidable, and late reconstructions may then be done under more adverse conditions, with established soft-tissue contraction When one is fortunate in treating the patient in a well-equipped unit from the beginning, the two strategies can be harmonised to facilitate a planned combination of the reconstructive and prosthetic options (Fig. 21.48).

Assessment

In the early assessment of a severe ablative facial injury, an examiner confronted with a mass of unidentifiable bleeding tissues will direct attention to the control of the airway, the circulation and the cerebral status, and other important effects of the injury may easily be missed. Nevertheless, as soon as possible, the systematic clinical, laboratory and radiological work-up described in Chapters 6–9 should be carried out. If there is any reason to suspect vascular damage, such as the trajectory of missile fragments in the lower face and neck, cerebral angiography should be done; if there is any suspicion of blunt or penetrating injury of the brain, computerised tomography (CT) scan is essential. Magnetic resonance imaging (MRI) may also be of great value, but should not be used if there is evidence of a ferromagnetic missile in a dangerous situation. Lead and cupronickel missiles can be disregarded, but the fragments of bombs and shells should be regarded with suspicion and it should be remembered that modern military bullets may have steel components.

In planning the definitive management of a severe ablative injury, the multidisciplinary evaluation and team discussion described in Chapter 9 is of great value. Throughout the course of management, the injury should be repeatedly reassessed. Marx & Stevens (1991) give special attention to:

1. The physiological state, as related to anaesthetic risk
2. The size of the residual soft tissue defects, and the extent of scarring
3. The size and nature of the bone defect(s)
4. The presence of residual pathological conditions, such as foreign bodies or bone chips, perhaps associated with foci of chronic inflammation or fistula formation.

First aid and life support

This has been discussed in Chapter 8; securing the airway and controlling haemorrhage are immediate priorities in most avulsive injuries of the lower and middle face. Clarkson & Walker (1955) vividly described World War II management on the battlefield, emphasising the occasional need for cricothyroidotomy (p. 226). They noted that injured soldiers often felt little initial pain from jaw wounds, and might survive transection of a major cervical artery: vascular spasm and retraction often provided very effective primary haemostasis. Banks et al (1985) have reviewed more recent wartime practice in Israel, Vietnam, Nigeria, Ireland and the Falkland Islands; they emphasise that early endotracheal intubation is today the mainstay of airway control. Intubation is especially necessary when the conscious level is impaired; cricothyroidotomy may be appropriate when intubation is made difficult or impossible by massive orofacial damage.

Haemorrhage is controlled along the lines set out on p. 227. Banks et al (1985) reported that exsanguinating haemorrhage was not very common in uncomplicated battlefield wounds in the CMF area; however, blood transfusion was often necessary. Delayed bleeding may follow resuscitation and it is likely

that emergency carotid angiography and embolization of bleeding arteries will be used increasingly in the management of primary and secondary haemorrhage.

Timing of repair

This is somewhat controversial. There is no doubt that open wounds can and should be closed as soon as possible in civil practice, and also in war surgery; CMF injuries provide one of the few exceptions to the general military rule that soft-tissue wounds should not be closed at the time of the initial wound care (Australian Army Manual of Land Warfare 1985, Whitlock 1985). If properly debrided, the vast majority of CMF wounds will heal well.

There is also little argument when the avulsive wound exposes or penetrates the dura mater. Early closure of the dura and skin is essential, though repair of a subfrontal dural defect may be deferred if there is brain swelling (p. 377). Calvarial bone defects can wait for a much longer period.

It is over the timing of definitive facial reconstruction that discordant opinions are expressed. Many authors see the definitive repair of massive defects as best done late, and only after careful preparation; Marx & Stevens (1991) see reconstruction as 'tertiary care', carried out in stages: in their practice, avulsive missile injuries required 'an average of 7.3 surgeries..., and avulsive motor vehicle injuries... an average of 5.7 surgeries.' Our philosophy is somewhat different, and closely parallels the views of Gruss et al (1991) summarized above.

It is hard to set out a rule of thumb for ablative injuries of the CMF region because each case is unique, and because avulsive trauma has no respect for anatomical boundaries. We talk of the avulsive injuries of the upper middle, and lower thirds of the face, but many such injuries involve all three regions; the treating team is faced with the need to set priorities and to design and implement plans for any permutation of defects. Nevertheless, there is an increased tendency to do more and more, earlier and earlier. It is often necessary to operate on patients in the first few hours after trauma to cover vital structures and to stem haemorrhage and to perform other life-saving manoeuvres; at such operations, as much as possible should be done to facilitate secondary surgery, for example by stabilising bony fragments of the mandible (Figs 16.1 and 21.86). In less extensive injuries, if all of the surgery can be done in this first stage with little or no extra stress on the patient, then this should be done. There remains a group of patients who will require extensive, and perhaps complex reconstruction of the bony and soft tissues of the face and skull. We see these, not as late reconstructive procedures, but as the continuation of the initial treatment inaugurated by the definitive multidisciplinary team. Where possible, reconstruction should be conducted in the first 7–21 days after injury but there are no hard-and-fast rules, especially when local facilities are inadequate and transfer to a specialised unit is necessary. For example, the microvascular reconstruction of the symphysis and body of the mandible, together with skin and oral lining repair, might well be started very early whereas a cranioplasty for ablated frontal bone in the same patient, if covered with a suitable scalp flap, might be left for some months (Figs 21.1 and 21.73).

The often quoted dictum: 'first-the bone and then the soft tissue' attributed to Pichler & Trauner (1948) is a good principle; however, with modern techniques of microvascular flap repair, microvascular tissue transfer and osseomusculocutaneous flaps, much of the reconstruction of bone as well as soft tissue can be performed simultaneously at a single procedure. Where this is done, early problems of scar contracture and difficulty in recreating the defect are not encountered. In all reconstructive procedures, what is done at the first operation often sets the limits on what can be achieved. The first chance is often the best chance. If the patient is fit and free of infection, every effort is made to produce a definitive result at this early phase. However, early operation need not mean immediate operation — it is often prudent to delay surgery for a few hours to stabilise the patient and to prepare a clear, well-planned operative strategy based on good radiological investigations.

Management of soft-tissue avulsions

Because of the favourable blood supply, most CMF wounds can be closed early and will heal uneventfully. This has been the experience in recent war zone surgery (Chapman 1985) as well as in civil practice. However, close-range gunshot wounds with widely disrupted soft tissue may need more extensive treatment by packing, cleaning and drainage, and implanted bone chips or tooth fragments may need to be located and removed.

Cleaning is done chiefly by copious washing with normal saline and a good deal of time should be spent in this, using large syringes with compressive bulbs. The initial debridement may be followed by partial skin closure and packing, with delayed complete closure within a week, at which as much of the definitive reconstruction as possible is carried out. If exposed areas of muscle a bone are left after debridement and suture of skin to mucosa, they should be packed with saline-soaked ribbon gauze. The other indication for packing is bleeding not controllable by diathermy or ligation of vessels. In such cases, the airway is secured by intubation or tracheostomy, and the nasal oral and pharyngeal spaces are firmly packed with 2.5 cm ribbon gauze dampened with saline. The taped ends of the packing are brought out through the mouth and the nose. This packing can be changed at 24–48 h or at the next available opportunity depending on the treatment schedule, e.g. the need for a general anaesthetic for other injuries. Removal of packing shout be done under general anaesthesia in the operating theatre with the capacity to control further bleeding and repack if necessary. The insertion of Foley catheters through the nostrils and inflated in the postnasal cavity is a popular way of controlling bleeding in the area. Traction may need to be applied to the catheter to produce the desired effect and this should not be done by tying the catheters together across the columella, which may undergo necrosis—a very unpleasant complication!

In the head and neck, minimal debridement is appropriate because of the good blood supply. The wound edges are excised with a blade and trimmed square with sharp tungsten carbide inset scissors. An experienced surgeon can usually mobilize soft tissues in debrided wounds to produce an effective closure. Equal attention should be paid to suturing the oral mucosa as well as the skin. Mucosal lining must often be sutured to skin in the region of the nose and mouth. The branches of lacerated nerves should be identified and sutured if possible (p. 440); if microsurgical facilities are not available the nerve ends are tagged with black silk sutures in the epineurium, care being taken to avoid damage to the funicles of the nerve.

When there is a raw surface wound uncovered by skin or mucosa which cannot be closed primarily, the exposed tissue may be dressed with gauze soaked in saline, changed every 2–4 h, and later covered with skin grafts when the surface is clean and well vascularized. However vital structures must be covered as soon as possible. Exposed dura mater is usually covered with a scalp flap (Fig. 21.1); this is even more necessary when brain is exposed, but to minimise adhesions to the damaged cerebral cortex, a fascial or pericranial graft is first interposed. Bare vital structures in the neck, such as carotid vessels, are covered by a local flap or by a distant pedicled flap; bare bone may also need to be covered by a local or distant flap (Fig 16.2). Soft-tissue defects resulting from the transferring of local flaps or from residual soft-tissue injuries in the face can be closed by split skin grafting.

The advantage of having primary closure performed by members of an experienced multidisciplinary team is that at this stage with the team assembled, as much as possible of the reconstruction can be done at once, and an appropriate plan can be made for the intermediate and later treatment.

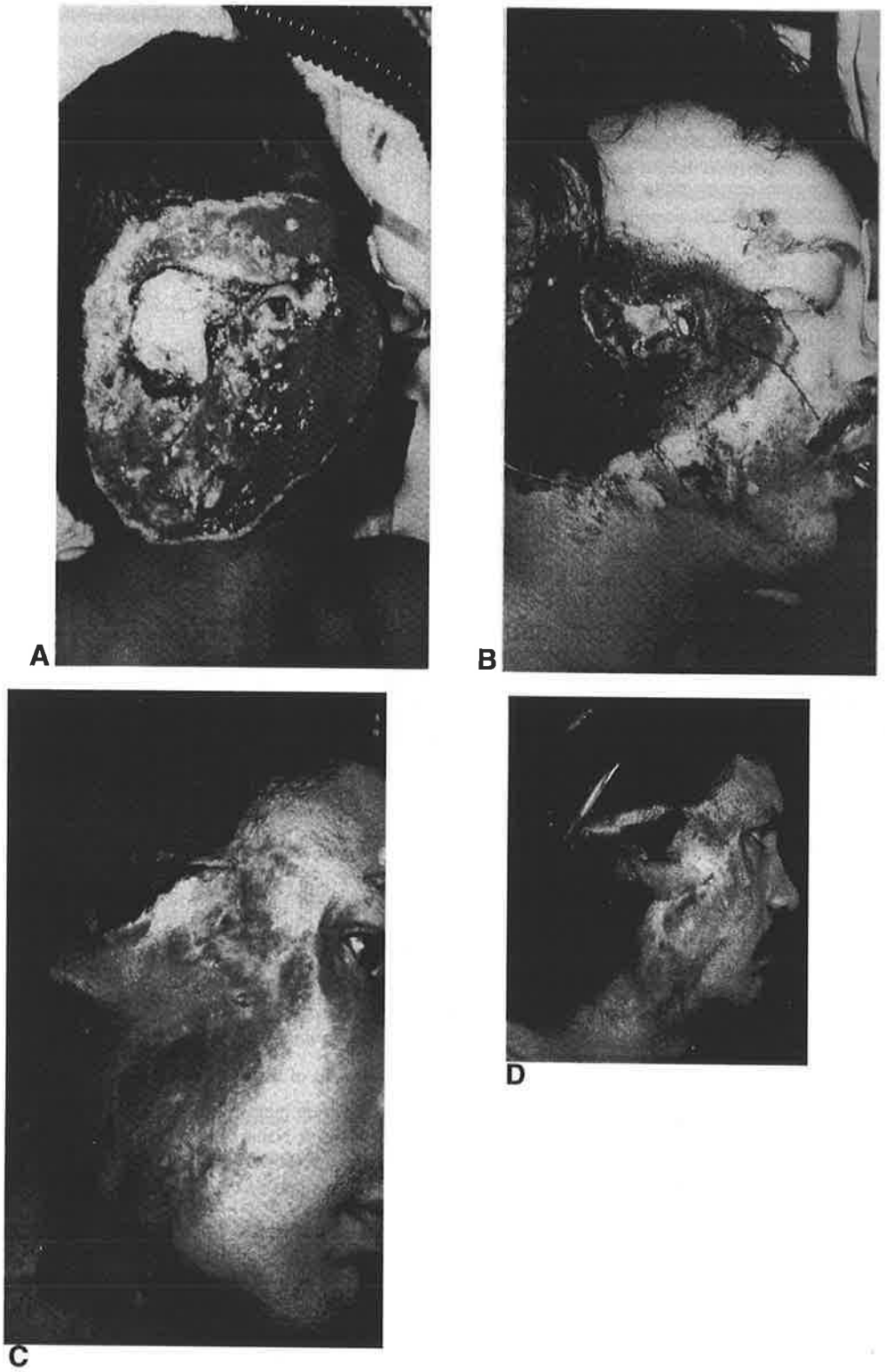

FIG. 16.2. ***Secondary treatment of an avulsive injury.*** *An Asian boy sustained a massive avulsive injury of the right side of the face including the right ear which was not treated initially.* **A.** *Exposure of the zygoma and temporal bone with extensive soft-tissue loss.* **B.** *An initial attempt was made to cover vital structures with a local flap.* **C.** *The result of inadequate debridement and inadequate soft-tissue cover: multiple sinuses and underlying dead bone.* **D.** *The patient presented for surgery after healing by secondary intention, with some flap cover.*

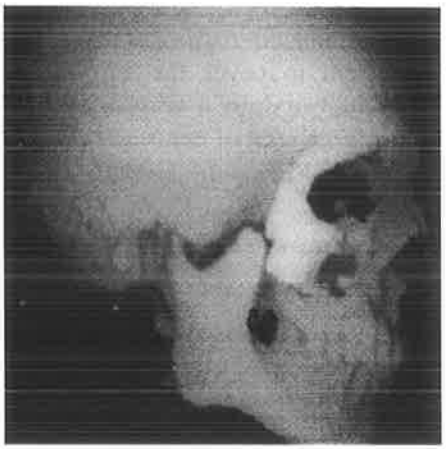

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FIG. 16.2. ***Secondary treatment of an avulsive injury.*** *An Asian boy sustained a massive avulsive injury of the right side of the face including the right ear which was not treated initially.* **E.** *Three-dimensional CT showed loss of most of the body of the zygoma and its arch.* **F. G.** *The face in lateral view and anteroposterior views after tissue expansion to advance the scalp hair, free flap (lateral arm) reconstruction of the soft tissues, and*

Replacement of skin in avulsive injury

Restoration of the integument is crucially important to protect vital structures and to cover or form a bed for bone grafting; it is also important in restoring the aesthetic appearance of the face. To this end, the face should be treated as a series of aesthetic units (Fig. 17.10). If possible, muscle and nerve should be replaced, skin giving good colour match should be sought, and a sound skeletal basis should be provided either before or at the same time. An example is seen in total nasal reconstruction: the bony structure is essential to support any soft-tissue reconstruction and planning should be so as to minimize the number of stages. The tissues available for reconstruction of the face are any and all that can be chosen from the entire armamentarium of plastic and reconstructive surgery.

Free grafts

Split-thickness grafts and full-thickness grafts are applicable in the reconstruction of certain cosmetic units such as the upper eyelid, lower eyelid, and the nose where there is a good soft-tissue base; such grafts may be suitable when there is need for replacement of superficial loss of the cheeks and lips.

Wound contracture after early grafting may result in an unsatisfactory result; it may be possible at a later date to excise the grafted area and replace it with skin obtained by tissue expansion (p. 583).

Flaps

The workhorses of traditional plastic surgery have been used in almost every conceivable way to reconstruct various parts of the face. While these find their chief applications in the elective correction of deformities (p. 621), closure by a pedicled or free microvascularized flap is sometimes necessary in the acute phase of management of an avulsive injury.

Scalp flaps

The prodigious blood supply of the region allows the whole scalp to be moved on even one of its major vessels and there are many variant techniques, e.g. the crane principle of Millard (1969).

Forehead flaps

The central forehead flap is made to include the supratrochlear vessels in its base; it may be used to provide a lining for the nose or a cover for the nose. Recently this flap has gained much advantage with the advent of tissue expansion to provide a large amount of skin for nasal reconstruction. In some cases adjacent tissue can be gained by using tissue expanders to advance forehead tissue after the hairy flap has been returned. Tissue expansion is a valuable method of gaining additional skin of good quality and appropriate colour, with or without hair. However, tissue expansion requires some weeks' delay, and this makes it useless in the early closure of avulsive injury; tissue expansion finds its chief roles in postacute reconstructive surgery. Techniques of tissue expansion, and the occasional complications, are discussed on p. 583.

The 'lateral' forehead flap is based on the anterior branch of the superficial temporal artery; Banks et al (1985) note that this branch alone is inadequate in some 20% of cases, being partially supplanted by the zygomatic branch of the superficial temporal artery (In Fig. 2.16 this vessel is designated as the zygomatico-orbital artery.) They stress the need to assess these vessels before operation by palpation; Doppler ultrasound examination may be more reliable. The value of the lateral flap can be extended by tissue expansion.

The Converse (1942, 1977) scalping flap can be used to provide thin skin for nasal reconstruction. This elegant procedure entails raising the forehead as a full-thickness bicoronal scalp flap; one-half of the forehead is first raised as a thin flap by dissecting it off the frontalis muscle. The thin flap is based superiorly on the main flap, and can be doubled under it to reconstruct the nose. The postauricular flap of Washio (1972) can be used to transfer postauricular skin and some cartilage for reconstruction of the nose. Once again, tissue expansion has enabled small portions of skin to be expanded and transferred on flaps.

Replacement of muscle and skin

There is a wide range of muscle and musculocutaneous flaps; Mathes & Nahai (1979) describe the classical procedures, some of which can be used in avulsive injuries in the CMF region.

Temporalis muscle flaps

The temporalis muscle can be used to cover bone or restore contour to the face; it can be also used to transfer calvarial bone on a vascularized pedicle (Fig. 21.78).

Galeal flaps

These can be used to cover exposed ear cartilage and to support skin grafts and temporal fascial flaps. The galea must be dissected away from the scalp skin, and its viability depends on the adequacy of supply from the major arteries of the scalp: as they approach the midline, the branches of the superficial temporal artery may fail to meet this need (p. 47).

Deltopectoral flap

First described by Bakamajian (1965) for pharyngo-oesophageal reconstruction, it was for many years the workhorse in the reconstruction of the face and mouth. The flap is transferred on a pedicle raised deep to the deep fascia and supplied by the perforating branch of the internal mammary vessels; it can be moved high enough to replace the cosmetic unit of the cheek. At the height of its usage this flap was manipulated in many ways: it could be tubed, de-epithelialized, turned upon itself and reinnervated by the incorporated supraclavicular nerves.

It is still worthwhile to have this classical flap in the armamentarium although it has been largely replaced by free tissue transfer.

Pectoralis major flap

This is a myocutaneous flap. It is often used in reconstructions after head and neck cancer excision, but not so often for traumatic ablations. The flap is based on the thoraco-acromial branches of the axillary vessels which reach the deep surface of the muscle just below the clavicle and medial to the tendon of the pectoralis minor. The flap may be raised as an island of skin and muscle, with a neurovascular pedicle; the flap may also incorporate bone from the rib or lateral sternal edge (p. 622).

Latissimus dorsi flap

This myocutaneous flap was popularized for breast reconstruction but has versatile applications. The predominant source of supply to the muscle is from the thoracodorsal branch of the subscapular artery; this artery forms a vascular pedicle which is separated from the muscle. The muscle is divided proximal to the insertion of the pedicle, which can support an overlying skin paddle to reach quite high up to the cheek, the pedicle being passed in a tunnel under the skin of the neck.

Trapezius flap

This myocutaneous flap is based on the deep transverse cervical artery and vein; it can be used for defects in the neck and lower part of the face. The lateral third of the clavicle may be incorporated into such a flap as well as the spine of the scapula (p. 622).

Free flaps: replacement of skin, muscle, bone and nerve

A free vascularized flap may be used in postacute reconstruction of an avulsive injury, or may even be performed as an acute procedure; there must of course be a suitable artery and vein in the area to be reconstructed.

Groin flap

This is based on the superficial circumflex iliac artery (SCIA), and is suitable to cover bare bone, dura mater or brain (p. 456). Unless the flap is very large (> 10 cm in diameter), the donor site can be closed directly and a good cosmetic scar can be expected; when this is impossible, a split-skin graft may be necessary. Flaps as large as 20 × 30 cm have been used, and two flaps will cover the entire calvarial vault. The vascular pedicle has a variable anatomy, which is described by Taylor & Daniel (1981). In 48% of cases, the SCIA rises conjointly with the superficial inferior epigastric artery (SIEA), in 35% the arteries arise separately, and in 17% the SCIA does not arise from the femoral artery. Nevertheless, the SCIA, whatever its origin, is usually 1–1.5 mm in diameter and adequate for an anastomosis. The pedicle tends to be short unless the surgeon puts the skin component of the flap more laterally, when the pedicle can be developed into a longer leash of blood vessels; however, this carries a higher risk of flap morbidity.

An important variant of this groin flap is based on the deep circumflex iliac artery (DCIA). This vessel provides a more dependable blood supply for

grafts taken from the ilium; larger bone grafts can be harvested when supplied by the DCIA, and in our practice this free flap is the chief means of reconstruction after massive avulsive injuries of the mandible (David et al 1988). Our operative technique is detailed in Chapter 21 (p.628).

Latissimus dorsi

This muscle flap, with or without a skin paddle, may be used as an alternative to the groin flap. The transferred muscle must be covered with split skin unless the skin paddle is sufficient to cover the defect. The latissimus dorsi muscle fans out into a wide triangle and may be used to cover a large area of exposed cranium; it is useful in other avulsive CMF injuries when bulk replacement is required.

Omental flap

Gruss et al (1991) have used free vascularized omental grafts to cover bone grafts and to provide bulk in reconstructing gunshot wounds of the lower jaw. We have little experience of the procedure, and have preferred to use the vascularized groin flaps described above.

Intestinal graft

It has been suggested that oral mucosal defects can be replaced with free vascularized intestinal flaps. Again, we have little experience of this technique. One case was treated with a jejunal free flap in this way; the graft survived, but was rugose, friable and productive of excessive mucus, and had to be removed.

The use of free flaps is further described in Chapter 21 and includes many applications of microvascular tissue transfer. The possibility of transfer of muscle, nerve and bone for the reconstruction of all layers and restoration of some function, together with tissue expansion, titanium osseo-integrated implantation and the closely related art and science of computer-aided design/computer-aided manufacture (CAD/CAM) prosthetic reconstruction has advanced reconstruction after ablative craniofacial injury both in quality, result and time saved.

Avulsive injuries of the lower jaw

Avulsive wounds of this region involve bone, teeth and soft tissue. Almost invariably, the bone is extensively comminuted. There is always doubt about the viability of the bone and tooth fragments. Initial debridement involves copious irrigation with normal saline and the meticulous removal of all foreign material; antiseptic solutions may also be used for this essential washing procedure. Loose bone is removed, attached bone is preserved. The dental team decides the fate of involved teeth, the emphasis being on retention, further dissection to remove tooth roots or questionably viable teeth being avoided.

The surgical aim is to prevent or control infection and to produce bone fragment alignment within a sleeve of relatively healthy periosteum. Antibiotics are given as early as possible (p. 236). Where there is extensive comminution, the fragments are united with titanium plates and screws. Where there is loss of bone, particularly in the symphysis, the two sides of the mandible are maintained in their normal anatomical relationship using a bridging metal implant. Long titanium plates (Fig. 16.3). are well suited for this purpose; they can be shaped to adapt to the contour of the missing mandible, and fragments of residual bone can be attached to the plate through the screw holes. Alternatively extraoral fixation devices can be used, but in our practice, these have been supplanted by the availability of a wide range of titanium plates. Maximum immobilization is necessary, and where there is an intact maxilla and enough remaining mandibular teeth, intermaxillary fixation can be added to give additional stability.

In ablative injuries of this region, there is often massive skin loss or disruption. Once the bone has been fixed either by reconstituting the mandibular arch or by interposing an appropriate spacer, a water-tight closure of mucosa

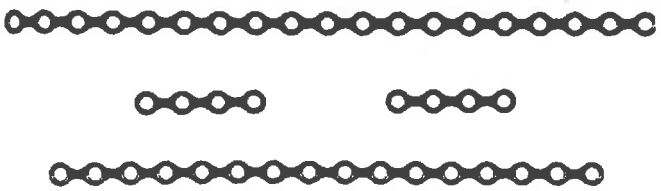

FIG. 16.3. ***Long titanium plates (AusSystems®).*** *These can be cut into smaller segments when necessary. The full length is useful to bridge gaps in bone for temporary reconstruction.*

and a covering of skin is necessary. For this, it may be necessary to undermine the mucosa of the cheek or the structures of the floor of mouth or to mobilize skin from the adjacent cheek and neck. The reconstructed bone must be protected in this way for bone healing to be possible. If skin closure cannot be achieved, then some form of flap repair, whether pedicled or microvascular, must be undertaken. This is probably best done at an intermediate or postacute stage. When hairy skin is needed for the beard or moustache area, tissue expansion (Ohana 1986) may make a scalp flap available.

Avulsive injuries of the midface

When debridement has been completed, the remnants of the maxillary arch should be aligned by manipulation, followed by application of appropriate arch bars and fixation to the mandible by some form of intermaxillary fixation if there is a good mandibular template intact. Titanium miniplates are helpful in stabilising the zygomas and in attempting to re-establish an attachment of the palatal fragments to the zygomatic buttresses; even at an early stage, the reconstructive principles of Gruss et al (1991) are applicable.

Oro-antral and oronasal fistulae often result from the frequent inability to close the palate. Loss of the specialized nasal tissue—the septum, nasal lining, and nasal cartilages—will necessitate long-term and often difficult reconstruction in this area. So one should be conservative and should reposition the remnants of these structures with as much care as possible in the primary repair. Where skin cover cannot be completely effected, mucosa to skin suture may be necessary and also grafting with split-thickness skin. The principle that applies in this area is to carry out all the reconstruction that can be done as early as possible, according to the state of the patient and the ability of the treating team; this will facilitate the later reconstructive procedures described on p. 633.

Avulsive frontal and fronto-orbital injuries

In war, many cases of massive injuries in this site survive in good state, and it is often possible to perform much of the definitive treatment as a primary procedure. In a classic review of World War II injuries, Stewart & Botterel (1947) reported on 25 cases of 'cranio-facial-orbital' wounds, and advocated:

1. Radical debridement of the cranial and cerebral wounds
2. Debridement of paranasal sinuses and establishment of drainage through the nose
3. Sealing the subdural and subarachnoid spaces
4. Closure of the scalp without tension and without drainage.

Modern experience has shown that these principles are still valid. However, it is now possible to be less radical in debriding the cerebral wound (p. 373), and to preserve even detached fragments of bone, especially in the fronto-orbital region; these can be replaced and fixed with miniplates, unless there is gross contamination or delay in operation.

If the frontal scalp defect cannot be closed by direct suture, a large frontolateral flap, based on the superficial temporal artery, is rotated forward to cover the sutured dural wound (Fig. 21.1). There must be no tension in the final

skin suture line; if there is risk of tension, the posterior margin of the wound is not sutured and the exposed pericranium is covered with a split-skin graft. We have done this even for relatively small (2 × 4 cm) residual skin defects (Fig. 13.1); more experienced war surgeons are sometimes able to cover fronto-orbital defects without flaps, by mobilising the scalp, but the novice is advised to use flap closure if there is any risk of tension. Emergency skin closure is disfiguring when hairy scalp is advanced onto the forehead or to cover an eye socket; however, the hairy scalp can be later returned using the crane principle described by Millard (1969). This uses the scalp to carry the galea forward. Scalp superficial to the galea can then be returned and split-skin grafts placed over the galeal layer. In some cases adjacent tissue can be expanded using tissue expanders to advance forehead tissue after the hairy flap has been returned.

Scalp avulsion

When a large piece of scalp has been avulsed and that tissue is available then it may be replaced by microvascular techniques (p. 437). Where this is impossible but the pericranium is preserved, split-skin grafts may be applied to the pericranium; care must be taken to ensure that the pericranium does not dry out—this can happen very quickly. When the pericranium is lost but the skull intact, the outer calvarial table can be removed and split-skin grafts placed on the diploe after granulations have appeared; this temporising procedure is discussed on p. 437. It is, however, better to cover the exposed bone with a scalp transposition or rotation flap, and this is essential when dura or brain are exposed. On the rare occasions when there is no local tissue with which to cover exposed dura and brain in an emergency, a pedicled flap or microvascular free tissue transfer may be indicated (Fig. 15.2).

Avulsive injuries of the frontal sinus and orbital rim

The general management of fracture of the frontal sinus has been discussed on p. 372. If there is loss of the anterior wall of the sinus or of the orbital rim the defect may sometimes be repaired by a calvarial bone graft at the time of the initial wound closure. However, it is our preference to replace any remaining segments by miniplate or microplate fixation, using bone grafting for an elective repair (Fig. 21.3).

Frontal bone defects

Cranioplasty is a standard neurosurgical procedure and the techniques and problems are well known (p. 548). But in the frontal region cranioplasty presents some special difficulties. The area is not covered by hair and unless contouring is perfect there will be a visible deformity of the forehead. The proximity of the air sinuses makes delayed infection a real threat, especially when a foreign implant is used. In our view, there is no place for cranioplasty in the early management of ablative injuries of the fronto-orbital region. The indications and techniques of frontal cranioplasty are considered in Chapter 21.

Injury to the eye and eye socket

Ablative injury in this site demands consultation with an ophthalmologist; management of the injured eye is considered in Chapter 14. The principles of reconstruction of the eye socket are that the rim should be reconstructed from what fragments are present and that the walls should be primarily bone-grafted, providing there is reasonable soft-tissue cover (Fig. 16.4). The use of foreign material such as Silastic® under these circumstances is not encouraged. When there is insufficient local soft tissue to cover a shattered orbit, it is sometimes necessary to employ a forehead flap or rotation scalp flap.

Ablative injury of the ear

Lacerations, avulsion and destruction of the external ear should be repaired after careful debridement; exposed cartilage should be covered by excising its margins

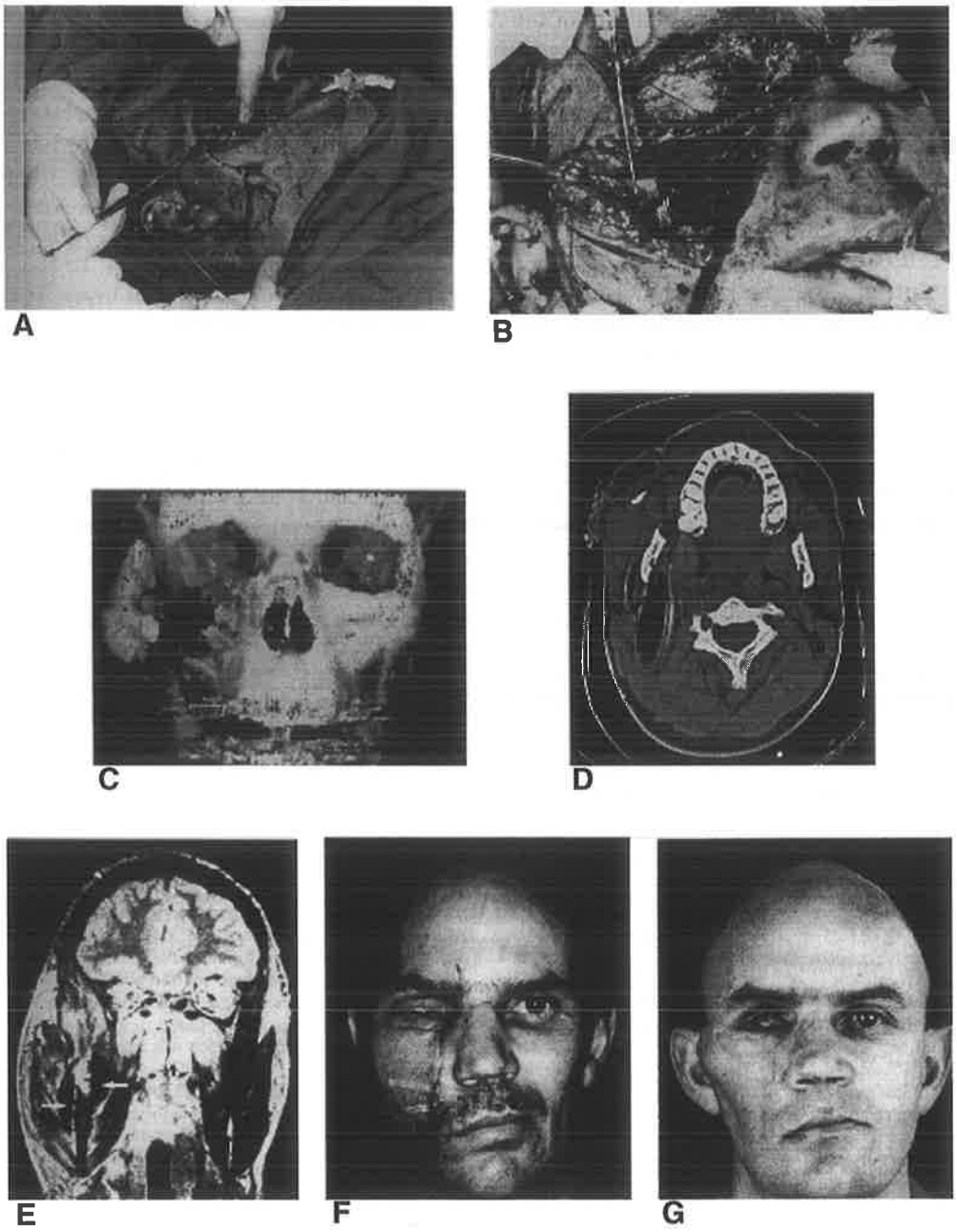

FIG. 16.4. ***Avulsive facial injury in a car crash.*** *A 39-year-old man was involved in a freak car accident: a tree fell upon his car and a branch penetrated the front windscreen, piercing the patient's face like a spear, avulsing the right zygoma and leaving it pedicled on soft tissue laterally. Although he was pinned to the vehicle, he was promptly rescued thanks to his mobile telephone. Examination showed that the branch had encountered the anterior border of the mandible, dislocating it laterally. The branch was split by the mandible, with shafts of wood passing medially and laterally into the neck. The medial fragment lay on the carotid vessels. The right orbital floor was destroyed, the right eye was lost, and the anterior wall of the right maxillary antrum was destroyed. There was an extensive soft-tissue deficit of the right cheek. The emergency management involved freeing the patient from the tree and transporting him to a central unit. MRI scan showed the exact relationship of the penetrating shafts of wood to vital structures, and angiographic enhancement indicated the relationships with the vessels. The patient was anaesthetised with care to avoid further damage. The cavities were cleaned and debrided, the zygoma was temporarily fixed and the skin was sutured in place. At a second operation the zygoma was further secured, and at the same time the remnants of the globe were removed and a conformer placed into the eye socket.* **A.** *Manipulation of the foreign body.* **B.** *The zygoma was turned back on a lateral myocutaneous flap, exposing massive destruction of the orbital floor and anterior maxilla.* **C.** *Three-dimensional CT showing the bony deformity.* **D.** *CT scan showing relationship of the shafts of wood to the mandibular ramus.* **E.** *MRI scan showing the relationship of the wood to the mandible and soft tissues (white arrows indicate the wood).* **F.** *The patient demonstrates the residual deformity.* **G.** *Appearance prior to secondary reconstruction of the zygoma, orbit and maxilla.*

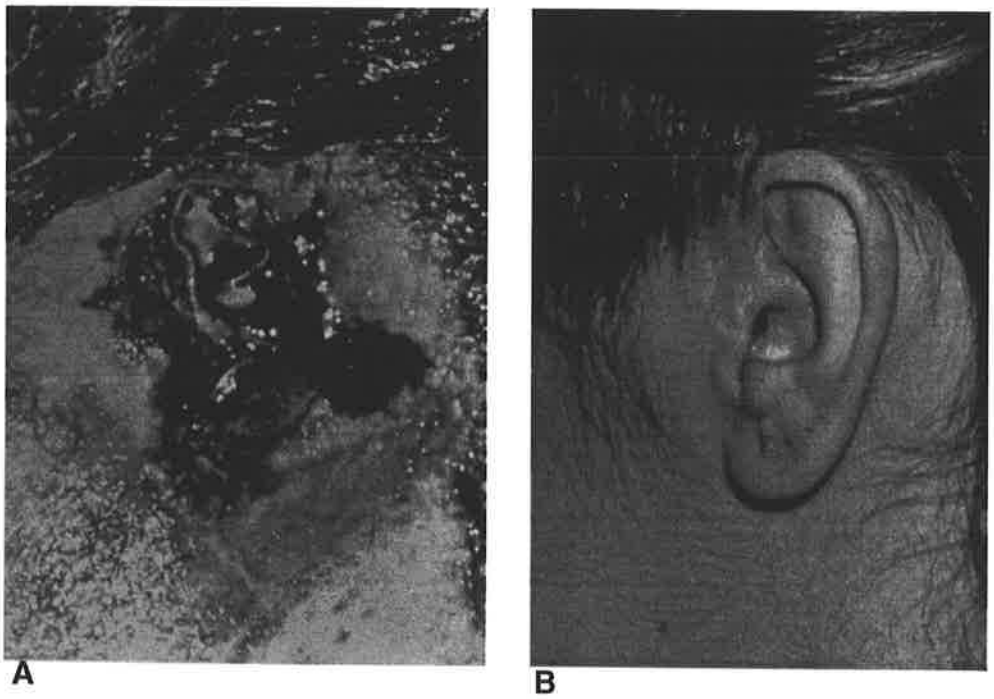

FIG. 16.5. ***Traumatic loss of an ear and microvascular replacement.*** **A.** *Initial appearance of the wound with road abrasion and avulsion of the ear.* **B.** *Appearance following reimplantation and successful revascularization.*

and suturing skin to skin. When an avulsed ear is brought with the patient, it may be possible to suture the ear in place using microsurgical techniques; our colleagues Katsaros et al (1988) reported success in two of three cases of avulsion sustained in road accidents. An artery must first be found in the avulsed ear; when this is perfused from a suitable artery (usually the posterior auricular artery), it is easier to find a bleeding vein. Intraoperative heparin and also haemodilution are advised (Fig. 16.5).

Follow-up care after avulsive injury

The next phase of treatment involves all those measures which are expected to promote mobility and rapid healing: these include wound care, both superficial and intraoral, nutrition, and control of infection. Management aims to prevent or to treat all the systemic problems that may complicate severe trauma and its surgical treatment, notably septicaemia, pneumonia, peripheral vein thrombosis and pulmonary embolism. Also to be treated are the local problems that may complicate these injuries especially secondary haemorrhage, infection and meningitis where there has been a cranionasal fistula in the anterior fossa (p. 387).

When these dangers have been circumvented, and the wounds are well healed, the stage is set for correction of residual deformities. This stage is discussed in Chapter 21.

Complications and results

Wartime experience has shown that if the victim of a massive gunshot wound in the CMF area does not die on the battlefield, the prospects of survival are surprisingly good. Banks (1985) reviewed mortality rates from series of military maxillofacial injuries, and these varied from about 1% in World War II to 26.3% in cases admitted to an intensive care unit in Northern Ireland. These statistics clearly express different chances of early survival and different criteria in selection.

The complications of massive avulsive injury are numerous and diverse. Many are beyond surgical control, but some relate to the quality of management. In the early management of injuries entailing massive tissue loss, it is necessary

to use strategies to save life, conserve tissue and commence reconstruction, with a full awareness of what future treatment may be available to complete the job. In a fully equipped craniofacial unit, the processes of management move smoothly from early management to definitive repair, and depending on the nature of the injury it may be possible to carry out the definitive reconstruction at an early date, as urged by Gruss et al (1991). Under other circumstances, the victim of an avulsive, injury may have early treatment in a less specialized centre and be transferred elsewhere for the completion of the reconstruction. In this case, a great service is done for the patient if the team giving initial care can adhere to the principles of treatment which will enhance the future reconstruction.

The procedures described in Chapter 21 are aimed to restore function and to correct deformity with as little delay as possible. But with severe avulsive injuries, the phase of reconstruction may be prolonged for months or years, and be subject to the changes of growth, age, degeneration and the emergence of new techniques of treatment.

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Deformities

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Introduction

Merville et al (1974) wrote that in the treatment of facial fractures, one should strive for rearchitecturalisation of the facial skeleton . . . one should try at the outset to reduce everything to perfection, since each osseous element determines the position of its neighbours . . . one should thus try to leave nothing for secondary restoration.' This view has been eloquently reinforced in the most up-to-date way by the work of Gruss, who goes to great lengths to achieve a perfect initial reconstruction. This very desirable strategy is exemplified in the management plans set out in Chapters 11–19; it is made easier by good X-ray assessment, good exposure, very good fixation, and modern grafting techniques including the use of free grafts vascularized by microsurgery. Nevertheless, serious residual deformities do occur either because of the severity of the initial injury, or because of inadequate primary reconstruction.

Sir Harold Gillies (1968) divided post-traumatic facial deformities into those with substantial loss of tissue and those without. When there is no serious loss of tissue, residual deformities are due to:

1. Failure to diagnose correctly, i.e. to assess the nature and extent of the original acute injury
2. Failure to disimpact or wholly replace displacements from such an injury and/or to provide adequate fixation.

Deformities often entail visible contour defects, even when there is no severe tissue loss. Contour defects may be treated by:

1. Osteotomy and repositioning
2. Onlay or inlay of bone grafts or alloplastic material
3. Prostheses which may be intraoral (maxillary or mandibular) or extraoral, such as prosthetic eyes, ears or noses.

This chapter deals with those deformities for which primary treatment might have been better, and with those where even, in retrospect, primary treatment was appropriate, but where there was still unavoidable uncertainty about the final result, such as enophthalmos, temporomandibular joint (TMJ) injury, childhood injury and many soft-tissue injuries with scarring. Also to be considered are those injuries which from the beginning were destined to need secondary surgery, such as the massive avulsive defects considered in Chapter 16, not all of which can be adequately corrected in the primary and postprimary reparative procedures.

The aim of secondary surgery for deformity is to restore the patient to functional and aesthetic wholeness, always taking into account the pretraumatic appearance. This may entail another sequence of operations, often performed over a long period of time. Although the ideal of full correction must be kept constantly in mind, complete restoration of form and function may in fact never be possible.

Classification of major post-traumatic deformities

These can be broadly grouped as:

1. Forehead contour deformities
The frontal sinuses may be involved, and loss of integument (bone and/or scalp) may expose the brain to risk of future injury (Fig. 21.1). Forehead deformities can be considered in two groups: frontal and temporal.

2. Orbital deformities
These include:

- enophthalmos
- exophthalmos
- orbitozygomatic and naso-orbital deformities
- telecanthus
- orbital dystopia (including hypertelorism)

These may be associated with nasolacrimal dysfunction, abnormalities of eye movement or impaired lid closure (Ch. 14). Orbital injuries sustained in early life are likely to result in deformities which become progressively more marked as growth advances. Many orbital deformities involve significant deformity of the anterior and/or middle cranial fossae, and can be described as cranio-orbital.

3. Nasal and naso-orbito-ethmoid deformities
In this category, which is poorly defined from the preceding group, one can identify a common type of post-traumatic deformity localized to the nasal bones and cartilages, and a group of more complex deformities involving the bridge of the nose and the ethmoid bow. There may be associated problems related to the nasal airway and paranasal sinuses.

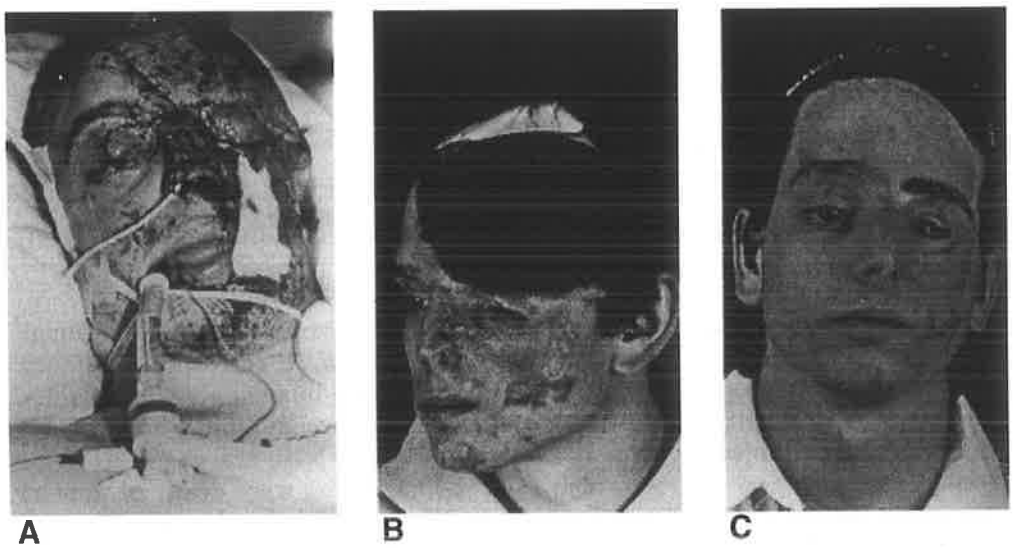


FIG. 21.1. ***Cranial trauma with exposure of the brain.*** ***A*** *A teenage male was involved in a vehicular accident, in which the frontal lobe was exposed and damaged on the left side. There was severe orbito-frontal skeletal damage and after debridement the brain required urgent skin covering. This was done by mobilizing a scalp flap which was brought forward to the eyebrow level. The posterior defect was covered with split-skin graft.* ***B*** *The flap, seen some months later as hairy scalp extending to the eyebrow. The nose and face are badly scarred.* ***C*** *The scalp has been returned superficial to the galea. The galea was left intact and the frontal defects grafted with iliac bone. The forehead was replaced as a cosmetic3 unit using a groin flap vascularized with microvascular techniques.*

4. Deformities of the facial skeleton involving the upper and lower jaws
These deformities are intimately related to occlusal problems and may be affected by post-traumatic TMJ pathology, including ankylosis. Distortions of the facial skeleton may be in three dimensions; in children, distortions are affected by time and growth, constituting fourth dimension.

5. Soft-tissue defects
Deformities resulting from hypertrophic scars and keloids are briefly discussed on p. 438. Other deformities result from avulsive soft-tissue injury (Ch. 16). The loss of a eye constitutes a soft-tissue deformity of much aesthetic and psychological significance (p. 641).

Our computerised records of post-traumatic deformities give details of 385 cases referred to the Australian Cranio facial Unit over a period of 18 years; although this figure underestimates the number of such cases seen in that period, it seems likely that the series is representative of the experience of a tertiary craniofacial unit such as ours. Of these cases, a detailed review of 205 records shows that approximately half were classed as orbital or cranio-orbital. Mandibular and/or maxillary deformities with malocclusion constituted ~24% and naso-orbito-ethmoid deformities only ~8%; in >10% the deformities were complex and had to be described as panfacial. The series excluded localized post-traumatic nasal deformities, but a separate series of 185 such cases has also been reviewed (see p. 579).

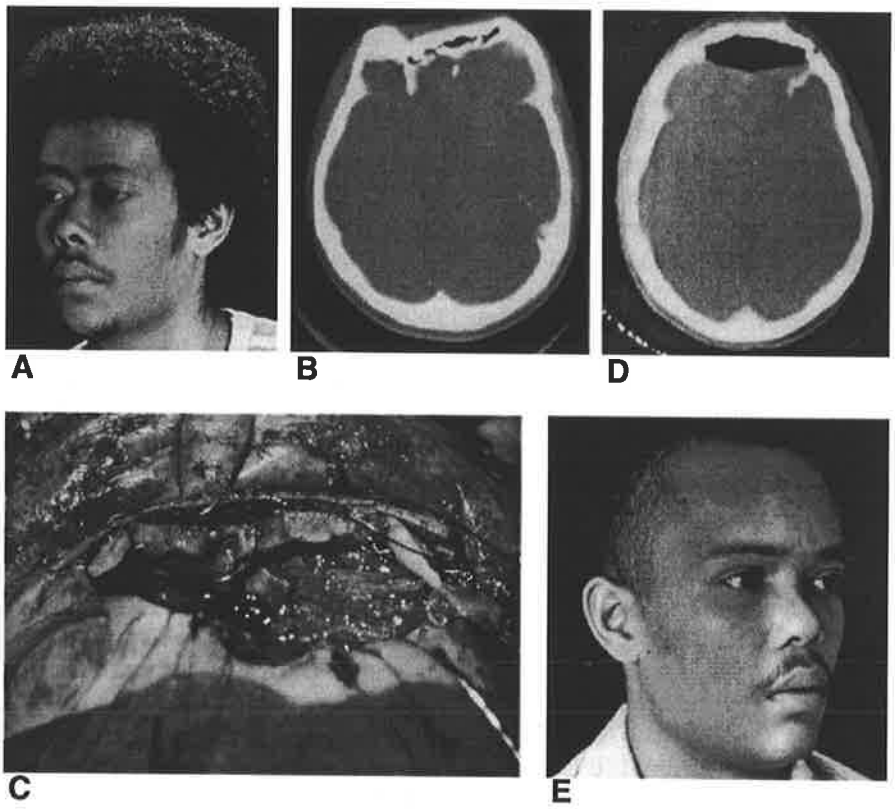


FIG 21.2. ***Delayed correction of cranial deformities.*** *A 20-year-old man was involved in an accident in a cane field in which his motorbike ran into a post. The post struck him in the forehead inflicting severe central frontal and frontonasal fractures. He did not receive any primary treatment, and presented some years later with an uncorrected fracture involving both walls of the frontal sinus and the glabella region.* ***A*** *Depression of the frontal bone, producing a contour defect and an obtuse frontonasal angle.* ***B*** *CT scan of the deformity as it affects the frontal sinus and its relationship to the frontal lobes.* ***C*** *Surgical exposure via bicoronal scalp flap and subperiosteal dissection to expose the fragmented and depressed skull. A craniotomy was performed above the affected region and the scarred dura was separated from the back of the damaged frontal sinus. Following repair of the dura, the frontal sinus fragments were replaced and augmented with split calvarial graft. All fragments were firmly wired into place and the frontal sinus was obliterated by cranialization. Additional bone graft was placed into the nose and the nasofrontal region.* ***D*** *Postoperative CT Scan with extradural air.* ***E*** *The young man at time of discharge with a properly contoured frontal bone and reconstituted frontonasal angle.*

Frontal Deformities

Surgical pathology

The most common post-traumatic frontal deformities are traumatic bone deficits, displaced supraorbital rims, flattening or concavity of a shattered frontal sinus and postoperative deformities such as sunken bone flaps and burrholes. Loss of bone secondary to osteitis is also a cause of disfigurement and often exposes the frontal lobes to the risk of future trauma. Wires or plates used in previous repairs may be prominent or even exposed by ulceration.

Management

Post-traumatic frontal recontouring can be achieved by bony advancement, autogenous bone grafts, bone allografts, metal plates or mesh, or implants of Silastic®, methyl methacrylate or other biocompatible material (p. 154). All methods of frontal cranioplasty have merits and demerits.

Correction by osteotomy
Forehead remodelling by osteotomy is often recommended for congenital deformities of the superior orbital margin and frontal bone resulting from craniosynostosis, and similar osteotomies can be performed for neglected post-traumatic deformities. But in traumatic cases, correction by osteotomy is a long operation that usually involves the adherent dura (Fig. 21.2) and occasionally the brain. The mucosa of the frontal sinuses must be eradicated down to the frontal recess; the osteotomized bone should be fixed rigidly and any defects grafted with calvarial bone graft. The potential for complications is high: these include infection in an inadequately vascularized bone, haematoma in the dead space between advanced bone and dura, and bone resorption from avascular necrosis. Epilepsy presumably caused by disturbing an underlying meningocerebral scar is a particularly serious complication.

It is not uncommon to see a lateral fronto-orbital contour deformity resulting from an uncorrected or poorly corrected fracture in this site. The fracture can be osteotomized and repositioned and fixed with miniplates. Exposure is through the bicoronal scalp flap with limited dissection of the temporalis muscles from the skull and lateral orbital wall to expose the old fracture. A small craniotomy or enlarged burrhole can be made to expose and protect the dura during the osteotomy. After contour restoration and fixation any remaining defect is filled with split calvarial bones.

Contour restorations by osteotomy can produce excellent results. The most frequent complications in our experience result from the dead space created by the advancement of the fronto-orbital margin when the connection with the nose has not been adequately obliterated. Care must be taken to cranialize the frontal sinus and to separate the extradural space from the nose. This can be effected by cutting a galeofrontalis flap from under the surface of the bicoronal scalp flap. If the dead space is small, it can be filled with this flap or by a pericranial flap based laterally on the temporalis muscle. More massive extradural dead space cavities may be filled with a free muscle transplant either from rectus abdominis or latissimus dorsi vascularized by microvascular techniques, (Fig. 21.3).

Correction by bone grafts
Bone is a logical material to use to fill a traumatic bone defect, and is often initially very successful. When the defect results from the removal of a frontal bone flap as an emergency neurosurgical decompressive procedure it is usual to preserve the flap in a bone bank and to replace it when the patient's condition permits. Frozen bone flaps sometimes resorb, but success is achieved often enough to justify this very simple and innocuous procedure.

Fresh autogenous bone harvested at the time of a reconstructive procedure gives early vascularization, excellent capacity for immobilization, lack of immune rejection and freedom from disease transmission. However, there is much increase in the operative time and some increase in morbidity (Laurie et al 1984). Small post-traumatic defects are commonly filled with calvarial bone, bone dust, rib or iliac crest (Fig. 21.4). Calvarial grafts can be taken through the exposure of the frontal bone defect for very large defects, parietal bone can be transferred and the posterior defect filled with another bone graft or with bone obtained by splitting the calvarial plate. When split calvarial bone is used, the inner plate insert in the parietal region and the outer plate is transferred to the frontal region. Split-rib grafts have been much used to fill large calvarial defects (Fig. 17.20). Medium-sized defects can be filled with a bone plug removed by trephine or a graft of cortical and cancellous bone cut to shape from the ilium (p. 240). Split inner table of ilium can used as a vascularized graft for very large defects where the vascularization is at risk because the overlying scalp has been compromised (Taylor et al 1979a,b, McCarthy et al 1987) (Fig. 21.5).

Apart from grafts revascularized by microsurgery, bone grafts are unpredictable and may resorb. A single grafting does not always suffice, though Phillips & Gruss (1991) have shown that fixation renders the survival of bone grafts more predictable than was previously thought. The degree of overgrafting necessary to produce an acceptable result varies and is hard to estimate. Rib and iliac crest grafts have been successfully used by many surgeons, and once they are incorporated, long-term infection or skin breakdown is unlikely. But it is not easy to get a perfect aesthetic result with split-rib grafts: Körlof et al (1973) reported uneven, depressed or prominent contours in half their cases. It is common experience to be obliged to reoperate to smooth the surface of a frontal bone graft and to remove wires and screws which have become prominent from partial resorption (Fig. 21.6). It is also questionable whether free bone grafts have the impact resistance of a metal plate: Timmons (1982) noted progressive loss of bone density in later X-ray pictures.

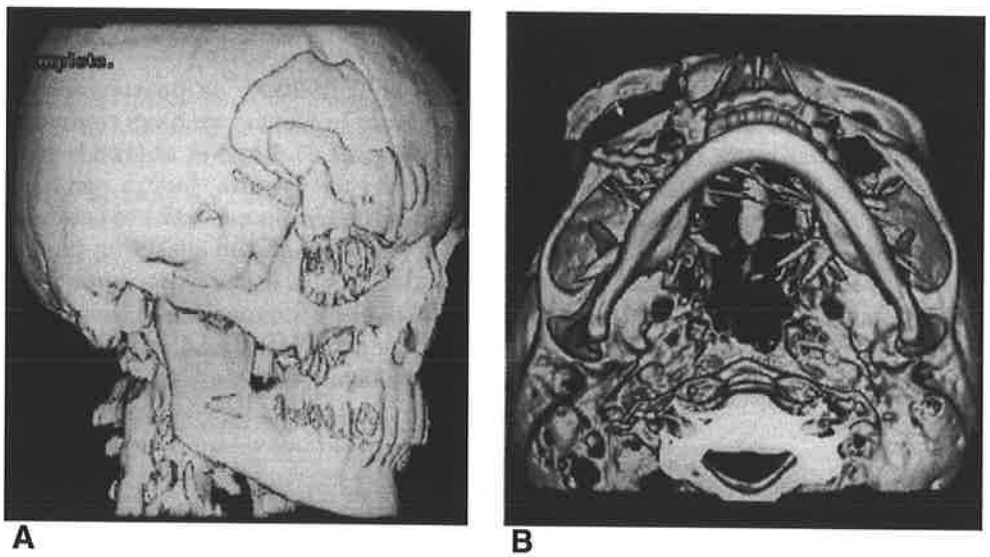


FIG. 21.3. ***'Dead space' from cranial fracture.*** *A middle-aged woman suffered a compound, comminuted fracture of the right fronto-orbital region affecting the shape of the orbit and producing a flattening of the right eyebrow region. A CT scan showing the relationship of the distorted fronto-orbital crown and orbit. B Fronto-orbital advancement was performed on the right side with an osteotomy, repair of the underlying dural defect, cranialization of the frontal sinus and blocking of the nasofrontal ducts. Postoperatively she blew her nose violently and produced an extradural air pocket which became filled with fluid.*

Metal and plastic implants

These are often used for large frontal cranioplasties (Fig. 21.7). Titanium plates offer excellent protection and are easily screwed to the skull. The Belfast technique of shaping titanium plates in a high-pressure hydraulic forming chamber (Gordon & Blair 1974, Blair et al 1980) makes it possible to give a nearly perfect aesthetic effect; the plates are radiolucent and we have not found the thermal conductivity of the titanium plate to be a cause of complaints. The application of three-dimensional computed tomography (3D CT) has provided an even more elegant method of producing titanium plates: with the aid of 3D CT data, a model of the defect is sculpted and from this a plate of the desired thickness and shape is cast in titanium (computer-aided design/computed-aided manufacture: CAD/CAM technique). Titanium has high biocompatibility (Fig. 5.28), and in the short term we have been well pleased with metallic cranioplasties. Unfortunately the long-term results are not always satisfactory. We have records of some 150 patients with post-traumatic skull defects repaired with tantalum or (since 1961) titanium plates, and in at least 16 (11%) it has been necessary to remove the plate because of infection or ulceration of the skin. The incidence of such complications may indeed be higher as many patients have left our area and their plates could have been removed by neurosurgeons too kind to tell us the bad news. In some cases the complication can be blamed on preventable errors in technique, but we have seen infection supervene 20 years after a satisfactory cranioplasty, from a superficial scalp wound.

Acrylic (methyl methacrylate: p. 155) is cheap and easy to shape; Ousterhout & Zlotolow (1991) have reported good results. Manson et al (1986) have found a low incidence of infection though, when this does occur, the results can be devastating. The plates are less resistant to impact than titanium and may fracture (Otto 1958, Henry et al 1976). This can be rectified by reinforcing the acrylic with metallic mesh (Galicich & Hovind 1967), though in our hands these reinforced plates have been aesthetically disappointing: when the skull is thin, the wire-reinforced plate margins are hard to inlay and are palpable and visible when fitted as an onlay prosthesis. Long-term complications, such as infection, are not rare and we have the impression that these complications are not less frequent than with titanium plates, though to our knowledge no comparative clinical trials have been reported.

We have only rarely used silicone rubber (Silastic®) as postoperative reconstructive material in the craniofacial skeleton; however, we have removed many pieces of silicone rubber used by others (Fig. 21.8). Lash et al (1964) and Courtmanche & Thompson (1986) have reported good results, but in some of their cases the follow periods were short. Silastic® implants are hard to scull do not unite with the underlying skull and often erode through the overlying tissue which may itself be compromised by the original injury. It is our experience that these implants are often placed within the soft tissues by inexperienced surgeons: a capsule forms around the imply which becomes prominent and may migrate under gravity producing an unsightly frontal appearance.

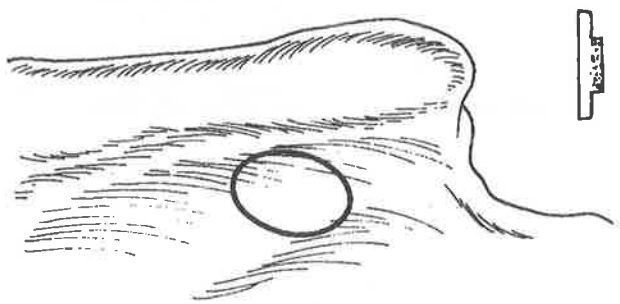


FIG. 21.4. ***Post-traumatic cranial defects.*** *Medium-sized post-traumatic defects in the frontal region can be effectively closed with a 'bath plug' graft from the inner table of ilium. Cortical and cancellous bone is harvested and shaped as a bath plug so that the cortical bone overlaps the frontal bone adjacent to the defect and the cancellous bone fits as a plug into the defect. Two small screws provide rigid fixation.*

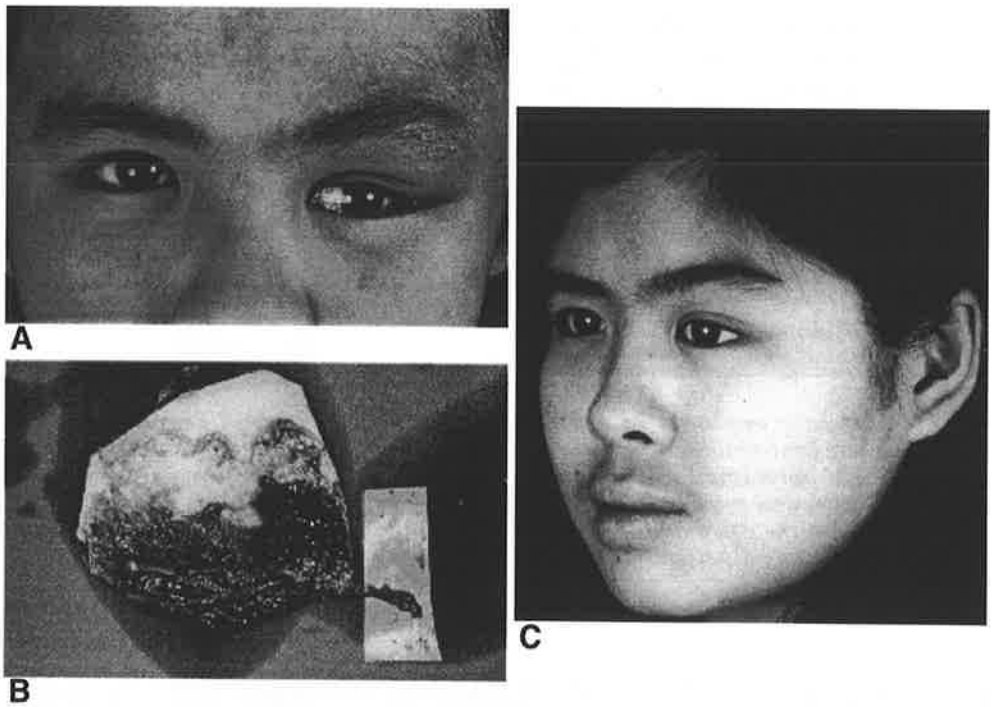


FIG. 21.5. ***Vascularized graft reconstruction of cranial defects.*** *Split inner table of ilium, in the form of a free flap based on the deep circumflex iliac vessels, has been used to reconstruct the frontal bone defect.* **A** *Distorted frontal region with some degree of global dystopia* **B** *Inner table of ilium with attached periosteum and vascular pedicle. The quite pliable inner table has been cautiously bent and fixed into the defect peripherally with titanium screws, the pedicle placed Within reach of the temporal vessels.* **C** *The patient 1 year after the reconstruction.*

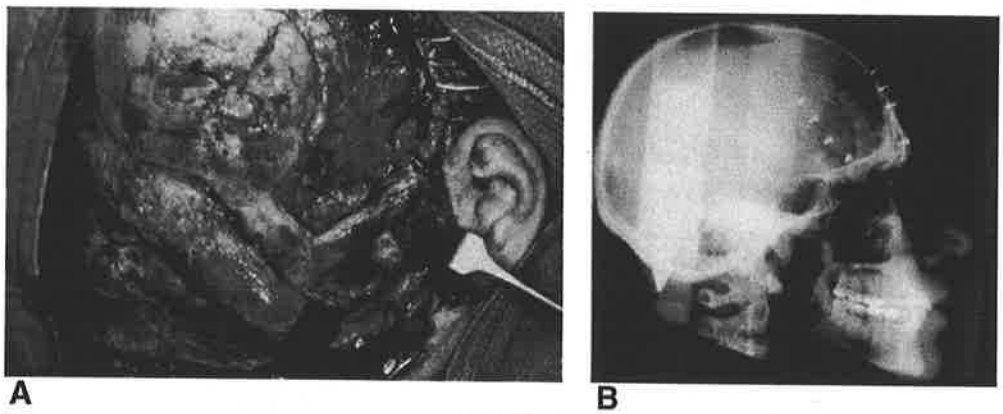


FIG. 21.6. ***Rib grafting for cranial defects.*** *A woman was involved in a vehicular accident and suffered severe fronto-orbital and left facial fractures.* **A** *Primary reconstruction performed with split-rib grafting of the frontal region. Later, the grafted area became rough and uneven, with resorption resulting in palpable grooves and wires.* **B** *Split-ribs used for frontal cranioplasty and fixed with titanium screws.*

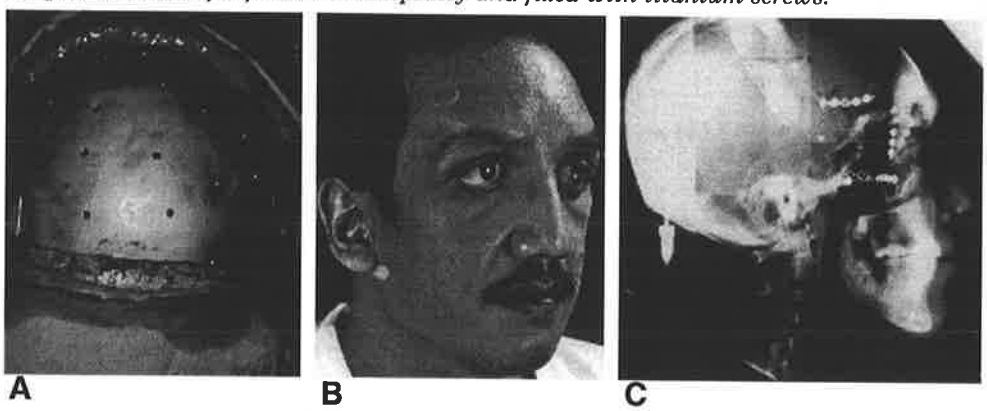


FIG. 21.7. ***Frontal cranioplasty using a large titanium plate.*** **A** *Operative view showing plate in position.* **B** *Half-face view showing the frontal contour produced by the plate.* **C** *Lateral X-ray view showing radiolucency of the plate.*

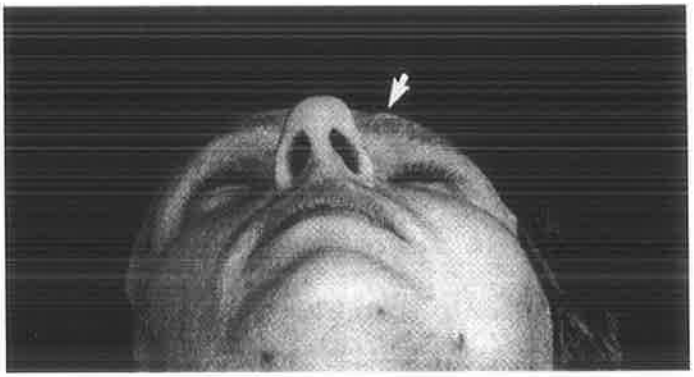
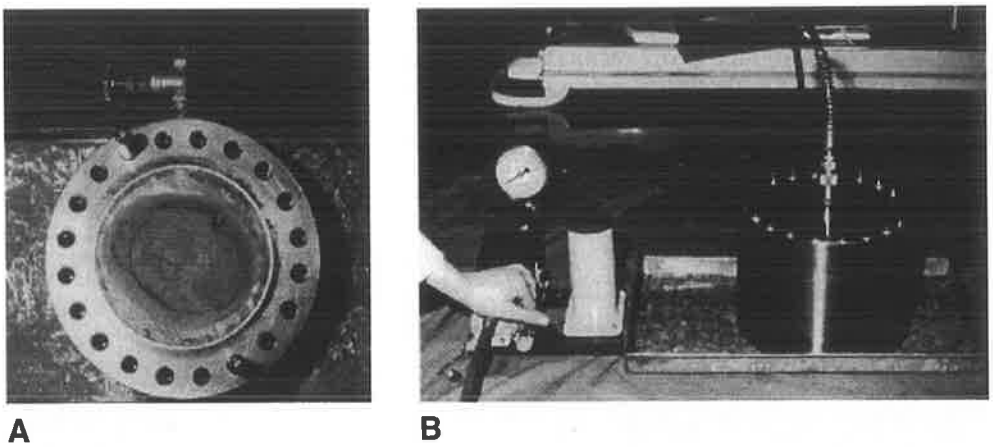


FIG. 21.8. ***Silastic implants in craniomaxillofacial trauma.*** *A 19–year old man suffered a severe left frontal–orbital fracture in a motor vehicle accident, leaving him with a depressed left supraorbital margin and frontal bone. This was inadequately treated at the first operation and subsequently managed by inserting a carved block of Silastic into the plane between the galea and the skin. The resulting soft–tissue capsule (arrow) was bulky and mobile on palpation.*



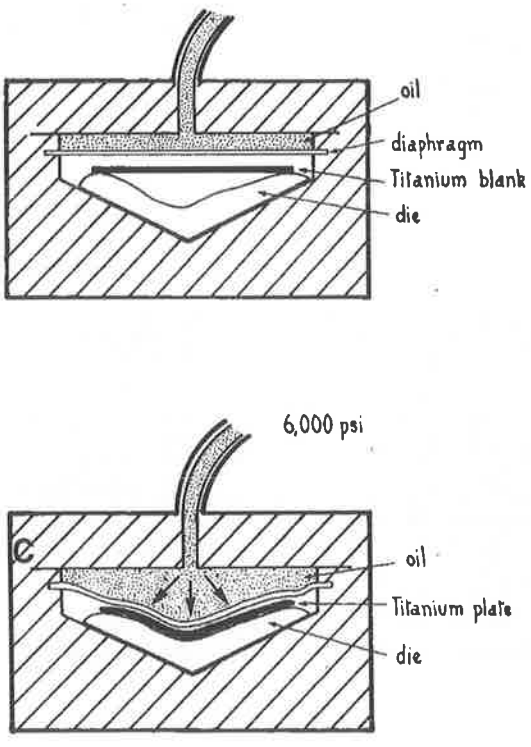


FIG. 21.9. ***Contouring a plate in a pressure chamber: Belfast technique.*** *An impression of the bone defect is taken in wax; from this a plaster model of the calvarial region is made.* ***A*** *A Matrix of the plaster model is made in dental stone and fitted into the cylindrical pressure chamber. A titanium blank plate, cut to be larger than the bone defect and of approximately the same shape, is then placed in chamber on the matrix.* ***B*** *The chamber is closed with 20 screws and a hydraulic pump raises the pressure to 6000 psi.* ***C*** *Diagram of action. Above, the titanium blank is lying on the matrix under a thick rubber diaphragm; below, it is forced by high pressure to conform with the contours of the matrix. The moulded plate is trimmed and polished and holes are drilled.*

Alloplastic cranioplasties will continue to show complications until a material is found which is permanently incorporated in the tissues. Hydroxyapatite may be such a material (p. 155). Waite et al (1989) have reported on a small series of cranioplasties done with blocks of hydroxyapatite; they report some difficulties in contouring the block, which can be overcome by preforming with the help of a model prepared from a 3D CT scan. Hydroxyapatite is brittle, but is said to become acceptable impact-resistant when invaded by fibrous tissue. Demineralised allogenic or xenogenic bone prepared by freezing, lyophilization and irradiation has been advocated. We have no experience of treating frontal contour defects with these substances. We note that Ousterhout (1985) reported that there was considerable implant resorption and that the results of contour restoration were unsatisfactory.

Implants are *contraindicated* within 6 months of any local infection; a previously damaged paranasal sinus increases the risk. No cranioplasty should be attempted unless there is good skin cover over the area to be repaired. The dura mater should be intact, and if the paranasal air sinuses were involved, then the area of involvement should have been covered with soft tissue. These requirements sometimes necessitate a staged sequence of operations over 6 months or more.

Cranioplasty with titanium

The plate is made before operation. Blake et al (1990) use plates shaped and contoured by hand, with the help of a model of the defect prepared from CT scan data; where necessary, plates are welded together. In our experience, plates made by hand are satisfactory for closing defects when simple contours are needed; a wooden mallet and a concave anvil can be used to form a convex plate, final shaping being done with pliers and a metal ball hammer. However, plates made in this way are sometimes unsatisfactory when complex defects have to be closed.

We now prefer the Belfast technique of preforming the plate in a pressure chamber (Blair et al 1980). When this method is used, an impression of the defect is made through the intact skin with dental alginate or wax, and a model of the forehead is then made in dental plaster. From this, a matrix is made in dental stone; the matrix is placed in the forming chamber. A sheet of 0.6 or 0.7 mm titanium is cut to cover the defect with a clearance of at least 15 mm, and laid on the matrix; the sheet of titanium is placed between two sheets of mild steel to avoid crumpling of the plate in the chamber (Fig. 21.9) The chamber is sealed and a hydraulic pressure of ~6000 psi is applied. The plate is moulded by the pressure to conform with the impression in the dental stone matrix. The shaped plate is removed from the chamber; screw holes can then be drilled and slots cut to allow bending of the plate margins, since a perfect fit is rarely obtained when the frontal bone has been modelled through the intact skin. Finally the plate is polished and its edges smoothed. It is sterilised by autoclaving. The bone defect is exposed by a coronal scalp flap, possibly modified to include an earlier scar (Fig. 21. 10A). The scalp is elevated by sharp and blunt dissection, care being taken not to open the dura or the frontal air sinuses. The plate is then made to conform with the exposed bone exactly; this usually entails some swaging and bending. Pericranial flaps are elevated to define the margins of the bone defect. The bone is prepared for the plate by raising a thin shaving of bone all around the outline of the plate; (Fig. 21.10B): this covers the edges of the plate and is intended to promote osseo-integration (Fig. 5.28). The plate is secured with at least four titanium screws; in the past, the slotted 5 and 6 mm screws made to our design (Simpson 1965) have been preferred to the smaller screws used to hold miniplates, but the heads of these smaller screws are less conspicuous and recent experience suggests that they may give adequate fixation. In drilling tapping holes for the screws, care is needed not to tear the dura; it may be wise to put oxycel gauze in the extradural plane under the site of the screw. The pericranium is pulled over the edge of the plate and the skin flap is replaced with or without suction drainage.

With the advent of 3D CT scanning and the ability to cast titanium implants using the CAD/CAM technique,* the plate can be shaped with much more precision (Fig. 21.11), and a perfect fit can be achieved (Fig. 21.12).

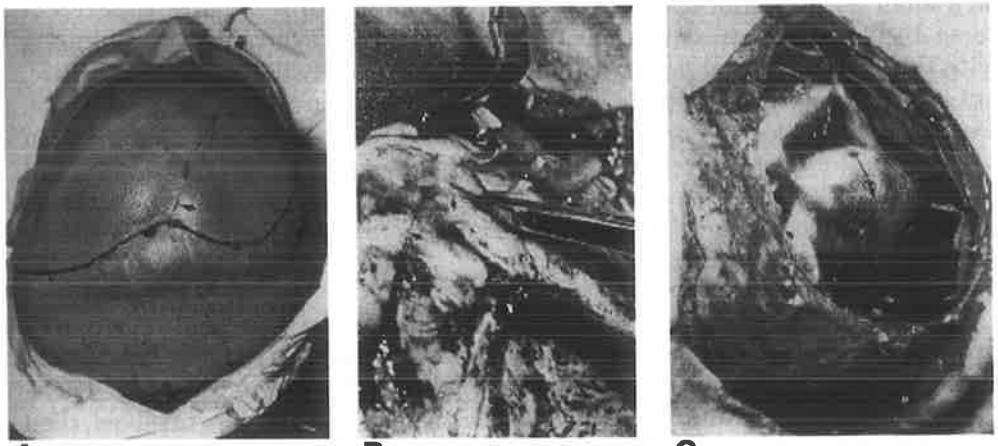


FIG 21.10. ***Fitting a moulded titanium plate.*** *A 4-year-old boy suffered a compound depressed frontal fracture. Scalp closure was difficult, and the bone fragments were not replaced. The wound healed well.* **A** *7 months later, the bone defect was explored through a coronal scalp flap, modified to ensure that the incision would not lie directly over the defect. One limb of the original scar (dotted line: arrow) was not reopened.* **B** *The defect was defined and pericranial flaps were reflected from it. The preformed plate was fitted to lie on the bone. A line (arrows) was marked on the bone, and shavings were elevated with a chisel along this line to overlap the margin of the plate.* **C** *The preformed plate was secured with four slotted screws and the pericranial flaps were brought to cover the edges of the plate. Recovery was uneventful. This technique has been shown to promote osseo-integration of the plate.*

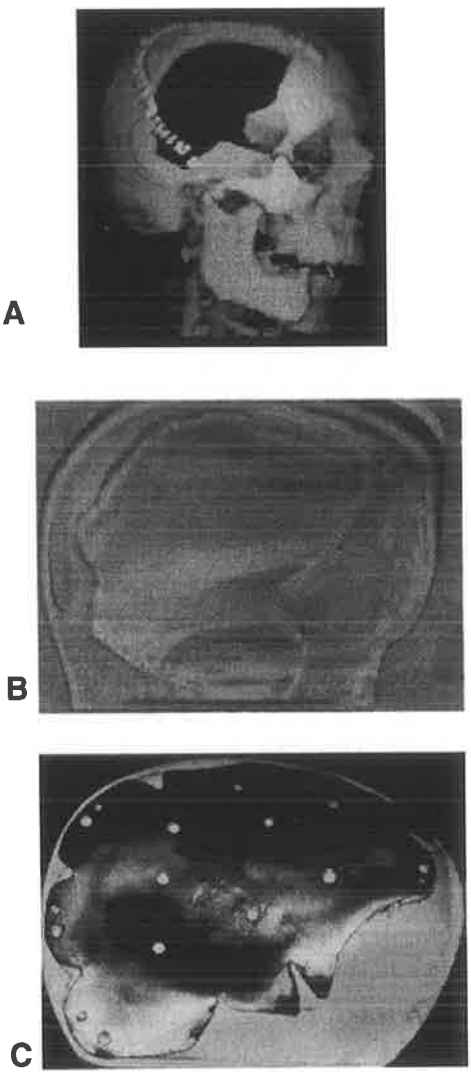


FIG. 21.11. ***Casting a plate: CAD/CAM technique.*** **A** *CT scan shows a post-traumatic frontotemporal craniotomy defect; the scan was obtained soon after operation. Scalp clips outline the wound.* **B** *Using PERSONA software analysis, a computer-milled model of the defect was obtained and from this a mould for a plate was made.* **C** *A titanium plate was cast in this mould in an inert atmosphere at 1720°C with Tycast 2000 centrifugal casting equipment. (Jeneric Pentron 53N, Plains Industrial Road, Wallingford, CT 06492, USA.) The cast plate is shown fitted to the model.*

* For modelling these plates, we have used Persona software, available from Maptek Pty Ltd. 350 Indiana St (Suite) 530, Golden Colorado USA.

Cranioplasty with autogenous bone

When a frontal defect is closed with autogenous bone, the exposure is similar. The bone edges must be more fully exposed and freshened with a chisel or large dental burr designed for shaping acrylic; bone wax is not used in situations where it might delay bone growth. If rib is to be used, a suitable number of ribs is harvested and the ribs are split as described on p. 241. The ribs are cut to appropriate lengths and secured to cover the defect: the end of the rib can be inlayed into the freshened margins of the bone defect, and secured with steel or titanium wire. However, small titanium screws make rigid fixation possible with greater speed and simplicity. Bone dust obtained from the calvaria near the defect can be used to improve the contour of the repair. A plate of iliac bone may be preferable for a smaller defect or may be used in conjunction with rib grafts for a larger defect.

Split calvarial grafts (p. 241) may be used for smaller defects when X-ray has shown that there is a good calvarial thickness. For larger defects and a superior cosmetic result, a temporoparietal bone flap is removed by a neurosurgeon and the inner table is split off in one or more segments (Fig. 21.13). These segments can be used to repair the secondary bone defect created by the craniotomy while the outer table can be used for the frontal defect. If a vascularized graft is needed, the bone of choice is the inner table of the ilium. This can be split, providing a large area of bone and leaving little contour defect in the hip. The indications are not common but occur in situations where the defect is large, the overlying scalp much damaged or after failure of other methods.

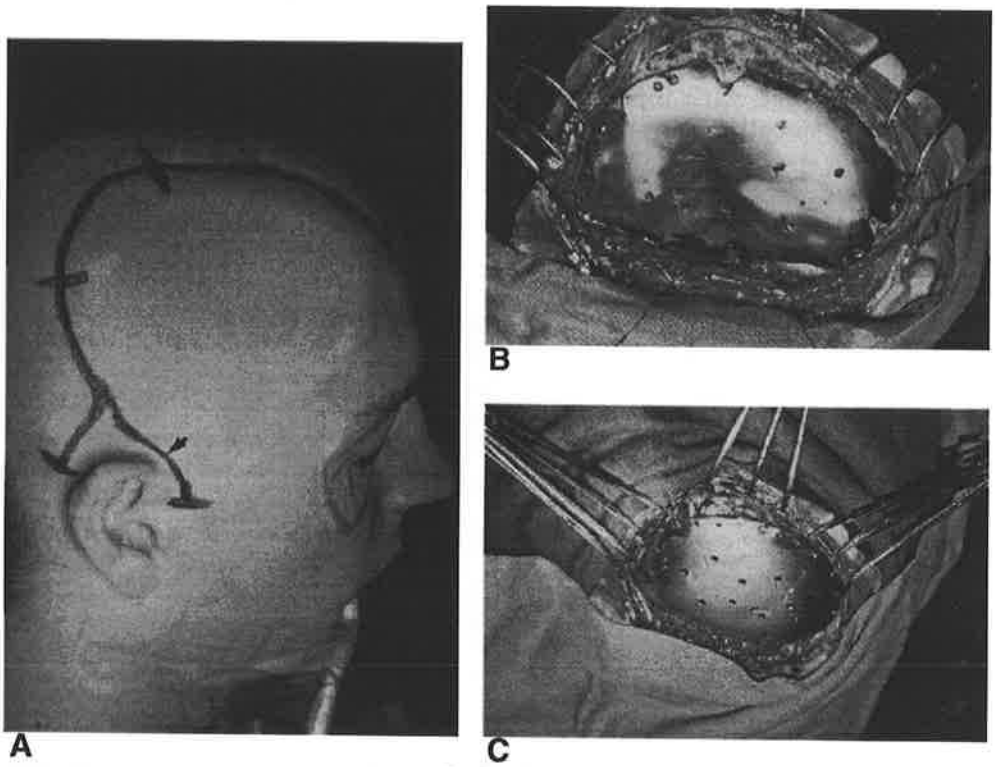


FIG. 21.12. *Fitting a cast plate made by CAD/CAM technique.* *A 19-year-old man suffered a compound depressed fracture in the right frontotemporal area. Decompressive craniotomy was performed to relieve cerebral oedema. The bone fragments were shattered and it was decided that they were unsuitable for a future cranioplasty. (Fig. 21.11 shows the resulting bone defect and the preparation of the plate) **A** 7 months later, the bone defect was explored through the original incision with a postauricular extension; the lower limb of the incision (arrow) was not reopened to ensure that the plate was not under the final scar. **B** The bone defect was defined, and flaps of pericranium and temporalis fascia were elevated. A pocket in the temporalis muscle received the lower margin of the plate. The precast plate conformed perfectly to the margins of the bone defect and was secured with screws. The concavity in the temporal fossa is seen as a shadowed area in the plate. **C** By contrast, a plate moulded in the pressure chamber is shown in the same patient: it had not been possible to make a matrix with such an exact replication of the temporal contour, and the temporal concavity was not reproduced. This plate was discarded. Case report by courtesy of Mr M Stoodley.*

The technique demands the skills and experience of a microsurgeon. A separate surgical team exposes the ilium (p. 240). A free flap of split ilium vascularized from the deep branch of the circumflex iliac artery is detached from the inner table of the bone and anastomosed to one of the scalp arteries; a cuff of muscle is transferred with the bone to protect the arterial supply. The flap of bone is firmly fixed in the defect. The maximum area of bone that can be taken is about 12 × 8 cm; however, larger defects can be filled by a combination of vascularized iliac bone and conventional rib or calvarial grafts. The procedure has the advantages of vascularized bone and the ilium gives a reasonably appropriate contour, which can be further improved by bending. But the dissection is difficult, and the need to establish an anastomosis with the superficial temporal artery restricts its use somewhat. It seems to be most useful in the frontotemporal area. We have done this in four cases, only one of which was post-traumatic.

Choice in cranioplasty

At present, our preferences are divided between autogenous bone and titanium for cranioplasty when a large frontal defect has to be repaired, but we are not blind to the occasional failures with both materials. Results appear to be better where fresh autogenous bone is used, and have been further improved with the use of rigid fixation by miniplates and screws. But in almost all cases, plates screws and wires become prominent when swelling subsides and bone resorption takes place. Except for small defects, the best aesthetic results have been obtained with large fragments of split calvaria and with titanium. Calvarial cranioplasty seems to be more satisfactory in the long run. Split-rib cranioplasty tends to remain uneven and this may show in the frontal region (Fig. 21.6)

Therefore, as a general rule, we favour autogenous bone grafts. Small defects are plugged with inner table of ilium, having some cancellous bone attached—the so-called bath-plug graft. Medium-sized defects are closed with split calvarial grafts. Large defects with compromised soft-tissue cover are repaired with the split ilium free flap. Nevertheless, titanium plates remain very popular, especially when the patient requires maximum protection against recurrent cranial trauma.

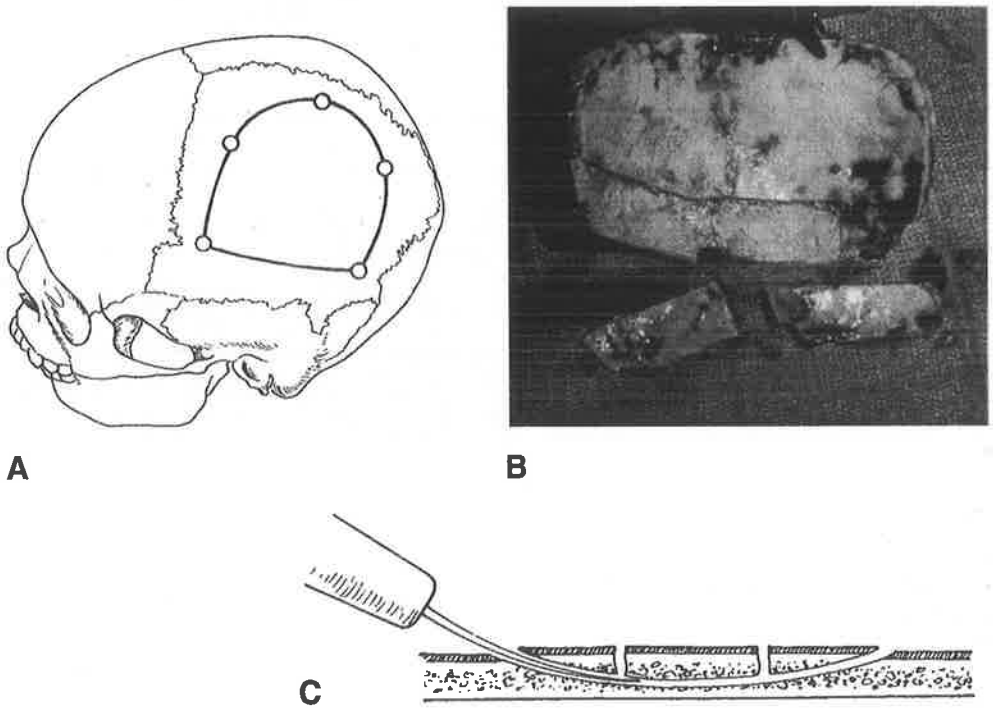


FIG. 21.13. ***Split calvarial bone grafting.*** ***A*** *Frontal defects are exposed by turning the bicoronal scalp flap forwards; the parietal region is exposed by turning the flap backwards. A parietal bone flap is raised and split.* ***B*** *Strips taken from the inner table are replaced to fill the secondary defect, and the outer table is placed into the primary defect and screwed or plated into position. Thin two-hole plates with low profile screws are used.* ***C*** *Smaller defects can be filled with the outer table of calvarium, split through the diplöe.*

FIG. 21.14. ***Temporal hollowing.*** *A 50-year-old man involved in a flying accident required a bicoronal scalp flap for exposure of the upper and middle face. Several months after the initial surgery, temporal hollowing resulted from displacement of the temporalis muscle from its normal attachments.*

Temporal Deformities

Surgical pathology

Hollowness in the temporal region can be due to a sunken or displaced temporal bone flap. More commonly in post-traumatic cases, the deformity follows a surgical procedure in which the temporal muscles have been elevated from their calvarial attachments above and from their orbital attachments anteriorly, when exposed through a bicoronal scalp flap (p. 238). When this occurs the lateral margin of the lateral orbital rim and the zygomatic arch often appear very prominent and angular.

Management

The temporal concavity resulting from elevation of the temporalis muscle from its attachments can be prevented by careful reattachment of the temporalis fascia to the superior temporal line, the lateral orbital margin and the superior surface of the zygomatic arch. When an established deformity has to be corrected (Fig. 21.14), the area is best exposed by the bicoronal scalp flap. The temporalis muscle is dissected out of the temporal fossa down to the level of the zygomatic arch. The temporalis muscle must be reattached, but it is often also necessary to fill the temporal hollow by placing bone or some other substance underneath the muscle. Bone is preferred, with rigid fixation. Repair is completed by suturing the superior margin of the temporalis muscle to the pericranium above as well as to the lateral orbital rim using a few holes drilled along that margin.

Orbital Deformities

Surgical pathology

These deformities generally involve some or all of the following:

1. Enophthalmos due to expansion of the orbital walls, floor and/or roof, expanding the orbital volume; there may also be damage to or loss of orbital contents (Fig. 21.15)
2. Rarely exophthalmos, with or without pulsation due to loss of the roof or caroticocavernous sinus fistula (p. 392)
3. Naso-ethmoid deformity, often with loss of function of the nasolacrimal apparatus (Fig. 21.16).
4. Extra ocular muscle dysfunction.

Orbital deformities established in childhood

Naso-orbital and orbito-zygomatic fractures are rare in young children, less so in the older child. Post-traumatic orbital deformities in the younger child have special features resulting from interference with normal growth.

True hypertelorism may result from an anteroposterior crush injury in infancy, causing genuine separation of the orbits (Fig. 21.17). More often, nave-orbital fractures cause traumatic telecanthus which tends to become more prominent as growth proceeds; there is often an associated depression of the nasal bridge and lacrimal drainage dysfunction (Fig. 21.18).

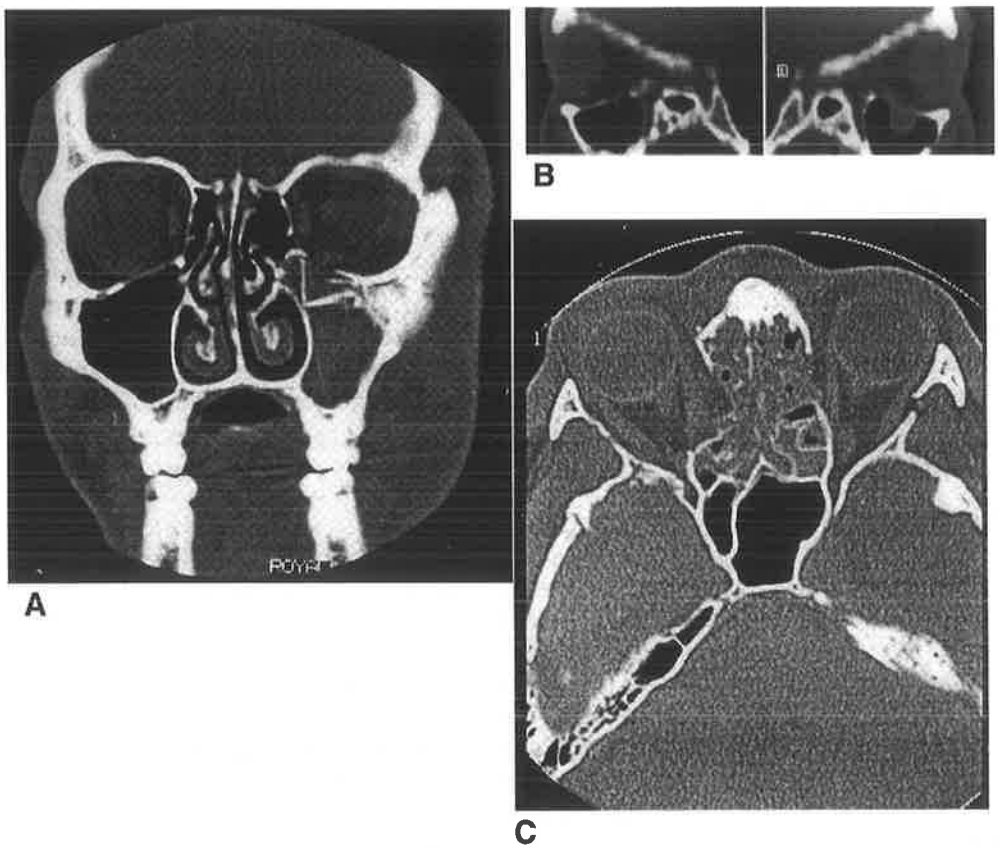
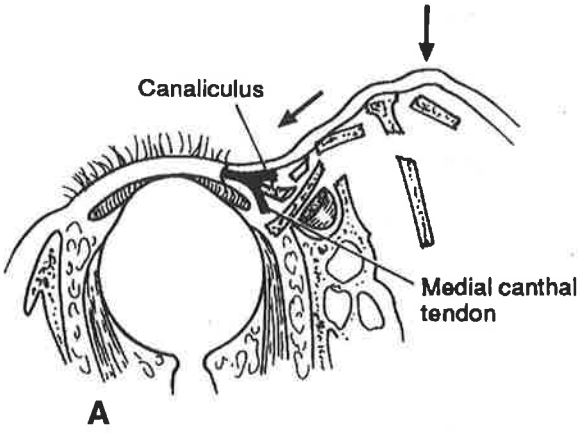


FIG. 21.15. ***Enophthalmos secondary to traumatically increased orbital volume.*** *A Coronal CT scan showing damage to the orbital floor, lateral orbital wall, and medial orbital wall, producing expansion of the orbit.* ***B*** *Sagittal reconstruction showing the expanded orbital floor.* ***C*** *Axial view showing the medial orbital wall collapsed into the ethmoid, expanding the orbit in this direction. The effect of the malar displacement on orbital volume is also evident.*



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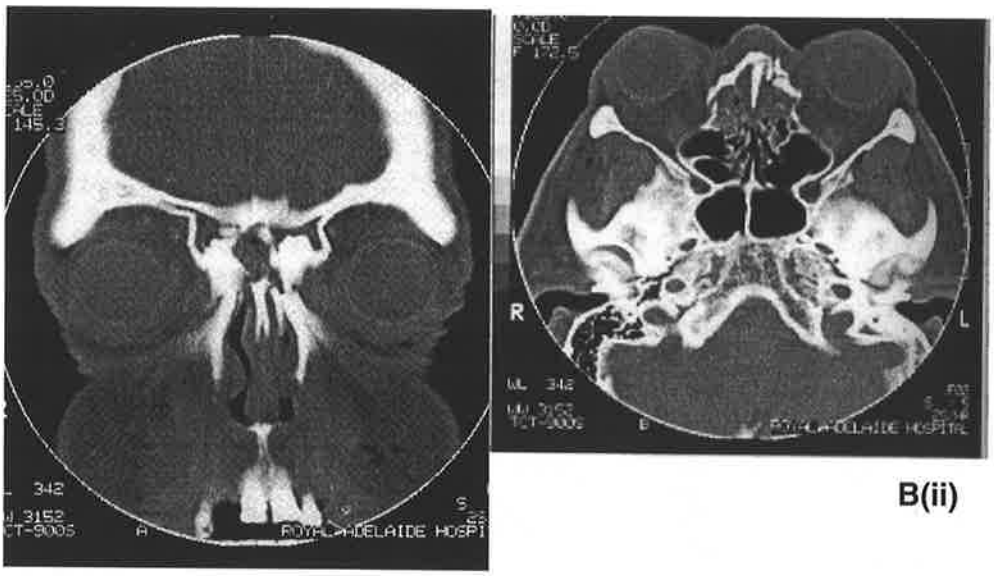


B(ii)

B(i)

FIG. 21.16. ***Injury to the nasolacrimal apparatus.*** *A Relationship of the nasolacrimal apparatus to the lacrimal fossa and the medial orbital walls in crush injury of this area. Not only is the bone disrupted but the delicate nasolacrimal apparatus is endangered.* ***B*** *CT scan of a crush injury to the naso-orbital-ethmoid region: (i) axial view showing the complete disruption at the level of the canthal attachment; lacrimal drainage is impaired; (ii) coronal view showing the buckle in component of the deformity.*

Loss of an eye during the phase of growth of the globe and orbit results in reduced orbital size, presumably because the orbit is deprived of its functional matrix (P. 77).

Management

The results of treatment of established post-traumatic orbital deformities in the paediatric population depend on the age of the patient and the timing of treatment. As a general principle, surgical treatment should be delayed until growth is complete; malunited fractures should not be treated in the period of craniofacial growth by refracturing or bone grafting (Manson et al 1986). Definitive surgical corrections are best performed when orbital growth is complete. If it is necessary to intervene earlier then multiple surgical interventions will probably be required. Most orbital growth is complete by the age of ~6 years and osteotomies in the orbital region can be performed at or after this time. However, onlay bone grafting of the region is likely to need later repetition because of resorption.

Enophthalmos

Surgical pathology

Residual orbital deformities can result from inadequate primary treatment or from massive loss or damage in the orbital margins and walls. Lesions of the orbital contents such as the globe, muscles, fat and lacrimal apparatus are frequently associated with malunited orbital fractures.

If the orbital deformity expands the orbital volume, or is associated with a bone defect into which orbital contents can herniate, then there will be enophthalmos; the recession of the globe will be exaggerated if there is loss or atrophy of any part of the orbital contents.

Management

The late correction of enophthalmos should be possible if the orbital contents can be replaced and the walls of the orbit reconstructed. This has in the past been considered extremely difficult if not impossible (Converse et al 1977, Dingman 1978). Today, the better understanding given by CT scanning and the capability of 360° circumferential access and full dissection of the orbit have together made the correction of enophthalmos possible— but not always certain. Prevention of orbital deformity by initial operative reduction is always desirable, and with good imaging, wide exposure and primary bone grafting, the need for late correction has receded.

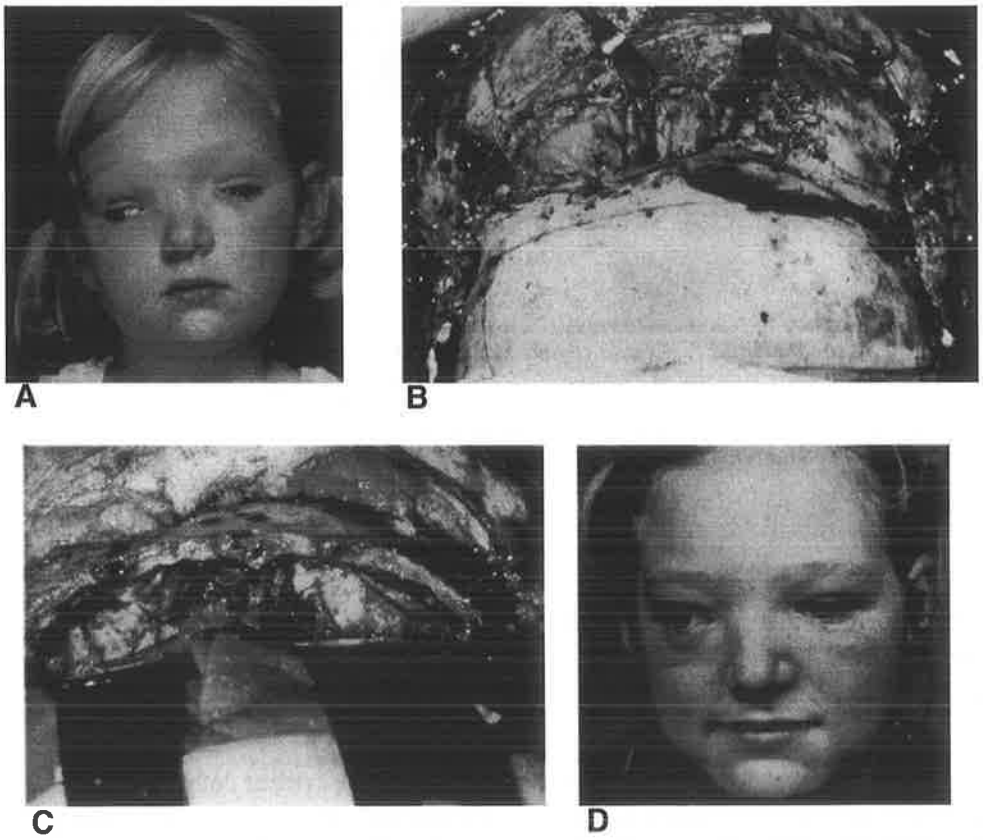


FIG. 21.17. ***Traumatic hypertelorism.*** *A 3-year-old girl presented with traumatic hypertelorism having been run over by a tractor as a neonate. In spite of the heavy weight, the pliable skull and soft ground saved her life but compression produced a splaying of the orbits, including the lateral wings of the spheroid bone, reminiscent of congenital defects with hypertelorism. This is a very rare situation and, we believe, only occurs under similar conditions.* ***A*** *The child on presentation with obvious traumatic hypertelorism and increased lateral canthal distance, medial canthal distance, and interpupillary distance.* ***B*** *The left orbit is displaced laterally.* ***C*** *When the anterior fossa is exposed the shape and direction of the roof of the orbit can be seen to be rotated to the left, and the widened interorbital distance is reflected in the midline of the anterior cranial fossa.* ***D*** *10 years later she still has a squint on the right side, widening of the intercanthal region and some tearing due to the disruption of the nasolacrimal apparatus on both sides.*

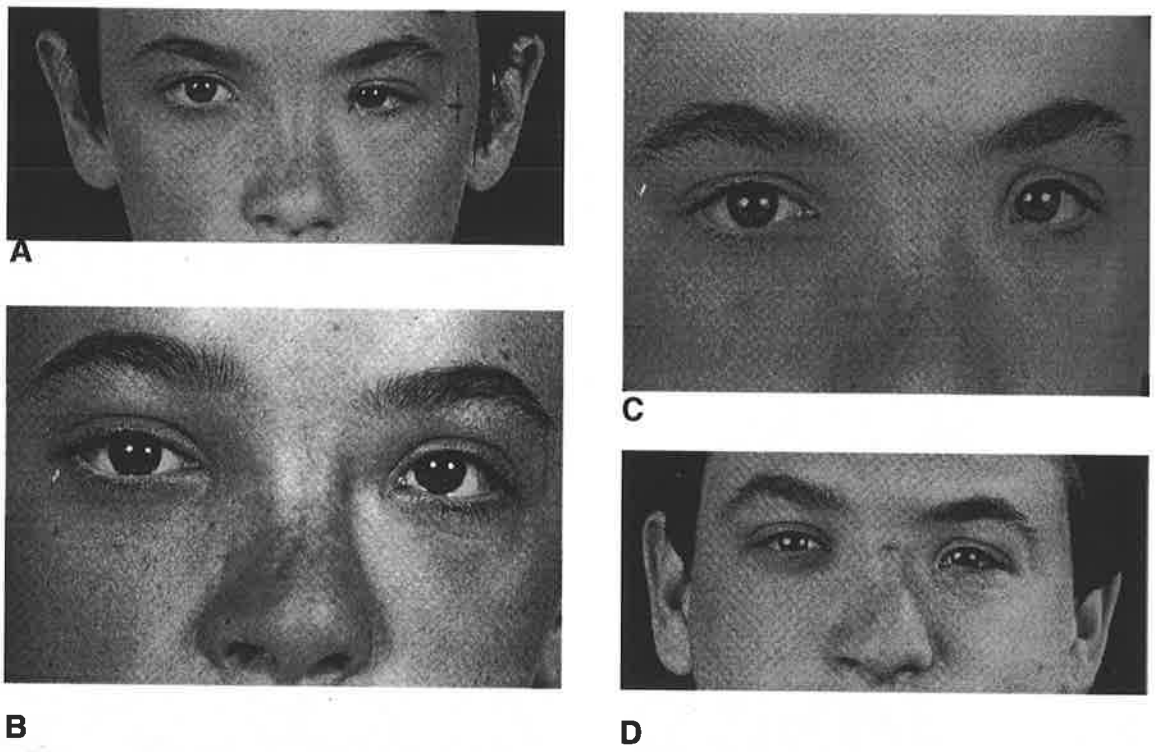


FIG. 21.18. *Orbital fracture in childhood.* *A 6-year-old boy fell from a cliff. He presented with a distorted nasal bone and medial orbital wall area around the nasolacrimal apparatus. In spite of appropriate primary correction the damage and obstruction persisted. When growth was completed he required definitive surgery.* **A** *Appearance shortly after medial canthopexy and orbital wall and nasal reconstruction.* **B** *The naso-orbital region a month later, with signs of tearing and canthal drift.* **C** *Appearance 2 years later with the canthal drift worsening and the shape of the palpebral fissure markedly changed.* **D** *Appearance 8 years after the original injury. Further corrective surgery has been performed to replace the canthus and build up his nasal bridge, as well as dacrocystorhinostomy.*

Orbital floor defects

Floor defects, on which all attention was previously focused, are often associated with medial wall defects or with an enlarged inferior orbital fissure; the orbital contents prolapse into these defects and adhere to them.

The damaged orbital floor can be exposed by the approaches described in Chapter 9 (Fig. 21.19). For the simplest problems, such as a small 'blow-out' fracture, the transconjunctival approach to the orbital floor is adequate; a proximal conjunctival flap is elevated and held out on a stay suture to protect the cornea (Tessier 1973, David 1974) (Fig. 9.5). For more extensive exploration when wiring or plating may be necessary, the incision may be extended by a lateral canthotomy. However, care must be taken in repairing this as inaccurate restoration can produce tethering and sometimes a thickened scar.

The blepharoplasty incision incorporating a musculocutaneous flap also gives wide exposure but results in an external scar albeit a usually excellent one. In this approach, a small incision 3–4 mm long is made in the crow's foot crease from the lateral canthus downwards and laterally, through skin and muscle. The points of curved iris scissors are inserted deep to the muscle and the musculocutaneous flap is developed across the whole width of the lid and up to the lid margin. The scissors can then be inserted with one blade deep to the musculocutaneous flap and a cut made 1–2 mm below the lid margin. This musculocutaneous blepharoplasty flap provides very wide exposure to the upper anterior maxilla and the orbital floor. We rarely use lower lid incisions because of the resultant scarring and potential lower lid oedema.

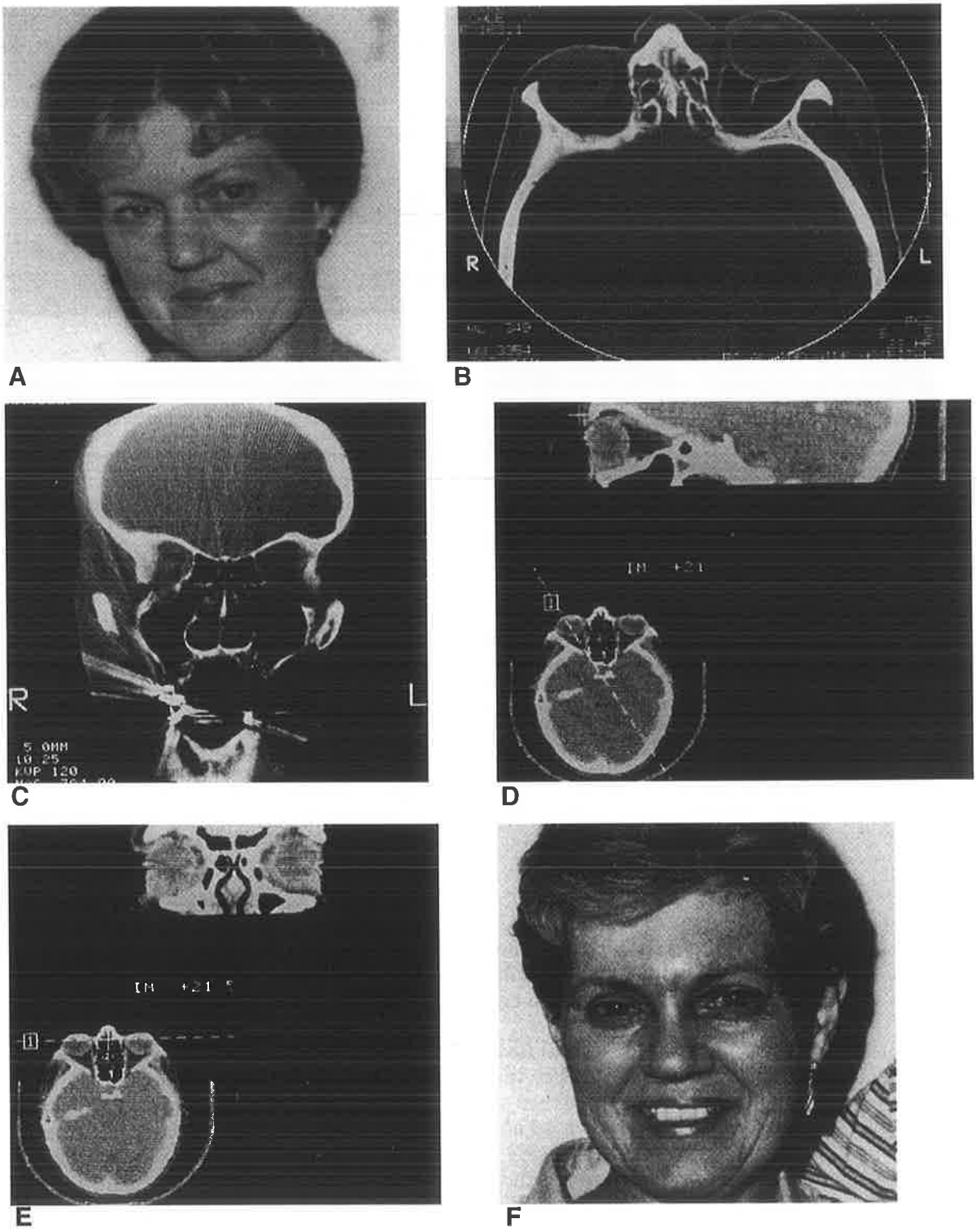


FIG. 21.19. ***Complications of orbital trauma. Depressed orbital floor, uncorrected for almost 10 years.*** *The patient had enophthalmos and persisting diplopia.* ***A*** *Initial presentation with enophthalmos, slight global dystopia on the right side, and the head tilted to compensate for diplopia.* ***B*** *Axial CT shows the enophthalmos, with intact medial and lateral walls.* ***C*** *Coronal CT shows the downward expansion of the orbital floor with orbital contents prolapsed into the antrum.* ***D*** *Sagittal reconstruction demonstrating how iliac crest cortical bone graft to the orbital floor sits over the defect and supports the globe.* ***E*** *2D reconstruction in a coronal plane, showing the globe supported on the newly grafted orbital floor.* ***F*** *Postoperative appearance. There is still a slight deepening of the supratarsal groove, but the enophthalmos and diplopia are almost completely resolved.*

Where the orbital floor exploration is part of an orbitocranial operation, an experienced surgeon can approach the floor via the bicoronal scalp flap. The periosteum is mobilised from the lateral orbital rim and the zygomatic arch. The periorbital dissection is as extensive as possible, though of course avoiding the apex of the orbit. The margin of the defect must be clearly defined; the misplaced orbital contents are often fibrotic and dissection may be difficult. Care is needed to avoid excessive pressure on the globe, yet the surgeon must be able to extend the exploration to the posterior aspects of the orbit and discover the posterior margin of all defects. Considerable experience is needed for competence in this. Wolfe & Berlowitz (1989) enthuse over Tessier's technique for inferior orbital marginotomy to facilitate this difficult dissection (Tessier 1982) and to allow the defect in the orbital floor to be bone-grafted precisely under direct vision (Fig. 21.20). We rarely find this procedure necessary.

When the defect or defects are fully visualized, the defect is repaired. The architecture of the orbital floor may be restored by means of bone grafts, inorganic implants, or osteotomies. Except when a malunited orbitozygomatic complex can be corrected by osteotomy alone, we unreservedly support the use of bone grafts alone for reconstructing the orbital floor, having removed all shapes and sizes of Silastic® and other foreign material inserted in earlier days by ourselves and latterly by others. Silastic®, in particular, in time seems to find its way into the paranasal sinuses.

Medial orbital wall defects

These are identified and defined exactly by CT scanning. Often, some of the orbital contents are prolapsed into the ethmoid sinus, and the volume of the orbit is thus enlarged. The intact ethmoidal sinus with its 'Chinese lantern' structure can be seen on the normal side and its delicate contribution to the orbital wall contour and hence the volume can be estimated and compared with the injured side.

The approach is best made via a bicoronal scalp flap, giving wide exposure of the fracture in the lateral plate of the ethmoid; the defect can then be dissected and the contents reduced. To do this effectively the posterior aspect of the fracture must be identified. The anterior ethmoidal artery is encountered en route to the fracture and should be coagulated. Defects can then be repaired, usually with a thin piece of split calvarial bone, harvested through the bicoronal scalp flap, which allows exposure of the parietal region (Fig. 21.21). Not only must the defect be bridged but the volume of the orbit must be reduced; however, the normal anatomy is not easy to reconstruct and bone graft replacement is always a little imprecise.

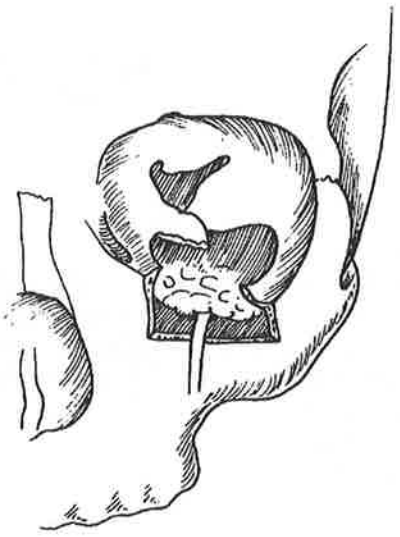


FIG. 21.20. *Surgical exposure of the orbital floor.* *Technique recommended by Tessier (1982) for resection of the inferior orbital margin above the infraorbital nerve, providing increased exposure for retrieving orbital contents from the antrum and subsequently reconstructing the orbital floor.*

Lateral orbital wall defects

Isolated fractures of the lateral wall are rare: damage in this area is usually associated either with total orbital disruption with multiple wall defects, or more commonly with an uncorrected or inadequately corrected fracture of the zygomatic complex. Enophthalmos resulting from the displacement of the zygomatic complex is caused by downward and outward displacement of the bone, enhanced by the pull of the masseter muscle, producing an expanded orbit. The zygomatic component of the orbital floor may be disrupted, allowing the orbital contents to prolapse into the maxillary antrum. Similarly the thin bone behind the lateral orbital rim, which articulates with the greater wing of the spheroid bone, may be disrupted with prolapse of orbital contents into the temporal fossa. Once the malar fracture has been reduced by osteotomy (Gruss et al 1990) then any persisting enophthalmos is due to the depression of the orbital floor (see above) or to outward bowing of the lateral orbital wall. Surgical strategy and operative techniques are as for enophthalmos caused by massive destruction of one wall only: wide exposure of the orbital skeleton, restoration of the zygomatic complex, rigid fixation, identification of areas of bone loss in the walls of the orbit, reduction of all herniated orbital contents and restoration of orbital volume, with careful reconstruction of the orbital wall defects by autogenous bone grafting. Usually this is done with split calvaria but rib and iliac bone can be used. If more projection of the globe is necessary smaller grafts can be placed behind the axis of the globe to produce forward projection. In the past diced cartilage and other materials have been used to pack the orbits, and even glass beads, Elastics blocks, Teflon®, Supramid® etc. They are no substitute for the direct visualisation and accurate repair of the defects by bone graft. However, we are beginning to use porous polyethylene (Medpor®) in the hope that this will obviate the problems of harvesting and resorption of bone grafts (Nguyen & Sullivan 1992).

There are some rare cases where the globe and the surrounding orbit are retrodisplaced but the relationships of globe to orbital rim are maintained (Fig. 21.22). Such a deformity is the result of injury in childhood, causing posterior displacement of the whole orbital box: correction is properly done by transcranial advancement of the orbit with the contained globe, using the osteotomy patterns described below for the correction of orbital dystopias. After such an osteotomy it is likely that the orbital contents will need some further augmentation by bone grafting the defect and packing some bone on the walls behind the axis of the globe. This troublesome deformity has received insufficient attention in the literature.

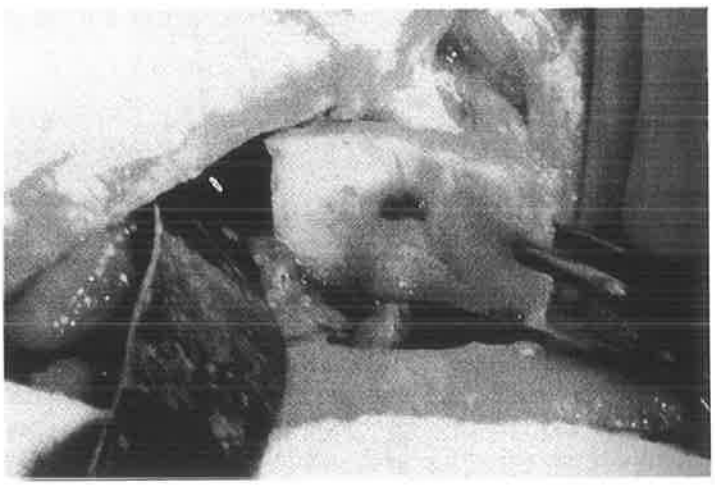
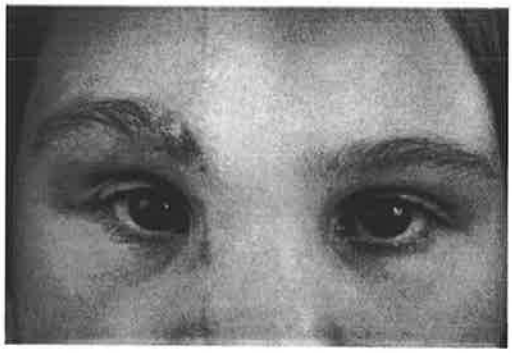


FIG. 21.21. ***Post-traumatic enophthalmos.*** *Patient presented with enophthalmos following orbital trauma. Split calvarial bone graft is placed into the medial orbital wall via the bicoronal scalp flap.*



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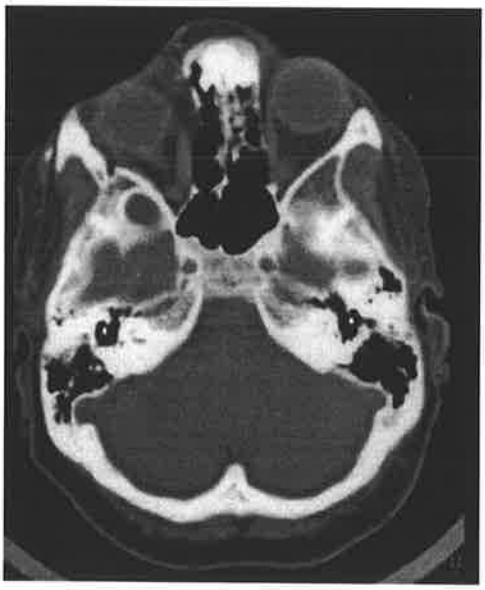


B

FIG. 21.22. ***Displacement of the orbital complex.*** *There are rare cases in which the globe and orbital box are displaced but the relationship between the globe and box is maintained. It is our experience that this occurs when there is an injury early in childhood with force directed to one side such that a 'unilateral hypertelorism' results.* ***A*** *Distorted asymmetrical retrodisplacement of the orbit and globe.* ***B*** *CT scan showing some retrodisplacement of the globe within the expanded orbit, with the whole complex positioned posteriorly.*

Deformities Of The Orbitozygomatic Complex

Surgical pathology

These deformities commonly result from untreated or inadequately treated depressed fractures of the zygomatic bone and lateral wall of the orbit. The zygomatic bone makes a key contribution to facial morphology, connecting as it does by its radiating processes to the frontal, maxillary and temporal bones, and depressions of the zygomatic bone are often very noticeable. The clinical signs and symptoms of secondary deformation of the orbitozygomatic complex are similar to those following acute injury:

1. Alteration of facial contour
2. Displacement of the orbit and/or globe
3. Diplopia
4. Residual sensory nerve damage
5. Limitation of mandibular movement.
6. Aesthetic problems from a damaged globe, absent globe or empty socket; these are considered on p. 641.

Alteration of facial contour

This may be due to bony displacements and/or soft-tissue damage (Fig. 21.23). The range of deformity varies from a hardly noticeable bony displacement which blends with natural facial asymmetry, to gross displacement of the whole bone with or without comminution, overlying scarring and loss of the fibrofatty malar cheek pad.

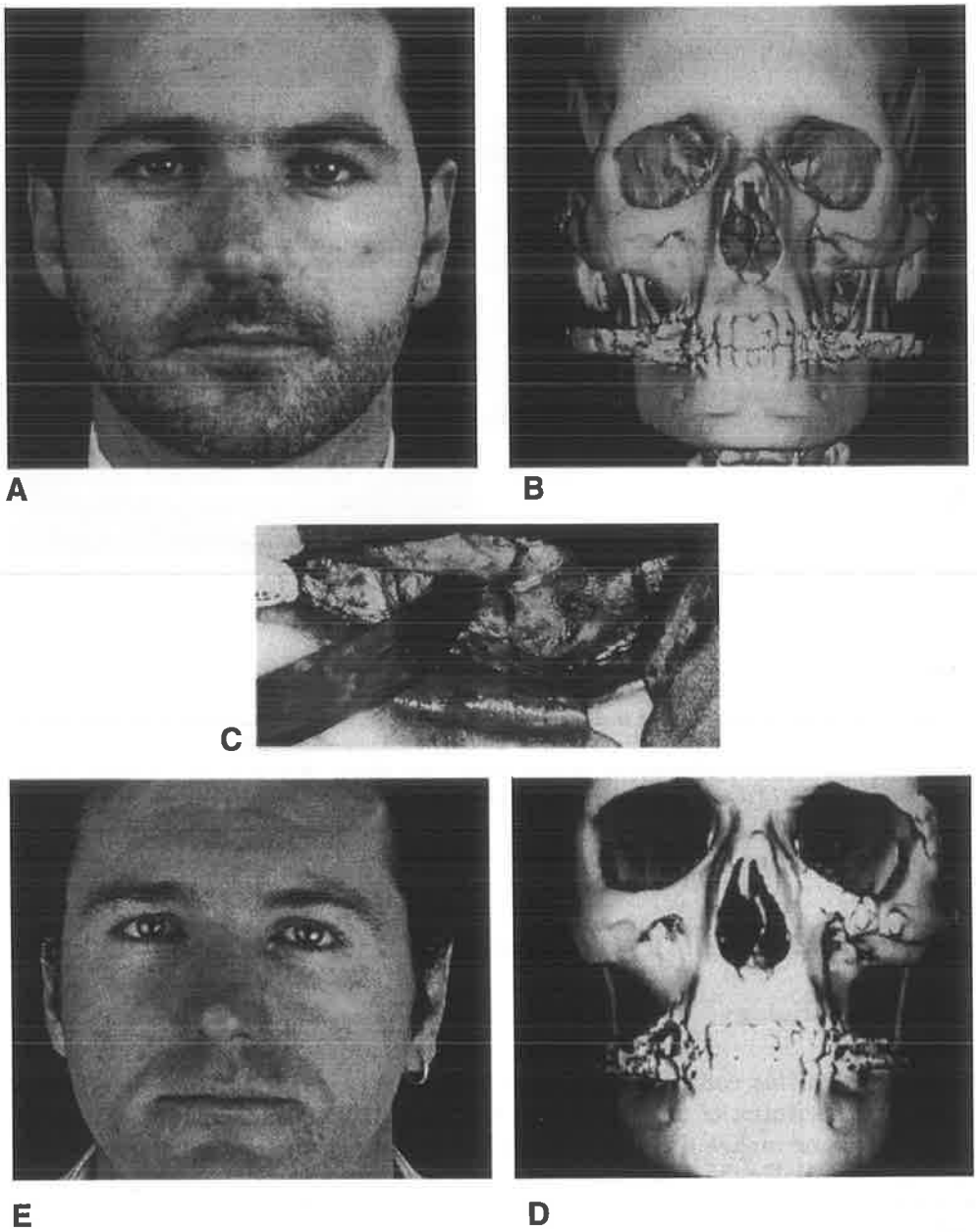


FIG. 21.23. *Zygomatic osteotomy.* ***A*** *Patient with a fractured zygoma expanding the orbit and producing flattening of the cheek.* ***B*** *CT scan showing the bony deformity in three dimensions.* ***C*** *Operative photographs showing deformity of the zygoma.* ***D*** *Postoperative CT scan.* ***E*** *Postoperative appearance.*

Bony deformity may result from severe comminution and subsequent resorption of small fragments of bone, or from displacement caused by scar contracture in skin and muscle. Inadequate primary fixation may result in displacement due to the action of the masseter muscle during mandibular movement. Segments of bone may be lost from the orbital rims and zygomatic arch. The zygomaticomaxillary buttress area is often subjected to compression forces, resulting in movement. Segments of bone may be lost from the orbital rims and zygomatic arch. The zygomaticomaxillary buttress area is often subjected to compression forces, resulting in comminution and later fibrosis: these, if uncorrected in a primary repair, add to the orbital deformity.

Orbital and/or ocular dystopia
In these deformities, the orbit and globe may be displaced in any or all of the three dimensions, and displacement of the globe may be independent of orbital rim displacement. Horizontal and vertical dystopias are especially disfiguring, as is orbital hypertelorism, which is a form of horizontal dystopia. Hypertelorism was one of the first challenges for pioneers in the surgery of craniofacial deformities (Fig. 21.24).

The whole orbital complex may be enlarged inferiorly or inferolaterally by a downward and lateral displacement of the zygoma. The floor of the orbit is thereby involved and there is vertical orbital dystopia. The globe itself may be displaced downwards when the floor of the orbit is deficient as in a 'blow-out' fracture. Lateral displacement of the globe may occur in fractures of the naso-orbito-ethmoid complex, but true lateral displacement of the whole orbit is in our experience a rare deformity. Also rare is an inferior displacement of the orbit due to trauma. When the compressive forces occur in infancy, the bones are soft and injury results in a splaying out of the orbits, which is maintained or accentuated with further growth (see below). Superomedial displacement of the malar bone is rare but may produce a decreased orbital volume with superior positioning of the globe and exophthalmos.

Diplopia
Double vision may result from displacement of the globe, damage to the cranial nerves, damage resulting in scarring of the extraocular muscles or prolapse and entrapment of orbital soft tissue. Because there are so many factors involved in diplopia, reconstructing the orbit and repositioning the globe may not in themselves overcome double vision. It is wise to wait a considerable time (at least 1 year) after reconstructive surgery before surgery on the extraocular muscles is undertaken. Multiple surgical interventions may be necessary and the muscles of the other eye may indeed need adjusting to achieve fusion. The assessment and management of post-traumatic diplopia are considered in Chapter 14 (p. 418).

Sensory nerve damage
Anaesthesia, paraesthesia or hyperaesthesia of the zygomaticofrontal, zygomaticofacial and infraorbital nerves may express nerve injury resulting from the initial trauma or from dissection during the surgical correction.

The nerves passing through the body of the zygoma (p. 61) may take well over a year to recover and the numbness of the cheek and temple must be explained to the patient. The recovery phase is often accompanied by dysaesthesiae — 'funny feelings' — and shooting pains. Recovery is usually complete after 2 years.

Infraorbital nerve lesions affecting the gums and teeth are present in a minority of patients and in a very few give rise to neuralgia which is persistent and hard to treat (p. 659).

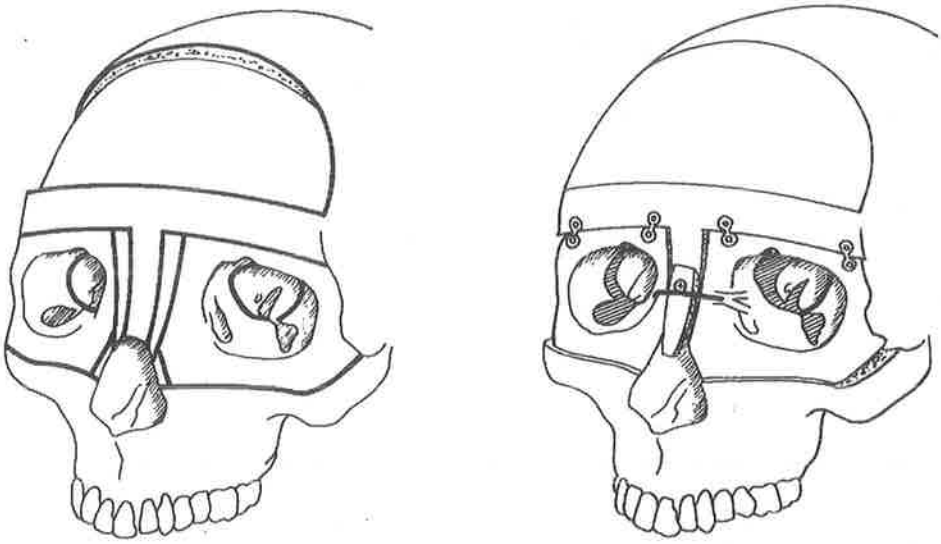


FIG. 21.24. *Hypertelorism correction.* *Hypertelorism correction according to Converse et al (1970). Midline structures are preserved wherever possible. The cuts are made behind the axis of the globe so that the globe is translocated with the orbital box.*

Limitation of mandibular movement
This occurs when the zygomatic arch impinges on the excursion of the coronoid process of the mandible.

Assessment

Nothing surpasses thorough clinical assessment and the critical eye of the skilled surgeon. In addition to the plain radiographs listed in Chapter 7, namely the occipitomental view, postero-anterior views of the orbit, submentovertical view and orthopantomogram, the CT scan has given a new dimension to assessments of deformities of this region. The ability to view the orbital walls in detail has been supplemented by attempts to quantify the orbital volume on the basis of CT data (Manson et al 1986). The techniques for this have their limitations but do indicate that changes in the size and shape of the bony orbit constitute the most important factor in the problem of enophthalmos (p. 193). Whitaker (1987) has given an excellent analysis of the midface malar region in three zones, which is very applicable to the assessment of contour restoration after trauma, no matter what technique of repair is used. These three zones extend from the nasolabial fold outwards and onto the zygomatic arch. The medial zone is just lateral to the nasolabial furrow. The middle zone is the prominence of the zygoma and the outer zone is the zygomatic arch (Fig. 21.25).

Management

The indications for surgical correction can be inferred from the pathology. The chief is obvious facial deformity resulting from bony displacements and/or soft-tissue scarring. Diplopia due to orbital or global dystopia is an indication when persistent. Enophthalmos, often combined with deformity and diplopia, may be an indication, as is impingement of the zygomatic arch on the coronoid process.

Long-term paraesthesia or hyperaesthesia of the infra-orbital nerve may be an indication when the patient persists with the complaint, though the likelihood of symptomatic relief may not be good.

Operative correction
Success depends largely on determining whether the deformity results from a deficit in the bone, or from a pure malposition, or from a combination of both.

Segmental bony deficits are usually corrected by bone grafts, whereas malposition of the orbitozygomatic complex with normal adjacent bones is corrected by osteotomizing the bone, thus recreating the fracture, and then repositioning the bone with or without onlay grafts.

The most appropriate exposure must be used: the choice of exposures includes the bicoronal approach, the periorbital (eyebrow, lid and conjunctival) incisions, and approaches through existing scars. Whichever approach is chosen, access should not be compromised. When contour restoration by onlay or inlay techniques is indicated, a choice of material needs to be made. Segmental bone losses around the orbital rims, zygomaticomaxillary buttress and zygomatic arch are replaced with autogenous bone and/or inlay, fixed with titanium screws or miniplates (Gruss 1992). The bone graft of choice is split calvarial bone (p. 241), but rib or hip bone can be used. The bone may be screwed into place and recontoured in situ or preshaped. Maejima et al (1993) and Yab et al (1993) have developed 3D solid models. which can be used to prepare templates from which bone grafts can be modelled in a precise fashion to repair defects in the cranium and orbit. Figure 21.26 shows such models being used in a case of neurofibromatosis, by a method equally applicable to post-traumatic deformities. Countersinking screw heads adds to rigidity and makes the head less palpable through the skin when the inevitable — though usually slight — bone resorption occurs. The finest possible plates consistent with stability should be used in this region. While we prefer bone even for the most subtle onlay restoration of the malar prominence, other substances are available. Silastic® has been used for this purpose, but it is difficult to carve exactly; the tendency to migrate has been noted. Hydroxyapatite is likewise difficult to contour to the complex curves of the cheekbone. Whitaker (1991) championed the use of Proplast® (polytetrafluoroethylene combined with aluminium oxide or other structural substance) in this area and supported its use in spite of some claims of increased infection. However, recent studies suggest that Proplast® is often associated with adverse local reactions (p. 155). Polyethylene (Medipor®) may be more satisfactory. The frequent necessity of having to remove previously placed plates and screws, the distortion or loss of tissue planes, the presence of fragments of bone and scar often containing sinus mucosa, all combine to make the use of alloplastic material very problematic.

In almost all cases, operation is done under general anaesthesia with oral endotracheal incubation. The surgical field is cleaned with povidone-iodine solution; local anaesthetic with adrenaline (p. 255) is injected at the subperiosteal level. The patient is given intravenous antibiotics before the incision is made.

When the lateral rim of the orbit and zygomatic arch are involved then the bicoronal approach is preferable; this can be combined with buccal and eyelid incisions if necessary. The upper buccal sulcus incision can be used in cases where there is an aesthetic deformity affecting the prominence of the zygoma and the anterior maxilla. Exposure from above allows detachment of the temporal fascia from the lateral orbital rim and the zygomatic arch, and this enables the periosteum to be elevated from the orbitozygomatic complex (Fig. 21.23). Exposure from below is done via an incision in the buccal mucosa 5 mm above the apex of the sulcus, and the cut is made directly to bone beneath the infraorbital nerve: the periosteum and attached muscles can then be elevated with a sharp periosteal elevator. The right-handed surgeon can protect the globe of the eye by grasping the orbital margin, zygomatic arch and body junction between the finger and thumb of the left hand, dissecting between the two digits (Fig. 21.27).

When bone is used to re-establish a pre-existing contour by replacing lost bone, there is always a problem as to how to shape the graft and how thick it should be. Even when appropriate bone, e.g. calvaria, is secured with rigid fixation, judgement at operation is often compromised by soft-tissue swelling, and as there is always some resorption of bone there is a necessity for overgrafting. It is this uncertainty that inspires the continuing search for a satisfactory bone substitute.

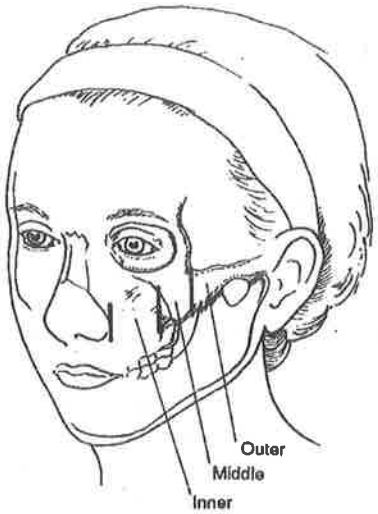


FIG. 21.25. ***Midface malar region.*** *The three regions of the malar midface region as identified for the purposes of augmentation by Whitaker (1987). By courtesy of* Plastic and Reconstructive Surgery.

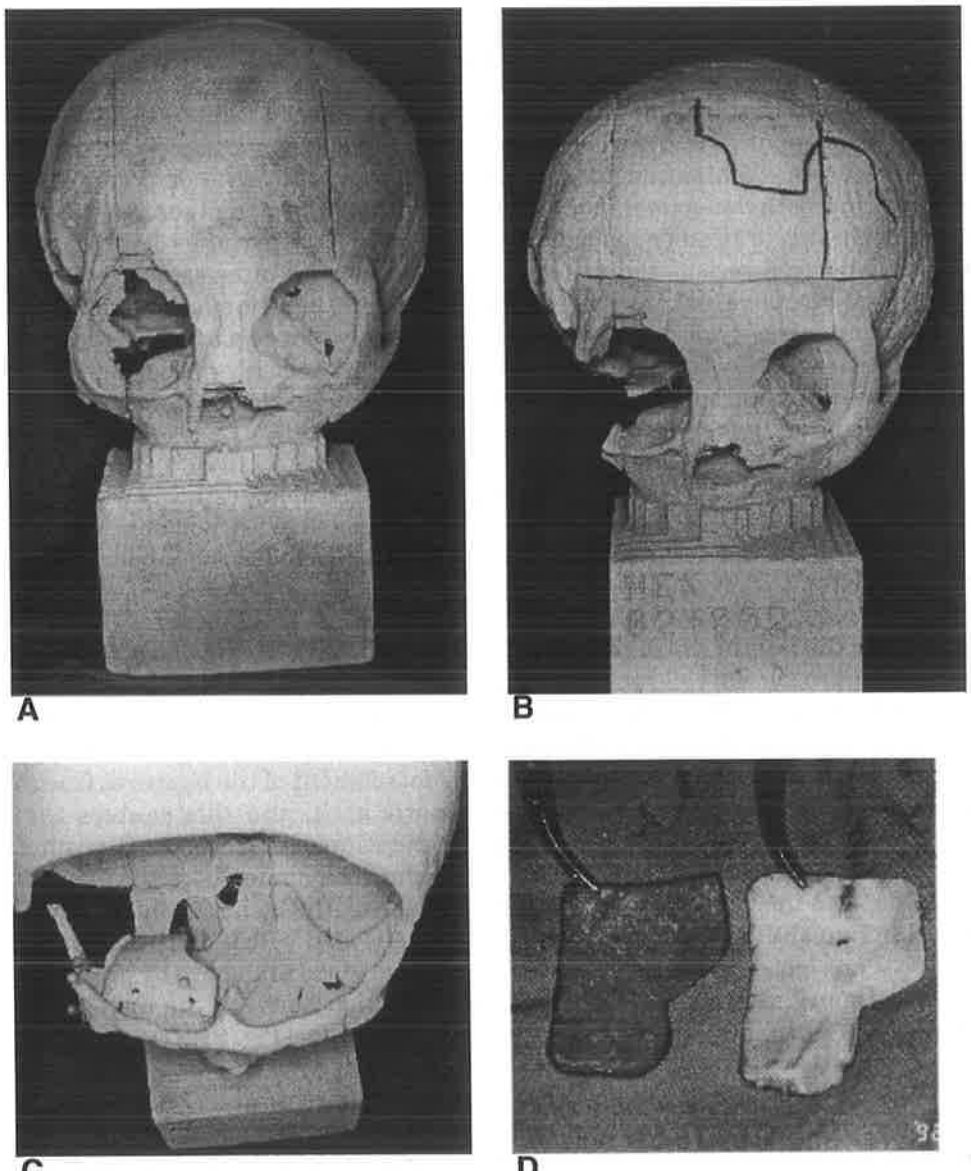


FIG. 21.26. ***Solid models for preoperative planning.*** ***A*** *Solid model of a case of sphenoid resection due to neurofibromatosis. (The technique may well be used for trauma).* ***B*** *Exterior view.* ***C*** *Anterior fossa view, enabling templates to be prepared so that the bone can be cut absolutely accurately at operation.* ***D*** *Templates with the carved bone at the time of surgery. (Photos courtesy of Dr S. Tajima).*

When the whole orbitozygomatic complex is malpositioned, treatment is by osteotomy. Wide exposure is desirable, and the bicoronal scalp flap is preferred. Osteotomies are made at the frontozygomatic suture, and in the lateral orbital wall, inferior orbital rim, lateral maxillary buttress and zygomatic arch (Figs 21.28 and 21.29) To perform these cuts, the periorbita is stripped from the lateral wall and floor of orbit. Additional exposure may be necessary through the eyelid (p. 238). The exposure extends to the infraorbital fissure and orbital floor. The periosteum is stripped from the arch and body of the zygoma, avoiding and protecting the infraorbital nerve. The masseter muscle may be detached from the body of the zygoma to achieve movement after osteotomy. In old traumatic cases this dissection may not be easy. The tissue planes that are so readily stripped in congenital or purely cosmetic deformities are lost or interrupted and scar from trauma or previous surgery bleeds unduly. Fragments of bone are often displaced into soft tissues; on the orbital side of the dissection the periorbita has often been previously breached and there may be little but scar between the orbital contents and the maxillary antrum. Careful persistence is needed, with good surgical assistance, taking care to avoid too much pressure on the globe but not failing to get the adequate exposure that the surgery demands.

The osteotomies are made with power saws; a reciprocating saw is preferred, held like a pen. Malleable metal retractors protect the orbital contents. Osteotomies are made through the arch, through the frontozygomatic Suture and from the temporal fossa into the orbit; in cutting into the orbit, the saw blade is held flat against the temporal fossa surface, and the surgeon views the point of the blade on the orbital side and the base of the blade on the temporal fossa side, to avoid entering the middle cranial fossa. The cut extends to the inferior fissure and across the orbital floor to the rim, respecting the infraorbital nerve; it is extended laterally to separate the zygoma from the maxilla. Once these cuts have been successfully made, a slightly curved osteotome is inserted into the section of the cut in the zygomatic buttress; gentle rotation of the osteotome will usually separate the bone quite easily.

As Gruss (1992) has rightly pointed out, the key to the correct positioning of the orbitozygomatic complex is the zygomatic arch. Once the distorted arch's shape and length are restored, an accurate overall positioning is possible with restoration of correct orbital volume. In old malunited fractures the shape of the arch may have to be changed by osteotomy; appropriate basal CT cuts will show the distortion of the arch and guide the surgeon as to how it should be cut and reshaped. Without the wider exposure given by a coronal flap and complete arch dissection, this manoeuvre is very difficult. Long fine miniplates or microplates are used to stabilize the newly shaped zygomatic arch and bridge the zygomaticotemporal gap; similar plates are applied to the frontozygomatic suture, lateral maxillary buttress and inferior orbital margins. Autogenous bone grafts are used to fill the gaps in the arch, buttress and orbital walls.

Correction of deformity in the orbitozygomatic complex is an essential first step in correcting a malunited panfacial fracture, utilizing the same planning principles that are applied in the primary management of a recent panfacial fracture (p. 339). In reconstructing such an extensive deformity, correct positioning of the orbitozygomatic complex on each side is followed by establishment of the occlusion and correcting malunited jaw fractures; this enables the relationship of the correctly occluded jaws to be established with the zygomatic complex. The naso-orbital component can then be attached medially, enabling the anterior maxillary walls to be built out and the nose constructed on the platform of the face (Fig. 21.29).

When there is massive disruption of the orbitozygomatic complex with extensive destruction of bone, then osteotomy is out of the question: the whole orbitozygomatic complex must be rebuilt. This can be achieved with split calvarial bone grafts. If enough bone cannot be harvested from the skull, then the calvarial grafts should be augmented with split-rib grafts. A long piece of bone is shaped

and fixed into position with plates and screws to replace the zygomatic arch, being attached medially to the maxilla and laterally to the zygomatic process of the temporal bone. The lateral orbital wall is reconstructed with a series of longitudinally placed struts from the frontal bone, attached to the newly formed zygomatic arch. Further onlay bone graft may be needed to shape the orbital rim laterally. The cheek prominence may be further enhanced with bone screwed to the longitudinal graft used for reconstruction of the zygomatic arch. Such reconstructions are often compromised by damaged skin and other soft tissues; further surgery may be necessary to smooth out the bone, to remove screws which have become prominent from resorption or to add additional bone or other material to enhance the contour.

Post-traumatic orbital dystopia may require correction by moving the 'whole orbit': in reality, this means moving the anterior two-thirds of the orbital pyramid. As has been previously stated, it is rare that post-traumatic hypertelorism or vertical orbital dystopia requires mobilization of the orbital box; in our experience this occurs only with deformities resulting from injuries suffered in infancy and early childhood. When such a mobilisation is required, it is to move that part of the orbit which carries the globe with it, and this is the anterior two-thirds of the orbital pyramid. The mobilisation is in part done by a transcranial approach, and the bicoronal exposure is of course necessary; the neurosurgeon is added to the craniofacial team. The distances of the planned orbital movement are determined by examination of the patient and by appropriate radiography.

In vertical dystopia (Fig. 21.31), a frontal craniotomy, in size less than the width of the orbit, is performed on the side to be displaced, and the intracranial extradural dissection is limited to part of the anterior fossa to be protected during the cutting of the roof, and the medial and lateral orbital walls. Additional incisions in the eyelids or upper buccal sulcus can be performed, but usually the whole osteotomy can be done from above. The upper osteotomy is made 1 cm above the supraorbital rim and the lower is made across the anterior maxilla just beneath the infraorbital foramen. The posterior lateral orbital cuts are made vertically through the lateral orbital wall, flush with the temporal fossa base. The lateral orbital wall osteotomy is extended across the orbital roof and inferiorly across the orbital floor into the inferior orbital fissure. The medial orbital wall is cut at about the level of the anterior ethmoidal foramen and we now prefer not to dissect the medial canthal ligament as it is difficult to reattach this ligament again.

If the orbit is being repositioned superiorly, a measured amount of bone is removed from the frontal bone. The orbit is gently mobilized and fixed superiorly. The removed bone can then be used to graft the horizontal defect in the maxilla beneath the infraorbital nerve to maintain the vertical translocation; additional bone grafts are placed in the inferior defects if necessary. The new position can be determined very accurately.

When the orbit is being moved medially, as in hypertelorism, we prefer to maintain the midline structures wherever possible, namely the vertical plate of the ethmoid and the central part of the anterior fossa (Converse et al 1970), and in our experience this has always been possible in post-traumatic hypertelorism. Medial translocation of the orbits is achieved after the removal of the appropriate amount of nasal bone, frontal bone and anterior fossa, parasagittal to the cribriform plate. The medial translocation is stabilized by insertion of bone in the lateral osteotomy. It is prudent to place a thin overlay of bone on the horizontal maxillary cut, to prevent prolapse of soft tissue into the defect, which might result in a sunken appearance in the mid-face. The medial canthus must be aligned as accurately as possible and if necessary canthopexies are performed (Fig. 21.24). It is our experience, and we believe this is generally shared, that vertical dystopias are easier to correct than horizontal dystopia. Hypertelorisms, particularly in the rare post-traumatic cases, are likely to require bone grafting to the nose, canthopexies and often secondary surgery to the nasolacrimal apparatus. In our

experience, multiple operations are necessary during the growth period and even later to overcome the persistent telecanthus component of these horizontal dystopias.

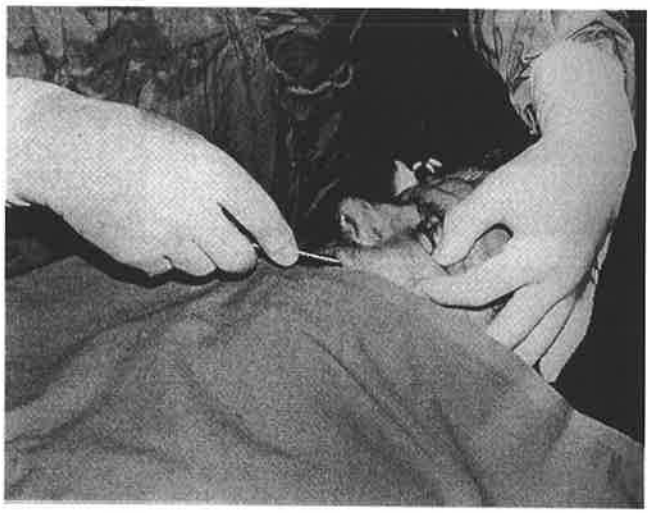


FIG. 21.27. *Dissection of the orbitozygomatic complex via the transoral approach.* *The forefinger and thumb of the non-dominant. hand protect the orbit during the dissection.*

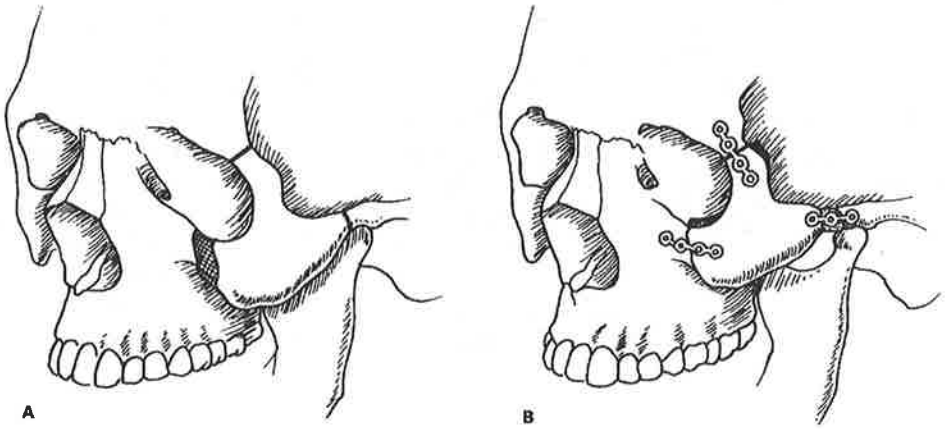


FIG. 21.28. *Zygomatic osteotomy.* ***A*** *Depressed and malpositioned zygoma.* ***B*** *Three-point osteotomy and repositioning with miniplate fixation and bone grafting.*

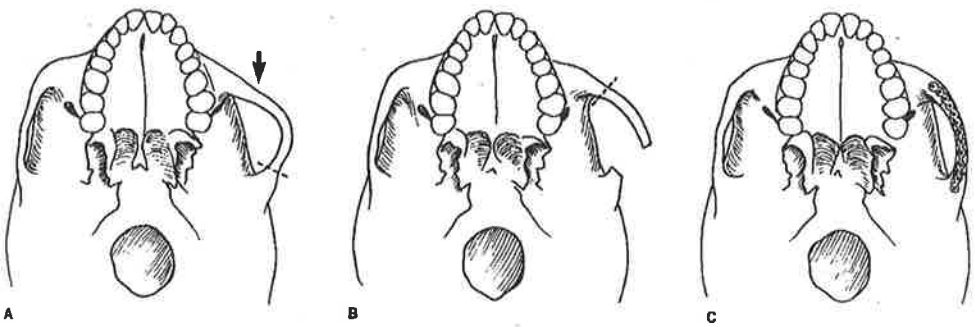


FIG. 21.29. *Zygomadc osteotomy.* ***A*** *Depression of the zygomatic body and bowing of the zygomatic arch requiring osteotomy for correction.* ***B*** *The orbitozygomatic complex is freed at the frontozygomatic suture, the infraorbital margin, and the lateral maxillary buttress.* ***C*** *After repositioning the arch, forward projection is maintained by grafting the gap in the arch and fixation with a long miniplate.*

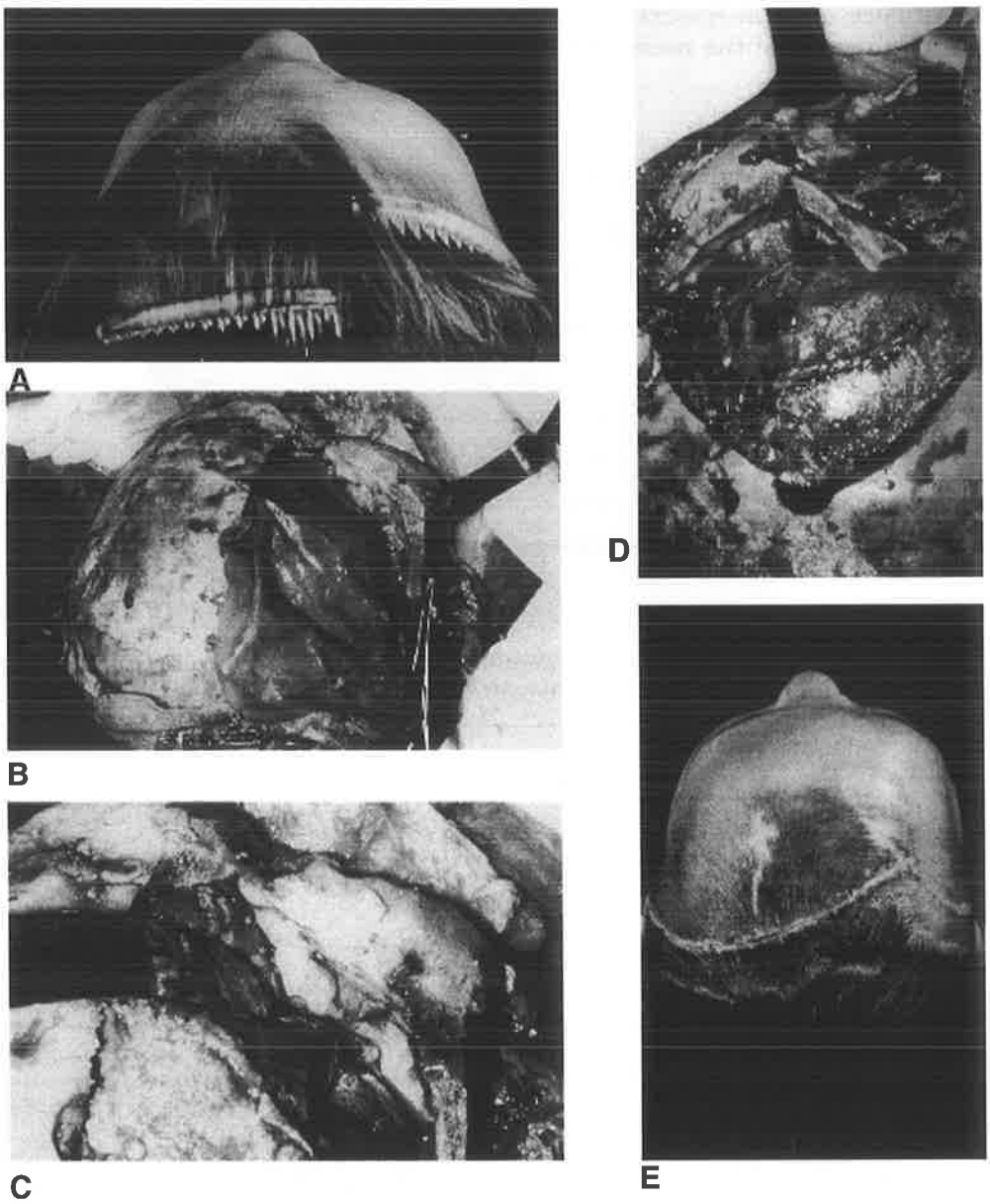


FIG. 21.30. ***Secondary alterations in facial contour.*** *Patient shown in Fig. 21.22, who had a severe post-traumatic deformity of the right side of the face following a complex fronto-orbital, orbitozygomatic and naso-ethmoid injury sustained in a vehicular accident 6 years earlier. The initial treatment consisted of a frontal craniotomy, reduction and fixation of the zygoma, and soft tissue repairs.* ***A*** *View from above, illustrating deficiency of the forehead and right side of the face. Enophthalmos is mild because the position of the globe within the orbital box has been relatively well maintained while the whole of the orbital complex is displaced posteriorly.* ***B*** *Operative appearance of the deformity with the frontal craniotomy removed to reveal the shattered and depressed fronto-orbital complex.* ***C*** *Dissected anterior fossa with the old fracture represented at the junction of the anterior and middle cranial fossae.* ***D*** *Fronto-orbital complex replated in its new position.* ***E*** *The postoperative appearance highlights the repositioning of the zygoma and forehead to restore and slightly overcorrect the contour on that side.*

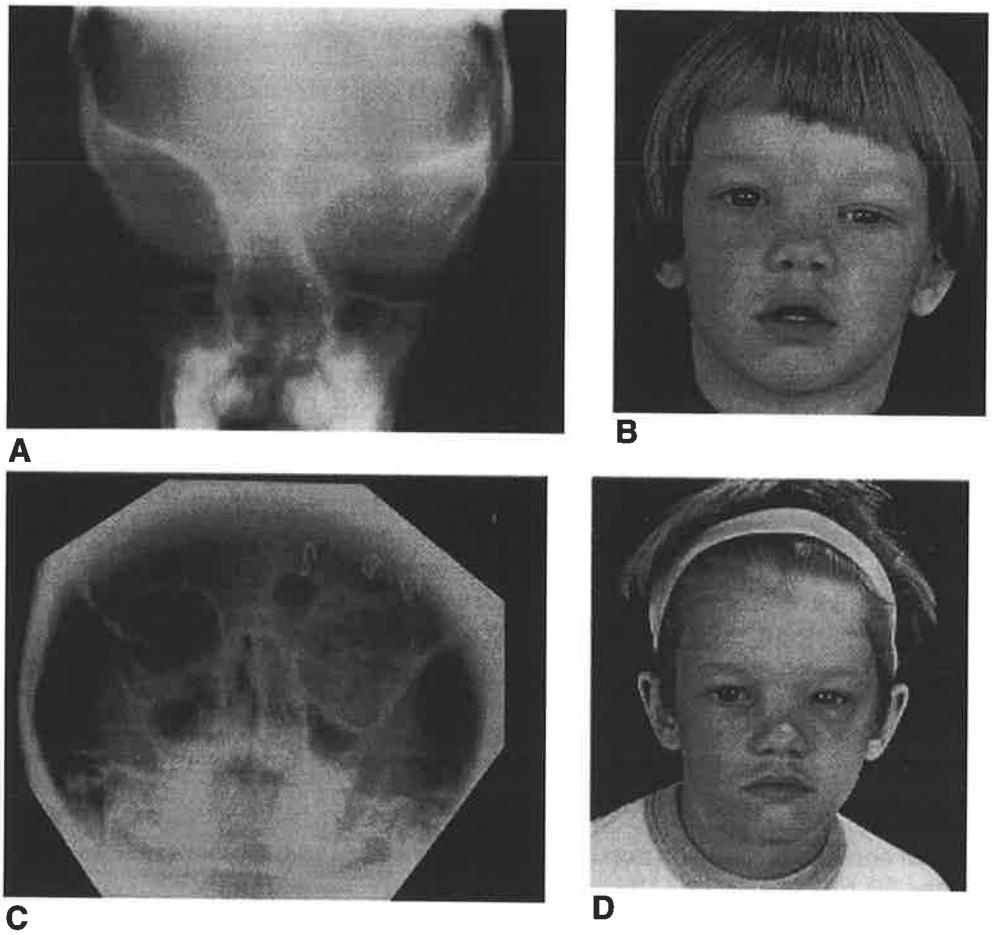


FIG. 21.31. ***Translocation of the orbit.*** *A 6-year-old boy fell 20 feet from a balcony at his home onto cement, suffering concussion, nasal fracture, and left orbital fracture. He developed a CSF leak into the orbit which was repaired by a neurosurgical team. He presented two years later with left orbital dystopia of 1 cm and mild traumatic hypertelorism with a left divergent squint.* **A** *Tomogram taken at initial treatment showed a depressed and expanded left orbit. A residual fracture was evident in the orbital roof.* **B** *Photograph at the time of his presentation for craniofacial surgery showed orbital dystopia.* **C** *Post-operative X-ray picture showed the orbit after elevation, with three wire ligatures holding the elevated orbit to the frontal bone.* **D** *Post-operative photograph of the child showed the correction two years later with virtually no residual enophthalmos or orbital dystopia.*

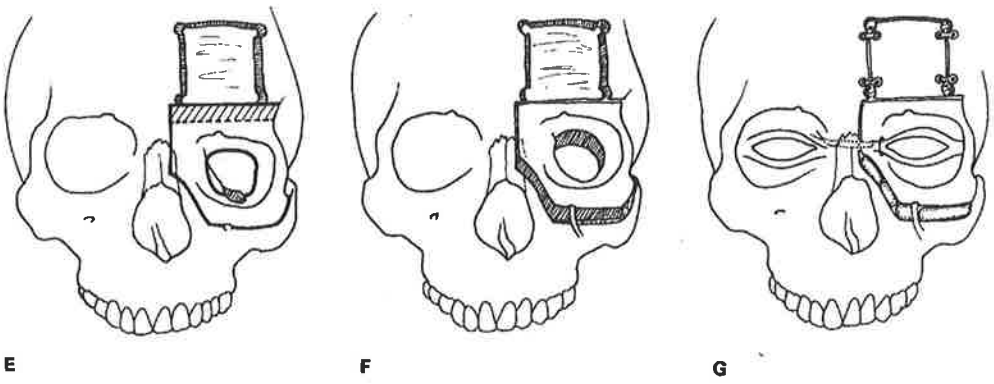


FIG. 21.31. ***Translocation of the orbit.*** **E** *Diagrammatic representation of the small frontal craniotomy and removal of bone superior to the orbit used in such cases. Intraorbital and anterior cranial fossa cuts are made behind the axis of the globe to facilitate movement of the globe with the orbital box.* **F** *After repositioning, the bone removed superiorly can be used to augment the maxilla inferior to the orbit. The bone removed for the frontal craniotomy can be split to provide additional graft if necessary.* **G** *Canthopexy is necessary to re-establish the lid shape and relationships.*

Complications and results

Haematomas and infection may occur. Infection is often associated with implanted materials such as plates and screws, and with operative exposure of the maxillary sinus. Pre-existing sinusitis should always be treated before reconstructive surgery; any indications of sinusitis from the history or preoperative X-ray findings may be an indication for endoscopic examination and subsequent drainage of the infected sinus, especially when the maxillary antrum is involved. In our experience screws presenting into the maxillary antrum after primary surgery have sometimes appeared to be a focus of infection.

More serious complications relate to eyesight. Compression of the optic nerves may result from implanted bone, from manipulation of the fracture or from retrobulbar haematoma. In our experience, the patients susceptible to this complication are those with massive destruction of the orbit, where there has already been some compromise in optic nerve function; such patients are in danger of losing some or all of their residual vision. Fortunately, this is not common but does occur. Wolfe & Berlowitz (1989) refer to one case of blindness in 1600 orbits dissected and one case of loss of visual field. We have had one case in which vision decreased after an orbital dissection, in an experience of more than 100 translocations for post-traumatic orbital dystopia. This misfortune occurred in a patient with pre-existing visual impairment; eventual visual loss was not complete. It appears that, in traumatic cases, it is the damaged optic nerve that is more at risk. Trauma to the sixth cranial nerve is also common in cases where there is much scar and dissection is difficult.

In our experience, the aesthetic results are often indifferent, and often the reasons for this are beyond the control of the surgeon.

The controllable factors are the repositioning of the zygoma by osteotomy and the reconstruction of the orbital walls by bone graft. The variables are the restoration of the orbital volume to produce accurate positioning of the globe in three dimensions and the status of damaged soft tissues; loss of orbital fat, damage to muscles, eyelids and lacrimal drainage apparatus may impair the correction.

Initially good results can deteriorate with time. Resolution of postoperative oedema confounds early assessments. Bone grafts used to increase the projection of the globe may slowly resorb; tissues scarred by trauma and/or surgical intervention go through phases of hypertrophy and maturation. Even when a good early result has been obtained, and with the best possible surgical experience and judgement, the ultimate result with respect to the position of the globe may leave something to be desired. Because of these unpredictable factors, it is necessary to monitor the results of correction for a number of years.

Some patients have prolonged postoperative infraorbital nerve anaesthesia. The area of sensory loss usually extends from the lower lid over the cheek and lateral alar region to the upper lip. If sensation has not recovered after 3 months then it is most likely that the nerve is permanently damaged. We have not found release of the nerve as a secondary procedure to be of any benefit. In those cases where the numbness has progressed to a neuralgia — a very rare but distressing situation — exploration of the nerve and relief of pressure in the canal has not produced satisfactory symptomatic improvement in our hands (Fig. 21.32). The management of intractable facial neuralgia is further discussed in Chapter 22 (p. 557).

Nasal and Naso–Orbito–Ethmoid Deformities

Surgical pathology

Secondary deformities of this type range from malunited fractures involving the nasal bridge (i.e. the nasomaxillary region) together with the nasal cartilages and septum, to complex lesions involving the entire naso-ethmoid and orbital regions (Manson & Sargent 1992). The symptoms may be functional as well as aesthetic: nasal airway patency and sinus drainage may be affected, the sense of smell is often lost and impaired nasolacrimal drainage may cause epiphora. There may also be risks of late intracranial infection, arising from proximity to damaged meninges (pp. 147 and 379).

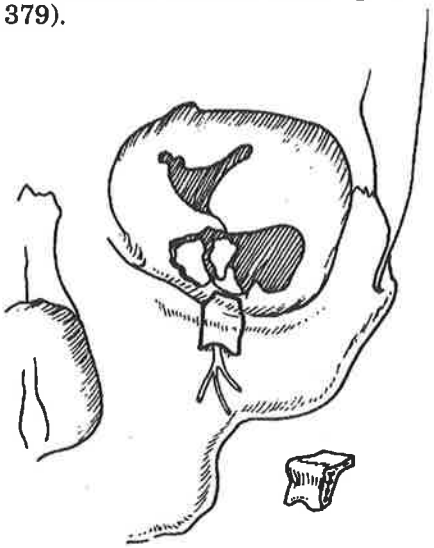


FIG. 21.32. ***Technique for decompression of the infraorbital nerve.*** *A portion of the bone is removed above the nerve and into the orbital floor. The nerve may be traced back to the fracture area and dissected from the orbital content, and the fragments of bone removed or replaced. A bone graft may also be used to overlay the area (after Converse et al 1977). This approach observes the principle that nerves should be explored from a normal area to the abnormal. It is not a very satisfying procedure as far as results are concerned.*

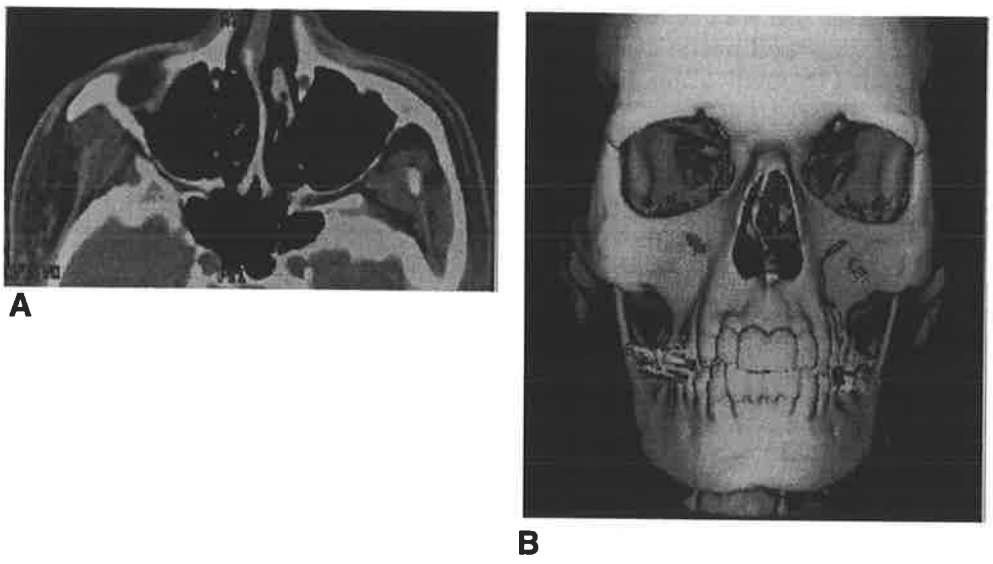


FIG. 21.33. ***Nasal septal deformity.*** ***A*** *Septal architecture shown on axial CT.* ***B*** *Deformity of the bony septum.*

Localized nasal deformities

These deformities may be confined to the nasal bones and cartilages or may be seen in conjunction with a more extensive deformity. When untreated or inadequately treated there may be resorption of bony fragments, fibrous union or malunion with excessive callus formation. Depending on the direction of the deforming force, the nasal pyramid may be deviated or flattened. An anteroposterior defect results from crushing of the supporting walls and collapse of the supporting bony structures. The nasal septum may be buckled by the impact and displaced according to the direction of the force, and buckling may obstruct one or both nasal passages.

An unacceptable appearance due to distortion of the nasal pyramid is the most common presenting symptom. The nature of the injury determines the type of deformity, which may involve only the bony portion of the nose above, or both bony and cartilaginous parts (Fig. 21.33).

The bony nasal skeleton may be deviated to one side, or one nasal bone and maxillary process may be depressed more than the other; the cartilaginous lower nose may be displaced in conformity with the displacement of the bone, or may be deviated in the opposite direction. The cartilages may be torn from the bone and displaced. The dorsum may be depressed in either its bony or its cartilaginous portions.

Malposition of the septum is responsible for deformity, not only in the dorsum but also at the columella level. There may be retraction of the columella or displacement of the caudal septum so that it presents in one or other nostril. The septum is often responsible for alteration in the patency of the nasal airway, the other common presenting symptom. It is the underlying deformity of the nasal septum which produces airway obstruction; obstruction may also be due to adhesions between the middle and inferior turbinates, and the septum. Underlying developmental variations of the middle and inferior turbinates, particularly bullous enlargement on the contralateral side of a pre-existing deviation, will further complicate the obstructive picture. Septal damage may also be associated with a nasoseptal perforation, giving a whistling noise in breathing, and causing crusting in the nose.

Disruption of the lateral wall of the nose and consequent stenosis and obstruction of the normal ventilation and drainage of maxillary ethmoidal and frontal sinuses can lead to infection. This usually becomes apparent some time following injury and needs active treatment only if symptoms persist that are not controlled by medication, or if further surgery is planned.

The nasal valve area is the narrowest part of the nose and nasal airway and is the most important site of airway obstruction. Trauma can produce deviations of the septum in this area and also stenosis and webbing of the attachment of the upper lateral cartilage to the septum producing very troublesome obstructive symptoms. Trauma to the lower lateral cartilages may cause not only cosmetic deformities but also collapse of the lateral vestibular wall particularly on inspiration which is a most distressing symptom.

Naso-orbito-ethmoid deformities

Where a nasal fracture extends into the orbit, the term naso-orbito-ethmoid fracture is appropriate (p. 308). Malunited fractures of this type are complicated by the presence of the lacrimal system and the medial canthal structures; they also cause problems with orbital volume and contour and may result in an abnormally shaped palpebral fissure by disturbing the attachments of the medial canthal ligaments. There may be posterior displacement of the comminuted fragments of the nasal bones and the frontal process of the maxilla which may protrude into the orbital cavity as well as injure the lacrimal sac.

There is characteristically a flattened bridge of the nose and telecanthus; the eyes appear to be apart, there is often canthal dystopia, and if infection is present the tell-tale swelling of a blocked and infected lacrimal sac is seen. There may be cutaneous scars and prominent epicanthic folds. The deformity may be unilateral or bilateral (Fig. 21.18A). The abnormal 'eye' slope is due to the disconnected medial canthal ligament (and attached bony fragment) being distracted by the continuous pull of the orbicularis oculi muscle (Fig. 21.34).

Localized Nasal Deformities

Assessment

These deformities present with a history of nasal injury, often not of great severity. Examination reveals a malunited fracture involving the nasal bridge and the lateral walls of the nasal cavity, with no extension into the adjacent frontal or orbital regions. The causative fracture involves the nasal bones, the frontal process of the maxilla and the septal cartilage, and these are deformed in varying degrees (Fig. 21.35).

A complete internal examination by the team ENT surgeon is mandatory. The internal physiology of the nose and paranasal sinuses must be examined, as well as the external form of the nasal pyramid. Photographs are taken to show front profile and basal views of the nose. Plain X-ray pictures demonstrate the anatomy of the septum and the state of the paranasal sinuses (p. 19S). CT scan shows the details of septal deformity and nasal architecture in general very well (Fig. 21.33); CT scans will also show the details of the distorted architecture in the medial orbital walls in the canthal region.

The ENT assessment may indicate a need for sinus drainage; chronic suppurative disease of the sinuses needs to be dealt with before major reconstructive surgery, particularly if it is intended to insert free grafts of cartilage or bone.

In the more complex injuries, assessment of the lacrimal drainage apparatus is important and a dacryocystogram may be useful (p. 178).

Management

Emphasis must be placed on the correction of both function and form. Ideally, both dysfunction and deformity are corrected in a single operation. It is our experience, however, that 10–20% of cases will need some later 'touch-up' operation because of the tendency for scar tissue to produce unpredictable results as it forms and matures: there is much variation in the endpoint of healing in this facial region. In very badly damaged nasal pyramids where the anteroposterior crush element is combined with a nasoseptal deviation, it is often wise to plan a two-stage operation: a first stage to centralize the nasal pyramid and reconstruct the septum and a second stage to graft the nose to obtain the desired definitive shape.

Correction of deviation

The nasal skeleton must be exposed and this can be done intranasally (Fig. 21.36). In severe deformities of the septum and nasal skeleton, the open external approach provides the necessary exposure, not only to appreciate all the underlying deformities but also for their correction. For lesser deformities the intranasal or classical aesthetic rhinoplasty incisions suffice. The greater the deformity the more extensive the exposure necessary.

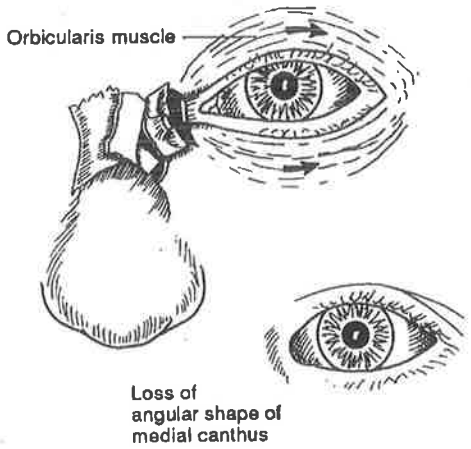


FIG. 21.34. ***Canthal dystopia.*** *The shattered medial canthal region is distracted laterally by pull of the orbicularis oculi muscle. giving the rounded appearance of the medial canthus which is typical of the deformity (after McCarthy et al 1990a).*

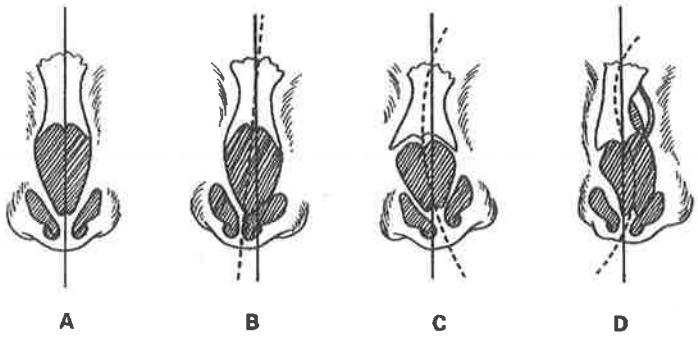


FIG. 21.35. ***Post-traumatic nasal deformity.*** ***A*** *Normal shape of the dorsum of the nose.* ***B*** *Lateral deviation after trauma. The whole nose is deviated to one side and the displacement is uniform.* ***C*** *Deviation with convexity of the crest on the right side, while the tip of the nose is displaced to the opposite side.* ***D*** *A 'double curvature' of the dorsum.*

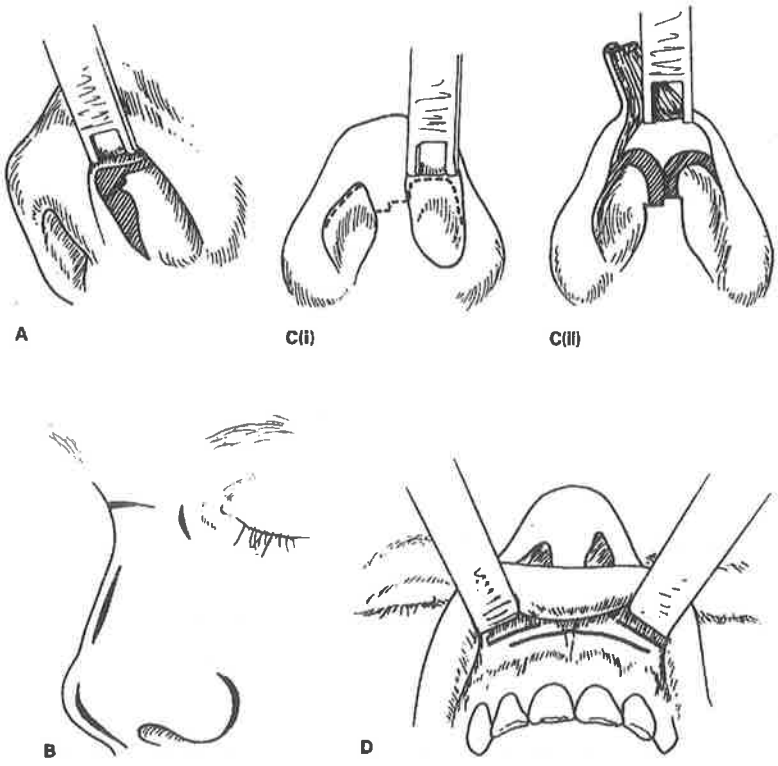


FIG. 21.36. ***Surgical approaches to the nasal skeleton.*** ***A*** *Transseptal incision extended into the intercartilaginous plane.* ***B*** *Various skin incisions available for achieving complete dissection of the nasal framework (Tessier 1991).* ***C*** *(i, ii) Open rhinoplasty technique with a transcolumella step incision leaving the nasal cartilages intact.* ***D*** *Intraoral transbuccal approach for exposure of the nose, either bilateral or unilateral.*

Dissection is facilitated by careful injection of 2% lignocaine and 1/80 000 adrenaline solution through a fine needle into the appropriate tissue planes, producing hydrodissection as well as vasoconstriction. The skin is widely freed from the bony and cartilaginous skeleton. Wherever possible the upper lateral cartilages are left attached to the septum and the septal mucosal dissection is not carried over onto the lateral cartilages. However, in more severe cases the upper lateral cartilages are freed from the septum, leaving the upper lateral cartilage attached to the nasal lining mucosa.

The mucoperichondrium and periosteum are dissected off both sides of the nasal septum through an incision at the mucocutaneous junction or via a hemitransfixion incision at the caudal border of the cartilaginous septum. This will give complete exposure of the entire septum, and correction of both anterior and posterior deformities can be addressed. Removal of non-supporting parts of the septum can be undertaken. These include part of the posterior cartilaginous septum as well as most of the perpendicular plate of the ethmoid and vomer. As much as possible of the anterior cartilaginous septum is preserved as it has important supportive and physiological functions. It is most important that even minor deviations of the septum in the region of the valve are corrected. Small amounts of cartilage in this area can be removed but enough must be left to provide satisfactory support for the anterior nose. Occasionally the entire cartilaginous septum may need to be removed and then replaced in a number of smaller but straight pieces. At the end of the procedure, replacement of bone and cartilage between the mucosal layers reduces the likelihood of perforations, stabilises the septum by reducing lateral movement or flapping and enables any subsequent surgery on the septum to be performed without too much difficulty.

Fracture lines in the cartilaginous septum should be excised; there is no place for scoring of cartilage on one or other side of the deviation in the expectation that this will correct the deformity. Adhesions or scarring in the region of the nasal valve can usually be treated by simple division and insertion of Silastic® stents between the septum and outer lateral cartilage. These are left sutured in situ for ~10 days to prevent recurrent adhesions.

There are many variants on the basic operative plan. Occasionally split-skin grafts or composite grafts of ear cartilage and skin may be needed. The septal incision can be extended into an intercartilaginous incision, through which the skin can be separated from the bony and cartilaginous framework. A direct approach can be made through the nasal skin by midline incisions through the columella, dorsum of the nose, glabella, alar base and lateral nose. We frequently use the dorsal nasal incision for fixation of bone grafts and the columella incision for reconstruction of the cartilaginous nasal framework. The vestibular incisions are very useful to completely free the scarred soft tissues from the piriform aperture; this is necessary when a columella strut is being used to obtain nasal projection. Once the airway is restored and the septum centralised by septal resection or septoplasty, the bony skeleton is osteotomized to reduce any displacement, and the nasal cartilages are repositioned and reshaped to produce a new centralised and symmetrical appearance (Fig. 21.37).

Osteotomies are made laterally at the base of the nasal pyramid and medially parasagittal to the midline. The superior attachment of the nasal bone can be osteotomized percutaneously with a tiny 2 mm osteotome or broken across by in-fracture or out-fracture. The lateral osteotomy is performed via a stab incision in the nasal vestibule laterally; the soft tissues are elevated from the frontal process of the maxilla with a Joseph elevator. The cut from the piriform aperture to the glabella is made with a saw and/or an osteotome; the thickening and distortion of previously fractured bones often make this manoeuvre difficult.

The paramedian osteotomies are performed with the saw and/or osteotome; the superior bridge of bone is cut with a small osteotome percutaneously or fractured by digital pressure. When the cuts have been made the nasal pyramid

can be centralised. Any bony or cartilaginous excess is then rasped or trimmed away. The central position of the septum is maintained with two Silastic® sheet splints, fixed with two sutures through the septum to produce a sandwich effect. The internal support of the nasal pyramid is provided by packing and the external moulding by a plaster of Paris splint (Fig. 21.37).

Correction of depression

The treatment will differ according to the degree and the site of the depression. Depression of the bony dorsum demands bone grafting. Depression of the lower cartilaginous part of the nose may also benefit from bone or cartilage grafting but other procedures can be used.

In principle, three strategies are available for repair of the lower cartilaginous part.

1. The bony pseudo-hump may be removed from above the dorsal depression, thus flattening and smoothing the profile. It is often also necessary to perform an osteotomy of the nasal pyramid to narrow it.
2. Small cartilage grafts taken from the septum, ear or costal cartilage can be used to build up the lower depression as well as to augment a recessed columella and deficient alar cartilages.
3. After flattening the bony dorsum with a rasp a very fine split-rib and costochondral-junction graft can be fashioned, the graft being fixed above to the nasal bone with a single screw for rigidity.

The principles for correction of total depression of the nasal body are well described by Tessier (1991). He stated that this reconstruction should always be done with bone, and that the bone must be fixed in the right environment, namely bone-to-bone surface. This dictum has been given scientific substance by Phillips & Rahn (1988, 1990). The nasal mucosa must be intact.

Bone grafting in the nasal region may be carried out through a variety of incisions, each with its merits:

1. An intercartilaginous incision is suitable for smaller distal deformities and does not facilitate rigid fixation of the grafts; it may need to be combined with a dorsal puncture or incision, to allow insertion of screws for fixation.
2. A vertical columella incision gives access to the alar domes and one can insert grafts of costal cartilage, septum or ear to augment the recessed columella.
3. An open rhinoplasty incision gives wide access to the lower cartilages as well as the bony dorsum.
4. An oral vestibular approach helps to free the piriform aperture.
5. A midline incision on the nasal dorsum: in our practice, this is the most common route by which bone grafts are inserted and fixed with screws. Pre-existing scars on the nasal dorsum may be used as access incisions. The bicoronal flap when used for other purposes provides good access.

To reconstruct the depressed nasal dorsum we agree with Tessier (1991), Merville et al (1483) and others in preferring to use autogenous onlay bone grafts taken at the time of operation. Iliac crest is preferred by Tessier, calvarial bone graft by Gruss et al (1985). Our preference is for rib and cartilage taken at the costochondral junction. Through the dorsal midline incision, together with a columella incision if necessary, the nasal bones are freshened and the graft is fashioned so that the rib bone overlies the nasal bone and the shaped cartilage lies at the nasal tip. The graft is secured with one or two countersunk titanium screws (David & Moore 1989), giving rigid fixation and allowing bone-to-bone union, while the cartilaginous component of the graft overlies the cartilaginous area of the nose (Figs 21.38 and 21.39).

Tessier and Merville both use transnasal wire to fix a shaped iliac bone graft, which is usually slotted into the glabellar region (Fig. 21.40). When the bicoronal scalp flap is used, or if an extended glabella incision has been employed, a titanium plate and screws can fix the graft and give the appropriate projection. An L-shaped graft may be preferred (Fig. 21.41), with a columella strut placed via the buccal sulcus incision or through a columella incision to help with projection of the nasal tip and to give bulk to a recessed columella. Since the advent of miniplate and screw fixation in the glabella region we do not use this technique.

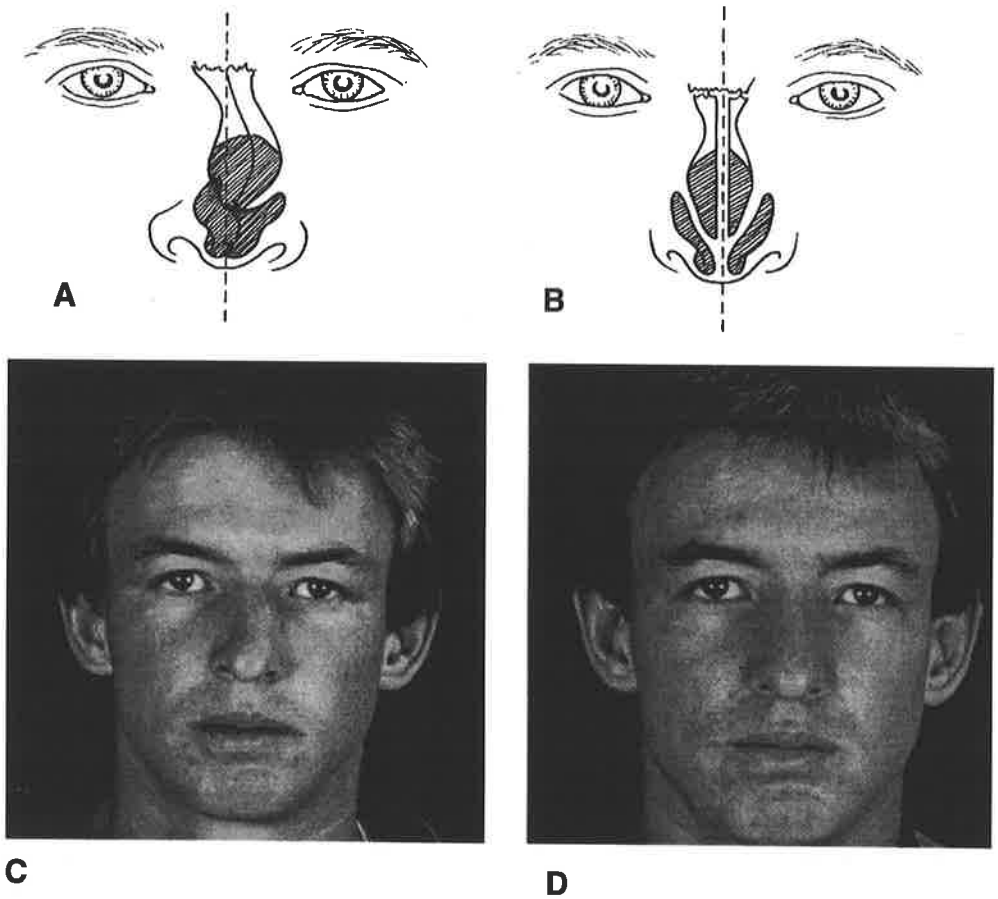


FIG. 21.37. ***Post-traumatic nasal deformity.*** *A Diagrammatic representation of the nasoseptal deformity.* ***B*** *The desired correction.* ***C*** *Appearance of the post-traumatic deformity.* ***D*** *Appearance 6 months after correction.*

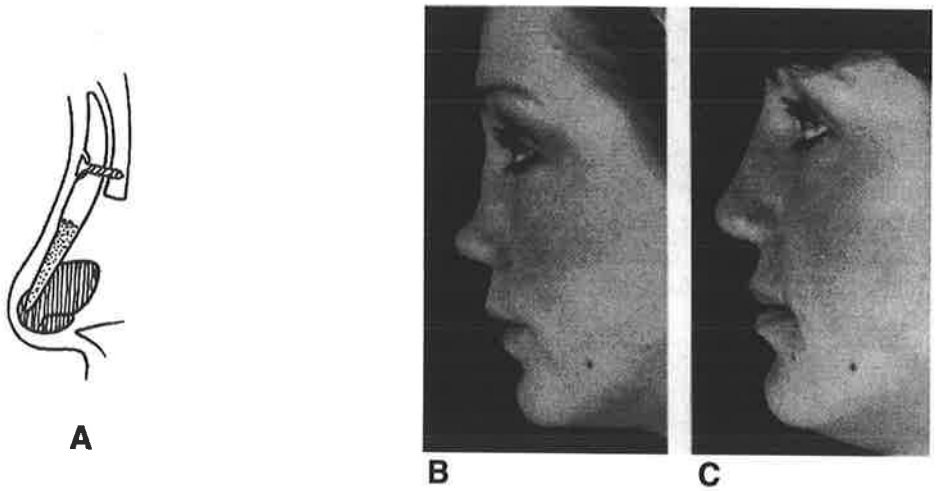


FIG. 21.38. ***Nasal bone grafts.*** ***A*** *Diagrammatic representation of the bone graft overlying the freshly rasped nasal bones as far as possible. This is fixed with a countersunk screw or, if required for stability, two screws.* ***B*** *A typically depressed nasal septum.* ***C*** *Appearance after bone grafting.*

Complications and results

Minor post-traumatic nasal deformities are common, and can be corrected by relatively simple surgical techniques. Rhinoplasty for localized post-traumatic nasal deformities is generally a satisfactory procedure. In a personal series of 185 consecutive rhinoplasties for post-traumatic nasal deformities of all types, early complications were listed in only 4.9%; these were chiefly secondary haemorrhage and persistent blockage of the nasal airway. Of those patients who required a nasal bone graft, it was necessary to remove the screw in some 10%; in 2.8% removal of the bone graft was necessary.

Complex Naso–Orbito–Ethmoid Deformities

Management

Correction of an established deformity is often very difficult. Once again, an early operation provides the best opportunity for repair (p. 311). Fundamental to the correction is restoration of the bony contour of the region. As has been pointed out by Gruss (1992), in most cases the canthal ligament is attached to a large fragment of bone. In these cases, restoration of the bony contour involves osteotomy, repositioning of the bone, fixation with miniplates or microplates, possibly bone grafting of the dorsum of the nose and reconstruction of the medial orbital wall where this is either protruding into the orbit or is absent (Fig. 21.42). The dorsum of the nose often needs bone grafting; this can be done with the costochondral graft described below, screwed into the underlying residual nasal bone or attached by an angulated plate to the frontal bone. Where it has been judged that there is too much scarring and the skin over the dorsum of the nose is tight, we have preceded this manoeuvre by inserting a small wedge-shaped tissue expander of 1–2 cc capacity which is blown up slowly over the next month. This has given very satisfactory results (Fig. 21.43).

Where there is an infected mucocele caused by obstruction of the nasolacrimal drainage it is important to clear up any infection beforehand. Having done this, it is probably best to proceed with the correction of any epicanthic folds which can be done by the Converse technique (Converse et al 1977) or the Mustardé 'jumping man' technique (Mustardé 1959). When these techniques are employed in post-traumatic cases, they are invariably associated with a canthopexy; if this cannot be achieved by repositioning the bone, then transnasal insertion of the canthus into the bone will be necessary. At the same time, all residual scar tissue around the medial canthus should be removed. It may be necessary to pass the canthal tendon into or through a bone graft and fix it to the other side of the nose (p. 312). Complete periorbital stripping is necessary to achieve movement of the medial canthus in post-traumatic cases.

Operative correction

The preferred approach for secondary deformity is a one-stage procedure in which osteotomies, bone grafting, rigid fixation, canthopexy and dacrocystorhinostomy are performed in a single operation. Surgical exposure may be via existing scars or through a glabella incision, vertical or horizontal, together with lower eyelid incisions. However, these are now almost invariably supplanted by the bicoronal scalp flap.

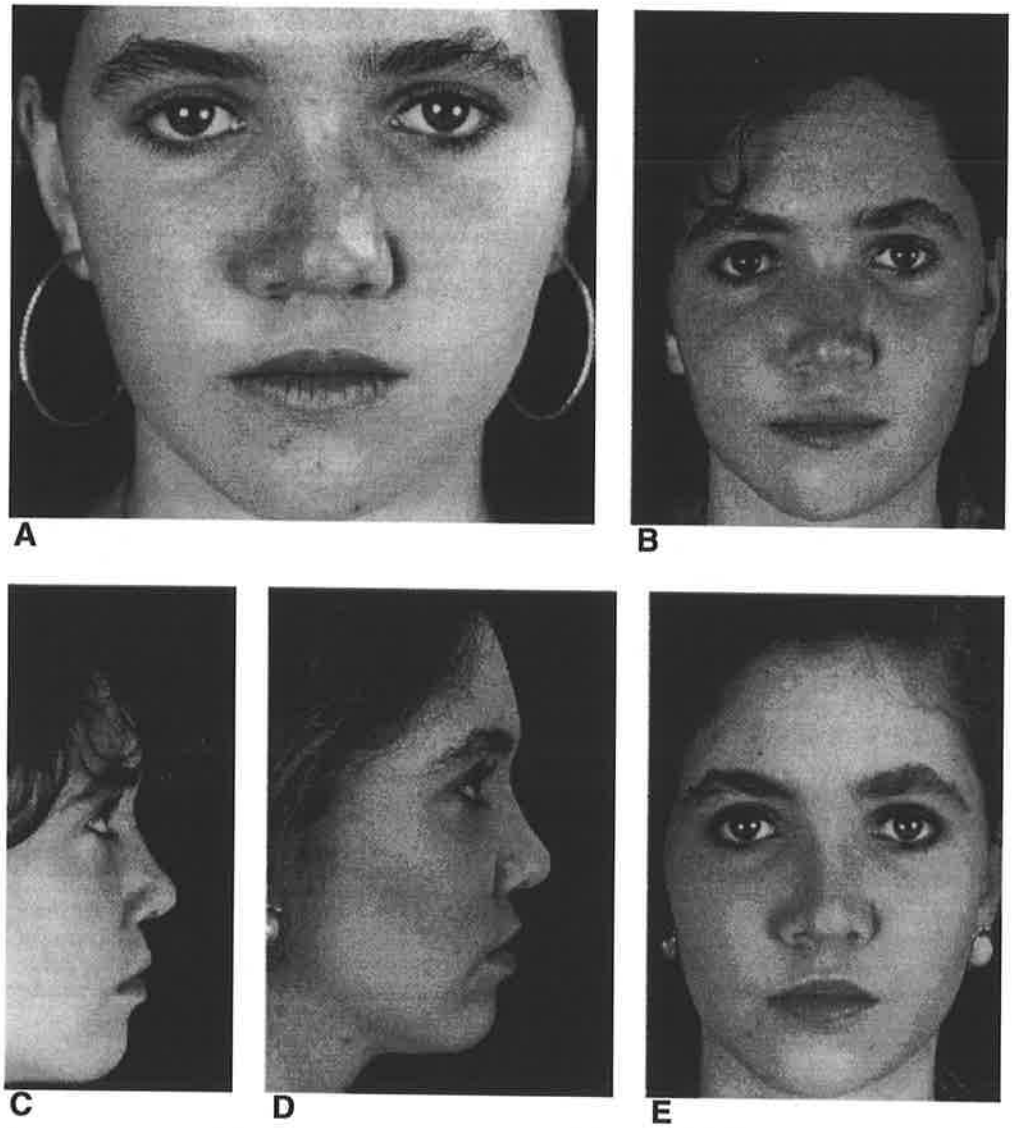


FIG. 21.39. ***Post-traumatic nasal deformity.*** *A teenage girl sustained a severe nasoseptal crush earlier in life with nasal deviation and blockage.* **A** *Initial presentation with a flattened, aesthetically unacceptable nose and blocked nasal airway.* **B** *Appearance following septorhinoplasty which has produced a narrower nasal pyramid, some projection of the pyramid, and a clear airway.* **C** *Postoperative lateral view.* **D** *Profile view after the next stage, in which a very fine costochondral graft was placed onto the dorsum of the nose as described.* **E** *Final appearance, with the small midline incision hardly visible.*

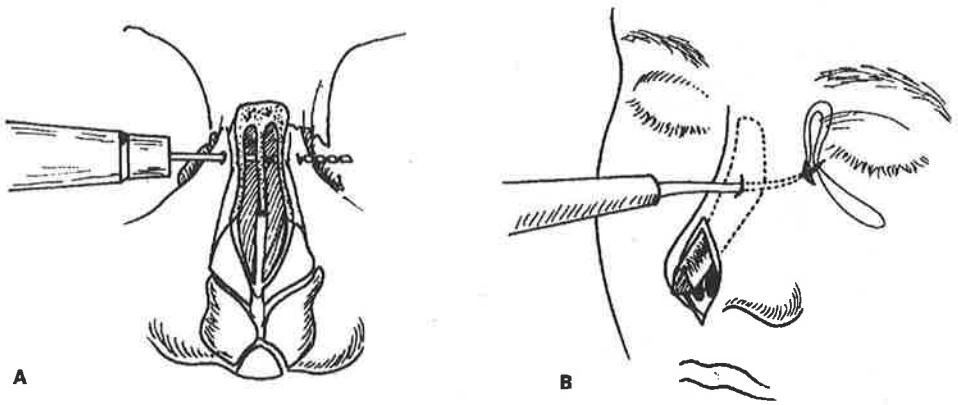


FIG. 21.40. ***Bone grafting the dorsum of the nose.*** *This technique is described by Tessier (1991).* **A** *Burrholes are placed in the frontal process of the maxilla. The bone graft, fashioned to produce a natural looking hump, is secured with a wire passed through the burrholes.* **B** *The graft is seated on freshened dorsal bone of the nose. The alar domes may be sutured over the bone graft.*

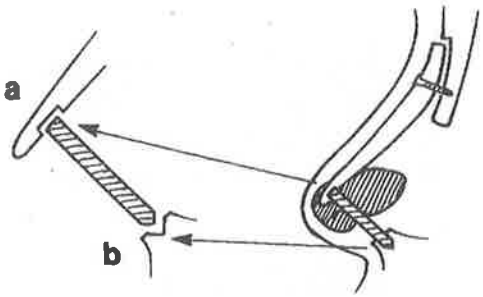


FIG. 21.41. ***Nasal columella support.*** *If the surgeon contemplates the need for a columella support, it is formed from cortical bone which may be embedded in a small groove* ***(a)*** *at the lower end of the graft. The base of the supporting strut is firmly embedded in a hole* ***(b)*** *prepared at the level of the anterior nasal spine. The screw fixation technique has obviated the need for this more cumbersome and less rigid form of reconstruction.*

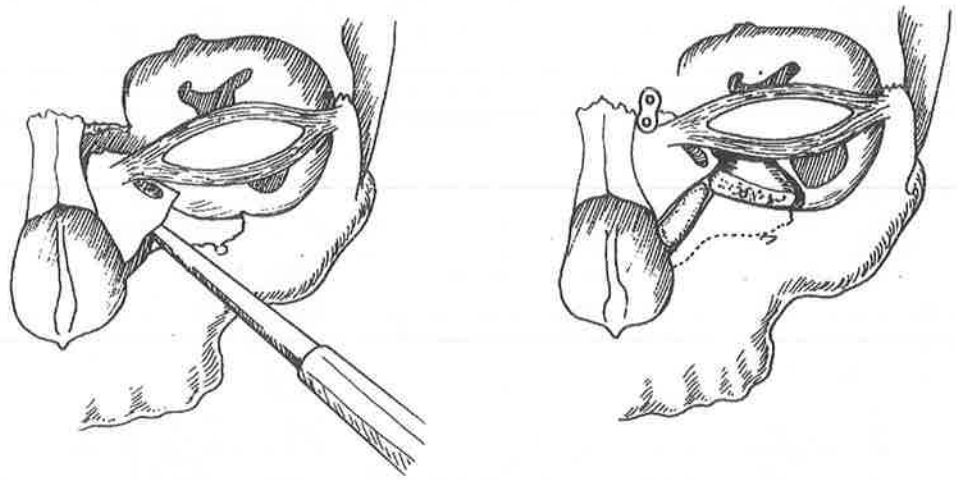


FIG. 21.42. ***Displacement of the medial canthal ligament.*** *When the medial canthus is distorted and the canthal ligament is attached to a displaced bony fragment, it may be possible secondarily to identify the fragment and perform a repositioning osteotomy. After repositioning, the fragment is best stabilised with a mini- or microplate, and any resulting defects bone grafted.*

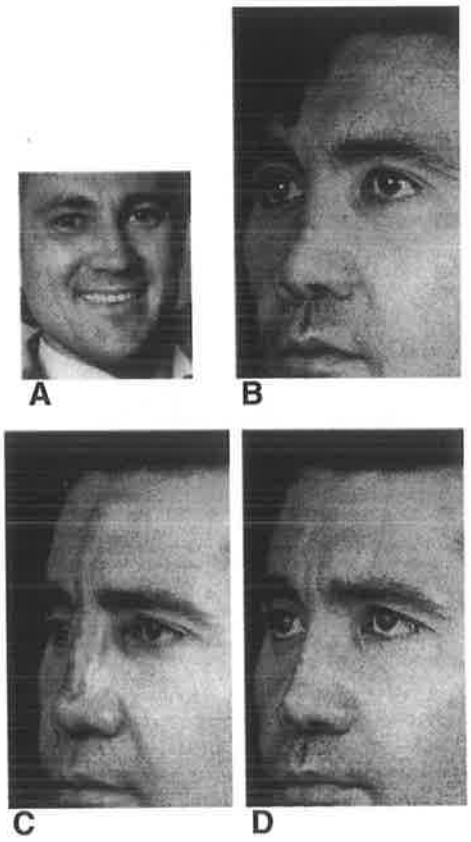


FIG. 21.43. ***Tissue expansion prior to nasal bone grafting.*** ***A*** *Patient seen before his massive naso-orbital-ethmoid fracture.* ***B*** *Postoperative appearance. He complained of a change in nasal shape.* ***C*** *Due to the tight skin and scalded dorsum, a small tissue expander has been placed under the nasal skin and gently expanded over a month.* ***D*** *Replacement with a costo-chondral graft fixed at the glabella.*

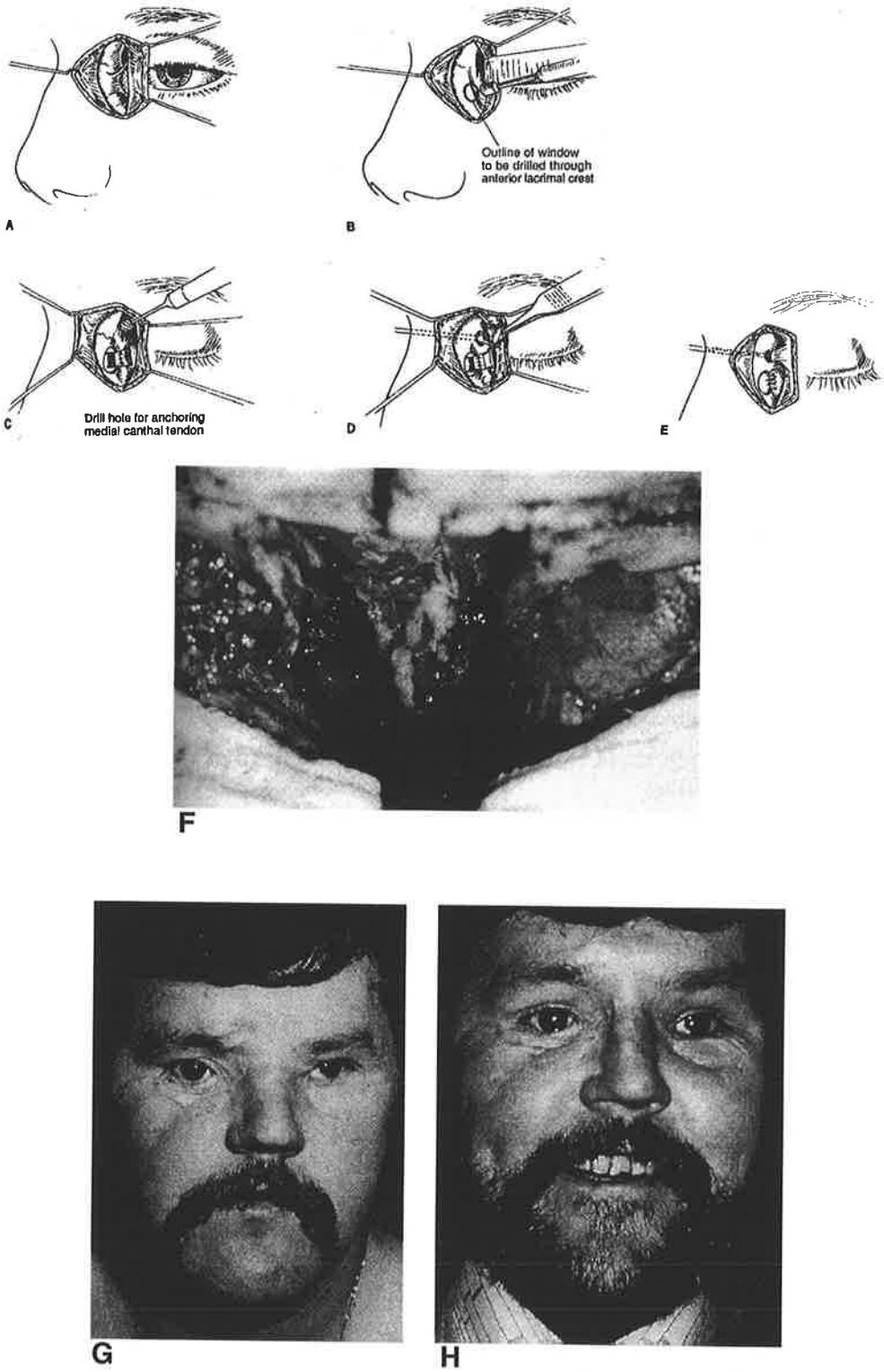


FIG. 21.44. ***Combined dacrocystorhinostomy and medial canthopexy*** *(after McCarthy et al 1990a).* ***A*** *Medial canthal incision. Alternatively, access can be gained from a bicoronal scalp flap.* ***B*** *Window to be drilled through the area of the anterior lacrimal crest, which may be significantly distorted.* ***C*** *When possible, the sac lining is sutured to nasal lining and a drill hole for anchoring the medial canthal tendon is performed above. This is done usually transnasally.* ***D*** *Medial canthal ligament fixed into a burrhole in the bone by wire passed across the other side.* ***E*** *Reattached canthus after the completed procedure.* ***F*** *Bicoronal view indicating the exposure for bone grafting the medial orbital wall and dorsum of the nose, canthopexy, and dacrocystorhinostomy. It is important that the operative sequence proceeds from the back forwards: reconstruction of the medial orbital walls and orbital floors completed, canthopexy started, repair of the lacrimal passages completed, canthopexy completed, dorsal nasal bone grafting completed, and skin closure with appropriate Z-plasties or W-plasties.* ***G*** *Patient with a gross naso-orbital-ethmoid fracture. Approached by the bicoronal scalp flap, the medial orbital walls and dorsum of the nose were grafted and medial canthopexies and dacrocystorhinostomy were performed on the left side.* ***H*** *Appearance 1 year after surgery.*

It is often wise to inject the nasolacrimal drainage apparatus with methylene blue. The medial orbital walls are dissected, the orbital floor is dissected in its medial part, the canthal ligament is identified where possible, and if attached to an identifiable fragment of bone, it is osteotomized. Those who have experienced this form of surgery know how tedious it is and how unrewarding the dissection of the scar and fragmented bone can be. The reconstruction proceeds from the posterior aspect forwards, the medial orbital wall being repaired with bone grafts first, and the dacrocystorhinostomy is performed before the medial canthus is attached (Fig. 21.44). If canthus is not attached to a fragment or fragments of bone which can be plated back into place, then the canthus itself is plicated with wire, which is then passed well posterior to the lacrimal groove or its supposed position, to anchor the canthus. In the past, the wires have been used to assist in the fixation of the dorsal bone graft; however this has been supplanted by screw fixation It is often necessary to correct a downward displacement of the medial canthus, particularly in long-standing cases, and this may be corrected by repositioning a bony fragment. Often a Z-plasty of the soft tissue is required (Fig. 21.45).

Complications and results

It is our experience that satisfactory results in these complex deformities are rarely achieved in the ideal of a single corrective procedure. Complications include:

- canthal drift, giving a wide flattened appearance between the eyes
- prominent wires and screws
- persistent infections resulting chiefly from blockage of the nasolacrimal drainage system.

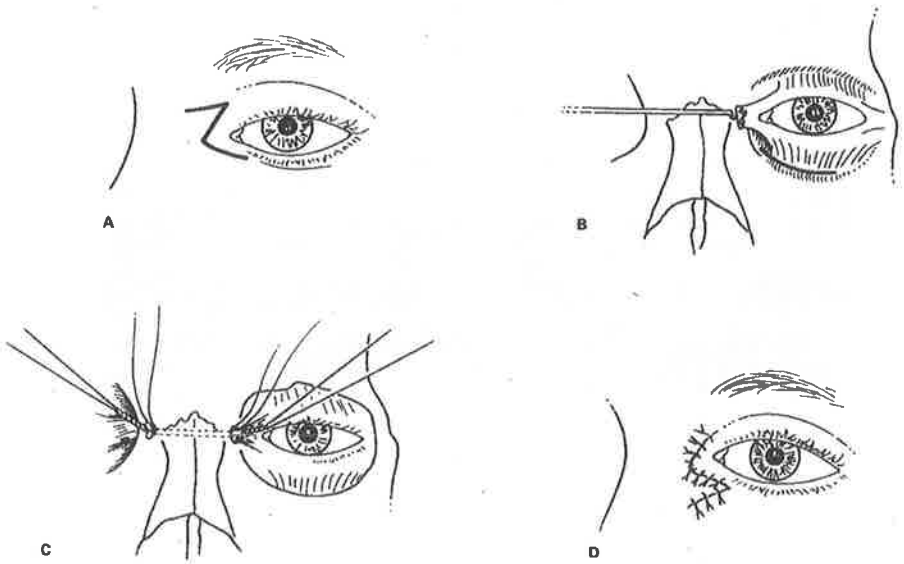


FIG. 21.45. ***Medial canthus repositioning with Z-plasty.*** *Z-plasty in the medial canthus for correction of canthal dystopia. The medial canthus is reattached into the bone and through to a toggle on the other side, thus creating an inferior defect which is filled from the flap above.* ***A*** *Design of the incision.* ***B*** *Repositioning of the canthus.* ***C*** *Medial canthus attached to the toggle on the opposite side, but well inserted into the bone.* ***D*** *Completion of the procedure with closure of the Z incision*

Massive Ablative Deformity Of The Nose

Management

Massive deformity of the nose involves significant loss of cover, support and lining. Techniques to achieve reconstruction under these circumstances are as old as plastic surgery itself, nasal reconstruction using the forehead flap being first mentioned in the Sanskrit writings of Susruta, thought to be no later than 600 BC and very possibly earlier (p. 8). Ever since, the forehead has been the most common source of skin cover; an excellent historical review of the development of techniques of nasal reconstruction of the forehead has been made by Adamson (1988). Gillies' 'up and down' flap was further developed by Converse (1942) as a scalping flap (p. 453). Since Tagliacozzi's brilliant use of the arm in the sixteenth century (p. 12), other sources of skin have been used. Distant flaps have been advocated by Washio (1972), bringing postauricular skin onto the nose. Free flaps have been used, vascularized by microsurgical techniques, nice results being produced at the 9th People's Hospital in Shanghai with radial forearm flaps (T. S. Chang, personal communication, 1986). Nevertheless, forehead skin remains the best substitute for nasal skin replacement as it is adjacent, of good colour and good texture though often of limited area (Fig. 17.18). The use of tissue expanders in the forehead as part of total nasal reconstruction has been advocated by Adamson (1988) and is now widely accepted.

Tissue expansion

This has been with us since the beginning of the human species, in the form of the expansion that results from pregnancy. The phenomenon is also seen when skin, nerves and muscles and even bone expand over an underlying haematoma or a slowly growing tumour. Neumann (1957) first used artificial expansion to facilitate a surgical reconstruction; more recently Radovan (1982, 1984) introduced surgical expansion for a wide range of reconstructive purposes. The technique enables tissue adjacent to a defect to be expanded, thus providing donor tissue of similar colour, texture and sensation with minimal scar formation. A temporary expander, capable of being filled with fluid by injection through a valve system, creates a pressure which exerts force on the overlying tissue; this gradually expands, providing the additional tissue for reconstruction. In replacing traumatic defects in the craniomaxillofacial (CMF) area, this technique may be used to expand scalp, face and neck tissue or tissue around the ear for secondary auricular reconstruction. Tissue expansion may also be used for the preforming and pre-expanding of a distant flap prior to its transfer by microvascular techniques to the head and neck region (Homma et al 1993).

Tissue expanders are now produced in a wide variety of sizes and shapes. The first stage is the insertion of the expander. Radovan in his earlier articles indicated that the base of the expander should be of similar diameter to the defect which is to be closed. A smaller pocket is developed for the reservoir dome. Initially, a small amount of normal saline is injected into the expander through the connecting tube — approximately 10–15% of the capacity can be introduced. Further injections are generally commenced after 10 days; each is monitored by watching for blanching of the skin and by the pain tolerance of the patient (Marks et al 1987). When the tissue has been completely expanded, and after allowing ~14 days between the last expansion and the planned time of operation to consolidate the effects of the expansion, the expander is removed and the tissue is transferred to close the defect.

Haematoma, infection and tissue necrosis are the chief complications (Austad 1987, Steenfos et al 1993). Insertion of a suction drain into the cavity will help to avoid these problems. If infection develops, it normally spells the end of that phase of the expansion, which may have to be abandoned. Necrosis is a rare occurrence; monitoring the expansion and deflating the expander if there appears to be a threat of ischaemia will usually prevent or control this complication.

Nasal lining

There are many techniques for providing nasal lining. They include inversion of the forehead flap to produce the nostrils and columella, lining the inner surface of the forehead flap with medium-thickness split skin, turning down local flaps of residual nasal skin, and the use of local flaps from the face, such as nasolabial tissue in patients where this is abundant. In total nasal reconstruction, it is essential to have good cover and good lining, and the skeleton must be reconstituted to provide good support: expanded forehead skin and free flaps will contract mightily if not draped over an adequate skeleton. With the use of plates and screws, the cantilever costochondral graft secured to the glabella region has proved in our hands to be an excellent method of securing skeletal support.

In post-traumatic cases, complete nasal reconstruction requires multiple interventions over a long period of time. There are often difficult scars in the region which make tissue expansion more hazardous, and may indeed necessitate a free flap reconstruction, which itself is often made difficult by damaged recipient vessels. Therefore there is need for precise preoperative planning.

Timing of reconstruction

When the patient has been managed by the CMF team since the time of injury, it may be difficult to decide when to do the definitive nasal reconstruction. Initially, the patient's life must be preserved and the principles of acute care of a massive avulsive injury are set out in Chapter 16. Nasal reconstruction is best performed on a face that has been otherwise fully reconstructed: the procedure is therefore done towards the end of a reconstructive programme. In our practice, the decision is usually easier, because the vast majority of patients present late, with well-established problems for secondary correction—unhappily often after many earlier attempts at reconstruction.

Operative correction: rhinoplasty

The preferred technique of rhinoplasty is a staged sequence of procedures, using forehead skin with tissue expansion of this region where necessary and where wound patterns permit.

In the first stage, the forehead skin is expanded by inserting a 200–300 cc rectangular expander in the forehead under the galea via a bicoronal approach where the scar is sited well behind the hairline to ensure that the suture line is not compromised by tension generated by the expander. The valve of the expander is placed over the temporoparietal region. Haematoma formation around the expander is reduced by a suction drain left for 48 h postoperatively, and generally brought out on the side opposite to the valve. After 3 weeks, expansion is begun and continued over the next 2–3 weeks until enough expanded skin is obtained. Necrosis and exposure of the expander are avoided by careful siting of the scar and by great care in expanding the prosthesis, using pain and skin blanching as indications of the limit of saline injection. Superior results are obtained if there is a further delay of 2–4 weeks after full expansion.

Nasal reconstruction can now be planned 'in reverse'. The nasal lining is often fashioned from a residual nasal skin flap based on the margins of the defect and turned over so that the raw surface is exposed. The forehead skin flap is planned to be based on one or other of the supratrochlear vascular pedicles.

In the next stage, the forehead flap is raised. The skin is cut with a knife and the capsule that forms around the expander is incised with cutting diathermy to avoid rupture of the expander. The expander is then removed and the flap is cut so that it can be rotated downwards to cover the nose on one or other of the supratrochlear vascular pedicles. The fibrous capsule that has formed around the tissue expander is carefully dissected away to provide a thinner flap and to mobilize the rest of the scalp for straight-line closure.

The next stage involves fashioning of the nasal lining. Our preference is to use local nasal remnants where possible. The distal flap turnover techniques, in which the distal end of the flap is turned in to form the nasal lining, nostril margins and columella, work quite well. Skeletal support is usually a costochondral graft, two-thirds bone, one-third cartilage, screwed into the glabella with one or two titanium screws sunk into the bone. Where there is not enough residual bone to give the right angle of the cantilever, an angled titanium plate can be used with the plate bent to give the right projection. The flap is then set in over the graft and the nostrils are held patent by shortened Silastic® 'trumpet' nasal airway tubes. After a few weeks these can be replaced by Silastic® nasal splints used for cleft palate nasal reconstruction. The forehead defect is closed by direct suture; if necessary a small split-skin graft can be used to cover any raw area at the glabella where the base of the flap may be exposed.

There is always a third stage, and possibly more stages, necessary for division of the forehead pedicle, thinning of the flap, possible re-augmentation of the skeleton and re-establishing an adequate nasal airway. The third stage is best left at least 3 months after the reconstruction and ideally for much longer if possible.

Complications and results

Complications of rhinoplasty include skin necrosis and recurrent deformity. To minimize the risks of skin necrosis as a complication of tissue expansion we choose the subgaleal plane and thin the flap later, though Adamson (1988) reports superior results by subcutaneous dissection. During the expansion process care must be taken to avoid haematoma formation, which is generally disastrous. If skin breakdown and expander exposure are threatened one may retrieve the situation by slowing the rate of expansion without abandoning the procedure.

The cartilaginous tip of the graft may produce necrosis of the overlying nasal skin. This should not cause alarm as the necrosed skin can be excised and the underlying cartilage pared down to a suitable size.

Recurrent deformity may result from shrinkage or collapse of the reconstructed nose. To prevent this, skeletal reconstruction is mandatory as the flaps, whether expanded or free, will shrivel if there is no underlying skeleton. Rigid fixation is necessary and bone grafting may need to be repeated to get the required nasal projection. Nostril contraction is a considerable problem requiring secondary surgery; this complication is best obviated by the use of nasal splints for up to 6 months (Figs 21.46–21.48).

Despite this intimidating list of possible complications, restorative rhinoplasty is in general a satisfactory procedure. Aesthetic perfection cannot be promised, but the result should be a great improvement on the appearance of a mutilated nose.

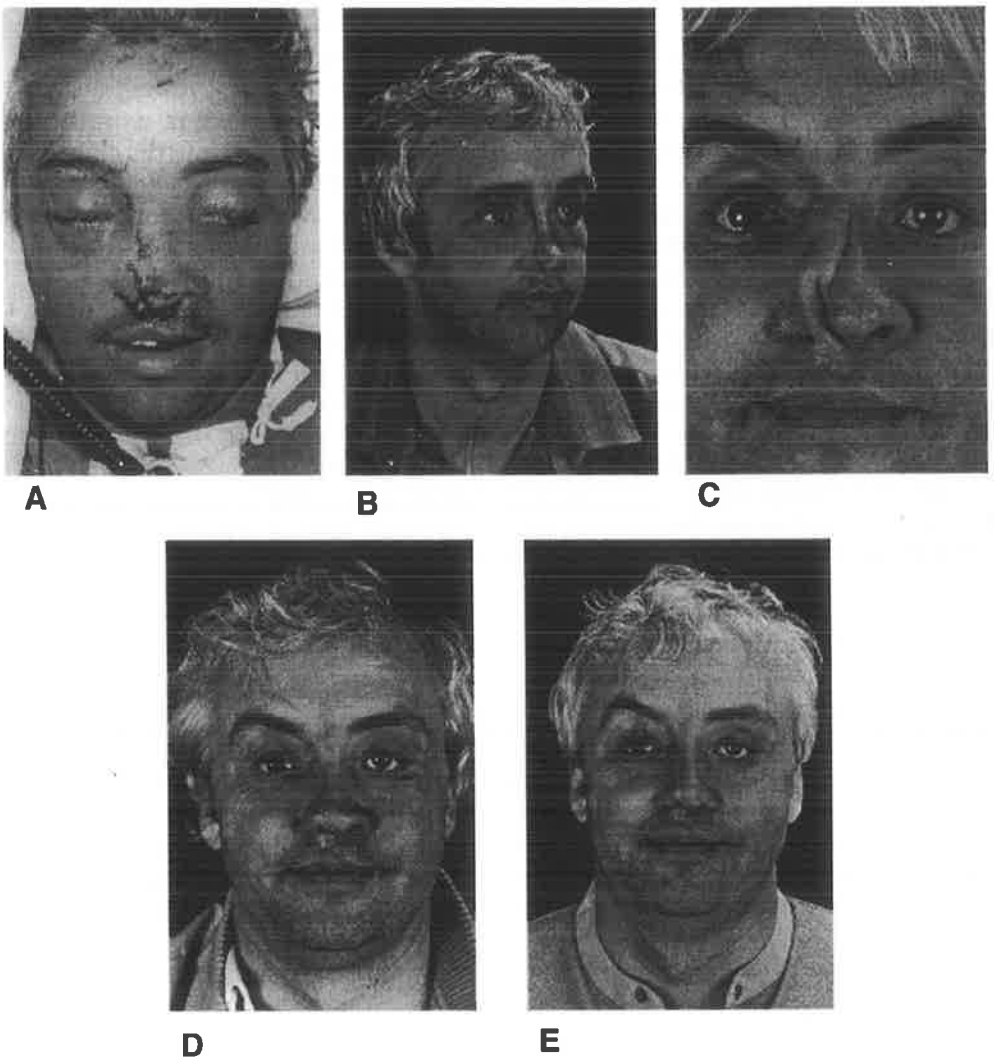


FIG. 21.46. ***Nasal reconstruction.*** *A 45-year-old male was involved in a motor vehicle accident in which he sustained multiple limb fractures and extensive fractures of the middle third of the face involving loss of nasal tissue and compound comminuted naso-ethmoid fracture, The soft-tissue loss included almost two-thirds of the right ala and part of the cartilage and skin of the right side of the nose with a laceration extending up to the medial canthal region. A series of operations were planned using a forehead flap based on the glabellar region, which was rotated down to provide extra tissue, turned over to provide an alar rim, and divided at a second stage. The local tissue was refashioned and ultimately the dorsum of the nose was expanded with a small tissue expander. A costochondral graft was fixed to the glabellar region with a single 9 mm titanium screw.* ***A*** *Initial appearance. Tracheostomy has been placed and the avulsion injury of the right side of the nose has been closed primarily.* ***B*** *Appearance following fixation of his facial fractures. The residual nasal deformity is evident.* ***C*** *Forehead flap rotated down to add soft tissue to the nose.* ***D*** *Penultimate stage in which the right ala has been refashioned and the nasal dorsal skin replaced in part.* ***E*** *After tissue expansion of the dorsal skin with a small cylindrical expander, a costochondral graft gives greater nasal projection.*

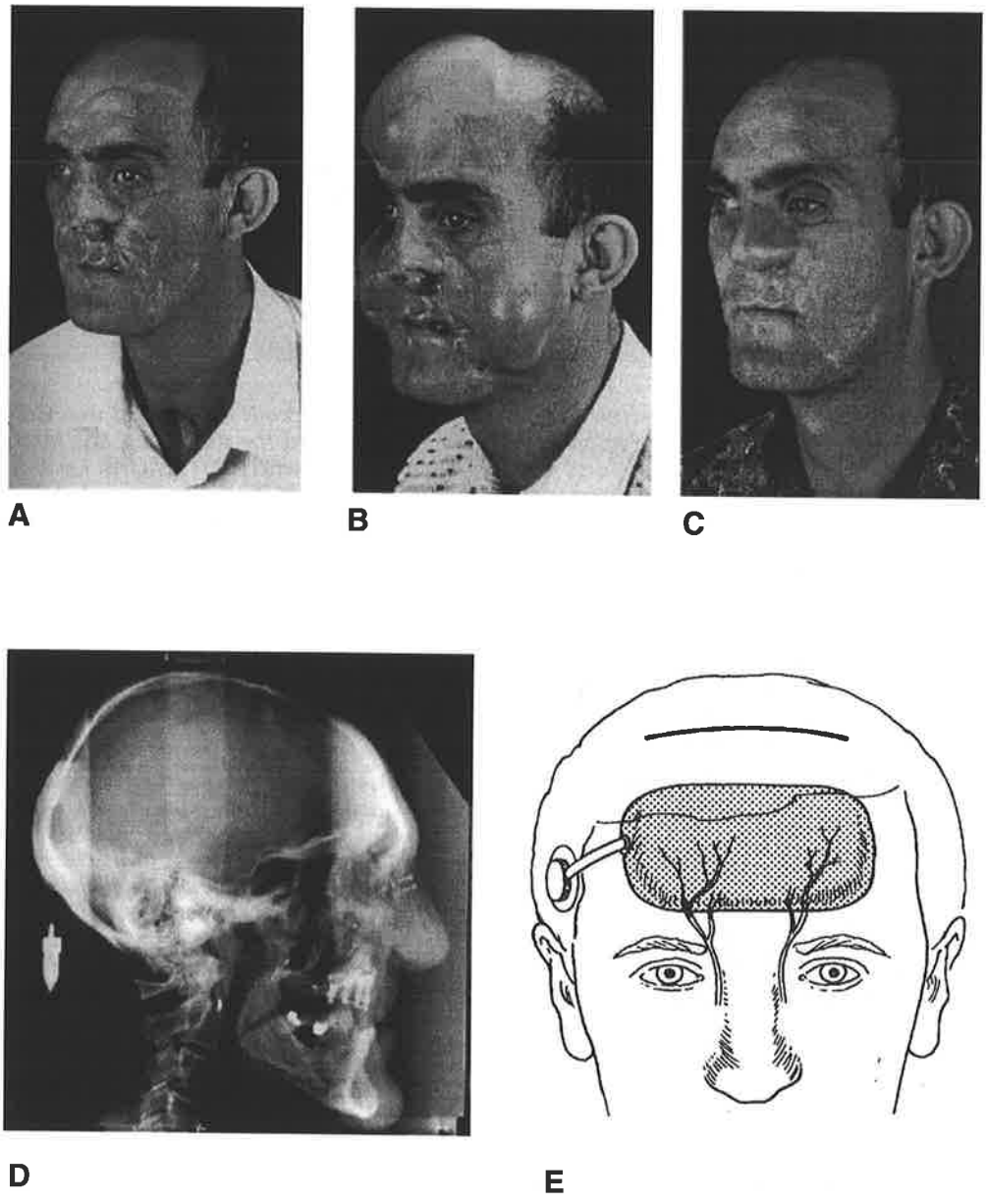


FIG. 21.47. ***Nasal reconstruction.*** *A young civilian male involved in a rocket attack in Beirut suffered a massive injury to the middle third of the face. While the reconstruction of the nose should not be seen in isolation from the rest of his deformities, it is illustrative of the use of tissue expansion, as there had been at least ten previous attempts to reconstruct his nose. A large tissue expander was placed into the forehead, with the port placed well into the parietal region, and the expansion was continued over 2 months. The whole nose was replaced as a cosmetic unit using the residual nasal skin for lining. The subsequent nostril patency was maintained by a pair of Silastic trumpet splints for 2 weeks postoperatively; then these were replaced by the smaller splints typically used for rhinoplasty associated with cleft lip repair.* ***A*** *Initial presentation.* ***B*** *Three tissue expanders have been placed to move undamaged skin medially from both cheeks and inferiorly from the forehead for nasal reconstruction.* ***C*** *Appearance following nasal reconstruction and simultaneous costochondral cantilever bone grafting.* ***D*** *Lateral radiograph demonstrating the nasal reconstruction as well as the subsequent ,CAD/CAM produced osseo-integrated implant in the deficient maxilla.* ***E*** *Position of a forehead tissue expander used for nasal reconstruction. The injection port can be placed in the parietal region, and the incision for insertion of the expander is kept sufficiently far away from the edge of the expander and behind the hairline if possible. Care must be taken to preserve the arterial supply to the expanded skin.*

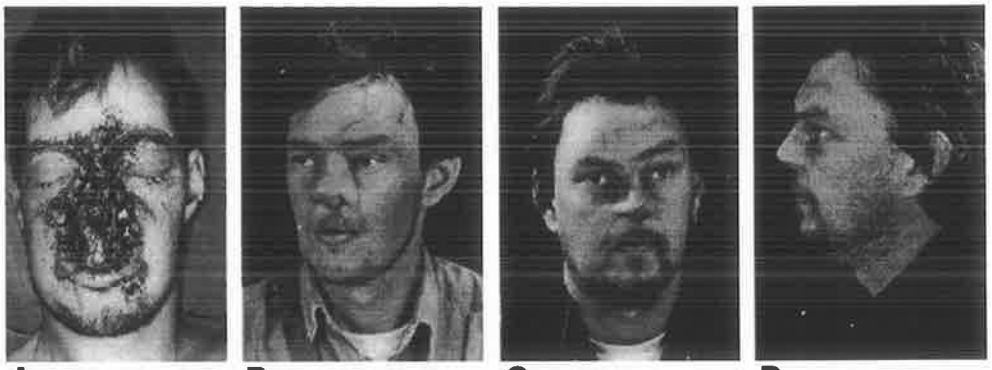


FIG. 21.48. ***Total loss of the nose and part of upper lip from a gunshot wound.*** *The nasal soft tissue has been replaced by a forehead flap and the distal end has been turned over to make the columella and nostril margin. Subsequently this complex needed to be radically thinned and supported with further bone grafting. This was secured at the glabella region with a four-hole titanium plate, with two screws into both the nasal bone grain and the frontal bone.* ***A*** *Initial presentation, with total absence of the nose and centre part of the upper lip.* ***B*** *Appearance after initial fashioning of the nasal soft tissue.* ***C & D*** *Appearance after subsequent thinning and bone grafting.*

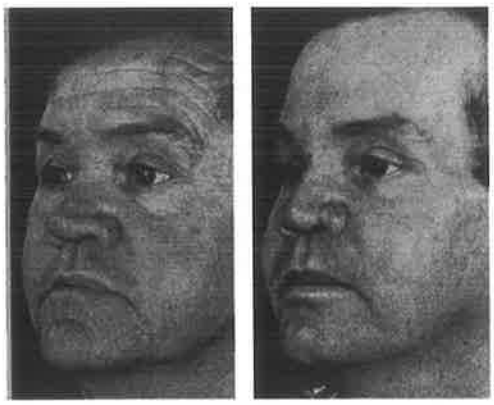


FIG. 21.49. ***Post-traumatic changes in nasal anatomy.*** ***A*** *A patient suffered a panfacial fracture in which most of the impact was taken on the middle third of the face. The midface is retrodisplaced and foreshortened, with widening of the alar base.* ***B*** *Appearance following advancement of the maxilla, replacement of the zygoma, and augmentation of his nose. The emphasis has been on elongation of the maxilla and autorotation of the mandible.*

Maxillary and Mandibular Deformities

Principles of management

The established post-traumatic deformity may be due to malposition of bone, loss of bone or soft tissue, loss of specialised such as teeth, or these in any combination. The result is a loss of form, function and aesthetic appearance.

The aim of aesthetic reconstruction is of necessity measured against the pretraumatic appearance of the patient and this is often impossible to restore (p. 83). The functions of speech, swallowing, mastication, and respiration may be compromised in a way that makes comfortable living very difficult. The complex anatomy of the region, interwoven with vital functions, and the need for aesthetic acceptability make secondary surgical correction of deformity in this part of the craniofacial skeleton very challenging, and nowhere are the needs for precise planning more evident.

Initial failure to achieve a correct functional and cosmetic restoration of the dentofacial complex can be caused by at least three factors:

1. The severity of the initial injury may necessitate prolonged maintenance of life support systems, and healing can occur before definitive surgical steps can be taken to restore normal form and function to the face. This is seen particularly in the maxilla and in young patients. Untreated displaced fractures of the jaws can be regarded as united at any time after 3 weeks from injury.
2. Accidents often occur in remote areas so that early treatment is not carried out in units that have the necessary expertise in the different specialty areas to provide the optimum result. Often the appropriate diagnostic radiographs are not easily available and so the extent and severity of facial injuries is not correctly assessed.
3. Finally, poor initial management can result from incorrect assessment of the extent of injury, or failure to reduce and fix bone fragments adequately.

Hopefully, we believe that the integrated multi-disciplinary approach to initial CMF injury management is today minimising the frequency and severity of secondary deformity. Improved fixation methods reduce the number the of patients requiring secondary facial correction: the use of miniplates rather than interosseous wires gives very accurate union of fractured facial bones. Again hopefully, the number of secondary deformities resulting from delayed or incorrect management should become less as better retrieval systems are in place to serve remote areas and as the primary treatment of trauma by general surgeons and general medical practitioners is improved. But it is important to understand that with almost all severe injuries, some residual disabilities will result. This must be accepted. Failure to recognize and to accept some reduction in the cosmetic and functional result may lead to repeated wasteful and futile surgical interventions and other treatments.

While one of the most important aspects of reconstruction of the middle and lower face is occlusion of the teeth, the more complex comminuted fracture may produce noticeable aesthetic changes even after malocclusions have been corrected.

Maxillary Deformities

Surgical pathology

The most common deformity caused by a comminuted fracture of the upper jaw is flattening of the profile and shortening of the vertical dimension. There is often associated perialar flattening, widening of the alar base of the nose and flattening of the nasal bridge. The result is a significant change from the premorbid appearance which is most distressing to the patient (Fig. 21.49). Less common is the elongated face where the disconnected maxilla has been distracted downwards and fixed in a low position, seen occasionally when craniomandibular fixation has been misused. Asymmetrical deformities result from twisting of the maxillary segment as a whole or splitting of the palate with malposition of other segmental fractures.

Clinical assessment

The victim of an accident often has difficulty in adjusting to an altered appearance and frequently complains of feeling and looking like a different person. Malocclusion is a common symptom. This may be severe and obvious, expressing a wide fracture displacement. In other cases the changes may be subtle and limited to the dentoalveolar complex; malocclusion may result from loss of teeth and alveolar bone or from changes produced by prolonged application of interdental and intermaxillary fixation. One of the main difficulties is to determine what the occlusion was prior to the injury, and old dental records may be very helpful.

Radiological assessment

Complete radiological assessment is necessary before planning correction. This includes the views necessary for planning orthognathic surgery (p. 177), including an orthopantomogram and biplanar cephalometric X-ray analysis. The pattern of the fractures must be identified and CT scanning is indispensable in this. Fracture sites and the extent and nature of any previous surgical interventions must be assessed. 3D data obtained by CT can be used for diagnosis of the deformity and for surgical planning (Abbott et al 1990). These data establish the shape of the maxilla and its spatial position with respect to the adjacent bones, and define any absence of bone. Such data are also used in construction of a maxillary prosthesis when necessary, by the CAD/CAM technique (p. 637).

Dental assessment

Detailed assessment of the dentition is important, and includes testing for viability of the teeth. The condition of the periodontal tissues must be known and any necessary treatment of caries and other inflammatory conditions performed. Dental models are taken and mounted on an articulator to facilitate operative planning (Fig. 21.50).

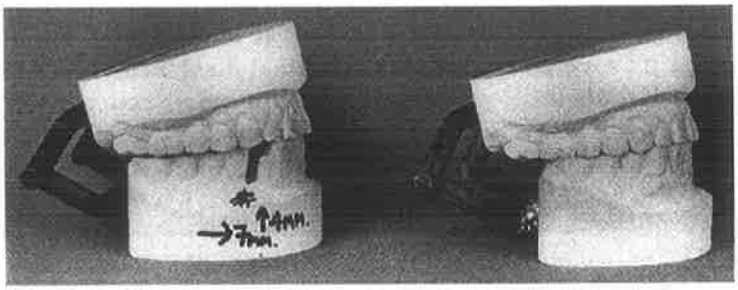


FIG. 21.50. ***Dental models mounted on disposable articulators.*** *The model on the left shows the traumatic occlusion, with written notes on the dental stone indicating the degree of displacement. The model on the right is produced by sectioning the lower model and re-establishing the occlusion. The interdental wafer is then produced from these prediction casts.*

Photographic assessment

This is an important record and is part of the routine work-up for any surgery on the face. Lateral, frontal, basal and occlusal views are taken. It is important, whenever possible, to have pretraumatic photographs of the patient. An understanding of the state to which the patient wishes to be restored is of vital importance to the treating team; examination of old photographs is quite often startling and the change brought about by the trauma and subsequent treatment may be alarming to the surgeon as well as the patient. Photography also provides the basis for a discussion about what is possible in each case.

Speech assessment

Trauma affecting the jaws, teeth, oral and nasal cavities must of necessity affect speech and mastication. The possible effects of nasal obstruction and neurological deficit due to head injury must also be taken into account. Each patient should have a speech assessment to document the situation with respect to hypernasality, hyponasality, lateral escape, and tongue position during speech; any problems with mastication and swallowing must also be recorded before secondary surgical intervention. A full ENT examination documents the state of the maxillary antrum, nasal physiology and middle ear function; if necessary endoscopic examination of the maxillary antrum may be done.

Management

Treatment aims at restoration of function (occlusion, airway and sinus drainage) as well as restoring the displaced or deformed maxilla to its correct position in relation to the other facial bones.

Planning
The information gathered from the clinical examination and special investigations is discussed at a multidisciplinary planning meeting which usually includes the patient. Decisions are made as to whether surgical intervention is needed or not, and whether orthodontic or prosthodontic treatment is needed, or combinations of each of these, in order to re-establish the occlusion and facial form. Priorities are established and a treatment plan formulated.

The first step may be dental restoration and orthodontic alignment of teeth, followed later by further dental model analysis and X-ray analysis in preparation for surgical movement of bony segments.

Surgery performed on the dental model precedes the actual intervention and determines the desired position of the maxilla or its segments; this is recorded with an occlusal wafer. The desired vertical dimensions are established in the model surgery but must be related to possible problems with overlying scarred soft tissue. For example, when lengthening an impacted maxilla, one must be careful when the lips are very scarred as the soft tissue will not follow the bone readily under these circumstances and an ideal maxillary reconstruction may expose too much tooth and gum. Compromises may be unavoidable.

At this stage the design of the fixation is determined (Fig. 21.51). The planning meeting also considers the possibility of secondary surgery, such as a second 'tidy up' operation after the initial major surgery with additional bone grafting or soft-tissue revision.

Correction of maxillary deformity
The pattern of osteotomy obviously depends on the pattern of the fracture and the nature of the deformity, and the surgical team should be prepared to do any manoeuvre necessary to restore the bony skeleton as nearly as possible to its original condition. To re-establish normal occlusion, surgical repositioning of the upper jaw may involve mobilising the whole maxilla, or only one or more segments of the dental arch. The whole jaw may be moved at the Le Fort I, II or III levels, with lengthening, rarely shortening, rotation or other segmental movement according to the needs established in the planning.

The most common surgical manoeuvre is the Le Fort I osteotomy. While it has been, and to a certain extent remains, convenient to describe a midface osteotomy by reference to the fracture pattern described by Le Fort (p. 18), post-traumatic deformities rarely correspond exactly to his classical lines.

Before operations of this type, most patients are fitted with orthodontic bands. Just prior to surgery, arch bars are fabricated for the intermaxillary fixation needed at the time of the operation. Sometimes orthodontic treatment is not possible, either because of the nature of the injury or because of the unsuitability of the patient for orthodontic treatment on the grounds of age, state of dentition, or personality; in these cases, the operation has to be done using arch bars designed by the maxillofacial technician. These are then wired to the upper and lower teeth after induction of anaesthesia to provide intermaxillary fixation. An occlusal wafer manufactured according to the model planning and pretreatment surgical; assessment determines the position of the maxilla in respect to the lower arch. When the face has been shortened the length must be increased, taking into account the relationship of the front teeth and the length of the upper lip, and bearing in mind where possible the premorbid appearance. Soft-tissue scarring of the lips and face may modify the surgical plan, as may loss of teeth.

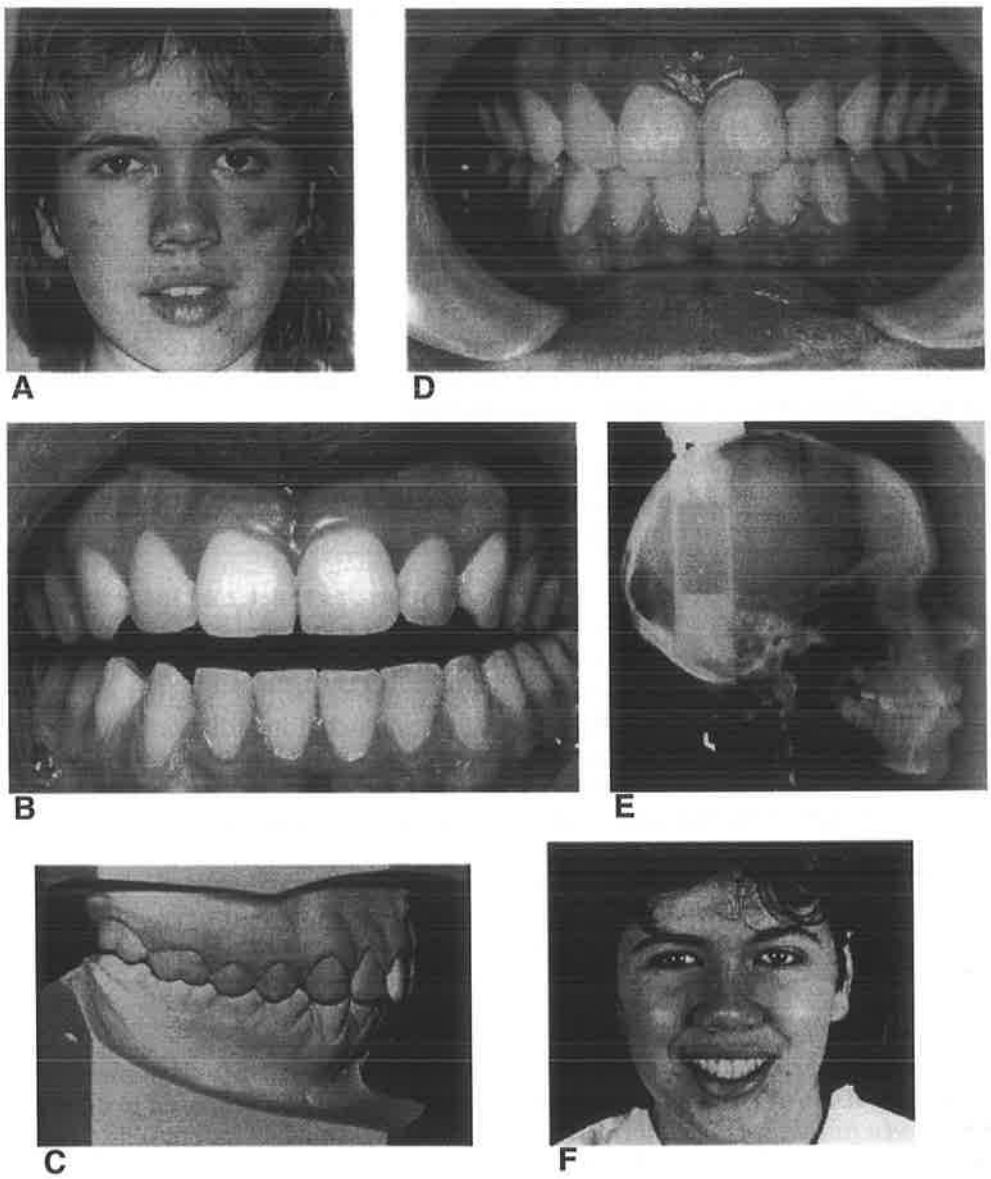


FIG. 21.51. ***Preoperative planning.*** *A 19-year-old girl was involved in a motor vehicle accident; she sustained a Le Fort I fracture which was initially not detected. 6 months later, she presented with a malocclusion. The models show the deformity and the planned surgical intervention.* ***A*** *Initial presentation with anterior open bite.* ***B*** *Close-up view of the malocclusion and anterior open bite.* ***C*** *'Plaster model surgery' predicts correction of the occlusion by 4 mm forward advancement and 5 mm downward displacement.* ***D*** *The occlusion 6 months postoperatively.* ***E*** *Cephalometric X-ray, taken at the same time, shows the restoration of her maxillary form.* ***F*** *Postoperative facial appearance. Nasal length and position have been restored.*

Correction of deformity is not easy. Measurements taken during the planning of surgery are transferred to the patient at operation by callipers. These measure from a fixed point on the forehead to the teeth; and the measurements are important to obtain a precise position of the jaw segments. But there is always a role for the eye of the surgeon who has been engaged in the work-up and who is closely familiar with the case—the art of surgery.

A horizontal osteotomy is placed above the tooth roots along the anterior wall of the maxillary sinus and carried through the buttresses at the piriform margin, zygomatic and pterygoid regions (Fig. 21.52). This enables 3D repositioning of the tooth-bearing part of the maxilla. Previous fragmentation of the area must be taken into account and the bony fragments are studied carefully on the CT scan. Scars of the buccal and palatal area must be noted lest a wide soft-tissue exposure compromise the blood supply of the osteotomized segments; a tunnelling technique with minimal soft-tissue elevation may then be necessary. The usual way to effect the Le Fort I osteotomy is to separate the maxillary tuberosity area from pterygoid plates; this may not be easy as previous trauma, often leaves very indistinct planes obscured by a mix of dense scar and bony fragments. After completing osteotomy in the anterior maxillary wall, medial wall of the maxillary sinus, nasal septum and pterygoid plates, the mobilising manoeuvre of down-fracture completes the osteotomy of the posterior sinus wall osteotomy. This manoeuvre must be done with care and patience as scar tissue is often very extensive and too much force may do unnecessary damage. We have long since ceased to use instruments such as Rowe's disimpaction forceps which grab the segment like tongs; in post-traumatic cases these forceps risk damaging soft tissues and compromising blood supply. Instead the fracture is gently levered open with an elevator or osteotome, or on occasion formally osteotomized to free the fragments.

Bone grafts (iliac crest, ribs, and/or calvarial bone) are used to fill the gaps in the osteotomy lines and fixation is secured with titanium miniplates of such malleability as to be easily bent without later recoil. If possible, the plates are placed at the zygomatic and piriform buttress areas on each side, with two screws on each side of the osteotomy, and are used to fix the interpositional bone graft as well to give rigid fixation. Extensive damage in the region of the osteotomy may make it necessary to compromise this ideal pattern of fixation. When this is so, segments of cortical bone can be fashioned as a 'bone plate' and used to bridge areas of the bone and scar inadequate for fixation, by securing the 'bone plate' to solid bone at each end. We have never used the pterygoid region for interpositional bone grafting.

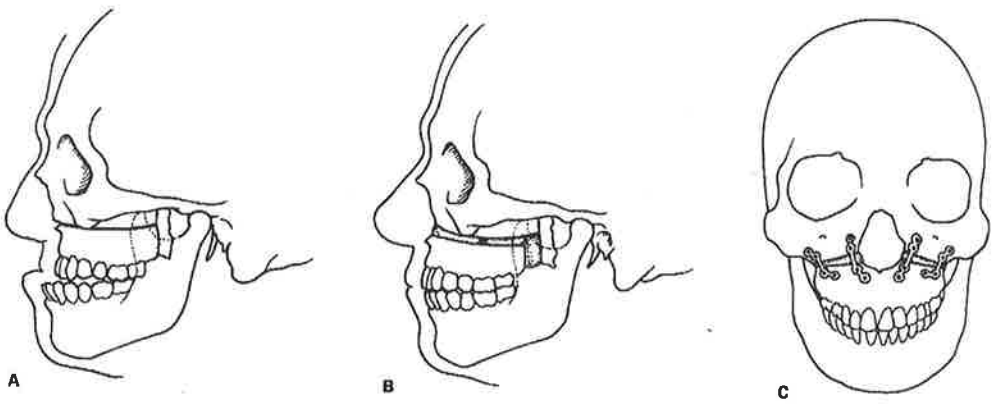


FIG. 21.52. ***Le Fort I osteotomy.*** ***A*** *The horizontal osteotomy is placed above the tooth roots along the anterior wall of the maxillary sinus and carried through the buttresses at the pyriform margin, zygomatic, and pterygoid regions.* ***B*** *Bone grafts are placed in the horizontal aspects of the cut if the advancement is more than a few millimetres. We have never placed these grafts between the maxillary tuberosity and the pterygoid plates although many surgeons follow this practice.* ***C*** *Four miniplates of the softer variety (without memory) are placed at the zygomatic and pyriform buttresses. These can be easily bent to shape.*

To establish the desired occlusion, the occlusal wafer is fixed to the maxillary segment. In most cases it is possible to dispense with intermaxillary fixation at the end of the operation; however, the occlusal status must be closely supervised and intermaxillary fixation re-established if necessary. Often intermaxillary elastic bands are useful to stabilize the occlusion and to give guidance on its long-term stability. This essential measure of control is lost if arch bars or dental appliances are removed too early.

In cases with a comminuted perialar extension of the Le Fort I pattern fracture, en bloc osteotomy is not only difficult to perform but does not produce good results. This type of deformity is therefore treated by a Le Fort I osteotomy plus onlay bone grafting in the region of the medial buttress; grafting extends in the perialar region from the lower part of the piriform aperture superiorly to the frontal process of the maxilla, with a bone graft to the nasal dorsum fixed with a screw in the glabella region. This can be combined with nasal septal surgery when necessary.

Secondary corrections sometimes require osteotomies more or less corresponding to the Le Fort II and III lines. However, as with the Le Fort I fractures, it is very rare for these lines to be well defined in facial fractures of the types seen today. The panfacial fractures for which secondary surgery is performed typically consist of a Le Fort I segment displaced to cause malocclusion, and a naso-ethmoid segment comminuted and retrodisplaced or concertinaed to produce the characteristic flattening and altered features, with one or both zygomas displaced. In fact, a deformity representing an en bloc Le Fort II or III fracture requiring the classical osteotomy is almost never seen.

If an en bloc osteotomy of the Le Fort II variety is to be performed, the area can be exposed through the buccal sulcus. Additional exposure may require vertical or horizontal incisions over the dorsum of the nose and lower eyelid or conjunctival incisions. Our preference is to make the approach through a bicoronal scalp flap in combination with the buccal approach. After forward advancement and any rotation or lengthening which may be needed, the gaps are filled with bone grafts and the segments stabilized with miniplates. The points of fixation are usually the zygomas laterally and the frontal bone medially. There have been many variations to these osteotomies. Where the Le Fort II osteotomy involves the orbital rim the cut must go behind the lacrimal fossa and this may cause a step in the glabella region which should be burred down (Fig. 21.53).

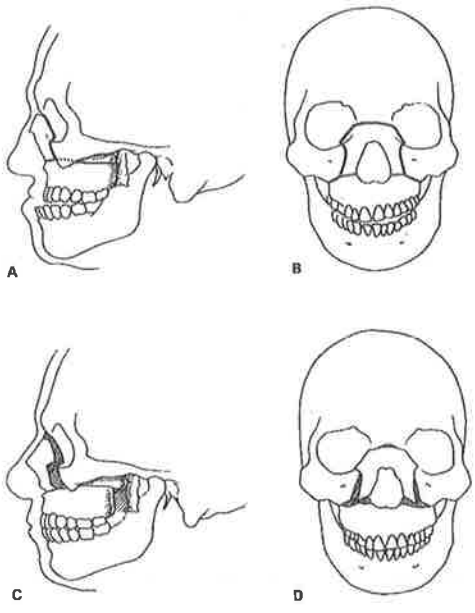


FIG. 21.53. ***Le Fort II osteotomy.*** ***A*** *The Le Fort II osteotomy can be done in combination with other osteotomies, either with the zygoma or a Le Fort I section.* ***B*** *We believe it important to have the upper part of the osteotomy behind the nasolacrimal apparatus to protect it, and to swing the osteotomy down medial and inferior to the infraorbital foramen to the line previously recommended for the Le Fort I osteotomy.* ***C*** *The gaps are bone-grafted and secured with miniplate fixation.* ***D*** *There is often a step in the infraorbital rim which needs to be grafted or smoothed over. The step at the glabella can be burred down and the gap grafted. Canthopexy and the addition of bone graft for nasal support are further options. In our experience it is best not to detach the canthal ligaments if they are reasonably well seated.*

The old displaced Le Fort III fracture is also a rare deformity (Fig. 21.54). The osteotomy lines have been described in the context of congenital deformities by Gillies & Harrison (1950), Tessier (1971) and Murray & Swanson (1968). The approach is via the bicoronal scalp flap and all the osteotomies are performed from above. In the frontal region care must be taken to cut below the level of the anterior cranial fossa, being mindful of the possibility that fragments of bone and scar tissue in this area may provide only a flimsy barrier between the nose and the cranial contents. Occasionally, the anterior cranial fossa needs to be exposed by the neurosurgeon to close a cranionasal fistula. A full transcranial approach is then necessary via a bifrontal or occasionally unifrontal craniotomy, with dissection of the anterior fossa and intradural (rarely extradural) repair of the fistula (pp. 339 and 379). This is done before the osteotomies are performed, and watertight dural closure is secured. After complete orbital dissection, each medial orbital osteotomy is made behind the lacrimal fossa, extended into the orbital floor and connected to the lateral osteotomy, which separates the zygoma from the greater wing of spheroid and extends into the inferior orbital fissure. The zygomatic arch is disconnected. The osteotomy between the maxillary tuberosity and the pterygoid plates is well done from above; a finger in the mouth placed over the maxillary tuberosity will feel the cut being made in the correct position.

It is more usual for the component bones to be separated and moved differentially according to the pattern of the deformity produced by the original fractures. Thus, the zygomas may be moved separately from the horizontal maxillary segment. Each zygoma can be repositioned and fixed with miniplates taking into account Gruss's strategy of accurate reduction of the zygomatic arch with attention to both its length and shape (Fig. 21.55). The segment mobilized by Le Fort I osteotomy can be located into the occlusal wafer and fixed to the mandible; the appropriate degree of lengthening is then produced by opening the mandible and so establishing the necessary downward displacement of the attached maxillary segment. The resulting gap can then be grafted. When dealing with the orbitonasal components of these complex midface deformities, there is a difficult judgement to be made in deciding whether to perform an osteotomy or to solve the problem with bone grafting alone. Similarly, the naso-ethmoid area can be osteotomized or more usually grafted (Figs 21.38–42).

Isolated dento-alveolar deformities

It is possible to reposition the anterior and lateral segments of a deformed maxilla as a secondary correction by applying the planning and operative manoeuvres described in Chapter 11. With appropriate X-ray examinations, the fracture site can be delineated; dental casts are cut and repositioned accurately and the appropriate osteotomy is performed on the model, from which a wafer is made. At operation, the segments are cut and located in the preformed wafer and fixed with miniplates and bone grafts where necessary. It is most common for such segments to derive their blood supply from the palatal segment and, as with other low maxillary osteotomies, great care must be taken to preserve this: there must be full knowledge of any previous scars in the mucosal and palatal attachments that might compromise the blood supply of the mobilized segments.

Maxillary deformity in edentulous patients

When the degree of displacement is minimal, then compensation can be built into the manufacture of new prostheses. There are, however, two situations that may call for secondary surgery for deformity.

The first arises in younger patients who have recently become edentulous, perhaps during the accident that has precipitated the call for treatment. Such patients may wish to have titanium implants for fixation of dentures. Dental titanium implants are a sophisticated form of denture fixation, best achieved when the maxillary (or mandibular) base bone is thick enough and so placed that the implants can be positioned vertically. If the maxilla is deformed significantly in any of its three dimensions then it should be repositioned with the aid of

miniplate fixation and Gunning splints (Fig. 21.56). A work-up is done as described above; model surgery on an articulator will indicate when the splints are located, what the position of the maxilla should be and what resulting bone defect needs to be grafted. If there is much loss of alveolar bone, autogenous bone graft(s) should be inserted. Planned insertion of titanium implants to fix a dental prosthesis (p. 637) can then be considered (Fig. 21.57).

Secondly, correction may be considered when there is an old and displaced fracture in an atrophic maxilla, so severe as to prevent successful denture retention whether with fixed or removable prostheses. Reduction is more difficult and repositioning of such a maxilla needs extensive bone grafting with the aid of miniplate fixation. The subsequent prosthetic management is the key to success of secondary surgery in such cases, whether it be by simple removable prosthesis, clip-on prosthesis with osseo-integration and a fixed prosthesis. In all such cases, patients must be fully informed of both the surgical and technical requirements that will be necessary to ensure a satisfactory result.

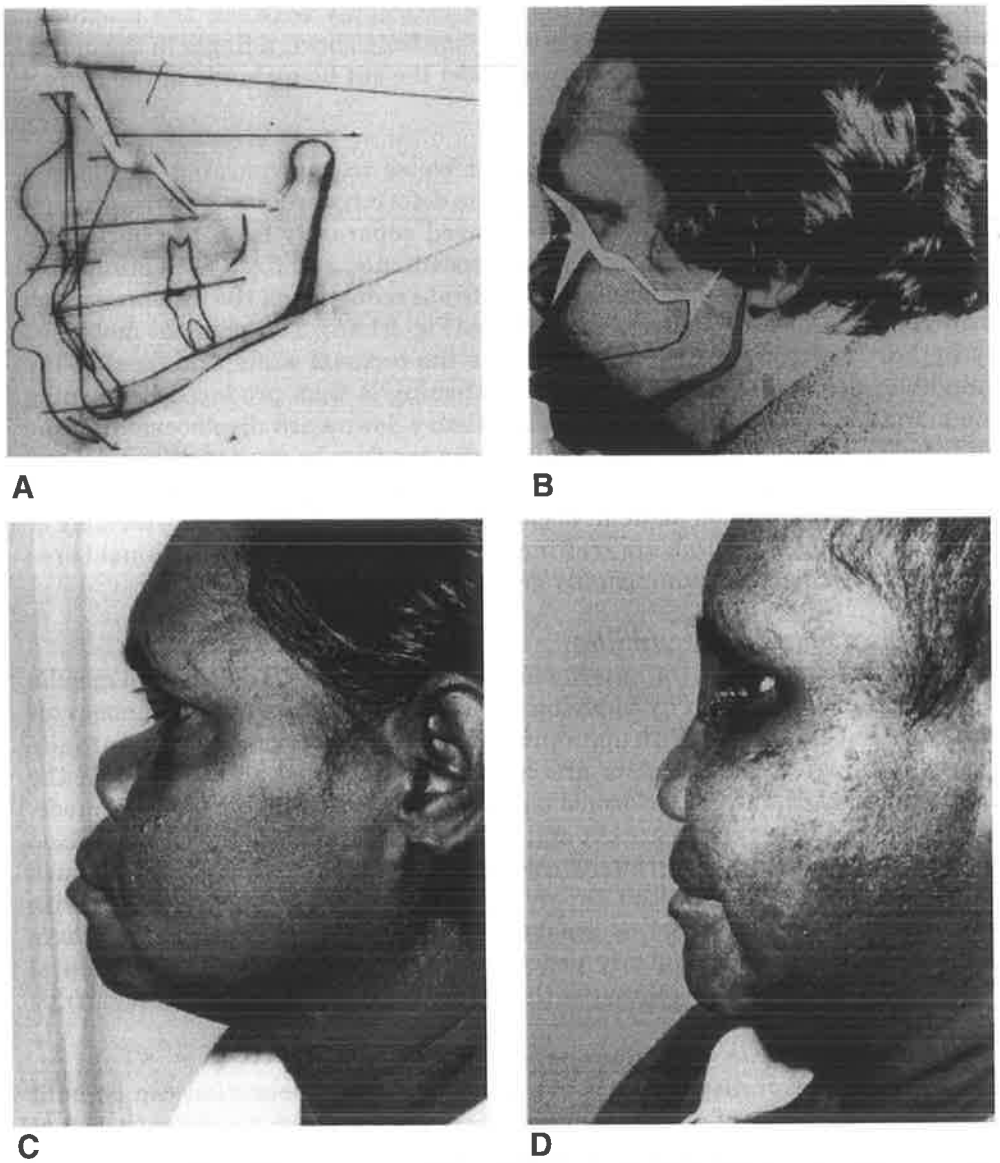


FIG. 21.54. ***Le Fort III osteotomy.*** *The midface can be moved forward en bloc as a Le Fort III osteotomy with the lines of disconnection from the upper skull made through the frontozygomatic suture, lateral orbital wall and anterior part of the zygomatic arch. More commonly the osteotomy is comprised of multiple segments, perhaps as many as a Le Fort I segment plus two zygomas and the naso-ethmoid complex. The latter procedure leaves much to be desired because of the instability of the previously fractured components.* ***A, B*** *Photocephalometric analysis has been used in this case (Henderson 1974), in which a life-size transparent photograph is superimposed on the cephalometric tracing and cut to reproduce the planned osteotomy and repositioning.* ***C*** *Patient with a previously uncorrected Le Fort III fracture.* ***D*** *Postoperative appearance, with the elongated and advanced middle third after subcranial Le Fort III osteotomy.*

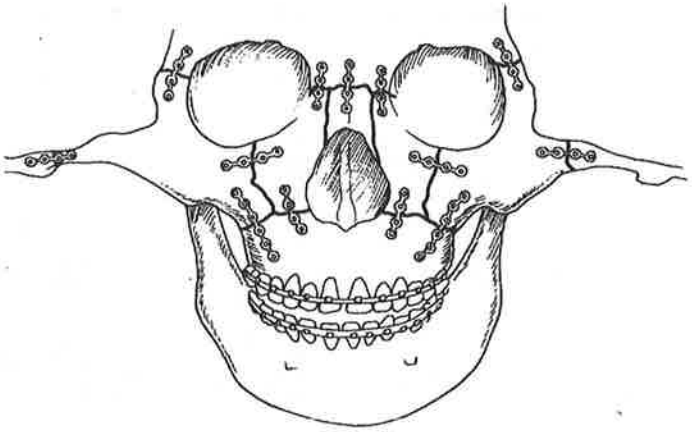


FIG. 21.55. *Reconstructive technique.* *After Gruss et al (1990). At the beginning the zygomatic arch and frontozygomatic suture line are secured giving anteroposterior projection and transverse facial width. The naso-ethmoid region may be reconstructed within this outer frame, and if the occlusion is then established onto a normal or accurately reconstructed mandible, the lower face will fit in harmony with this upper framework. If pieces of bone are missing, immediate bone grafts can be used to replace them with the same sort of rigid fixation.*

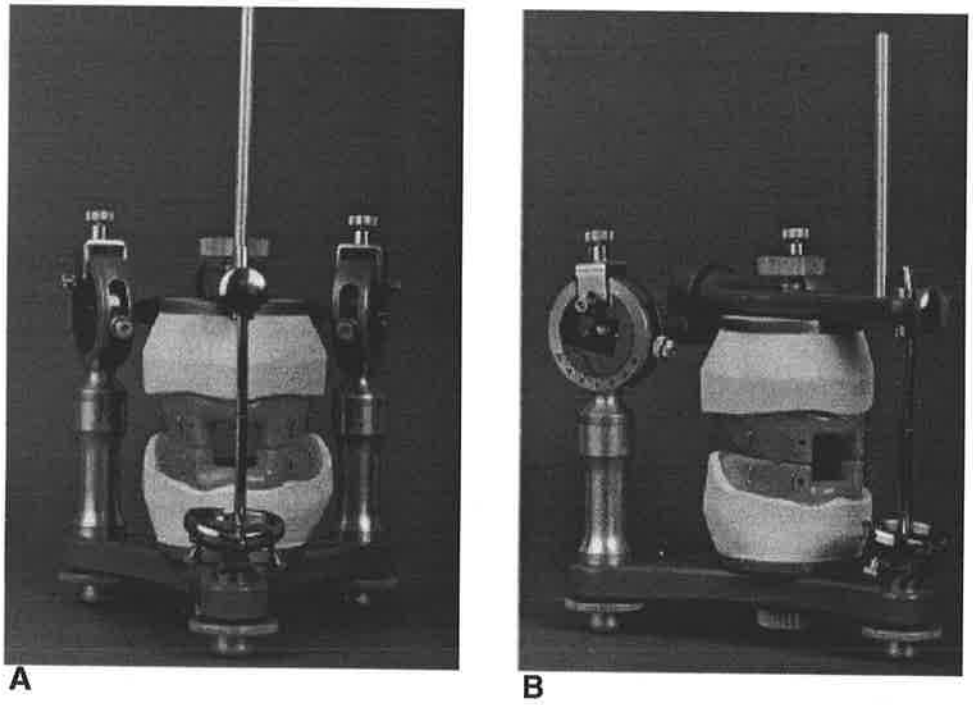


FIG. 21.56. *The Gunning splint.* *Example of a Gunning splint mounted on the articulator to indicate the planning necessary by the prosthodontist for the repositioning of the edentulous maxilla, with a goal of creating a denture suitable for cosmetic and functional purposes.*

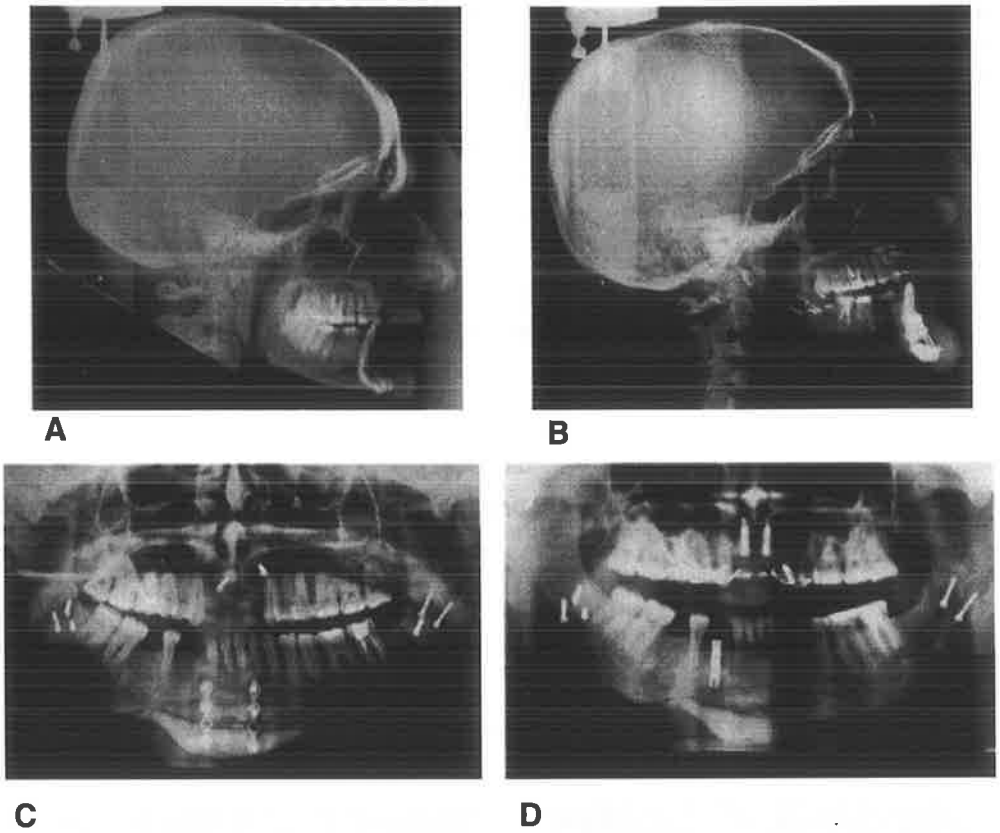


FIG. 21.57. ***Titanium osseo-integrated implants for dental rehabilitation.*** *A man with a previously fractured mandible, new alveolar fracture of the anterior maxilla, and loss of mandibular teeth.* ***A*** *Lateral X-ray before secondary treatment.* ***B*** *Appearance after advancement of the mandible and genioplasty.* ***C*** *Bone graft into the anterior maxilla held with two titanium screws to increase the bone mass in this area.* ***D*** *Osseo-integrated implantation supporting the anterior dental prosthesis as well as a single implant into the mandible.*

Temporomandibular Joint Ankylosis

Surgical pathology

Classification

Ankylosis of the TMJ may be fibrous or bony, intracapsular or extracapsular. Kazanjian (1938) was one of the first authors to classify these disorders in a logical way, and his terms remain useful. 'False ankylosis', called pseudo-ankylosis by many later authors, includes any mechanical obstruction to mandibular movement which does not represent pathology within the TMJ capsule. True ankylosis results from intra-articular disease processes which lead to bony or fibrous adhesions of the joint itself. Rowe (1982) also used the term pseudo-ankylosis to describe limited joint movement resulting from the indirect influence on joint mobility caused by depression of the zygomatic arch and subsequent fibrous adhesions; a similar situation arises where there is post-traumatic shortening of the ramus of the mandible, relatively greater length of the coronoid process and shortening of the temporalis muscle.

Pathogenesis and nature of deformity

Post-traumatic ankylosis of the TMJ may develop in childhood or in adult life (p. 286). The long-term consequences of facial skeletal trauma are nowhere more dramatic than the effects produced by fractures of the mandibular condyles in children, which may result in ankylosis and mandibular growth restriction (Fig. 21.58), often causing secondary deformity elsewhere in the face. In cases of traumatic ankylosis of the TMJ, secondary deformities of the upper jaw are admittedly not so common or severe as in hemifacial microsomia, where absence

or hypoplasia of the condyle is often associated with severe secondary deformities of the upper jaw. But in the few cases suffering from very early onset bilateral ankylosis, there is some deformity of the upper jaw also.

The pathogenesis is often controversial, and indeed there are many causes of reduced mobility of the mandible. Some cases of true ankylosis certainly result in early childhood from trauma to the developing mandibular condyle, perhaps complicated by secondary infection, and there is often an associated failure of mandibular growth (P. 504)

Intracapsular crushing and haemarthrosis in an area that is highly osteogenic and capable of being a focus of infection may produce a nasty combination of bony ankylosis and deformity. Not only does complete bilateral fusion of the bones lead to difficulty in opening the mouth but there is gross malocclusion and in extreme forms the 'bird face' deformity shown in Fig. 21.58.

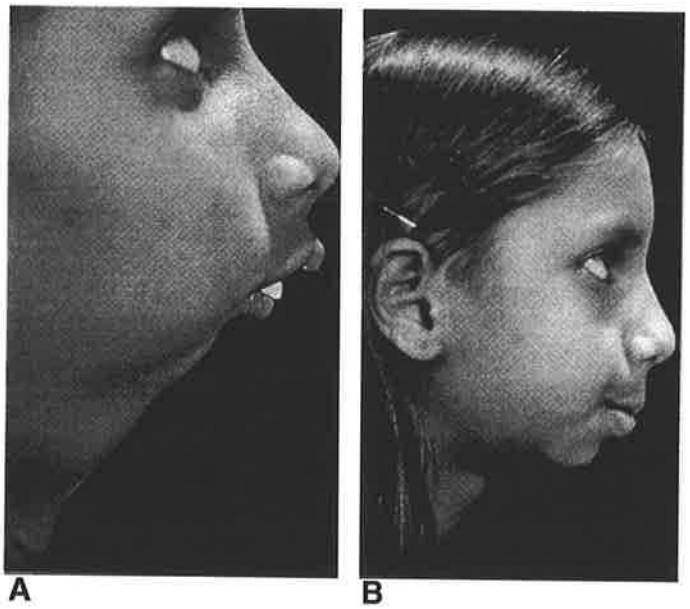


FIG.21.58. ***Temporomandibular joint ankylosis.*** *A 10-year-old child sustained mandibular trauma in infancy, with infection of the temporomandibular joints, giving rise to the dramatic 'bird face' deformity with severe retrognathia and trismus. This child was fed by forcing food behind the posterior dentition with a finger—first by the parents and later by the patient.* ***A*** *Initial presentation.* ***B*** *After reconstruction of the temporomandibular joints with costochondral grafts, inverted 'L' osteotomy, interpositional bone graft and genioplasty as a one-stage operation.*

The effects on airway and nutrition are significant as are the psycho-social problems. Less severe TMJ ankylosis can also occur in children. When seen late there may be some destruction of the mandibular condyles, trismus and a mild malocclusion, with varying degrees of growth disorder.

The relation between condylar damage and mandibular growth is still obscure (p. 80). There are cases in which, despite considerable condylar destruction, mandibular growth is almost normal. Perhaps the reason that such patients suffer minimal growth abnormalities following severe condylar injury is because ankylosis did not occur. It would therefore seem that the critical treatment objective in condylar fractures is early mobilization to prevent condylar ankylosis and therefore subsequent growth deformity.

The majority (69%) of cases reported by Lello (1990) were consequences of trauma, and this finding is consistent with most contemporary studies (Posnick & Goldstein 1993). Rowe (1982) supports the view that trauma has succeeded

infection as the principle cause of ankylosis in children. The cases that we have seen of this gross deformity have come from developing countries and we have formed the view that lack of health services resulted in failure to treat these children for an initial traumatic event.

Older authors have given more weight to the causative role of inflammation of one type or another (Topazian 1964). Norman (1978) suggested that the differential diagnosis of mandibular hypomobility may include pseudoankylosis (under this heading he includes fibrosis secondary to burns, radiation treatment, and infection), as well as union of an enlarged coronoid to the maxilla or zygomatic arch and a depressed fracture of the zygomaticomaxillary complex. Other less convincing causes include 'trismus' secondary to an elongated styloid process and an ossified sphenomandibular ligament.

Re-ankylosis after TMJ arthroplasty is held to be a postoperative consequence of the proximity of raw, uncovered bone surfaces as well as insufficiency or absence of immediate postoperative mandibular movement to disrupt the formation of bone bridges and maturing fibrous tissue.

In adults, TMJ ankylosis is usually due to intracapsular comminuted fractures. These may be associated with extra-articular fibrous ankylosis (Kazanjian's false ankylosis). Uncorrected fracture dislocations with subsequent fibrosis may also produce reduced mandibular mobility. Very severe crush injuries to the area can result in a fibro-osseous response of massive proportions with a block of bone extending from the ramus and coronoid process to the zygomatic arch and base of skull. Fortunately cases of such complete ankylosis in adult life are rare.

Finally, an unfortunate cause of post-traumatic iatrogenic ankylosis results from enthusiastic exploration of the joint for TMJ dysfunction, with a resulting increase in fibrosis.

Clinical assessment

The age of the patient at the time of the injury will determine the clinical symptoms and signs. In severe bilateral ankylosis, no movement of the jaws is possible: the victim may be undernourished and may suffer from chronic airway obstruction. In younger patients there may be characteristic secondary deformities. Symmetrical retrognathia may occur with bilateral joint involvement; with unilateral ankylosis, there is a variable degree of retrognathia and deviation of the jaw to the side of the ankylosis. There is compensatory adjustment of the occlusion and compensatory maxillary growth deformity. The birdlike facies with grossly retruded mandible, the small featureless chin and the antigonial notching of the mandible are characteristic. There is a Class II malocclusion. Dentoalveolar growth attempts to compensate for the lack of jaw bone growth and the mandibular teeth are proclined. In unilateral ankylosis in younger patients there is a marked degree of facial asymmetry, the ramus and body of the mandible being chiefly affected. The chin point is distorted in shape due to the asymmetrical muscular pull on it and is deviated to the affected side. However, the dental occlusion may show good interdigitation of teeth due to adjustment at the dento-alveolar level. Nevertheless, dental hygiene may be impaired, with all the consequences that this brings: caries, periodontal disease and dental abscesses have been recorded as the usual concomitants of complete TMJ ankylosis (Georgiade 1977). In cases of fibrous ankylosis, the signs are less obvious. The main symptom is reduced mobility and the growth deformity is not gross. In the adult the symptoms of lack of movement, possibly pain, and malocclusion are usual in both intra-articular and extra-articular ankylosis; the history and examination will help to make this important diagnosis.

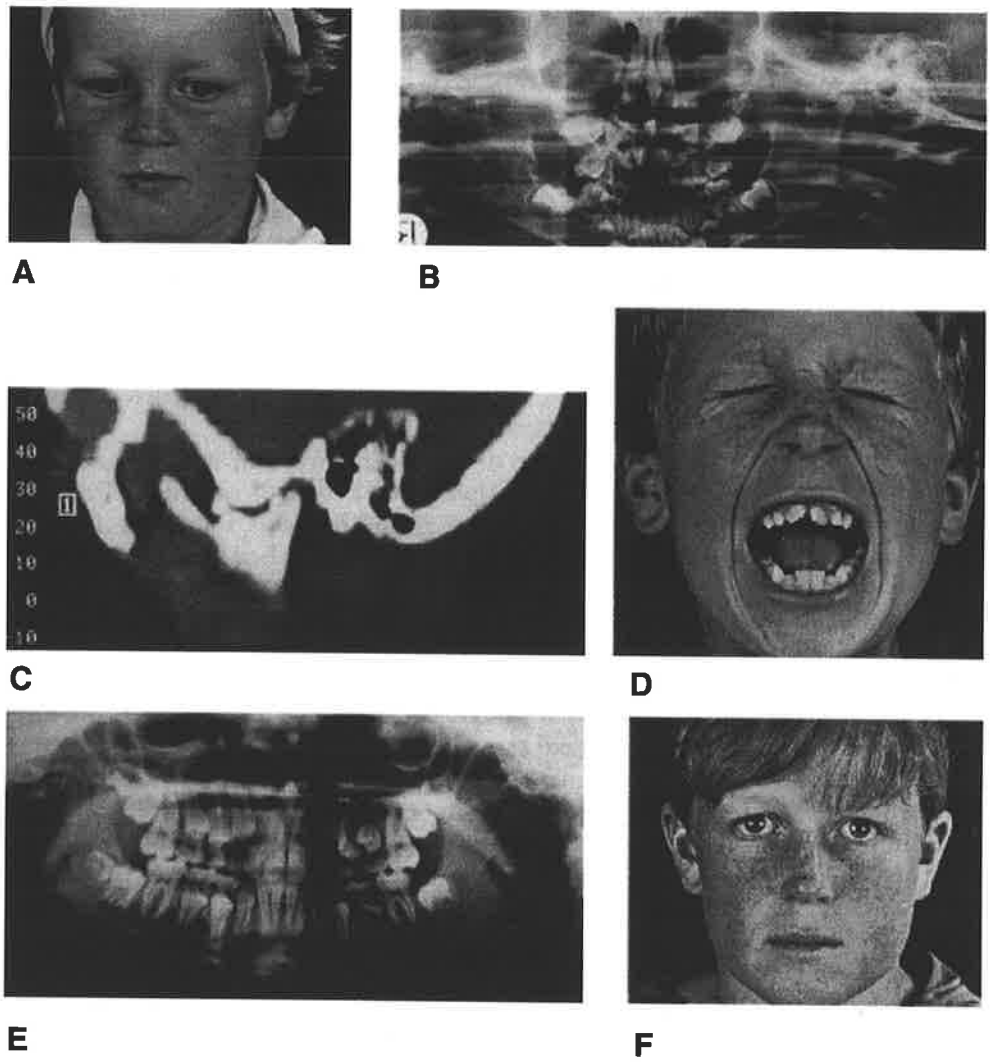


FIG. 21.59. *Temporomandibular joint ankylosis.* *A 3-year-old boy was run over by a tractor, suffering bilateral condylar fractures. These healed with a grossly shortened and flattened morphology. He was treated conservatively for 4 years but by then demonstrated restricted mandibular opening and X-ray evidence of bony ankylosis. He had problems when eating and cleaning his teeth. Removal of the bony ankylosis and interposition of perichondrium over the smoothed condyle restored the ability to open the mouth much wider. His facial growth has developed relatively normally in spite of the distorted temporomandibular joints.* **A** *Initial presentation.* **B** *Orthopantomogram (OPG) taken at the time of presentation, several years after the original accident.* **C** *CT scan showing the bony ankylosis.* **D** *Opening (with effort) after surgery.* **E** *OPG taken at 12 years of age with distorted temporomandibular joints.* **F** *Good facial growth has been maintained.*

Radiological assessment

Radiological investigations include plain X-ray views, orthopantomogram and CT scanning to demonstrate the nature and extent of the ankylosis and the presence of extra-articular components such as coronoid zygomatic bone fusion. The condylar fusion may extend medially, forming a bone block several centimetres wide, which may obliterate the sigmoid notch; it is therefore important to have 3D views of the region before any surgical venture, and modern applications of CT scanning are most helpful here. Magnetic resonance imaging (MRI) is useful in delineating the intra-articular soft-tissue pathology (p. 189).

Management

Principles of conservative and operative treatment

Treatment is indicated where there is a significant problem with function or form. In bilateral ankylosis in childhood, there are likely to be serious difficulties in feeding, airway obstruction and impaired dental hygiene, in addition to the deformed appearance resulting from cessation of growth. As has been noted above, the nature and degree of the secondary facial deformity are related to the time of onset of the ankylosis, its duration throughout the patient's growth phase and whether the lesion is unilateral or bilateral.

In less severe cases of fibrous ankylosis in an adult or older child, the indication for surgical or non-surgical treatment will depend on the outcome of a preliminary trial of exercise treatment. In some cases, progressive mobilization by conservative treatment may be successful: the gap is gradually widened with increasing numbers of wooden spatulas, or with an acrylic screw in the shape of a carrot (Fig. 21.60). In younger patients with fibrous ankylosis causing restricted movement but with no gross problem with growth, there is also a place for conservative management: this involves monitoring growth, physiotherapy and patience. However, as Munro et al (1986) have pointed out, the response to conservative treatment may be deceptive: even with complete ankylosis there is often sufficient elasticity in the jaw to allow some movement, and attempts to manage complete ankylosis by exercises, passive or active, are in vain.

Surgical intervention should not be undertaken lightly because the intervention itself may contribute to ankylosis. The aim of surgical treatment is to re-establish movement of the joint, to maintain movement, to facilitate growth and to reduce deformity, especially in the child since impaired mandibular growth may produce a cascade of secondary deformities in the upper jaw. In the adult a distinction must be made between ankylosis that has developed in childhood when there are secondary deformities of the chin, maxilla and dentition and

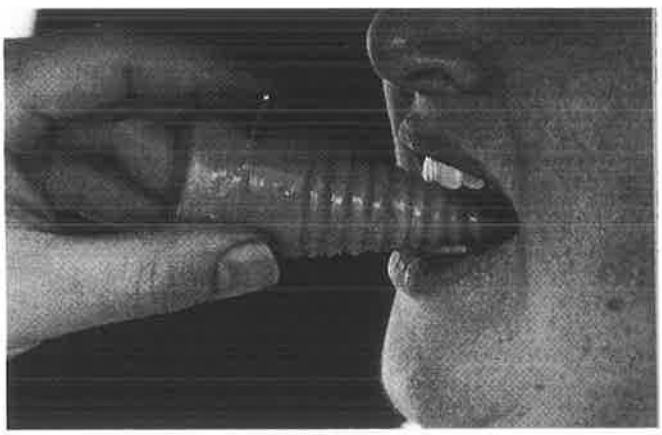


FIG. 21.60. ***Acrylic 'carrot'*** *used to assist with progressive mouth opening.*

ankylosis of recent origin when release of the joint will produce an acceptable occlusion.

The surgical challenge is not only to reconstruct the joint, but also to maintain function and preserve the reconstruction from postoperative re-ankylosis. The difficulty in producing satisfactory results from reconstruction of TMJ ankylosis is reflected in the many and varied procedures described to achieve these ends. Autogenous grafts have included metatarsals, calvarial bone, pieces of iliac crest, and costochondral grafts. Alloplastic substances used include metal (Vitallium) or acrylic prostheses, and sculpted Silastic® and Proplast® blocks, inserted to maintain joint mobility and mandibular height. MacAfee & Quinn (1992) used a polyoxomethylene condylar head fixed to a pure titanium mesh. The copious literature relating to alloplastic implants in the TMJ joint is reviewed by Kent & Misiek (1991) and there is a disappointingly high incidence of complications. Gallagher & Wolford (1982) reported on the better long-term stability of Proplast® when compared with Silastic®. However, Smith et al (1993) have confirmed what has become a common experience that Teflon®— Proplast® implants may generate an osteoclastic giant cell reaction that can erode bone even as far as the middle cranial fossa. The propensity for alloplastic materials to develop late problems including displacement, erosion of bone, and failure of the prosthetic joint as well as the suspected local and systemic responses to silicone, make autogenous costochondral joint replacement our procedure of choice (Kent et al 1983, Dolwick & Aufdemorte 1985).

Anaesthesia

In those cases where there is some movement of the jaw it may be possible to intubate orally. If it proves possible to ventilate the patient with a face mask then paralysis may be achieved with suxamethonium prior to incubation. A range of stylets, bougies and laryngoscopes should be at hand. The wide but flat Seward blade has been most useful.

If the teeth are firmly opposed to one another then the nasal route is used. The nose should be cocainized (10% plain cocaine paste) allowing plenty of time for good anaesthesia (20 min). Then 4% lignocaine (3 ml) is injected into the trachea through the cricotracheal membrane. The patient is encouraged to cough after the injection. Sedation with a narcotic (fentanyl) and a benzodiazepine (midazolam) is helpful. Anaesthesia is then induced with oxygen and a volatile agent. A fibrescope with an armoured tube passed over the scope is introduced through the nose and into the larynx and trachea. The tube is then slipped down over the scope and into the trachea.

Anaesthesia may then be formally induced. Blind nasal intubation is an alternative option (Edwards 1993).

While in general we do not advocate hypotensive techniques, it is desirable to suppress the reflex tachycardia and hypertension sometimes induced by dissection in the TMJ area. Occasionally the surgeon will be impeded by oozing from tissues around the joint capsule and modest hypotension may be induced for this portion of the operation.

If the facial nerve is at risk during the dissection then muscle relaxants should be omitted so that the nerve and the muscles it supplies are active and can respond to a nerve stimulator.

Exposure for arthroplasty

Surgical approaches to the TMJ are described on p. 285. It is very important to have wide access, and the approach offered by the bicoronal scalp flap, extended into the pre-auricular region and combined with release of the temporal fascia from the zygomatic arch and division of the masseter muscle, makes this the exposure of choice in difficult cases. In less difficult unilateral cases the flap need not be extended quite so far to the other side, though it must go far enough

to make access easy. The insertion of the costochondral graft has been described as best performed via a combination of the pre-auricular and submandibular approaches (Evans et al 1985, Crawley et al 1993); we have used this combined approach when inserting a costochondral graft. However, the extended bicoronal flap may offer sufficient exposure of the condyle and ramus to facilitate the whole operation. Where complex osteotomies are needed together with joint exploration and reconstruction, Crawley et al (1993) use a neck incision below and behind the angle of the mandible as well as the bicoronal approach.

The capsule of the joint is identified and divided. The bony mass enveloping the joint and condylar neck is exposed, together with the base of skull above and the condylar neck and ramus of the mandible and the sigmoid notch below. Subperiosteal dissection is continued down as far as the angle of the mandible and beyond if necessary. Where possible this dissection is extended to secure the posterior aspect of the condylar neck and a curved retractor is placed around the neck to protect the maxillary artery which lies medial to the joint (p. 47). However, this protective manoeuvre may not be possible and one must then proceed with caution in dividing the bone. When the retractor is in place, it is possible to use a large burr to release the bone at the level of the fused joint space which may be partly visible. The bone can be burred away medially leaving a thin shell intact above; lower down toward the condylar neck, the curved retractor protects the soft tissue and the cut can be made more aggressively. Once the bone is removed the ends are smoothed over and the base of skull is also smoothed. The mandible can then be tried for movement. Fibrous contractures may need to be divided or a coronoidectomy performed. To obtain sufficient mobilization, further dissection may be necessary to detach the medial and lateral pterygoid muscles, the temporalis muscle, the stylomandibular and sphenomandibular ligaments and the pterygomandibular raphe.

Interpositional arthroplasty

The aim of the operation is the creation of a gap, and it is in effect an extended condylectomy. There has been a persistent belief that this gap alone is sufficient treatment of the ankylosis. However, experience has shown that to prevent re-ankylosis, some form of interpositional arthroplasty is necessary. This may be with autogenous tissue, such as fascia lata, muscle, bone, cartilage or dermis (Tajima et al 1978), or alloplastic material, such as Proplast®, Teflon®, silicone rubber or metal.

In less severe cases we have inserted a sheet of perichondrium taken from the costal cartilage and sutured over the reshaped condylar process with small burrholes into the bone to aid fixation. Above this a disc of reinforced Silastic® sheet is similarly sutured into the area of the condylar fossa. Lello (1990) reported the successful use of a minimal gap arthroplasty with insertion of a graft of composite free auricular skin and a cartilage graft to prevent re-ankylosis; however, Yih et al (1992) report that auricular cartilage has proved disappointing in the long term. In the more severe cases the bone ends may need to be widely separated: Topazian (1964) recommended a gap at least 1 cm wide, and Rowe (1982) even more separation. Munro et al (1986) have stated that there should be a bone-free gap of at least 3 cm between the glenoid fossa and the ramus if recurring ankylosis is to be avoided. When a wide gap has been produced some form of support for the vertical height is necessary, or there will be deviation of the mandible and/or open bite: the wider the arthroplasty gap the more important it is that the material be of sufficient bulk and substance to support the ramus adequately and to maintain its vertical height.

Temporalis muscle can be inserted between the bone ends. A strip of temporalis muscle ~2–3 cm wide is mobilized up to the superior attachment of the muscle to the temporal bone, where it is detached. The muscle strip and its overlying fascia, now based inferiorly, are further mobilized and passed inferiorly to the zygomatic arch and interposed between the bone ends, where the muscle strip is kept in place by suturing it to tissue at the margins of the glenoid fossa

(Rowe 1982). The edges of the donor defect in the temporalis muscle are hard to approximate but success in doing so will reduce the eventual muscle defect and the associated aesthetic blemish. The results of this procedure are variable. Re-exploration has on occasion shown a mass of fibrous tissue, indicating that the muscle has degenerated: what was an initial good result from the point of view of a space occupier may with time give rise to recurrence of the facial deformity if not loss of joint movement. Certainly, if a considerable amount of mandible has to be removed, muscle interposition will not maintain the mandibular height (Fig. 21.61).

To obviate this, insertion of a bone graft or alloplastic material may be used. In children, a costochondral graft has theoretical advantages (Munro et al 1986, Crawley et al 1993). Not only is the height preserved, but in the growing individual one can hope that the grafted bone and cartilage will provide a new growing unit within the existing functional matrix (p. 77; Murray et al 1984). This thesis has been postulated for cases of hemifacial microsomia and the results are as uncertain in those cases as in post-traumatic ankylosis.

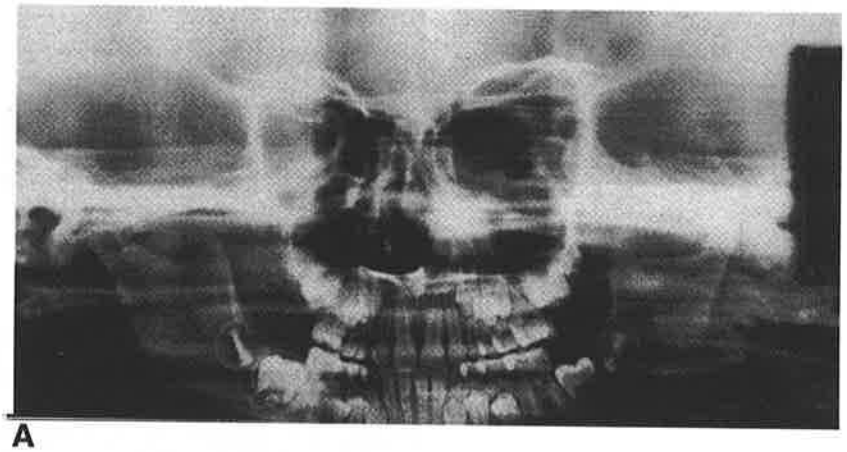
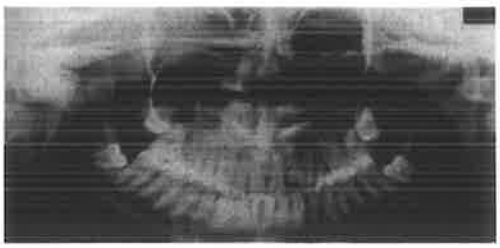


FIG. 21.61. ***Temporomandibular joint ankylosis.*** *This patient suffered an intracapsular disruption of his temporomandibular joint in early childhood which, in spite of early mobilisation, developed ankylosis and marked overgrowth of bone. A continuous mass of bone fused the condyle, coronoid process, zygomatic arch, and base of skull. A Disruption of the joint as the ankylosis is developing.*

Orthodontic management

When this procedure, whether unilateral or bilateral, is proposed in the growing child, it must be planned with a view to future orthodontic control and manipulation of facial growth. After creation of the gap and mobilization of the mandible, the jaw will be positioned by an acrylic occlusal wafer and wherever possible this is made before operation. Sometimes it has not been possible to make the wafer before operation, because of lack of jaw opening; impressions of the occlusion cannot be taken if the mouth will not admit impression trays. The appropriate positioning must be then determined at the time of operation and the device made postoperatively (Fig. 21.62).

The wafer serves as a splint and often has to be supplemented by vertical elastic bands. Oral hygiene is often a problem when this appliance is installed; the parents should remove it daily and replace it after cleaning the teeth and also the surface of the appliance as any residual food may cause enamel decalcification. The orthodontist can instruct the patient on how to remove the appliance from the mouth; the parents are given the necessary instruments for dental hygiene. The wire ligatures securing the acrylic wafer to the maxillary teeth must be readily accessible so that the patient and parents can carry out the



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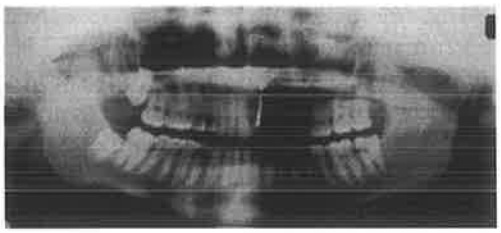

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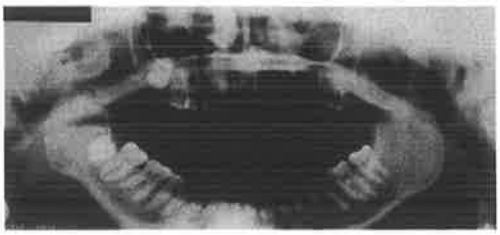

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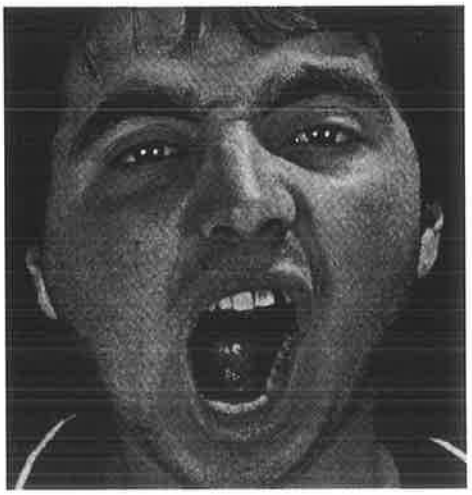


G

FIG. 21.61. ***Temporomandibular joint ankylosis.*** *This patient suffered an intracapsular disruption of his temporomandibular joint in early childhood which, in spite of early mobilisation, developed ankylosis and marked overgrowth of bone. A continuous mass of bone fused the condyle, coronoid process, zygomatic arch, and base of skull.* ***B*** *Further development of the ankylosis.* ***C*** *Block of bone encountered in the joint.* ***D*** *Appearance after resection and interpositional arthroplasty using temporalis muscle.* ***E*** *Full facial view, 1 year later.* ***F*** *Lateral view, 1 year later.* ***G*** *Wide opening with minimal jaw deformity; however, there is some deviation of the mandible to the right.*

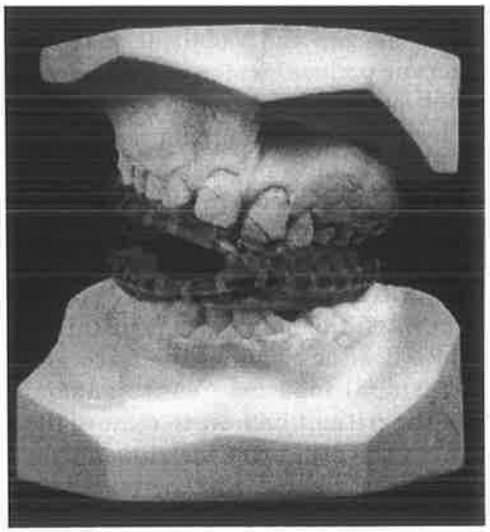


FIG. 21.62. ***Appliance designed to facilitate the gradual eruption of the maxillary teeth.*** *The acrylic is shaved progressively as eruption occurs.*

necessary hygiene. The wafer should be worn for at least 6 months after operation, and the longer it is worn the better: 18 months is the ideal.

Arthroplasty with costochondral graft

The contralateral chest is used for harvesting the sixth or seventh rib. More than 1 cm of costal cartilage needs to be attached to the rib, care being taken to preserve the periosteum and the perichondrium at the junction. The cartilaginous end is shaped nicely and seated into the glenoid fossa using a strong nylon stitch passed through a burrhole in the bone of the costochondral graft which is then sutured into the tissues of the temporal fossa. We have used a free piece of perichondrium from the rib to line the glenoid fossa on a number of occasions, or sometimes a small Silastic® disk, but because easy displacement has been noted, we have ceased to use this technique. Crawley et al (1993) use a vascularized segment of temporoparietal fascia. The ramus of the mandible must be shaped to receive the graft which is secured with three titanium screws, preferably placed in a triangle to give better fixation. Once the seating and fixation of the costochondral graft are achieved, occlusion should be released and checked. In the adult the costochondral graft can also be used, but like all autogenous implants it may undergo resorption, with or without re-ankylosis. This is not common and the costochondral graft has become our preferred implant in the treatment of severe cases. What is more controversial, however, is the combination of this form of reconstruction with other osteotomies of the facial bones at the same operation as advocated by Munro et al (1986). While we have done this to good effect, there is no place for hard and fast rules in this field of reconstruction. The view expressed by Lello (1990) is that when further growth retardation and skeletal deformity have been prevented by successful release of the ankylosis, secondary osteotomies for the correction of facial deformity can be undertaken later, with perhaps more precise planning. This consideration is only relevant in the period of growth; adult cases can be treated in one or two stages at the preference of the treating team (Fig. 21.63).

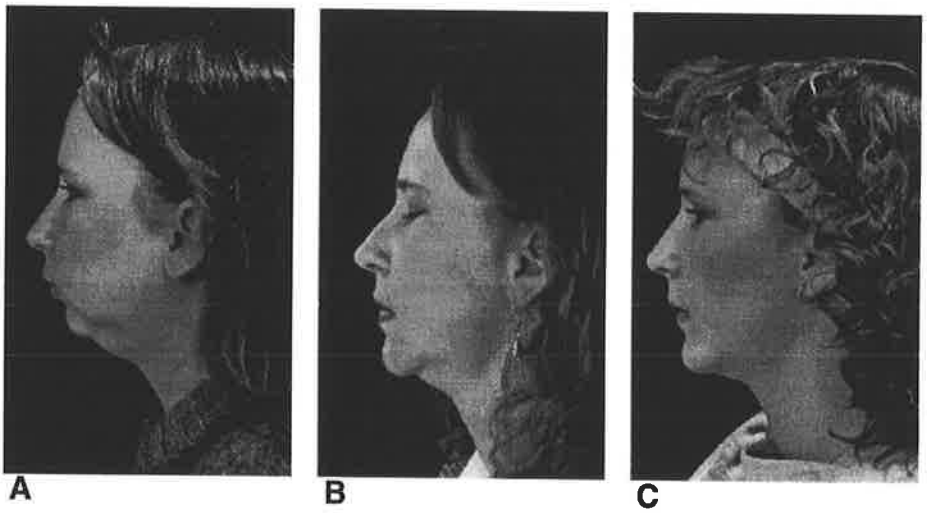


FIG. 21.63. ***Costochondral joint reconstruction. Case 1: ankylosis in early life.*** *A girl presented with mandibular hypoplasia and trismus as a result of a fracture sustained when she was 3 years of age. Multiple surgical interventions had been made to overcome her trismus. She had release of the left ankylosis, costochondral grafting, bilateral sagittal split osteotomies for advancement, and genioplasty. Osteotomies and costochondral grafting were done at the same time, an extensive but quite practical solution to such difficult problems.* ***A*** *Profile seen at presentation with mandibular hypoplasia.* ***B*** *Appearance 1 year after costochondral grafting plus sagittal split advancement and genioplasty.* ***C*** *The profile after 10 years. Slight relapse is noted but the profile is still acceptable.*

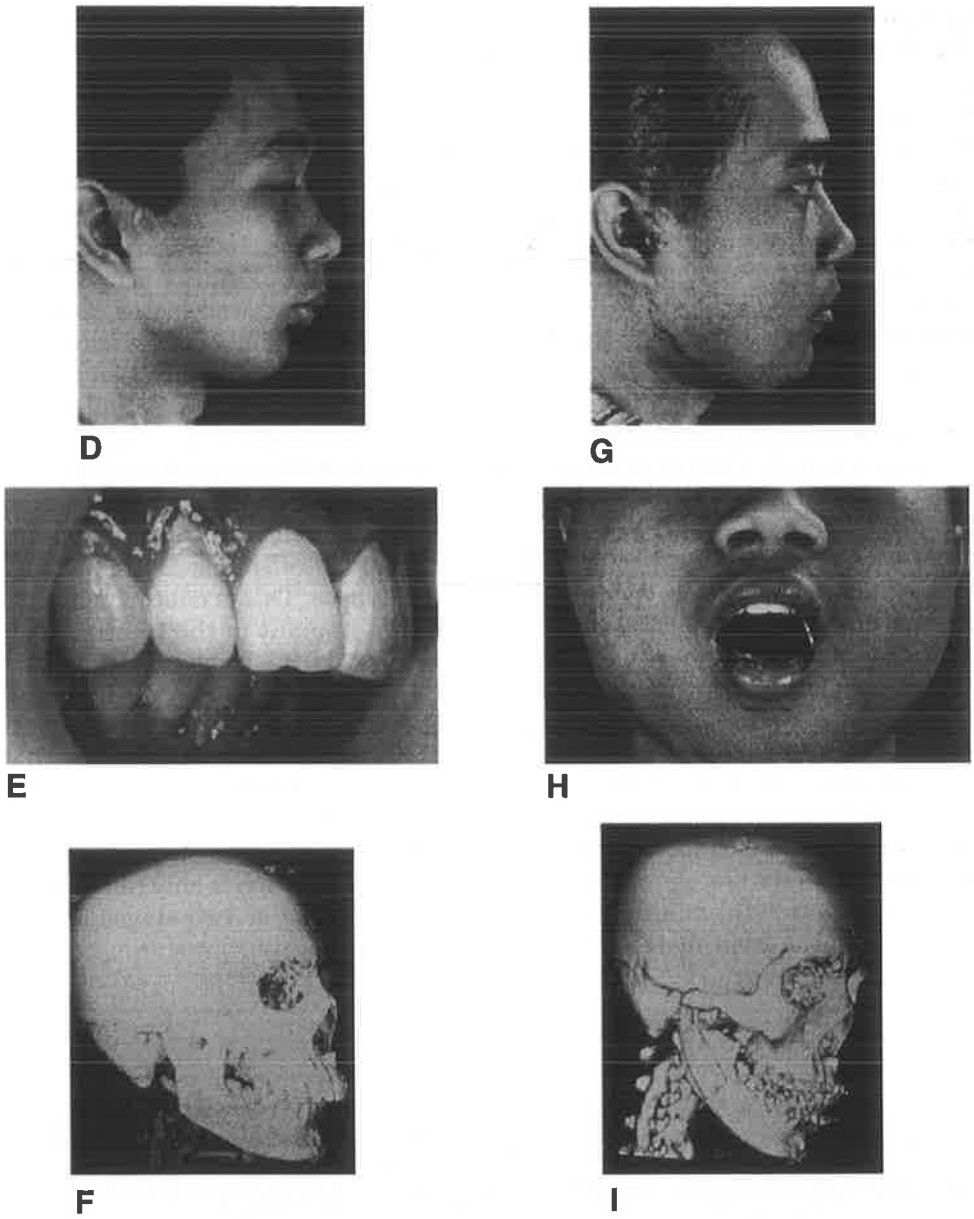


FIG. 21.63. ***Case 2: ankylosis in adult life.*** *A man was injured in a vehicular accident at the age of 21 years, in another country. He suffered an impact on his chin which drove both condyles into the mandibular fossae. He had no initial treatment and the condyles fused with the skull base, producing ankylosis and retrognathia.* ***D*** *The situation at presentation 10 months after injury.* ***E*** *The occlusion with no movement of the lower jaw.* ***F*** *The lateral 3D CT scan showing the foreshortening and fusion of the jaw with the base of skull.* ***G*** *The situation after costochondral graft.* ***H*** *The degree of opening, gently controlled with a very light rubber band.* ***I*** *Postoperative 3D CT scan.*

Postoperative care
After the insertion of a costochondral graft, it is often difficult to decide whether to re-establish the intermaxillary fixation for a short period of time. There is a delicate balance in planning the period of immobilization: immobility may promote adhesions and limit function, while early mobility may impair healing and promote graft resorption and future dysfunction. It was initially our practice to maintain maxillary fixation for at least a week, followed by controlled mobilization. However, as experience has been gained we have increasingly favoured immediate mobilization within the limits of discomfort. Munro et al (1986) advocate the early postoperative use of chewing gum as a form of immediate exercise and this is now our recommendation. The importance of postoperative oral hygiene has been emphasized.

Long-term care
This also demands judgement and resolution. Where the gap has been maintained with a costochondral graft in a young patient, the long-term supportive regimen is often very demanding. The patient is supervised by the team orthodontist and must often use the wedge-shaped occlusal splints mentioned above, which require frequent adjustments to encourage and control the eruption of the maxillary teeth to rectify the occlusal plane. It is an arduous programme for the child, the family and the treating team. Even with the best of techniques there may be collapse and shortening of the reconstructed ramus.

Complications and results
Good results have been achieved in less severe adult cases by release of the ankylosis, minimal reconstruction and the insertion of perichondrium and a thin Silastic disk to maintain the joint space, although the disk may need to be removed at a later date.

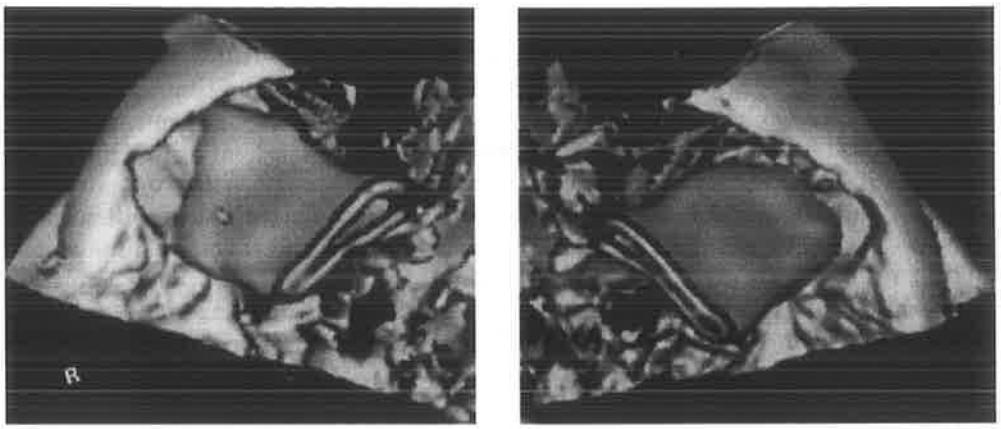
The more severe ankyloses, especially those with onset in childhood, tend to recur and the younger the ankylosis is operated on, the more likely it is to recur. Nevertheless Ohno et al (1981) favour early operation to restore normal growth. These two views are not incompatible. The cases described in Figs 21.58–63 exemplify our management of ankylosis in childhood and the results which can reasonably be expected.

In the adult, we favour the insertion of a costochondral graft as a space-filler after wide release of the ankylosis; however, we have observed changes in the shape of the cartilaginous component and a diminishing of the vertical height in some cases over time. Where pain has become part of the symptom complex, it may or may not be diminished; when it is not, we are faced with the ongoing difficulties of the patient who suffers chronic pain in this region (p. 659) (Fig. 21.64).

Mandibular Deformities

Surgical pathology

Residual deformities of the mandible may be extensive and grotesque, especially after gunshot wounds. Deformity may result from loss of tissue, from non-union or from malunion due to inadequate treatment—or no treatment at all. The consequences in terms of function and form depend on the extent of the malunion and on its site. The mandible supports the tongue through its extrinsic muscles, especially the genioglossus (p. 68); deformity of the mental region may affect speech and swallowing while complete absence of the mandible in this region may affect the airway by allowing the tongue to fall back. The aesthetic consequences of mandibular ablation or deformity can be appalling. For a century, surgeons have been looking for a suitable material to replace the damaged or absent mandible. Metals, acrylic and bone have had their advocates, but autogenous bone is of course the material of choice in replacing mandibular defects.



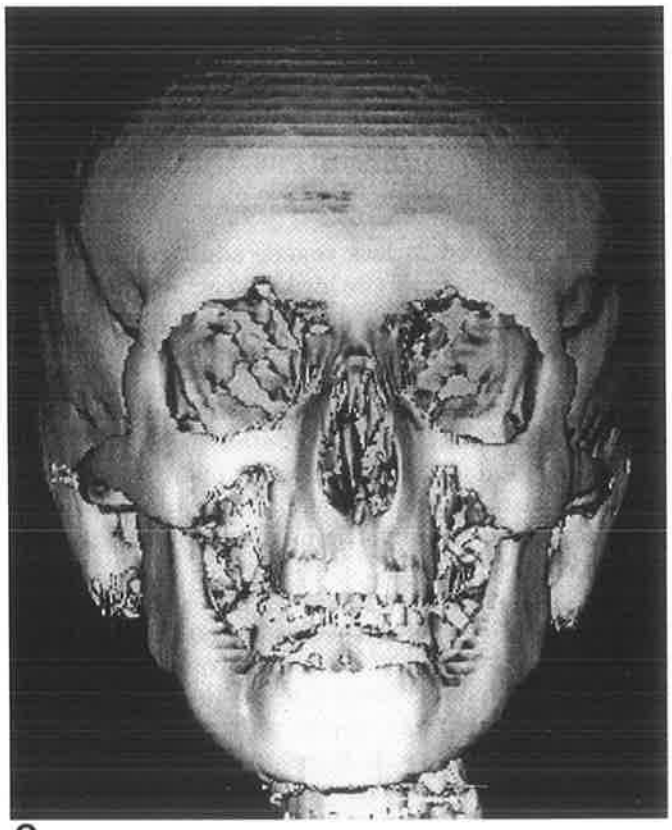


FIG. 21.64. ***Extensive surgical treatment of temporomandibular joints.*** *Three-dimensional reconstructions of the temporomandibular joints after radical excision in a woman who had undergone numerous operations for bilateral temporomandibular joint ankylosis. This is a cautionary tale, as the problem started with supposed minor trauma and pain with some joint clicking, and proceeded to increasingly radical excision of her jaw joints. The last procedure involved replacement with carved Silastic®, which was subsequently extruded. The woman presented complaining of persistent intense pain and further restriction of movement. Under general anaesthesia, however, the jaw opened easily to 4.5 cm.*

A *3D reconstruction of the right joint, seen obliquely from below where the mandible has been resected at the level of the sigmoid notch. The reconstruction has been cut away below for clarity*

B *Similar view of the left side.*

C *Frontal view 3D reconstruction, with the foreshortened mandible recessed beneath the zygomatic arches.*

If malunion occurs during the period of growth there may not only be malposition of the fragments but also deformity of the whole mandible, which is misshapen by altered growth dynamics: there is antigonial notching, deviation to the affected side and a shortened body. The chin may manifest the deformity most clearly and its shape depends on whether the mandibular lesion is unilateral or bilateral (Souyris 1990). Not only is the chin deviated to the affected side, but it is also retruded, with a secondary effect on the lower lip which may be everted. In the bilateral lesions there is typically a retruded misshapen mandible and deformed chin.

Assessment

In cases of malunion and non-union there is malocclusion, loss of chewing efficiency, pain, and perhaps stress on the TMJ. In cases with widely distracted bone ends and fibrous union, there is abnormal mobility. The symptoms relate to the malocclusion and to the malfunction of the jaw, as well as to the aesthetic deformity. On clinical inspection, the defects of the face may be obvious; palpation should confirm the abnormal mobility.

The work-up includes photography, radiology and dental assessment, which includes attention to the condition of the teeth, the taking of impressions, and the production of dental casts for study models used in multidisciplinary planning. In the preparation of a management plan, the opinion of the team prosthodontist will often be decisive.

Management

Principles of treatment
Surgical reconstruction may entail operations carried out on the alveolar process of the mandible, operations to change the shape of the body or ramus, operations to change the position and shape of the chin, and operations combining any or all of these.

Where there is loss of tissue a decision has to be taken concerning bone grafting of the mandible. If the defect is at the angle behind the tooth-bearing part of the bone, some small extent of approximation of the posterior and main fragments is permissible to close the gap. However, in the tooth-bearing part of the bone the fragments must be placed in their correct occlusion with the upper jaw and the defect must be grafted. This situation occurs most typically after gunshot wounds causing massive tissue loss.

Mandibular deformity in edentulous patients
Special consideration must be given to secondary deformity due to malunion in the edentulous mandible. Minimal deformity may be compensated for by the prosthodontist in the manufacture of a prosthesis. More severe malunion may make it impossible for dentures to be worn and the decision to offer surgical correction may depend on the ability to construct an adequate denture. The decision should therefore be made in conjunction with the prosthodontist. Surgical problems occur especially in the elderly where the bone is atrophic: it may be very difficult to treat a deformity which requires refracturing, plating and immobilisation with Gunning splints, all requiring the greatest possible respect for the poor blood supply of the ageing bone (p. 518).

Surgical correction
After the multidisciplinary work-up has been completed, three types of treatment are available:

1. Refracturing and resetting the bone with minimal bone grafting
2. Osteotomies to re-establish a suitable occlusion and/or facial shape
3. Bone grafting defects of the ramus or body of the mandible together with onlay bone grafts to recontour the misshapen bone.

Refracture and reposition
An operative plan is made after preliminary model surgery and subsequent preparation of an acrylic wafer. This wafer determines the correct relationship of the teeth and hence the new position of the jaw. The dental models are available in the operating theatre; from these models, preformed arch bars are manufactured before the operation. X-ray pictures show the position of the fracture and its relationship to the tooth roots. An appropriate surgical approach is made to the region of the fracture; this may be through either an intraoral or an external submandibular incision.

When the fracture site is exposed, the fracture is divided and the method of division depends on how solid the fracture is. If solid it may require division with a power saw; if not, mobilization by gentle insertion of an osteotome is sufficient. Callus and other interposed tissues are removed and the bone ends are freshened. Intermaxillary fixation is then established to place the jaws in predetermined position, the condyles being enlocated in the glenoid fossa. The bone fragments may then be plated. If there is still a small gap, cancellous bone from the hip is placed into the space. The plating technique employed in stabilizing osteotomies of the body and angle is usually monocortical (p. 271), using two plates in the body and one plate at the angle. In most cases, intermaxillary fixation may be removed at the end of the operation. In patients who are less likely to be compliant or who may be lost to medium- and long-term follow-up, bicortical plating system is used with a stronger plate and/or intermaxillary fixation, which is established for 1 month or as long as the patient's tolerance will permit within this time.

Osteotomies
Repositioning the body and ramus of the mandible deformed by trauma and malunion can be achieved by a variety of osteotomies. The workhorse in our unit is the sagittal split osteotomy of Trauner & Obwegeser (1957) and Dal Pont (1961). Corrections in the anteroposterior plane are well tolerated. Correction of asymmetry is less well tolerated as TMJ pain may result from angular and/or rotational stresses on the proximal fragment on the side to which the jaw is being moved. The mandible can also be rotated after a sagittal split osteotomy to correct an anterior open bite. This manoeuvre is said to be limited by the natural tendency for recurrence of the deformity from the pull of muscles opposing the correction. This tendency is recognized but has been diminished by the use of rigid screw fixation of the fragments. Side-to-side movements are well accomplished by the vertical subsigmoid osteotomy, which does not rotate or push the joint-bearing fragments laterally. More extensive lengthening of the ramus and angle region can be accomplished by the inverted 'L' osteotomy with interpositional bone grafting. This is the surgical treatment most often used in patients suffering from the 'bird face' deformity resulting from severe mandibular damage during growth.

Sagittal split osteotomy of the ramus
The technique develops the principle of the sagittal osteotomy which produces overlap of fragments. Trauner & Obwegeser (1957) applied the split to the retromolar region; Dal Pont (1961) extended the area of contact along the lateral cortical plate of the mandible (Fig. 21.65).

Like all intraoral osteotomy procedures on the jaws, the operation is very dependent upon the use of appropriate tools, adequate assistance and good lighting by a head lamp. An adrenaline-containing solution carefully injected under the periosteal plane on both sides of the mandibular ramus is very helpful in controlling bleeding and in defining the plane of dissection. The incision is small, ~3 cm in length, and runs a little lateral to the anterior line of the ramus. It extends downward over the buccal surface of the body of the mandible to the region of the second molar tooth; care is taken to keep the incision sufficiently far lateral to leave enough soft tissue for easy closure at the end of the operation. The cut is made down to the bone. The lateral aspect of the mandible is then

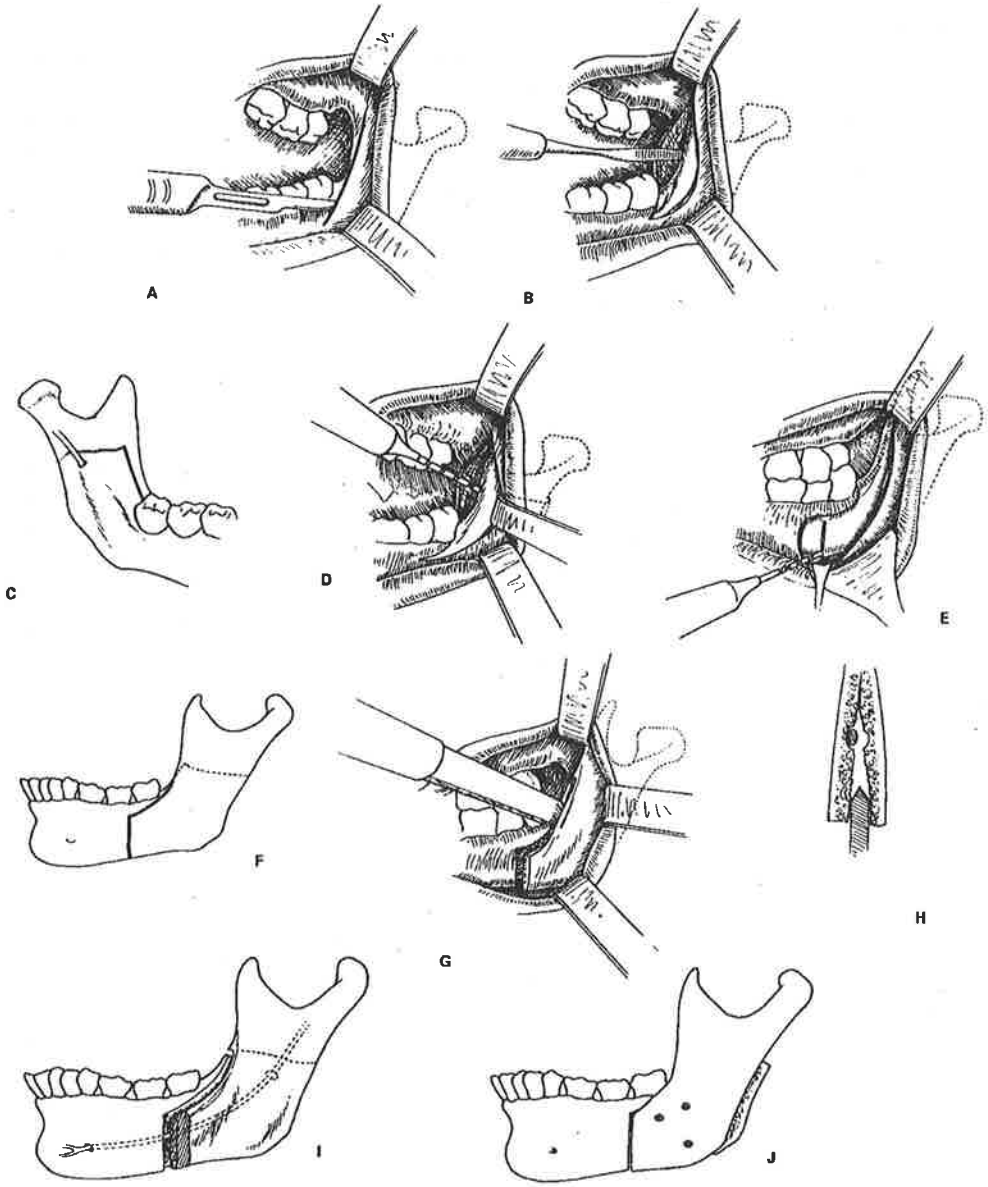


FIG. 21.65. ***Sagittal split osteotomy technique.*** ***A*** *Intraoral incision just lateral to the anterior border of the mandibular ramus, extended to the bone over the external oblique ridge.* ***B*** *Elevation of the flaps medially and laterally.* ***C*** *Schematic view of the osteotomy as if seen from the medial aspect.* ***D*** *Medial cut being made with a side-cutting burr. The soft tissues are protected by the channel retractor.* ***E*** *Vertical component of the lateral cut.* ***F*** *Schematic representation of the line of the split.* ***G*** *Cleaving or splitting of the fragments.* ***H*** *Separation of the lateral fragment (proximal segment) away from the neurovascular bundle.* ***I*** *Osteotomy segments in new position.* ***J*** *Positioning of the screws for rigid fixation.*

dissected in the subperiosteal plane. The masseter muscle is lifted away to allow the introduction of a Munro-Dautrey retractor, the lip of which fits around the ramus, angle or body of the mandible depending on the area being operated upon. The posterior attachment of the muscle sling must be dissected sufficiently to allow this protective metal retractor to be fully located. However, the surgeon must be mindful not to detach more muscle than is necessary to achieve this as every bit of blood supply to the bony fragments is helpful. The medial dissection is carried out above the level of the lingula; a channel retractor protects the neurovascular bundles.

The instruments and techniques for performing the osteotomy vary. The horizontal cut is made just above the lingula through the medial cortex: we prefer a Lindemann burr on a Hall drill to facilitate this, but some operators use a sagittal saw. The lower the cut the thicker the bone but the more risk to the neurovascular bundle, so good judgement has to be used in siting this horizontal cut. As originally described the cut extends to the posterior border of the mandible; Hunsuck (1968) made the cut to just beyond the mandibular foramen before the bone starts to thin off towards the posterior border, thus producing a more robust lateral fragment. The cut is extended down the anterior border of the mandible, medial to the oblique line. Classically, this is done by a series of burrholes which are then connected. With more experience the operator can dispense with burrholes and make the cut with a series of stroking movements of a sharp side-cutting burr. The lateral cortical cut is made vertically in the region of the second molar tooth. The lower border can and must be effectively cut through and care must be taken to 'pop' through the outer cortex in the region of the neurovascular bundle and the tooth roots. Throughout the procedure, the retractor is moved by the assistant to protect the soft tissues. The original splitting technique used a thick wedge osteotome inserted into the osteotomy and with a twisting movement split the two fragments asunder; insertion of the osteotome was done with the protection of a 'ring of steel' supplied by the retractors. Dautrey (1974) introduced an elegant osteotome designed to cleave the cortices apart with the curve towards the outer cortex. With this technique, the inferior dental nerve is safer from brutal surgery, but the outer cortex may be breached if it is fragile and great care must be used to position the protecting retractors to prevent damage to the adjacent soft tissues. Unless the protection of the soft tissue is meticulous, the use of this sharp osteotome in a cleaving technique makes it possible to damage the facial nerve by plunging the osteotome into the substance of the cheek (Jones & Van Sickels 1991). After the split has been achieved the fragments are gently separated and the neurovascular bundle is preserved and if necessary teased out of any attachments to the lateral fragment.

When the fragments have been freed on both sides, the tooth-bearing components are fixed into position with the maxilla using the preformed acrylic wafer. For fixation, it is our custom to use orthodontic bands to which are attached fine arch bars; this appliance is usually fixed earlier, thus saving time at operation. The wafer is tied to the maxilla with fine wires with the twists above the acrylic wafer for easy identification. The intermaxillary fixation is achieved with further wiring or tightly twisted rubber bands. The proximal fragment is then grasped with a long artery forcep and the condylar head properly located in the fossa. It is very important to do this lest there be movement and relapse postoperatively. Some surgeons have devised special techniques to ensure exact placing. A miniplate may be attached to the proximal fragment of the mandible prior to the osteotomy and the other end attached to an identifiable point on the maxilla, usually a slot in the maxillary appliance. The miniplate is then removed and replaced in the same situation after the osteotomy has been made; the jaw is thus repositioned to ensure that the proximal fragment is in its original position and well seated into the condylar fossa. We have not had the necessity for this or any other similar device. With the proximal fragment held in position and the teeth in occlusion the bone fragments are secured by percutaneous insertion of three screws, two above and one below the neurovascular bundle. To achieve this the operator needs good lighting, good assistance and the appropriate use of

retractors. The intermaxillary fixation is released to check the occlusion and to check that the mandible will easily come into the new position. It is a matter of judgement of the severity of the deformity as to whether the patient is left in intermaxillary fixation at this stage or not. The facial stab wounds used for percutaneous screw fixation of the mandibular fragments are closed with 6/0 nylon and the intraoral wounds with 3/0 chromic cat gut sutures. We do not dress these wounds. It is a common custom to use dexamethasone postoperatively to reduce swelling; this is not our practice. Ice-packs are used by some for the same purpose. The jaws may need to be supported with interdental rubber bands over the next few weeks and this need is monitored very carefully by the surgeon and orthodontist (Fig. 21.66).

Vertical subsigmoid ramus osteotomy (Fig. 21.67)
This osteotomy was originally used for the correction of mandibular prognathism (Caldwell & Letterman 1954) and has undergone a number of modifications. In post-traumatic cases we have used the subsigmoid ramus osteotomy when there is a tilting of the occlusal plane and where there is a necessity for significant lateral movement of the mandibular segment. Using this the condylar fragment can remain enlocated, unangulated and unrotated in the new position of the mandible.

An *intraoral approach* has become popular. The obvious advantage is the absence of an external scar, but others include simple and rapid dissection and virtual elimination of risk to the facial nerve. A 3 cm incision is made over the external oblique ridge of the mandible parallel with and just inferior to the mucogingival junction. This placement avoids formation of a scar in the buccal sulcus, which often creates an annoying 'food trap'. Blunt dissection reflects the periosteum from the entirety of the lateral surface of the ramus, which is fully

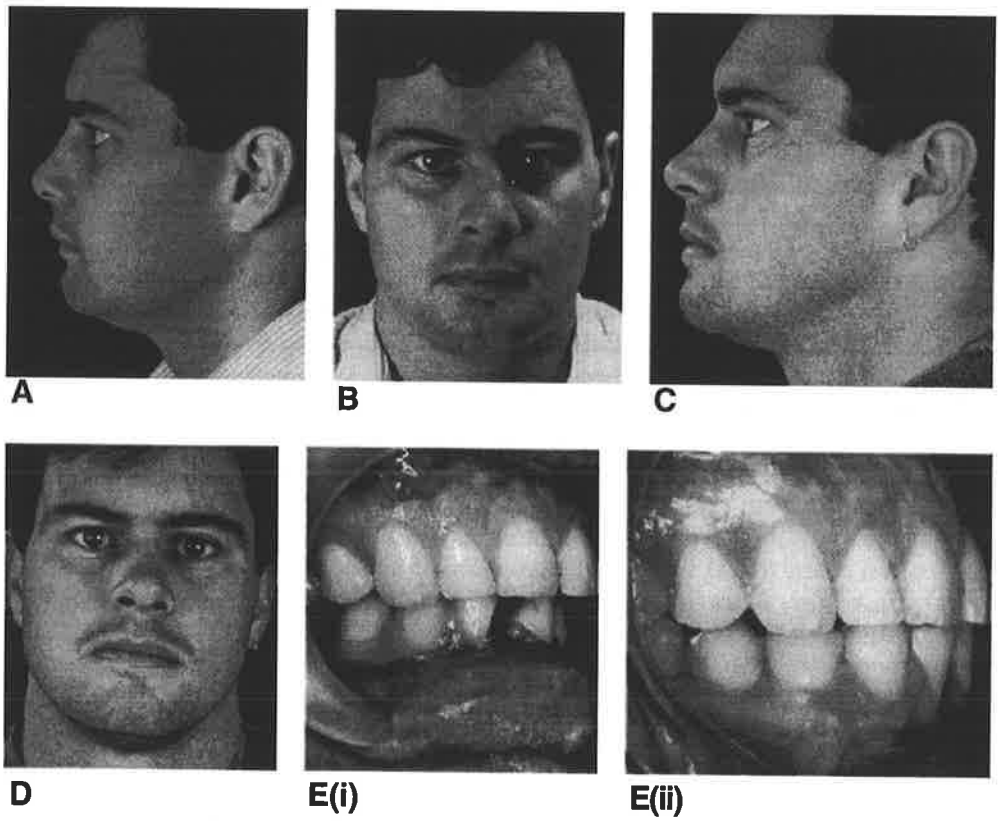


FIG. 21.66. ***Post-traumatic retrognathia.*** *A young man had a malposition of his mandible. He had subsequent advancement of 11 mm on one side and 7 mm on the other to centralise his facial skeleton and reconstitute the pre-injury facial appearance.* ***A*** *Retruded profile.* ***B*** *Facial asymmetry, including the nose.* ***C*** *Postoperative profile.* ***D*** *Facial symmetry improved in the final result.* ***E*** *(i, ii) Occlusion before and after surgery and orthodontic treatment.*

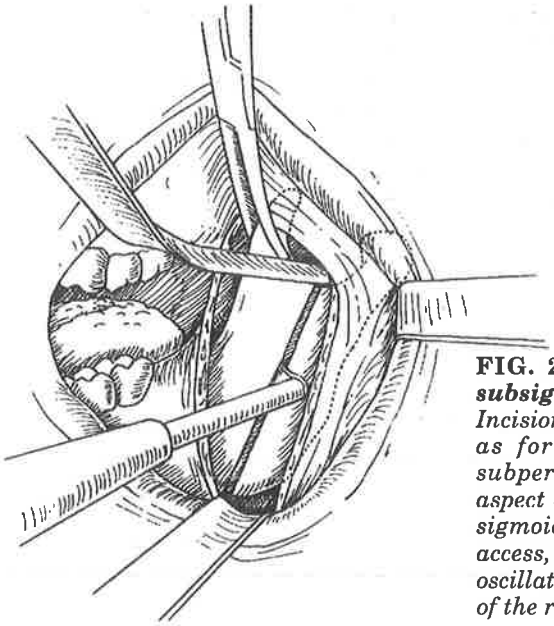


FIG. 21.67. ***Intraoral approach to the subsigmoid osteotomy*** *(after Bell et al 1992). Incision is made over the external oblique ridge as for the sagittal split osteotomy, and subperiosteal dissection exposes the lateral aspect of the ramus. Retractors secured at the sigmoid and antegonial notches allow broad access, and the osteotomy is performed with an oscillating saw 6–7 mm from the posterior border of the ramus.*

visualized with Bauer retractors placed in the sigmoid and antegonial notches. To afford the greatest protection to the inferior alveolar nerve, the initial osteotomy is made with an oscillating saw 6–7 mm anterior from the posterior border of the ramus, and therefore just posterior to the antilingula if one is discernible. From this starting point the osteotomy is carried superiorly to end at the sigmoid notch and inferiorly to end just forward of the gonial angle. Various authors have advocated trimming or grooving of the proximal segment to improve positioning during the healing phase. Although there has been a trend toward shorter periods of intermaxillary fixation, several weeks at least are necessary to prevent postoperative malocclusion.

Because rigid fixation and minimal use of intermaxillary fixation have become priorities in our unit, and a satisfactory technique for achieving this from the intraoral approach has yet to be devised, we have favoured the *external approach.* The incision is 2–3 cm in length and 1 cm below the angle of the mandible. The platysma is incised at a lower level than the skin and lifted superiorly with care to avoid the mandibular branch of the facial nerve (Fig. 21.68). The lower border of the mandible is approached by blunt dissection and the muscles and periosteum are elevated from the body up to the sigmoid notch on both lateral and medial surfaces of the mandible, care being taken to dissect posterior to the lingula. A malleable retractor placed on the deep surface and an angulated retractor with a reverse lip fitting into the sigmoid notch give together good protection and exposure. The osteotomy is made with a reciprocating saw. The cut extends from the depths of the sigmoid notch to a point in front of the angle of the mandible. When the osteotomies have been completed on both sides the position of the distal segment is determined by intermaxillary fixation with an acrylic wafer. Care is taken to have the posterior segments relatively unangulated and the condyles correctly placed in the glenoid fossa. The bone fragments can then be rigidly fixed with plates and/or screws. It has been said that rigid fixation of these fragments is not possible (McCarthy et al 1990b) but this has not been our experience. The wounds are closed in layers without drainage. The jaw is supported with intermaxillary fixation for a short period of time postoperatively; this may be achieved with rubber bands (Fig. 21.69).

Inverted L osteotomy of the ramus

This osteotomy is applicable in cases of severe mandibular hypoplasia resulting from trauma during growth (Fig. 21.70). Where significant forward projection has to be achieved and where the TMJ joint is functional or has been made to

function, the L-osteotomy of the ramus is performed at the point above the entry of the neurovascular bundle. The TMJ, the coronoid process, and the posterior part of the ramus are left in position, and the body and angle together with the nerve and blood supply are advanced and rotated. The intervening gap may then be bone-grafted. This approach leaves the opportunity for variation in the rotation of the tooth-bearing fragment and hence in the shape of the bone graft used to fill the gap. The bone graft is fashioned in such a way as to augment the shape of the mandible in the region of the angle. Wassmund described such an osteotomy in 1927 and Rushton (1942) also reported the osteotomy combined with bone grafting.

The ramus is exposed through the submandibular neck incision described above. The masseter and medial pterygoid muscles are elevated and the muscle sling is widely released. A horizontal osteotomy is placed above the entry of the neurovascular bundle and continued vertically to the lower border. After wide soft-tissue dissection, which is necessary to advance the mandible, the jaws are

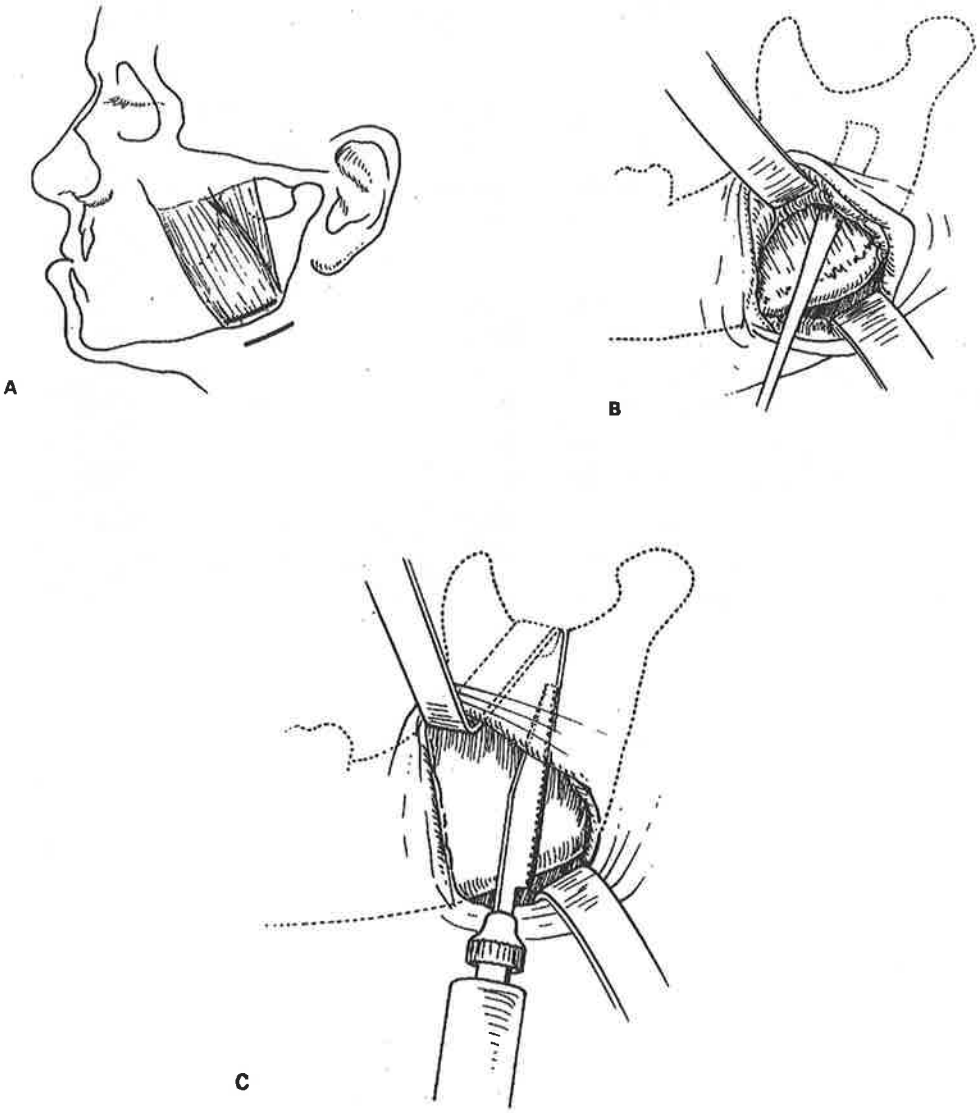


FIG. 21.68. ***External approach to the subsigmoid osteotomy.*** ***A*** *Incision is made below the angle of the mandible specifically to avoid the mandibular branch of the facial nerve. The masseter is divided from the inferior border.* ***B*** *Subperiosteal dissection on both sides of the mandible posterior to the entry of the neurovascular bundle.* ***C*** *Adequate protection with retractors allows the osteotomy to be safely performed with a reciprocating saw.*

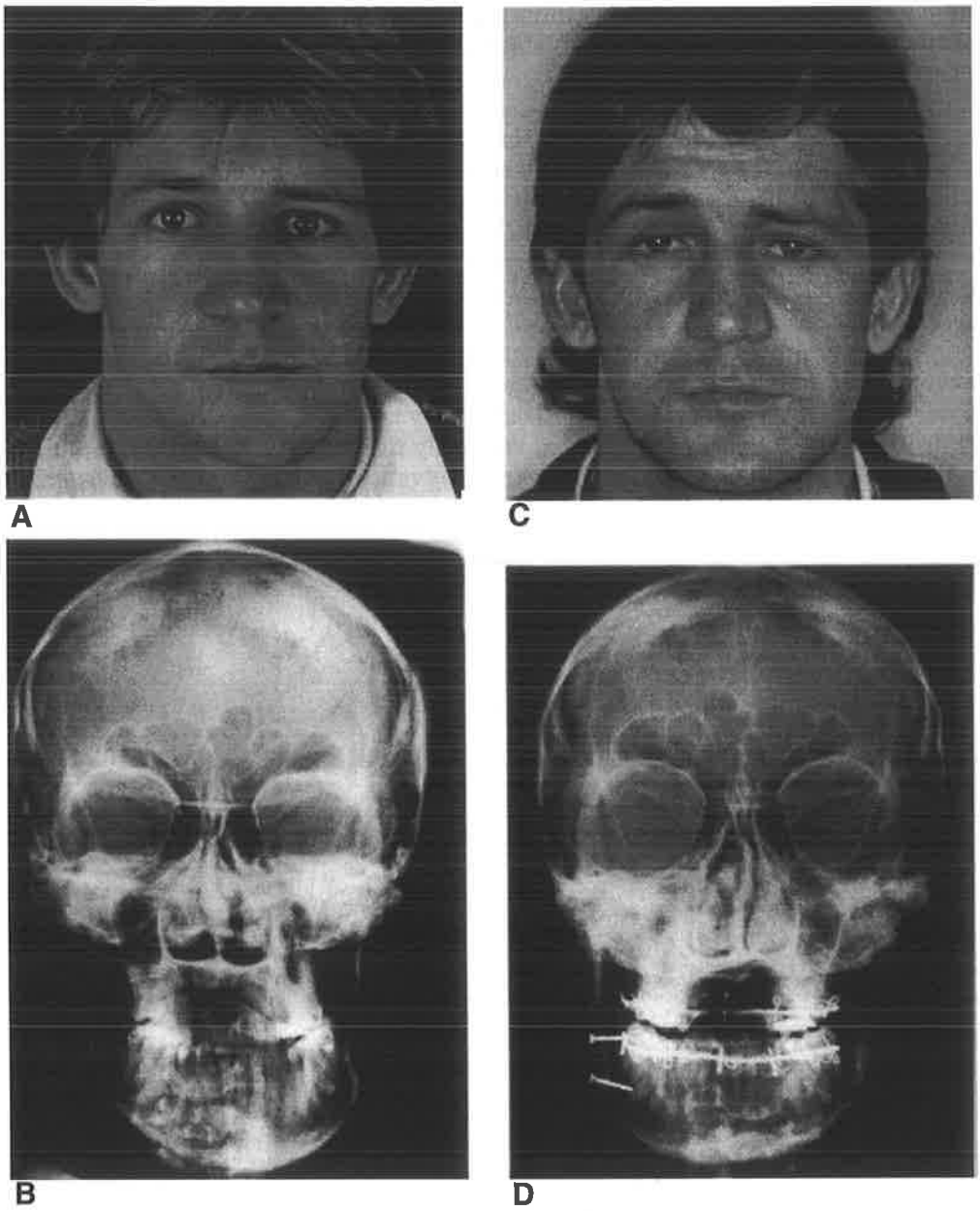


FIG. 21.69. ***Post-traumatic asymmetry with mandibular deviation.*** *A childhood injury led to asymmetric facial growth.* ***A*** *Asymmetry with deviation of the chin to the left.* ***B*** *X-ray further demonstrates the defect.* ***C*** *Postoperative appearance showing the restored symmetry.* ***D*** *Vertical subsigmoid osteotomy was performed on the right side and sagittal split osteotomy on the left. Intermaxillary fixation with lag screws fixing the two overlapping fragments on the right side.*

fixed into position with the preformed bite wafer and intermaxillary fixation. The bone graft is then inserted and fixed with miniplates. Further bone grafting may be needed to fashion the angle and enhance the stability.

Restoration of chin position and shape
In the post-traumatic setting, restructuring of the chin can best be made by osteotomies with or without bone grafting. There is an extensive literature concerning chin restoration with alloplastic materials such as silicone rubber; in our view, these techniques are best reserved for minor cosmetic deformities where there is no associated soft-tissue or bony damage (Fig. 21.71).

A horizontal osteotomy can be used to reshape the chin in three dimensions, moving the mobilized chin forwards, backwards, laterally and/or upwards, as well as downwards, when an intervening bone graft is needed. The surgical approach is made by a degloving dissection begun in the buccal sulcus. The incision

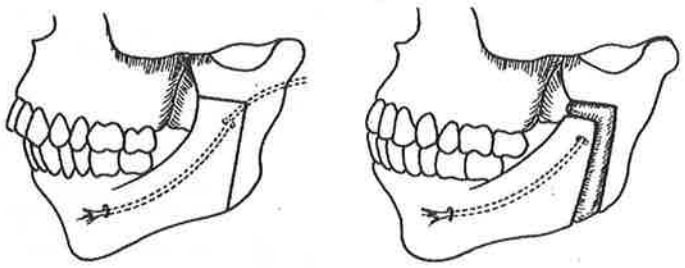


FIG. 21.70. *Inverted 'L' osteotomy.* *The inverted 'L' ramus osteotomy is performed above the entrance of the neurovascular bundle. After freeing the muscles, the distal segment can be distracted and bone graft inserted into the gaps. Plate fixation is preferred. There may be both onlay and inlay components in the bone grafting.*

is initially made through adequate soft tissue and passes obliquely down to the bone leaving some muscle on the dental side. Subperiosteal dissection leaves the soft tissue adherent to the chin point; the mental foramina are located and cleared. A reciprocating saw will easily make the osteotomy at the desired level, with wide clearance of the tooth roots. The posterior muscle attachments of the chin point can be retained for all but the 'jump' type of advancement, in which the distal segment is advanced and jumped entirely on top of the proximal segment. In all other chin reconstructions the attachments are left to ensure the blood supply to the fragment and to move the soft-tissue attachments with the bone. It is important when reducing the chin that the segment removed is taken from the body of the chin, not the chin point: if the point is resected, the soft tissue may fall away from its bony support, producing the 'witches chin' appearance (Gonzalez-Ulloa 1972). The fragment or fragments can be manipulated according to the surgical plan. Fixation is achieved by plates and/or screws. Two titanium miniplates may be used, but direct screwing is possible where there is an overlay of the apposed bones.

Contour restoration of body and ramus
It is unusual for the shape of the body of the mandible to need alteration after injury, but onlay grafts are sometimes needed to correct contour defects. Onlay bone grafting to the lower border of the mandible has been much enhanced in efficiency by the-ability to secure the grafts rigidly with screws. Exposure can be via the degloving approach or externally through the submandibular incision described on p. 612.

Complications and results

In our experience, osteotomies performed on patients who have acquired a mandibular deformity from trauma complicated by non-union or malunion and whose growth is complete, do not produce different complications or different

results from osteotomies performed for other reasons. However, there are two important points to make about the aesthetic outcome of these osteotomies.

First, the assessment of the patient must provide a detailed appraisal of the deficit that existed as a result of the trauma itself, and/or previous surgery, as the results of the current treatment must be seen against this background. Second, the treating team must have a clear knowledge of any pre-existing

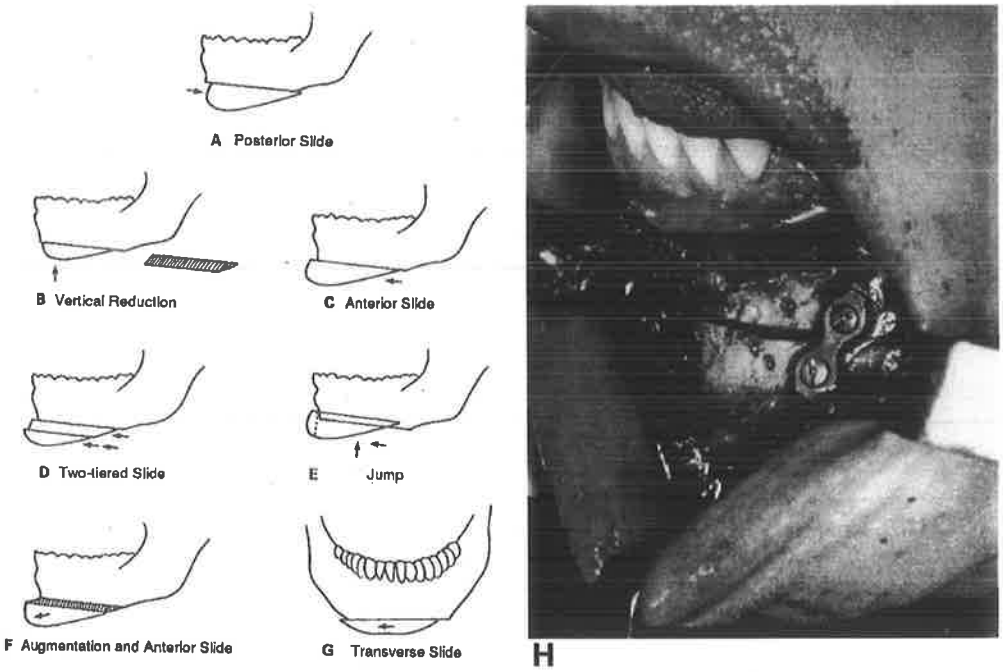


FIG. 21.71. ***Genioplasty. A–G*** *A variety of repositioning osteotomies can be performed on the chin using similar basic technique: retrusion, vertical reduction, advancement, 'jumping", side–to–side, and bond grafting as onlay or inlay.* ***H*** *Reduction genioplasty. The osteotomy is placed inferior to the mental foramina and the fragments are fixed with a pair of two-hole titanium plates.*

congenital deformity, and clearly separate, in the minds of both the treating team and the patient, what is to be achieved by the current treating plan. The question as to whether the aim is to return the mandible to its pretraumatic state or to an ideal state must be spelled out (Fig. 21.72).

The results are less predictable when the primary deformity has occurred during growth, for example the bird face deformity; in such cases, a compromise between ideal occlusion and facial form may be necessary.

The chief complications of mandibular osteotomy are infection, recurrent deformity and nerve damage.

Infection

Prophylactic antibiotics are used during surgery and for 24 h postoperative; nevertheless, and not surprisingly, infection is an important complication in most reported series. In 236 cases of mandibular osteotomy, our infection rate is 2.2%. Onlay bone grafts of the angle of the body of the mandible have been in our experience especially prone to infection. The use of rigid fixation with titanium screws has improved the infection rate and decreased the resorption rate; however, more than half our patients treated with onlay grafts have had trouble with infection and resorption in this site. If infection does occur it generally will not settle until the underlying screw, plate or bony sequestrum is removed.

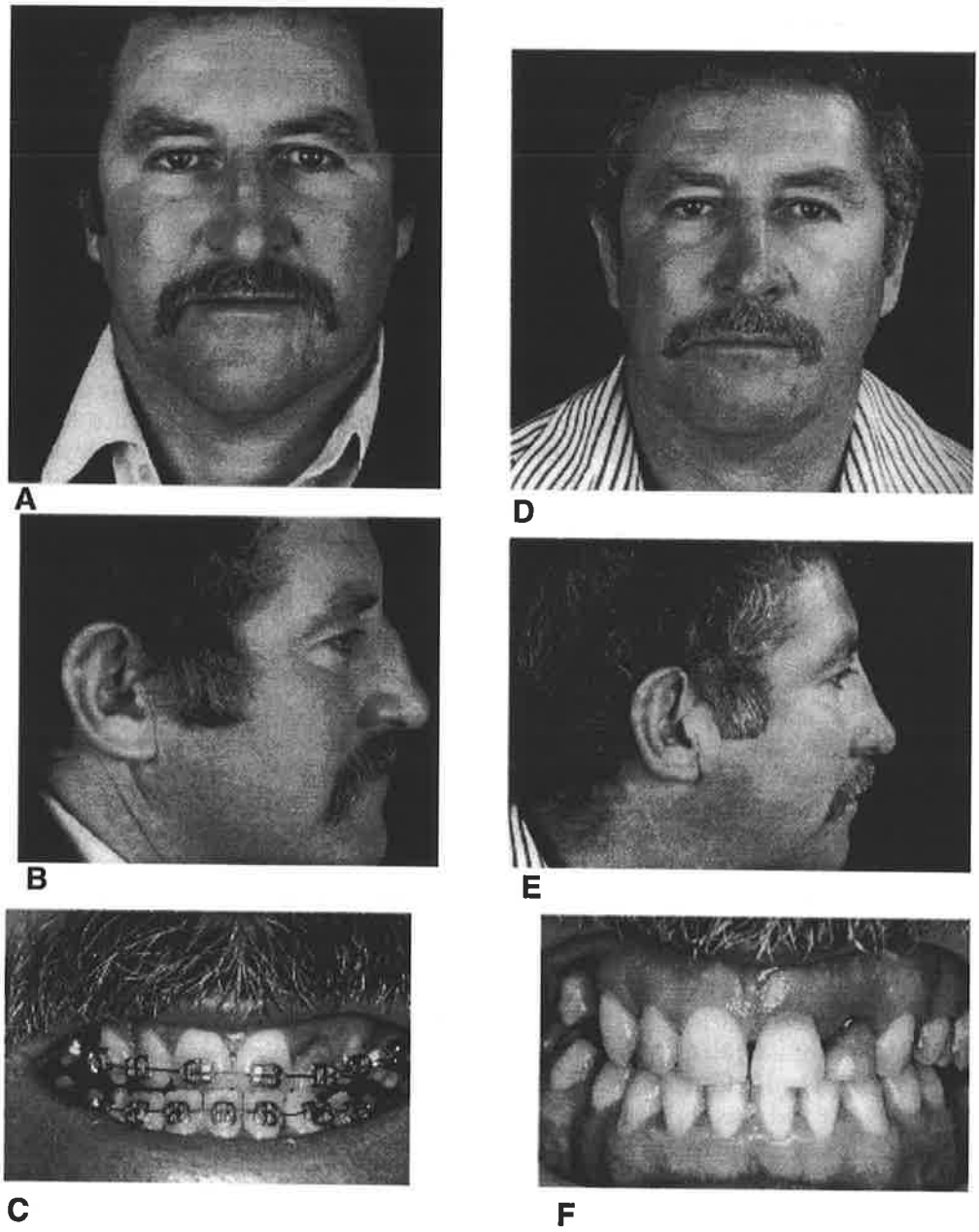


FIG. 21.72. *Traumatic displacement with pre-existing skeletal deformity.* *'It is an ill wind that blows no good.' A middle-aged man presented with a mandibular fracture which was accurately reduced, restoring him to his previous class III malocclusion. 2 years later he presented with a maxillary fracture and requested that his occlusion and facial shape be changed at this opportune time.* ***A*** *Anteroposterior view after the first mandibular fracture.* ***B*** *Profile view.* ***C*** *Close-up view of the occlusion with orthodontic treatment in progress.* ***D*** *Profile view.* ***E*** *Occlusion after the maxillary fracture has been repositioned anteriorly.* ***F*** *Facial appearance after the second fracture has been corrected.*

Recurrent deformity
This can be due to bad planning, bad execution of the surgery or massive infection and loss of bone.

Nerve damage
This may result from the original injury; it is therefore very important to examine the status of the fifth and seventh nerves before operation. The cervical branch of the seventh nerve may be damaged during the submandibular approach, although we have seen no occurrence of this in nearly 20 years of practice. The trunk of the facial nerve may be damaged as a result of poor technique in the sagittal split osteotomy. We have one experience where there was a temporary facial paresis. The nerve was explored and found to be intact but swollen and oedematous; spontaneous recovery took place. The inferior dental nerve is easily injured in mandibular osteotomy; in patients where this nerve was not been previously damaged almost 50% showed some sensory deficit for up to a year after definitive surgery and 15% have remained with some permanent sensory loss.

Massive Defects of the Mandible and Maxilla

Surgical pathology

Deformities entailing massive bone and soft-tissue loss are most commonly due to missile wounds; pathology, general management and treatment strategies are discussed in Chapter 16. The size of the bone defect needing replacement and the quantity and the quality of the soft tissues in the area are the pathological factors of chief relevance in planning a reconstruction. Also important are foci of chronic infection, which may form around indriven bone chips and tooth fragments.

The mandible has received prime attention. It is not only of importance for facial appearance but is also vital functionally since it supports the tongue; it is necessary for mastication, speech and maintenance of the airway. There appears to be relatively little written on the techniques of reconstruction for major deficiencies of the maxilla: nevertheless, these deficiencies are also very deforming and may be associated with defects of jaw function and airway, as well as with damage to the eyes.

Assessment

Investigations include the radiological imaging necessary to determine the size and shape of the bone defect and if necessary the nature of any associated TMJ dysfunction. In these, 3D reconstruction from the CT scan comes into its own, as it not only gives a very good image of the bone shape to be replaced but also displays the abnormal positions and relationships of the fragments. Programs are now available which are able to model the shape and size of the defect to produce a template upon which the bone graft can be modelled. Dental models help in surgical planning to restore whatever occlusion can be salvaged, and are also useful in defining the bony and soft tissue defects. Speech, swallowing and oral competence are necessarily affected by massive trauma of the jaws; the team speech pathologists are therefore involved in the planning, assessment and rehabilitation of these patients (Fig. 21.73).

Principles of management

The history of the repair of massive jaw injuries has until recently been intimately bound up with the history of war. Excellent historical reviews are presented by Rowe (1971) and McCarthy et al (1990b), and the theme is explored in Chapter 1.

Today, the options for jaw reconstructions are:

- Insertion of metallic spacers
- Insertion of alloplastic trays containing cancellous bone
- Replacement with heterogeneous treated bone
- Replacement with autogenous bone graft
- Insertion of a vascularized bone graft:
  - i pedicled, or
  - ii sustained by microvascular anastomosis.

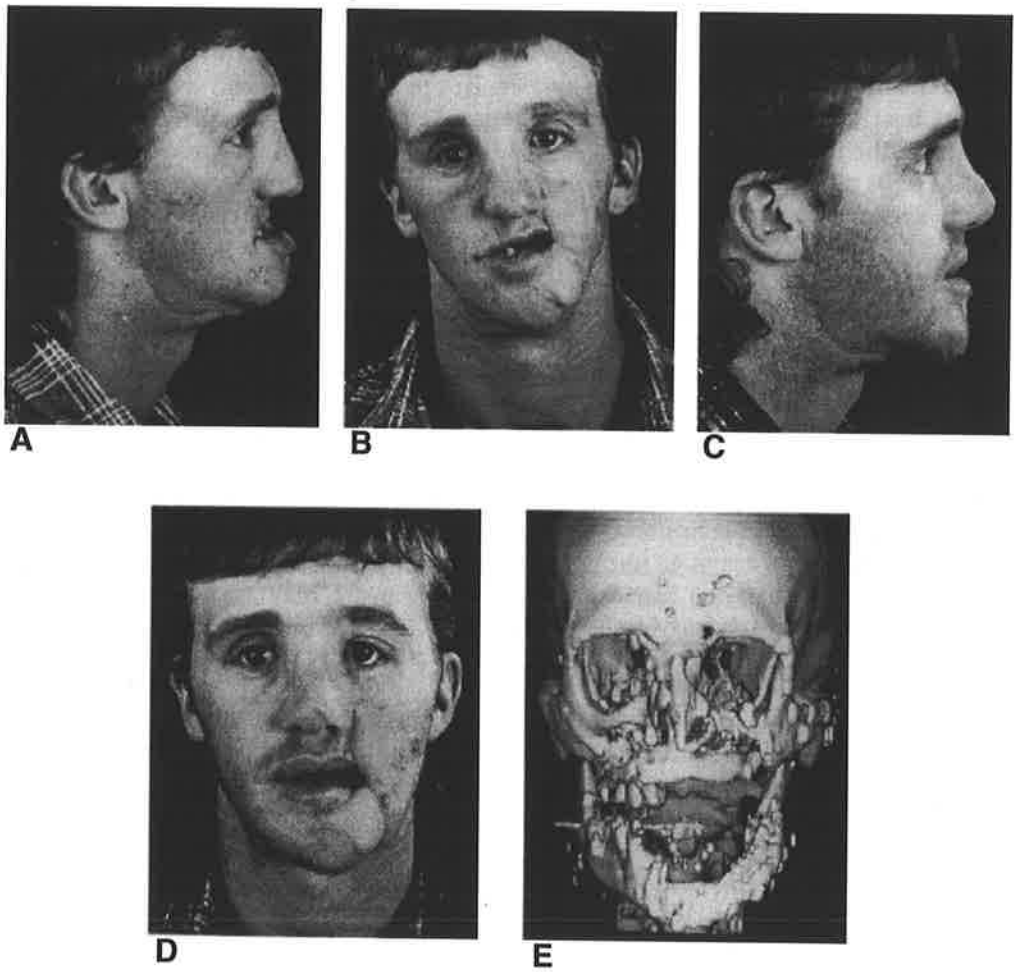


FIG. 21.73. ***Extensive trauma to the jaws.*** *A boy suffered a gunshot wound and was originally treated in an outlying community. He lost the upper segment of the maxilla leaving only a remnant on the right side. On the left side a large portion of the body of the mandible was absent, as was soft tissue of the floor of mouth and tongue. The mandible on the left side had been partially reconstructed by a free flap which had failed to heal properly. The lip was incomplete and a deficit remained in the floor of the mouth. X-rays indicated residual fragments from the gunshot. Treatment began with microvascular lower jaw reconstruction (iliac crest based on the deep circumflex iliac artery) and upper jaw reconstruction (vascularized rib). At the second stage the soft tissue from the floor of mouth was removed in preparation for osseo-integrated implants, on which a clip-on prosthesis will rest.* ***A*** *Initial presention, lateral view, indicating the absence of a significant amount of upper jaw.* ***B*** *Destroyed left cheek and remnant of the free flap covering the lower lip, under which was an unhealed remnant of the previous vascularized bone graft.* ***C*** *Lateral view after reconstruction of the upper jaw with vascularized rib and lower jaw with vascularized hip, and bone grafting of the nose.* ***D*** *Anteroposterior view indicating the remaining deficiency of the orbicularis muscle and soft tissue paddle on the chin.* ***E*** *3D CT scan indicating the reconstituted mandible and maxilla, repositioned left cheek bone, and the bone graft to the dorsum of the nose.*

The choice of procedure depends not only on the size of the defect in the bone but also on the quality of the soft tissue cover. A significant factor to be taken into account is the nature and the success of the primary treatment (p. 448). Unless it has an independent blood supply, a bone graft cannot survive without immobilisation, excellent soft-tissue cover and a good local blood supply. The assessment of the capacity of existing soft tissue to sustain a bone graft is a vital judgement that has to be made by any team treating such deformities. If it is thought that the soft tissue cannot do this, the bone graft must have its own blood supply from a pedicle or be supported by microvascular anastomosis. Alternatively new soft-tissue cover may be provided for the bone graft.

Insertion of metallic spacers

A good example of such devices is the Bowerman Conroy kit which became popular during the 1970s (Evans et al 1985). This kit provided large malleable titanium plates, which were used to bridge mandibular defects, being bolted to the lingual side of the mandible. This technique initially held some promise but experience has shown that exposure of the plate through the soft tissues often occurs, and leads ultimately to removal of the spacer. In the post-traumatic setting spacers are at best temporary manoeuvres to separate bone ends for subsequent bone grafting; the use of long titanium plates for this purpose is discussed on p. 455. In the light of modern techniques of bone grafting and its own poor record, this form of reconstruction is probably now obsolete as definitive treatment.

Insertion of alloplastic trays containing cancellous bone

The principle of this technique is that the bone chips act as a receptive scaffolding for the growth of new bone and the tray acts as support and spacer to facilitate this. We have no experience of this technique and reference is therefore made to the reviews by McCarthy et al (1990b), Evans et al (1985) and Marx & Stevens (1991). Taher (1990), reporting on a series of 128 mandibular defects sustained in the Iran-Iraq war, used titanium mesh trays containing bone and hydroxylapatite granules in some 34 cases, and achieved good results with a very low incidence of complications. Tayapongsak et al (1993, 1994) have also employed titanium trays to support autogenous bone chips and marrow, held together with autologous fibrin glue (p. 380) (Fig. 21.74). Other authors have used steel trays, which have to be precast, or plastic cribs.

Heterogeneous treated bone

Bone harvested from cadavers has been used. Marx & Stevens (1991) favour the use of freeze-dried ribs, ilium or even whole mandibles, hollowed and shaped to serve as trays to contain autogenous cancellous bone chips; these authors believe that with appropriate tissue bank precautions, the risk of viral transmission is negligible. We have no experience of this technique and believe that it has been supplanted by bone flaps and microvascular methods.

Autogenous bone grafts

For a century surgeons have been looking for material to replace the damaged or absent mandible. Autogenous bone is of course the material of choice in replacing a mandibular defect. Bone grafting is now the most commonly used method of reconstructing significant defects of the mandible. The bone can be harvested from the rib, hip or calvaria (p. 240). Although all of these donor sites are used from time to time, it is the hip that provides the most predictable source in terms of quality and quantity of both cortical and cancellous bone. We have found rib grafts to be less successful because they are often less robust and unpredictable in their shape and size. There is nothing more disturbing when harvesting ribs for jaw reconstruction than to find that the grafts are friable and lack substance. However, the costochondral junction is the preferred graft for TMJ reconstruction where there is a previous ankylosis (p. 604).

The general principles of bone grafting are very relevant in mandibular reconstructions. A bone graft will not 'take' unless it is placed in an adequate bed, hence the soft-tissue cover must be complete and of good vascularity. The

FIG. 21.74. ***Particulate bone and autologous fibrin polymer.*** *Particulate cancellous bone graft, in a matrix of autologous fibrin polymer, is used to reconstruct a large mandibular defect. A titanium crib replaces the inferior border only. (Photo courtesy of Dr Pairot Tayapongsak.)*

defect to be filled must be recreated by fixing the mandibular fragments in their predetermined correct position, usually by means of intermaxillary fixation and some form of locating splint. The bone graft must be harvested carefully, shaped in all of its three dimensions, inserted into the gap and fixed rigidly (Fig. 21.75). Watertight closure is important and great care must be taken to avoid haematoma formation. Under these circumstances quite large segments of mandible can be replaced (Manchester 1965).

Defects of the angle of the mandible behind the occlusion are best dealt with by fixing the lower jaw to the upper jaw by intermaxillary fixation, in a predetermined acceptable position. The posterior fragment is then manipulated into what appears to be an acceptable position and an iliac crest graft is cut and shaped to bridge the defect. There are a number of ways to fix this graft. Fixation can be done by overlapping the graft ends and fixing each end by direct screwing with at least a triangle of screws, or by abutting the ends and using plates, or by using a single long malleable plate with screws into the mandibular fragments and the bone graft. Except in rare circumstances the soft-tissue cover of skin and muscle in this region is very conducive to healing. Calvarial bone graft can also be used; it is possible to use calvarial bone pedicled on the temporalis muscle (see below).

Where large segments of mandible are missing and there is a good soft-tissue bed remaining then large bone grafts can be used as free grafts. Since World War I, restorative procedures have often involved the use of rib grafts (Gillies 1920). Ribs, however, are often of inadequate strength and when split to facilitate curving they become weaker. Levant (1977) has used whole ribs impacted into the mandibular stumps. Iliac crest can be used and segments of mandible may be carved out of the full thickness of the ilium. Various authors have suggested appropriate patterns. Dingman (1950) and Seward (1967) described U-shaped grafts to replace the anterior mandible. Manchester (1965) carved a half mandible and was able to retrieve it post-mortem many years later (Fig. 21.75). Alternatively segments of bone can be strung on a K-wire or in more modern technique united with a long titanium miniplate.

All these bone grafts depend on vascularization from the bed for survival; they require long periods of immobilization, vary in thickness and often do not become robust with time. They are subject to displacement by muscle pull and subject to the complication of infection and bone loss to an inordinate degree. Nevertheless the work of Manchester bears further consideration (Manchester

1972). His clinical studies showed that where there was an adequate soft-tissue bed and where the surgical technique was absolutely meticulous with respect to preparation of the bed, carving of the bone, fixation of the bone, immobilisation and prevention of haematoma, then large segments could be replaced which would survive. Indeed, there is strong evidence from his superb clinical studies to indicate that the transferred bone itself survived and underwent healing processes; healing was evident even in fractures in the bone made at the time of reconstruction for the purpose of shaping. Manchester was careful to point out that special circumstances must exist before these grafts would succeed; nevertheless his techniques and his subsequent theses for the success of 'bone taking' are worthy of note (p. 132). The majority of Manchester's cases came for reconstruction after excision of benign mandibular tumours and the application of his technique in the very different traumatic setting requires careful appraisal.

Vascularized bone flaps and transfers

In view of the problems that have always arisen in reconstructing large defects of the mandible with bone grafts, especially when associated with a compromised soft-tissue bed and cover, vascularized bone transfers and composite bone muscle and skin flaps have been used more and more frequently. There is now a vast experience of vascularized transfer of bone for restoration of the mandible in reconstructions carried out after tumour resections both benign and malignant. These techniques are eminently applicable to reconstruction after trauma. Vascularized flaps usually involve skin, muscle and bone, and numerous flaps of this type have been described. Initially these flaps were vascularized by pedicles dissected in continuity from parent arteries and veins.

Rib flaps

Pedicled flaps including a rib have been described by many authors, but the technique goes back to the very early part of the century when Vilray Blair described the transfer of rib as part of a complex flap for mandibular reconstruction (Blair 1918). Cuono & Ariyan (1980) used a part of the fifth or sixth rib associated with a pectoralis myocutaneous flap, using the pectoral part of the muscle based on its arterial supply from the thoraco-acromial artery. The transfer of rib as an island flap based on the internal mammary vessels alone was described by Strauch et al (1971). This was done first in dogs; the seventh rib was isolated on skeletonized internal mammary vessels and the clinical application was made by Ketcham et al (1974) who repaired a mandibular defect with a portion of the seventh rib and adjacent sternum. Rib transfer using the latissimus dorsi muscle and thoracodorsal artery has been reported (Schmidt &

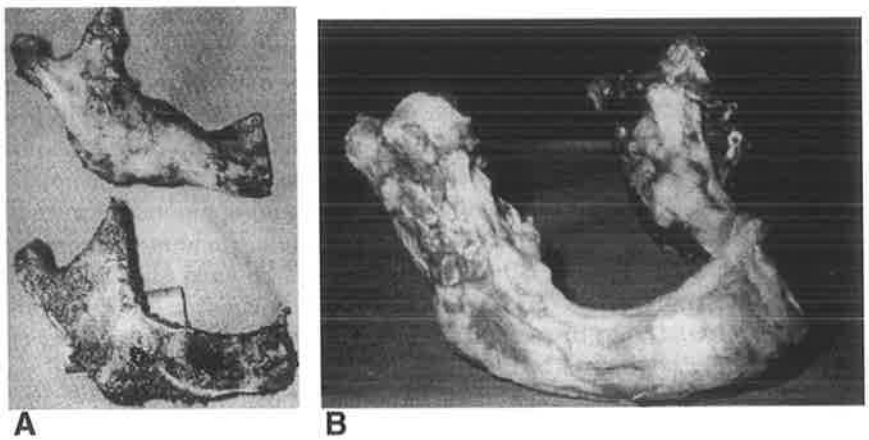


FIG. 21.75. ***Mandibular reconstruction with non-vascularized ilium.*** ***A*** *The desired mandible is carefully carved from the ilium to match the resected bone.* ***B*** *A similarly reconstructed mandible removed many years later at post mortem, showing remodelling. (Photographs by courtesy of Sir William Manchester and* Plastic and Reconstructive Surgery*.)*

Robson 1982, Maruyama et al 1985). Although Ariyan (1980) demonstrated that rib grafts can survive when transferred on their periosteal blood supply, this source of bone is very much second best in restoring a significant post-traumatic defect: the quality of the rib may be poor, its dimensions are inadequate for much more than a functional spacer, and it will not provide an adequate skeleton for framing a soft-tissue complex capable of fitting dentures, or stout enough to take osseo-integrated implants.

The clavicle flap

Conley (1972) described a compound flap involving the clavicle; Siemssen and his colleagues from Denmark (1978) also reported such a flap, a large segment of the whole clavicle being pedicled on the sternocleidomastoid muscle (Fig. 21.76).

The sternal flap

The lateral border of the sternum can be transferred using the pectoralis major myocutaneous flap as a carrier. Green et al (1981) described this procedure and Robertson (1986) also reported the results of using this technique.

The trapezius — scapula flap

A flap of trapezius muscle bearing the scapula spine has been described by Demergasso & Piazza (1979). The technique is also described by Panje & Cutting (1980) and Dufresne et al (1987) (Fig. 21.77).

Calvarial flap

All these flaps bearing bone to reconstruct the jaw are pedicled from below; in contrast, the calvarial full thickness or outer table graft is pedicled on temporalis muscle and rotates down from above (McCarthy & Zide 1984, Van Der Meulen et al 1984, McCarthy et al 1987). Where the bone has good diploe and can be split, cautious dissection can leave the inner table in situ. When this is not so, the aid of a neurosurgeon must be employed and the procedure becomes much more considerable, with all the problems attendant upon opening the skull, the least of which is the need to repair the defect with bone from elsewhere, usually split calvaria or bone from another site, hip or rib. The pedicled flap has been used to reconstruct the zygoma and orbital floor as well as the ramus of the mandible (Fig. 21.78).

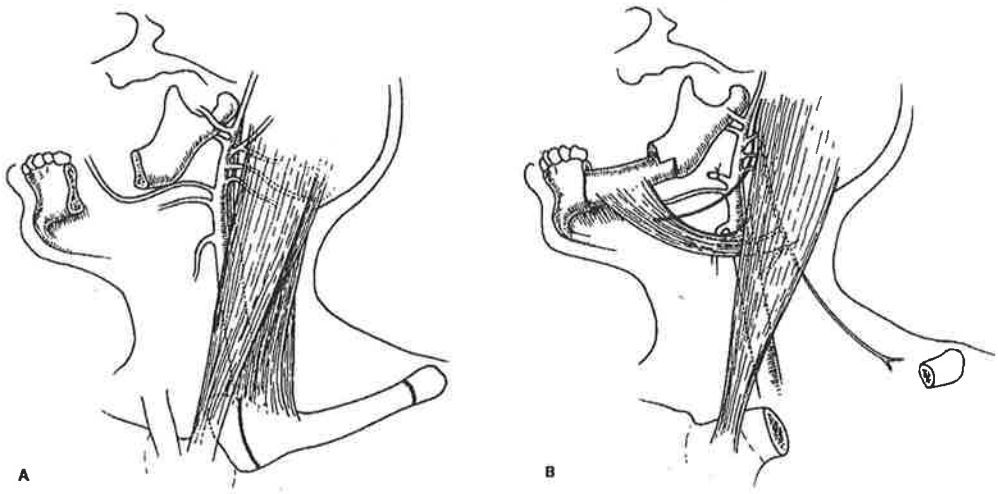


FIG. 21.76. ***Transfer of the clavicle on the sternocleidomastoid muscle.*** *After Siemssen et al* (1978), *by courtesy of* Plastic and Reconstructive Surgery. ***A*** *The mandibular defect.* ***B*** *Transposition of muscle and bone.*

Many of these techniques for pedicled bone grafting have grown out of procedures used in the reconstructions necessary after resection of the mandible for oral cancer, and have been applied to post-traumatic reconstruction. The calvarial flap had its origins in the need to reconstruct the orbit and the ramus of the mandible in Treacher Collins syndrome (Raulo & Tessier 1981). McCarthy et al (1990b) noted that bone transferred thus is best reserved for smaller segments; failures have occurred with more heroic attempts to fill large gaps. In our own practice the pedicled flaps have by and large been supplanted by more elegant and accurate reconstruction with microvascular anastomosis. They are described

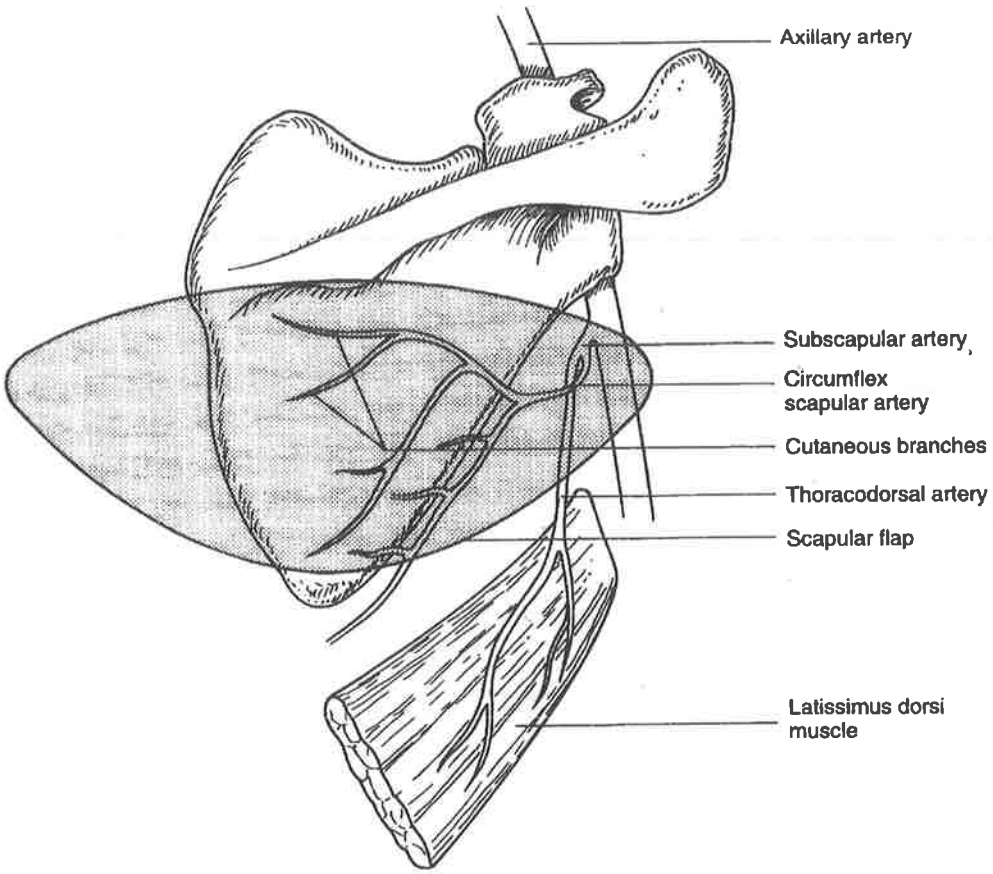


FIG. 21.77. *Scapular osteocutaneous flap.* *After Swartz et al (1986), by courtesy of* Plastic and Reconstructive Surgery. *Inclusion of the thoracodorsal pedicle with the scapular osteocutaneous flap allows the additional use of the latissimus dorsi muscle or myocutaneous skin paddle.*

here because they represent a stage in the evolution of techniques of reconstruction of the bony facial skeleton, and still remain relevant where microsurgical skills and technology are not available, or where less happily microvascular techniques have failed and some salvage procedure is necessary. All these techniques have been enhanced by the availability of rigid interosseous fixation through mini- and microplating.

Osseomyocutaneous vascularized transfers

Where microvascular anastomosis provides an intact blood supply to the bone graft, there is no loss of osteogenic potential in the bone, and less risk of compromise by an inadequate environment such as that resulting from scar or irradiated tissue. The historical progress of this technique from experiment to clinical application is one of the true success stories of modern plastic and reconstructive surgery. McCullough & Fredrickson (1973) inaugurated the use of microvascularized bone flaps in experimental procedures on dogs. Ostrup and his colleagues (Ostrup & Fredrickson 1974, Ostrup & Tam 1975) completed this

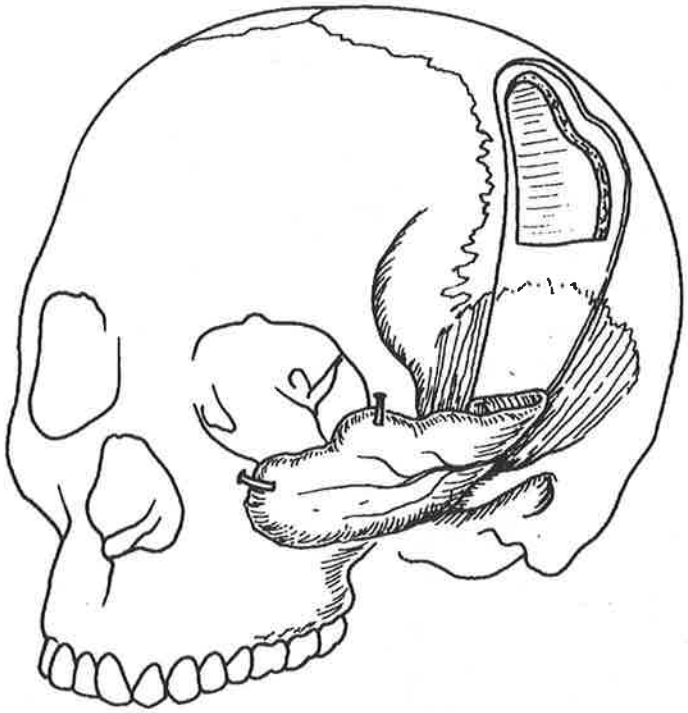


FIG. 21.78. *Pedicled calvarial flap.* *The calvaria can be pedicled on the temporalis muscle as described by McCarthy et al (1987), yielding an extremely versatile flap for the reconstruction of congenital deformities including Treacher Collins Syndrome. If the calvaria is thick enough to be split, the outer table can be carried in this way. More frequently it is a matter of carrying the full thickness, involving the neurosurgeon to protect the underlying dura. This pedicled flap can be turned down to reconstruct the ramus of the mandible as well as the areas around the orbit.*

work. In their 1974 study, angiography demonstrated that the flow of blood in the ribs was from the nutrient arterial supply. Doi et al (1977) showed that rib transferred by microvascular anastomosis to the femur of the same animal did not lose substance and rapid bone union took place. When Taylor et al (1975) successfully transferred a vascularized fibula in the human in a case of lower limb trauma, the crucial step of clinical application was put into place. Harashina et al (1978) reconstructed the mandible using a posterior rib graft. We (David & Tan 1979) reported a case performed 18 months previously of reconstruction of the hemimandible using iliac bone vascularized by the periosteal vessels from the groin flap as demonstrated by Taylor (1982) (Fig. 21.79). To this day we dissect both pedicles when using bone based on the deep circumflex vessel. There has been controversy as to the relative values of the blood supply taken from nutrient vessels or from periosteal vessels, but for practical purposes this does not seem to matter. Successful transfers with good long-term follow-up have been widely reported with bone flaps supplied from the nutrient vessels, or from the periosteum or from both, and the source of the blood supply to the bone no longer seems to be of great moment. The transfers used by us in mandibular reconstruction are described below; some of them have also been used in maxillary reconstruction.

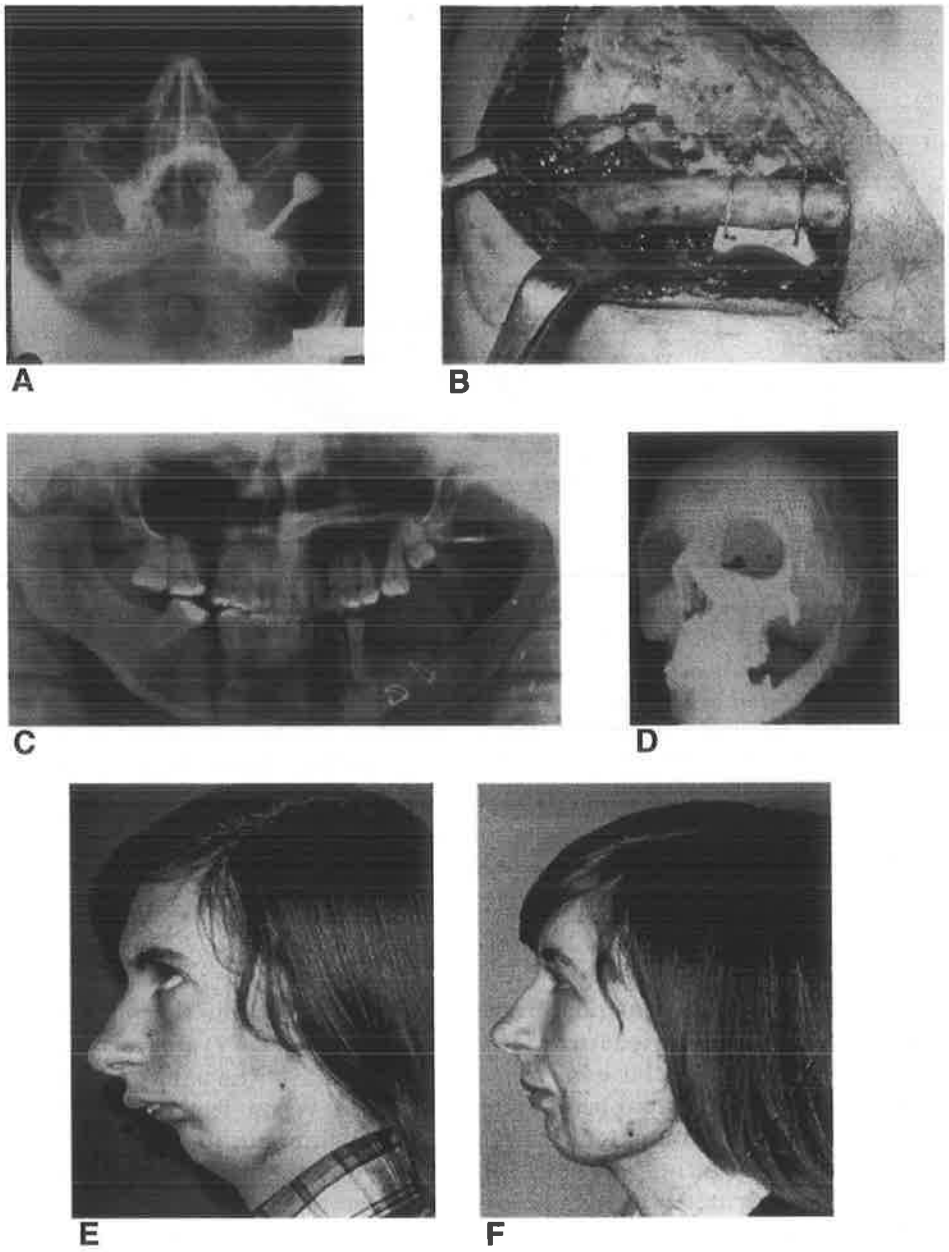


FIG. 21.79. *Iliac crest free flap.* *A teenage girl presented with craniofacial microsomia, having had a series of operations including a metatarsal transfer and a Bowerman's prosthesis which was subsequently removed. Non-vascularized rib was used to reconstruct her zygomatic arch and joint, and the hemimandible was reconstructed from iliac crest (supplied by the superficial circumflex iliac artery) with overlying soft tissue used for contour restoration. She is seen at the time of the operation and a decade later. It is notable that after 10 years the rib has disappeared but the ilium is intact.* ***A*** *Plain radiograph showing the Bowerman's prosthesis.* ***B*** *Operative photograph showing the zygoma reconstructed from rib with a costochondral condylar fossa suspended by wire.* ***C*** *OPG showing the reconstruction.* ***D*** *3D reconstruction made a decade later demonstrating the mandible intact but the zygoma having disappeared.* ***E*** *Initial presentation.* ***F*** *Final appearance.*

Reconstruction of Mandibular Defects

Management

Complex mandibular reconstructions require large bone replacements and multiple osteotomies in a compromised soft-tissue environment, and constitute a prime indication for microsurgical reconstruction. These procedures can be adapted for lesser deformities, tailoring the operation to the defect to create an elegant bone and soft-tissue reconstruction. Onlay and inlay techniques can be used and combined with miniplate and lag screw fixation; the grafts have the capacity to support osseo-integrated implants, and this versatile technique has greatly expanded our capacity to reconstruct the jaws successfully. A further advance is the use of multiple free flaps in a single procedure, taking the best from each flap: thus, an iliac bone graft supported by the deep circumflex iliac artery is used for jaw reconstruction and a thin hairless forearm flap for reconstructing the oral lining (David et al 1988).

Vascularized tissue transfers
The transfers now in use offer various possibilities in reconstructing mandibular defects. We review here the chief sources of vascularized bone, and describe in detail the operative techniques which have been developed by us in mandibular reconstructions for traumatic and non-traumatic deformities.

Rib transfers
Rib flaps pedicled on the nutrient vessel have fallen into disuse because an extensive transthoracic approach is necessary to harvest the rib. Free rib transfers were described by Ariyan & Finseth (1978) and Ariyan (1980) who showed that ribs can be transferred successfully if vascularized by a periosteal blood supply from the intercostal arteries, or by a muscular attachment to a revascularized muscle. Additional supply from a nutrient artery is not necessary, and this simplifies the dissection of the free rib flap. Anterior intercostal flaps (Ariyan & Finseth 1978, McKee 1989), posterior intercostal flaps (Daniel 1978, Serafin et al 1979, 1980) and lateral intercostal flaps (Badran et al 1984) have been used for mandibular reconstruction. We prefer to take a serratus anterior free flap as a carrier to vascularize a rib graft through the muscular attachment. One or two ribs can be raised, usually the fifth and/or sixth ribs, the length of rib being up to ~12 cm in an adult. A small skin flap can also be taken over the selected rib.

The patient is placed supine, arm abducted, with a sandbag under the shoulder. An incision is made in the posterior axillary fold, curved gently forward to include a skin ellipse if this is desired. The lateral border of latissimus dorsi is found; finger dissection opens the plane between this muscle and the lateral chest wall, and exposes the serratus anterior with its nerve and its vascular pedicle from the lateral thoracic artery. The nerve supply is segmental, and if the central slips of muscle are taken, it is possible to preserve the upper and lower slips and so to avoid winging of the scapula. The vascular pedicle can be elongated by dissecting to the thoracodorsal artery or even to the subclavian.

When the appropriate slip or slips of serratus anterior have been isolated, the rib graft is taken by incising the upper border of each rib and separating it from the pleura; the posterior periosteum is stripped. This lessens the risk of intrathoracic complications and promotes rib regeneration. The rib graft may be shaped by subperiosteal osteotomy (Fig. 21.80).

The scapula transfer
Swartz et al (1986) described a free osteocutaneous scapular flap which can provide a significant amount of bone for mandibular or maxillary reconstructions. This flap has been used for rebuilding the hard palate and anterior maxillary arch, when one or both sides of the oronasal cavity require lining tissue (Fig. 21.77). The flap may also be used in nasal or upper lip reconstruction in conjunction with repair of the anterior maxillary arch.

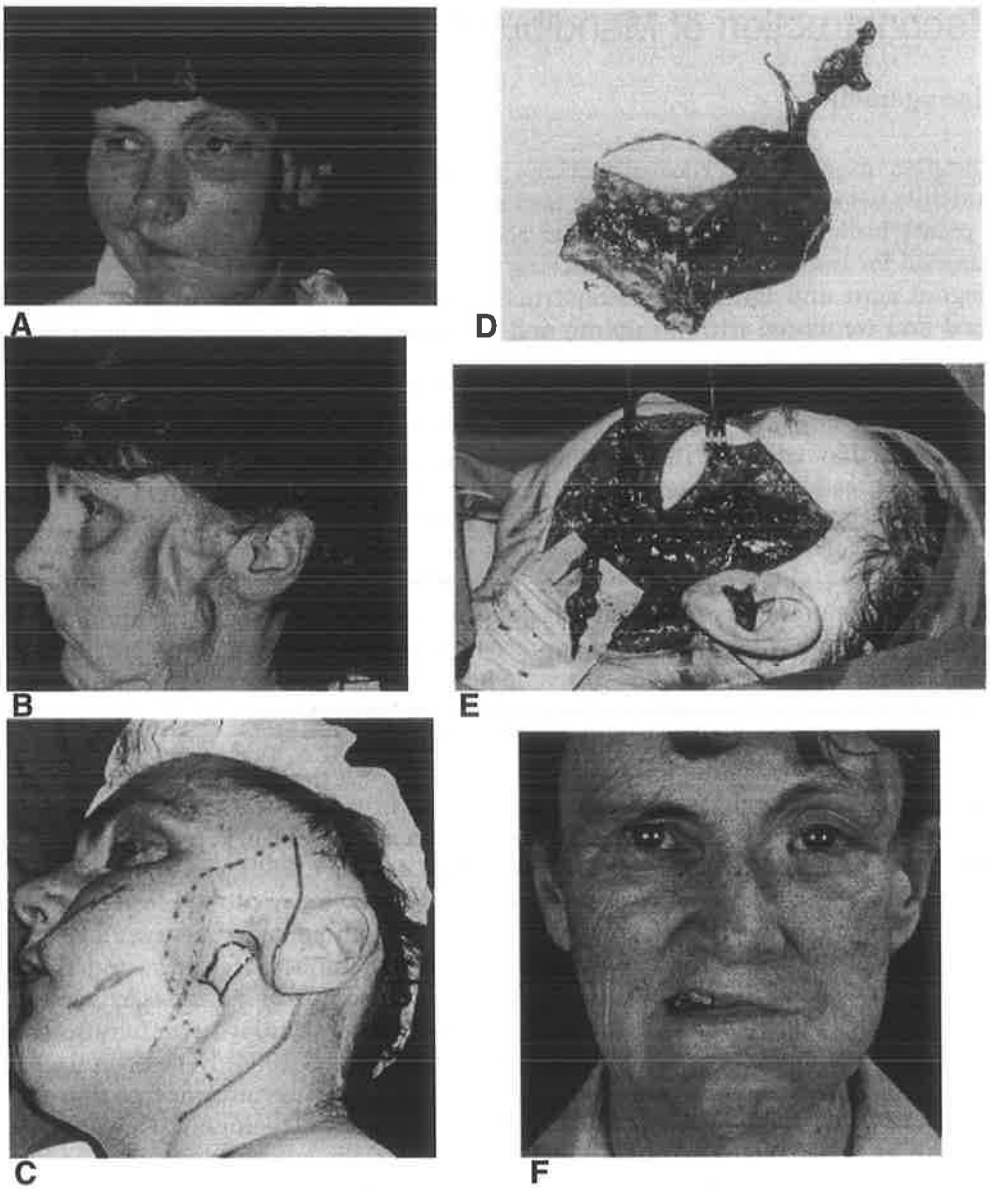


FIG. 21.80. ***Vascularized rib graft.*** *female underwent resection of a left parotid adenoidcystic carcinoma. Microvascular transfer of rib and innervated serratus anterior muscle was performed for reconstruction of the complex deformity in the left parotid area, including a left facial palsy.* ***A*** *Pre-operative view from the front showing the facial palsy and the contour defect.* ***B*** *Lateral view shows absence of the zygomatic arch, condyle, and condylar neck, as well as the effects of the facial palsy.* ***C*** *Design of the operation in which a composite flap of rib and serratus anterior with an overlying skin and fat paddle, together with nerve and artery, are to be placed into the defect. The muscle provides an innervated soft tissue bulk.* ***D*** *Disconnected flap.* ***E*** *Dissection at time of surgery. The flap is shown in situ prior to the anastomosis of the vessels.* ***F*** *The patient 2 years later with the capacity for facial mass movement, reasonable static tone, and good facial contour.*

The lateral edge of the scapula below the glenoid fossa is thick, but the bone thins to 2–3 mm thickness in most of its body; a scapula bone graft can be up to 10 cm long. The blood supply is derived from branches of the circumflex scapular artery, especially its branch to the teres major muscle. The skin over the scapula tends to be thick, with a considerable layer of subcutaneous fat, making it a difficult flap to use except in a slim individual. The skin is supplied by a cutaneous branch of the circumflex scapular artery which emerges through the triangular space bounded by the two teres muscles and the long head of triceps. The skin flap may be taken horizontally or obliquely in line with the lateral border of the scapula; in marking the skin incisions, the flap should be sited over its blood supply from the triangular space.

The flap is best raised with the patient in the lateral position and the arm supported in abduction. The skin flap includes the deep fascia, and dissection usually proceeds from medial to lateral, so that the cutaneous blood supply can be identified and traced to the triangular space. Further dissection requires deep retraction to identify the circumflex scapular artery and the subscapular artery from which it arises. If necessary, the subscapular artery is exposed by an additional anterior dissection — this helps to get a longer pedicle, and is especially useful in obese patients. The area of bone to be taken is left with some attached teres muscle and soft tissue around the circumflex scapular artery because small periosteal branches, especially one descending along the lateral border of the scapula, may contribute to the supply of the graft. If a long piece of bone, rather than a plate, is taken, it may be shaped by subperiosteal osteotomy as desired.

The lateral arm osteocutaneous transfer

This flap, described by Song et al (1983) and Katsaros et al (1984), is based on the posterior radial collateral artery, a branch of the profunda brachii artery. This vessel gives several cutaneous branches to the overlying skin and muscular branches to the adjacent brachioradialis and triceps muscles, through which a periosteal blood supply goes to the humerus in the area of the lateral supracondylar ridge. In an adult, this will provide a segment of bone 10 cm long and 1 cm wide. An osteocutaneous flap can be designed with a very small skin component based on a single cutaneous perforating artery and a small piece of bone which can be used to repair palatal or maxillary arch defects. A larger vascularized bone graft can be used in mandibular reconstruction.

The skin flap is centred over the surface marking of the lateral intermuscular septum — the lower half of a line drawn from acromion to lateral epicondyle. The flap is raised subfascially over the triceps tendon posteriorly and the brachioradialis anteriorly. As the dissection progresses proximally, the radial nerve is encountered anteriorly passing between the brachioradialis and the brachialis, together with the anterior radial collateral artery. Above this point, the pedicle can be developed by dissecting out the profunda brachii artery and its accompanying veins in the spiral groove, giving more length and larger vessels for the anatomosis.

In taking the bone graft, a 1-cm-wide cuff of muscle is preserved on each side of the supracondylar ridge. The selected piece of bone is cleaned, marked and then cut, preferably with an electric reciprocating saw, in the desired shape — usually gently curved. Further shaping may be done by subperiosteal osteotomy.

The radial forearm flap

Since its introduction by Yang (1981), this flap has found a firm place in intraoral reconstructions as a source of chin skin for lining and as an osteocutaneous flap for mandibular reconstruction. The blood supply to the bone is periosteal through the attachment of the lateral intermuscular septum. Up to 10 cm of the radius can be taken between the insertions of pronator teres and brachioradialis, but no more than half the thickness of the bone can be taken to avoid the risk of fracture. A very large skin flap can be raised if necessary, from the elbow to the wrist, or alternatively a very small flap can be designed, provided that a larger fascial component carrying a cutaneous branch is taken.

An Allen test (p. 254) is done to ensure that the hand will be viable without the radial artery. The course of the artery is marked between the palpable brachial artery and the radial pulse at the wrist. The desired skin flap is raised subfascially, leaving intact paratenon to receive a skin graft. Care is taken to preserve the lateral intermuscular septum and its attachment to the periosteum of the radius on its lateral aspect. Medially a cuff of flexor pollicis longus and pronator quadratus is left attached to the area of bone to be taken. Many large deep muscular branches of the vascular pedicle must be carefully ligated or clipped, as these may cause excessive bleeding when the flap is revascularized. The bone is then removed with an electric saw; if necessary it can be shaped by subperiosteal osteotomy in one or two places. The donor area usually requires a spilt-skin graft. A plaster splint is applied for 3 weeks to protect the weakened radius.

Ulnar and fibula transfers

Lovie et al (1984) have described a forearm free flap based on the ulnar artery (Fig. 17.17): its utility is discussed on p. 21.86. The fibula free flap has advocates, but we have not found it helpful in facial reconstruction.

The second metatarsal transfer

This was first described by O'Brien et al (1979). The authors described this composite flap apparently at the suggestion of another prominent Australian microsurgeon, D. W. Robinson of Brisbane, whose help was acknowledged. The second metatarsal gains its blood supply from the dorsalis pedis system; the venous drainage is via one of the accompanying veins which communicates with the superficial long saphenous vein. The second metatarsal is removed with its articular surfaces; remnants of the small muscles are left attached to produce soft-tissue connections to the overlying skin paddle.

In the original report, a reconstruction was undertaken to restore the mandible after a gunshot wound. The central mandibular bone was missing as well as soft tissue. Facial vessels were used for the anastomosis; interosseous wire fixation was used at one end and a plate fixation at the other. Union was reported as sound. The contour of the mandible achieved with this technique was deficient and needed further augmentation to produce an aesthetic result. Other authors have reported their experience with this technique (MacLeod & Robinson 1982, Duncan et al 1985) and it has proved possible to give adequate chin prominence. MacLeod & Robinson (1982) believe that this transfer is the technique of choice for reconstructing the anterior mandible; the pedicle is long and the vessels of large diameter.

Iliac crest transfers

In recent years, vascularized iliac grafts have been accepted as one of the methods of choice for achieving a satisfactory reconstruction of the mandible, especially when larger bone segments are required (O'Brien et al 1979, Taylor et al 1979a, b, Taylor 1982, Salibian et al 1985, David & Tan 1979, David et al 1988). In our unit, this is indeed the technique of first choice and it has been used and developed in clinical practice for the last 15 years.

In our initial experience with microvascular jaw reconstruction, segments of iliac crest based on the superficial circumflex iliac artery (SCIA) were used. This vessel anastomoses with perforating arteries emerging from the iliac crest in the line of the external oblique attachment (Fig. 21.81). When raising this composite flap, the initial dissection is the same as that used for raising a free groin skin flap (p. 454). Attachments of the skin to the fascia are preserved to retain a periosteal blood supply to an estimated 2-cm-thick segment of bone. Thigh muscle fascia and the external and internal oblique muscles are then incised to expose the desired height of the iliac crest which is then sectioned. This composite flap is now rarely used because of the inadequacy of the bone segment that can be attached to the flap and the unpredictability of the vessel size. Retrograde blood flow to the underlying iliac crest via small vessels in the fascia may be inadequate to sustain a large segment of bone. The perforating vessels

from the deep circumflex iliac artery (DCIA) are more reliable. These vessels emerge 2–3 cm medial to the iliac crest to anastomose with branches of the SCIA, and an adequate cuff of external fascia and external and internal oblique muscles must be taken if blood flow is to be possible in either direction. With the advent of the DCIA flap it became possible to provide a large amount of vascularized iliac crest for reconstruction of extensive defects (Taylor et al 1979a,b) (Fig. 21.82). Further development of the technique enabled the ilium to be split, thus improving the cosmetic result in the hip. As this procedure has become our workhorse for mandibular reconstruction, it is described in detail.

The operation is planned so that the shape and size of the mandibular bone defect can be predetermined either from study of the dental model or from 3D CT reconstruction (Fig. 21.91), which has proved very helpful in preoperative

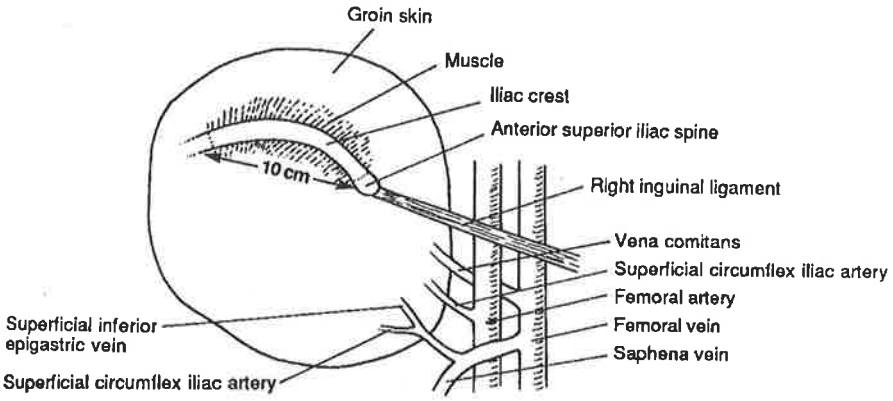


FIG. 21.81. ***Osteocutaneous flap from the right groin.*** *After O'Brien et al (1979), by courtesy of* British Journal of Plastic Surgery. *Also see Fig. 21.80 for its early application.*

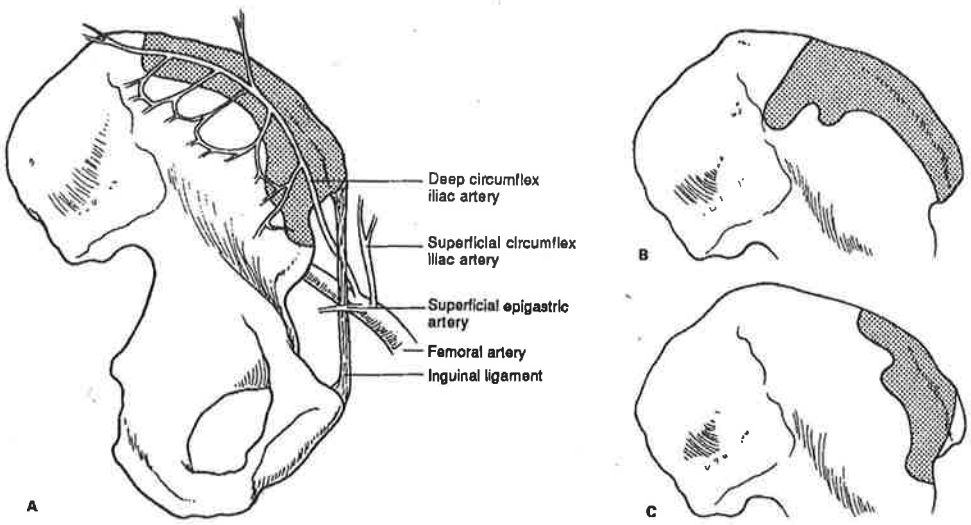


FIG. 21.82. ***Fashioning the deep circumflex iliac artery (full or split thickness) flap for mandibular reconstruction from the contralateral ilium.*** ***A*** *The bone with vessels superimposed.* ***B & C*** *Alternative methods.*

planning (Rose et al 1993). An acrylic template is made to assist in cutting out the exact shape of bone required for the graft. The appropriate intermaxillary fixation is determined in advance and a bite wafer is used to locate the mandibular fragments. Intraoperative organisation is carefully planned beforehand: usually two teams are deployed, one to prepare the jaw and the other to harvest the bone.

The team dissecting the jaw prepares the ground, recreates the defect and exposes the recipient vessels. Simultaneously, the DCIA flap is harvested. The DCIA runs a course 1 cm above the inguinal ligament from the external iliac artery towards the anterior superior iliac spine, deep to the transversalis fascia. The markings for a skin paddle are made over the anterior superior iliac spine and the iliac crest, usually as a lentiform shape, the medial end of which can be continued to the origin of the DCIA. The skin incision starts medially, and the external oblique aponeurosis is exposed; its fibres are split parallel to and 1 cm above the inguinal ligament. The internal oblique muscle fibres are cut transversely, and the DCIA is then identified. This is usually easy, first by palpation and then by sharp dissection through the transversalis fascia. The vessels are then followed medially to the external iliac artery and its accompanying vein, and laterally towards the anterior superior iliac spine. The ascending branch of the DCIA is encountered at a variable position in this dissection, and is divided. The main vessel lies between the iliacus and transversalis fascia, ~2 cm below the iliac crest. The skin paddle incisions are then completed and deepened to the aponeurosis of the external oblique muscle, which is divided 2–3 cm medial to the crest. Care is taken to preserve any perforating vessels appearing in the vicinity. The dissection is further deepened until the DCIA is seen on the iliacus; the iliacus is then incised and pushed away medially, leaving the periosteum of the inner table of the ilium intact. Further laterally and posteriorly, the external oblique aponeurosis becomes muscular, and a small (2 cm) cuff of the muscle is taken with the flap as more perforators often come through in this area. The line for splitting the iliac crest is marked by incising about midway along the top of the iliac crest; periosteal elevators are then used to push away the muscular and fibrous attachments from the lateral half of the iliac crest, the medial attachments being preserved. Near the anterior superior iliac spine, it is often necessary to divide the lateral cutaneous nerve of the thigh, though it is sometimes possible to identify and save this nerve when the DCIA pedicle is divided from the external iliac vessels.

The segment of the ilium is harvested according to the shape and size of the mandibular defect. Manchester's inspiration (Manchester, 1972) has influenced Taylor and others (Fig. 21.83) in the designing of the bone graft to be cut from the iliac crest. With the wide exposure obtained, the acrylic template is placed on the medial side of the iliac crest and the area to be resected is delineated with a marking pencil after the periosteum has been stripped away from the part of the ilium that will not be harvested. Often, the amount of iliac bone to be taken is estimated from measurement of the length and height of the bone defect, by now exposed by the other surgical team, taking into account possible osteotomy and reshaping. Similarly, the size and shape of the soft-tissue elements of the graft will have been determined by the need for, soft-tissue, external skin and mucosal replacement.

The iliac crest is split along its rim with a reciprocating saw (Fig. 21.83), and along the outline of the graft on the inner table With either an oscillating or a reciprocal saw. The final stages of removing the bone graft are facilitated by cuts with curved or straight osteotomes. It is helpful to divide the vascular pedicle before the saws are used; at all stages of the operation this pedicle must be safeguarded. The vessels are clamped with Ligaclips® and divided between the clips. If it is intended to use the grafted bone to support osseo-integrated implants, then the harvested split iliac crest must have a minimum Width of 8 mm at the projected upper border, which is usually the deeper part of the crest, So that the

splitting has to be executed with care to preserve that width. In most cases, the height of the mandibular replacement is 2 cm.

Closure of the donor site is carried out with careful approximation of the internal oblique muscle to the residual fascial attachments on the outer border of the iliac crest; the external oblique aponeurosis is attached to the superior

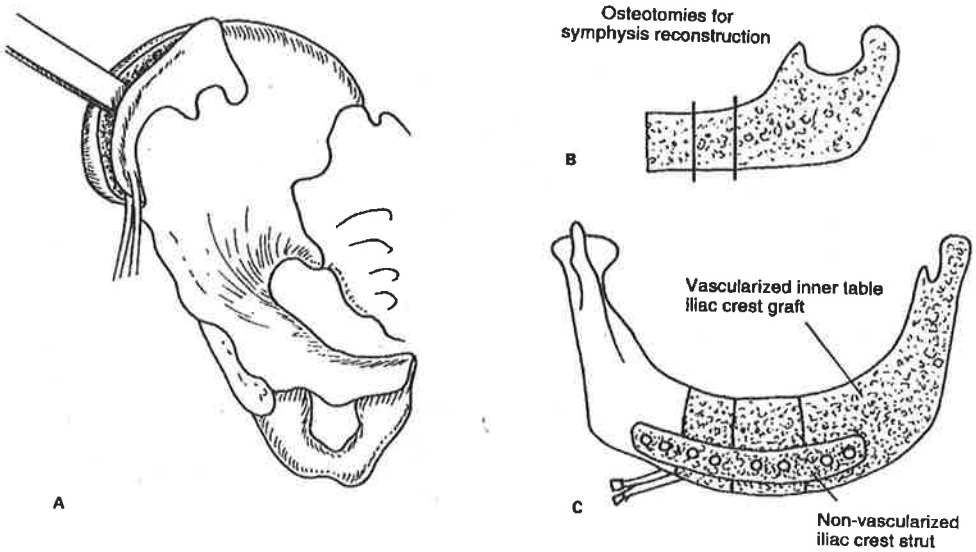


FIG. 21.83. ***Contouring the iliac crest graft for mandibular reconstruction.*** ***A*** *The iliac crest is split at its rim with a reciprocating saw, and the cut is completed with an osteotome which will lever the fragment away gently with the overlying soft tissue and vessels intact.* ***B*** *Saw cuts are made through the substance of the graft subperiosteally so that the desired curvature can be produced.* ***C*** *Reconstruction of the symphysis is achieved with vertical osteotomies, preserving the periosteum on the lingual side. Non-vascularized iliac crest can be shaped and placed on the facial side to secure the segments and augment the chin prominence. If the vertical osteotomies have opened they can be filled with cancellous bone.*

gluteal fascia and the fascia over the tensor fasciae latae with 2/0 PDS or Maxon®. Before closure, any sharp spicules of bone on the ilium are removed or smoothed by burring, as perforation of large bowel has been known to occur after this operation. This careful closure of the oblique layer should prevent herniation of large bowel through a weak point in the abdominal wall.

When the desired graft is removed, together with its vascular pedicle, it may be shaped by subperiosteal osteotomy. The use of a burr in sculpting the graft is very dangerous—one slip may avulse the pedicle in a split second! The shaping of the graft is done on a side table; the surgeon is helped by the prepared template. Osteotomies can be made in the graft; the symphysis can be built from the centre of the crest or the whole hemimandible may be shaped together with its condyle. The osteotomies performed to shape the flap leave the inner periosteum intact; the osteotomized bone is fixed with miniplates and screws. Strips of non-vascularized iliac bone can be added to secure the segments and shape the bone grafts (Fig. 21.84). Additional bone can be placed on the flap or into the osteotomies as they are opened by bending; the additional grafts are fixed with long shaped titanium miniplates. The graft is so easily shaped that either step joints or end-to-end apposition can be effected, and the ends can be very strongly secured with plates and screws to obviate the need for intermaxillary fixation. In cases of anterior mandibular replacement, the graft must be made secure against downward rotation. If the infrahyoid or other upper cervical muscles still exist they can be attached to the bone graft. Where a cuff of suprahyoid musculature still exists it can be anchored to the muscle attached to

the ilium to give anterior support to the tongue. If epithelial replacement is needed, the skin of the vascularized transfer can be used as oral lining or as additional skin cover. Careful planning ensures that the vascular pedicle lies in appropriate juxtaposition to the recipient vessels.

When intraoral lining is required the split iliac crest graft may be combined with a free forearm flap. The ulnar rather than the radial forearm flap is preferred as it provides more hairless skin and the donor site is less conspicuous (Lovie et

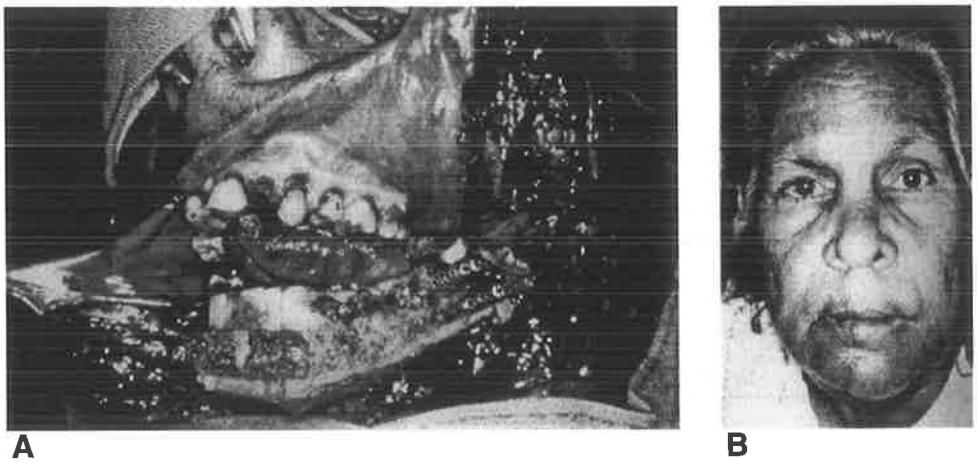


FIG. 21.84. ***Iliac crest graft for mandibular reconstruction.*** ***A*** *Reconstructed mandible with strips of non-vascularized bone overlying the vascularized bone to enhance contour.* ***B*** *Post-operative appearance with a very acceptable mandibular contour.*

al 1984). This thin flap helps to recreate the contours of the floor of the mouth, the labial sulcus and the alveolar margin; this may facilitate the fitting of dentures. It is often necessary to resect some of the soft tissue and reapply it to the underlying bone or to resect the skin from the flap and skin graft the underlying soft tissue as a secondary procedure prior to the fitting of a superstructure to support osseo-integrated implants (Fig. 21.91).

After the bone graft has been fixed with miniplates and the lining flaps stitched in place, the microvascular anastomoses are carried out. For arterial supply of the flap, the facial artery may be used, or the superior thyroid artery; for venous drainage, the anterior facial vein or the superior thyroid vein may be selected, though any sizeable venous channel in the submandibular region can be used. Rarely, we have used the superficial temporal artery and its accompanying vein. The choice of vessel depends largely on what is available, and in post-traumatic mandibular defects, the regional blood supply may have been devastated by the causative missile injury. It could be helpful to visualise the vessels by preoperative external carotid angiography; however, we have never had occasion to do this. Nor have we been prevented from performing a DCIA anastomosis because of destruction of recipient arteries, even in cases of wartime missile mutilations, though occasionally it has been necessary to use a vein graft to obtain arterial supply from a distant vessel. The microvascular anastomoses are performed with 10/0 nylon sutures on a 70 mm tapered needle. Interrupted sutures are first placed on the posterior walls of the approximated vessels, and the suture line is then brought forward around the circumference of the vessels to meet in the midline anteriorly. In the past, we began with stay sutures on each side, and completed the anastomosis by rotating the vessels in microvascular clamps, but we now prefer the sequential procedure. When the anastomoses are completed, the skin wounds are closed.

In massive defects, secondary surgery is usually necessary to trim excess soft tissue or skin. Sometimes soft tissue can be advanced into areas of contour

deficiency. Excess bone is easily sculpted away. These secondary procedures are best done after 3–6 months; then, even if the vascular pedicle is inadvertently cut, the bone and soft tissues will survive the secondary surgery provided that this is not excessive or associated with too much dissection of soft tissues.

Complications and results

The success of jaw reconstruction supported by microvascular anastomosis has transformed expectations of surgical reconstruction of this area. Flap failure has been rare in our experience (David et al 1988). In more than 100 of these mandibular reconstructions performed for traumatic, neoplastic or developmental conditions, only four flaps have undergone complete necrosis; in two others, there has been partial bone necrosis and in one there was necrosis confined to the skin.

In our view, all large post-traumatic mandibular defects are best treated by the DCIA flap. In our recent experience of patients wounded in the Iran–Iraq war, we used microvascular free flaps for both upper and lower jaw defects; of special value was the capacity of the free flap to add soft-tissue cover for bone grafts and to maintain grafts without later resorption.

Although the use of DCIA microvascularized grafts seems to us the treatment of choice for massive defects of the mandible, it must be said that good results have been achieved by other methods. The work of Taher (1990), cited above, and other surgeons has shown that cancellous bone supported in titanium trays will often give a satisfactory aesthetic outcome.

Reconstruction of Maxillary Defects

Assessment

Nowhere is multidisciplinary planning more relevant than in this area of reconstruction. The radiological assessment, particularly sophisticated 3D imaging of the defect, allows the missing bony maxilla to be considered as part of the organ complex involving the orbit, nose and mouth. The multidisciplinary team must not only contain surgeons with maxillofacial and microvascular expertise but also dental and prosthetic experts who can argue the case for prosthetic replacement of defects. The modern technology of osseo-integrated implantation now brings these specialists closer together; surgeons can provide not only tissue for total reconstruction but also tissue into which osseo-integrated implants can be placed to give more adequate support for whatever prosthesis may be necessary.

Prosthetic correction

Some upper jaw defects can treated by obturation of the defect with a prosthesis, constructed of silicone rubber, acrylic or a combination of such soft and hard materials; the techniques are well described by Conroy (1985). The prosthesis is designed to engage in a palatal defect or other ledge or pocket in the damaged maxilla; the prosthesis may be extended to support the anterior cheek and the orbit, sometimes by fitting in several component parts. Dentures may be fitted to the prosthesis. In mastication, the forces are considerable and the supporting tissues may suffer; surgical attention has been focused on providing a suitable lining to the defect to withstand the pressure of the prosthesis. Maxillary prostheses need to be changed with the changes in the supporting tissues which result from healing and ageing.

It is possible that the potential benefits of surgical replacement of the upper jaw have been disregarded in the past, because the defects were most often created by excision of the maxilla for cancer and there was a subsequent need to inspect the area for recurrent disease: hence the preference for prosthetic replacement.

Additional problems arose from the connection of the sinus cavities, oral cavity, orbital cavity and nasal cavity once the maxilla was removed. Such complicated anatomy made this area of surgical reconstruction even more difficult than that of the mandible.

Surgical correction

Simple onlay bone grafts can be used to restore contour to a maxilla deficient in shape. But more difficult problems arise when the entire framework structure needs to be restored to support the eyeball above and the teeth and roof of mouth below. The inherent difficulty in performing this type of reconstruction from autogenous tissue has always been to provide soft-tissue cover on both sides of the grafted bone.

Smaller wounds of the alveolus and hard palate may be dealt with secondarily by mobilising local mucoperiosteal flaps and inserting cancellous bone grafts, much as when a cleft lip and palate demands prosthetic bone grafting because the alveolus has resorbed due to loss of dentition and ageing. Operations of this type are often performed with a view to future osseo-integrated implantation of the area to support dental or orbital prostheses (see below). Obwegeser (1973) described how the hard palate and alveolar components can be reconstructed in two stages. In the first, a two-layer soft-tissue closure is provided by buccal flaps: (i) from the nasal septum above joined to flaps mobilized from the rim of the defect, and (ii) large rotation flaps from the remaining oral lining from below. At a second stage bone is inserted between the two layers and fixed to the remnants of the bony palate. This method should be kept in mind because trauma has no respect for time, place and the availability of surgical expertise: all techniques, old and new, may need to be used at some time. However, this technique was described for a pre-planned maxillectomy and not for a traumatic case.

Losses of major segments of the midfacial skeleton are not common except as a result of high velocity missile injuries, whether military or suicidal (Moore et al 1991). This represents a vastly different scenario from that which pertains after selective dissection of the maxilla for cancer. The defect reflects the missile cavity (p. 115) and may extend from the base of the skull through the orbits, nose and both jaws (Fig. 21.85). Reconstruction of such major composite tissue loss demands the provision of adequate intraoral soft-tissue coverage together with restoration of the midfacial skeleton. Modern microvascular composite free-tissue transfers and osseomuscular flaps provide the basis of reconstitution of the missing elements with autogenous tissue. In this way, efforts to restore form and function (including aesthetic balance, speech, mastication, swallowing and occlusion) are maximized.

The strategy of repair is to reposition the existing tissues and to recreate the defect according to the fundamental principles of plastic surgery, and then to transfer vascularized autogenous tissue, if possible at one stage, to rebuild the bony structures of the orbit, alveolus, anterior maxilla together with soft tissue, lining and cover.

Timing

If one is seeing such an injury de novo then the timing of the secondary reconstruction poses an interesting question. While it is not appropriate to do this reconstruction as part of the initial surgical intervention it should be performed before really dense fibrosis has developed, as there is nothing worse for the surgeon than trying to recreate the defect from what may be a 'porridge' of scar, bone fragments and foreign material.

Surgical techniques

It may appear trite advice, but it is important to stress that the deeper reconstructions must be done first and the more superficial procedures such as scar revision of the face and reconstruction of the orbicularis oris and nasal

pyramid must come later. These reconstructions used to involve many stages over a long period of time. The advent of microvascular tissue transfer has cut the stages down (Fig. 21.86). The techniques have already been described in the section on orbital and mandibular reconstruction: it is a matter of how to organize them. Cases of avulsive injury in the midface are so diverse in their anatomical deficiencies that it is impossible to give a stepwise guide to their repair: even more than in the mandibular replacements, each case is unique and requires its own operative sequence.

The orbital floors can be reconstructed from above using the temporalis muscles to support the globe on their own or with bone attached as in the pedicled calvarial flap (p. 621). Gillies (1920) described the use of the temporalis muscle for repair in this region, particularly for defects in the orbital floor. McCarthy & Zide (1984) described a calvarial bone flap pedicled on the temporalis muscle to be used to reconstruct the orbit and zygoma (Fig. 21.78).

The microvascular osseocutaneous flap has now assumed a prime role in the restoration of the upper jaw and indeed the rest of the face in massive post-traumatic deformities. The alveolar arch can be reconstructed by microvascular techniques; the buttresses between the zygoma and the palate and alveolar arch can often be rebuilt with free bone. Once this is achieved, it is possible at another stage to reshape the reconstituted tissue. All the osseomyocutaneous flaps described for reconstruction of the mandible can be used in maxillary reconstruction. Reference has already been made to Swartz et al (1986) and the use of the scapula flap. When free flaps are combined with rigid fixation, osseointegrated implants, CAD/CAM produced titanium attachments and tissue expansion, we are able to advance the reconstruction of this difficult area very significantly.

When the transferred bone is supported by microvascular anastomosis it will provide the arch for the alveolus and the supporting strut to the base of skull; if sufficient bone is not readily available from the same flap, the orbital floor can be reconstructed either by ordinary bone grafting if the soft-tissue support is still intact, or by the above-mentioned osteomuscular calvarial flap.

Osseo–Integrated Implants In Secondary Reconstruction

Osseo-integration

Titanium and hydroxyapatite can become integrated into bone, forming a solid and permanent structure into which can be inserted pegs which pass through skin or mucosa and form a basis for prosthetic superstructures which may be permanent or detachable. The biocompatibility of these substances is discussed in Chapter 5 (p. 155). However, biocompatibility is not the only factor relevant in the process of osseo-integration. The acceptance of the osseo-integrated implant depends on the viability of the bone, and this can be prejudiced by heat. Above 56°C, irreversible bone damage occurs; alkaline phosphatase and other enzymes are denatured, and bone synthesis is prevented, thus preventing the process of osseo-integration. Mechanical force effects are also important; if the implant is loaded during the process of osseo-integration, the balance between bone deposition and resorption is disturbed, new bone is not laid down, and the implant is not integrated. In the maxilla, a minimum healing time of 6 months is required, while in the mandible a period of three months may be sufficient. This variance in healing times is related to differences in bone quality.

Techniques for intraoral implantation

In post-traumatic cases, the alveolus may need to be reconstructed with bone grafts to ensure suitable depth and width of bone to receive the metal implant.

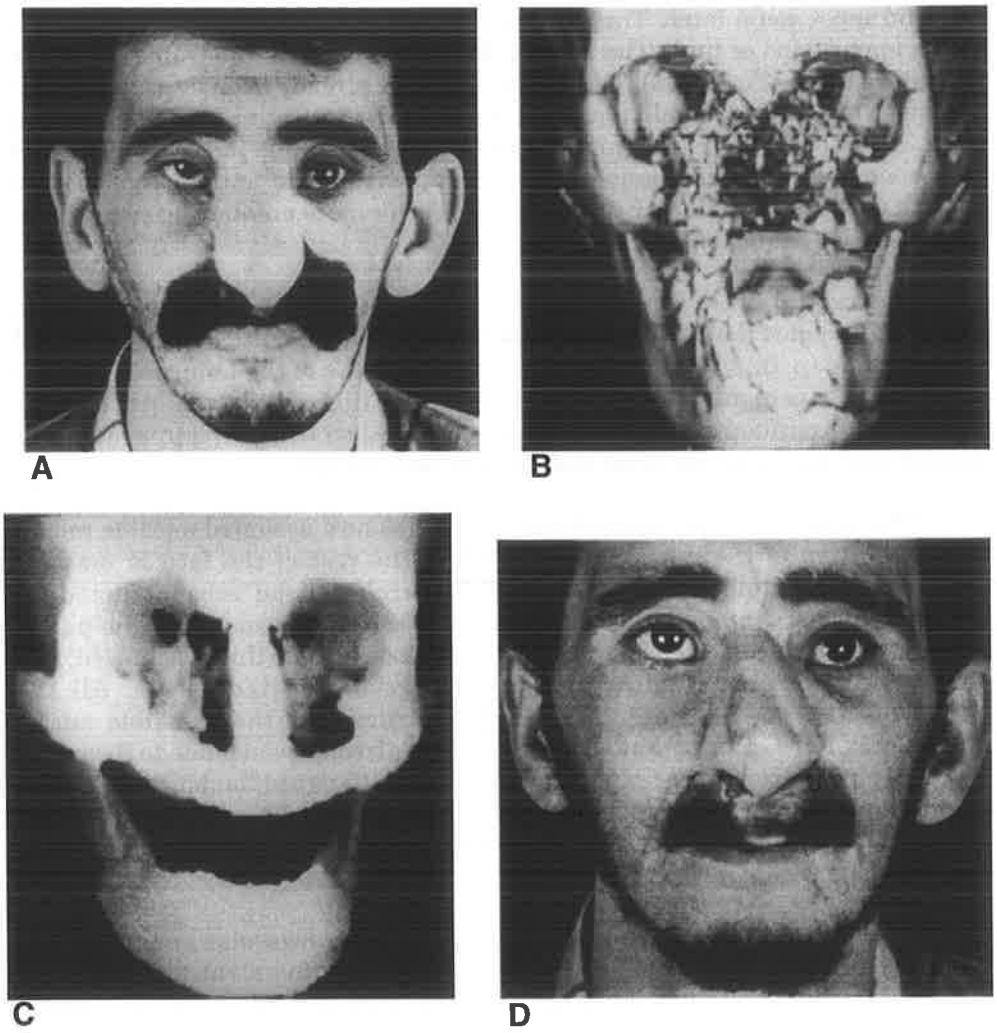


FIG. 21.85. ***Avulsive midfacial injuries.*** *A soldier suffered a missile injury in the middle third of the face.* ***A*** *The nose has been reconstructed from a distant flap and the upper lip from a bipedicled scalp flap.* ***B*** *Three-dimensional reconstruction shows the massive bony defect affecting most of the maxilla to the base of skull.* ***C*** *The central buttress of the nose and the maxillary arch were reconstructed with a microvascular free flap to give support for the middle third of the face and provide a basis for further prosthetic restoration.* ***D*** *Postoperative appearance.*

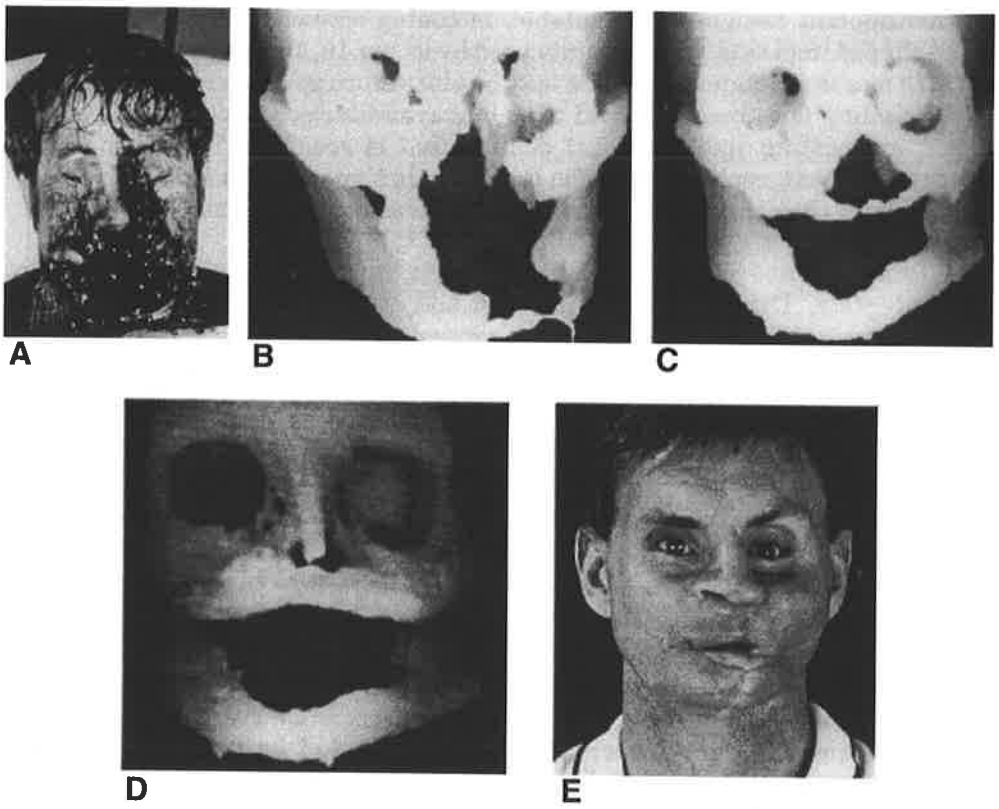


FIG. 21.86. ***Reconstruction of massive avulsive injures.*** *A young man suffered a shotgun wound which destroyed the mandible from the right ; of the symphysis to the left angle, carved a swathe through the upper jaw leaving a small remnant on the right and a tiny bit of buttress on the left, removed the left nasal bone, avulsed the soft tissue to the glabellar region, and destroyed the left eye. He had a retroclival subarachnoid tear and a number of pellets in the frontal intracranial region. Cardiovascular stability was gained and an urgent tracheostomy was done. A subdural catheter was inserted for intracranial pressure manometry. The mandible was stabilised with a K-wire, the wounds were debrided, and primary closure was performed where possible. Several days later his left eye was enucleated and an implant was placed into the socket. The first reconstruct on his mandible was performed using a vascularized iliac crest, with jejunum employed for restoration of the oral lining. 5 months later he underwent further reconstruction of his lower lip by cheek transfer, and rib grafting to his maxilla inserted under the mucosal lining. The first 6 months provided this man with some stability of his lower jaw, lining of the nose and mouth, and a tracheostomy. At the end of this he was able to be nourished and had an intact integument. The jejunum gave a very unsatisfactory florid lining which wept inappropriately and looked dreadful.* ***A*** *Initial presentation with a massive disruption of the face. The blast has cut its way through the mandible and removed a good deal of the middle third of the face.* ***B*** *CT scan shows the situation with the temporary spacer of the mandible and the absence of the upper jaw and the defect extending into the left orbit.* ***C*** *Subsequent CT scan shows the mandible reconstructed and a rib graft supported by bowel mucosa in the upper jaw. 2 years later he had insertion of tissue expanders in both cheeks and a forehead flap to reconstruct the left side of the nose.* ***D*** *A third CT scan shows the stage of removal of the intraoral jejunal tissue and replacement of the upper jaw with superficial circumflex iliac artery flap with overlying skin. He required further fashioning of flaps and releasing of scar inside his mouth, and further widening of the nostrils.* ***E*** *His current situation is reflected with the iatrogenic scar on the left forehead from whence the nasal reconstruction was taken, a prosthetic left eye, a misshapen mouth with deficient orbicularis oris muscle on the left side, and a deficiency of tissue on the left alar base. He is capable of feeding and speaking.*

Various implant designs are available, including screw implants and simple bullet-shaped implants. The implants used by us are 10, 13 and 16 mm in length and 3.75 mm in diameter. Implants may be placed into previously reconstructed jaws including jaws reconstructed with microvascularized bone grafts. A high torque, low cutting speed surgical drilling unit is required. To prevent bone damage from heat, copious irrigation is needed; in some drill systems, the longer drills embody the capacity of internal irrigation at the cutting surface.

Implants are allowed the appropriate minimum period of time to osseo-integrate. They are then uncovered and attachments are made for the superstructure. Where wider and more extensive prostheses are required and where solid bone is at a distance from the site of the necessary prosthesis, an appropriate structure can be milled in titanium from CAD/CAM planning; this is screwed into the bone, and when covered with mucoperiosteum will achieve enough osseo-integration to support a prosthesis (Figs 21.87 and 21.91).

Techniques for extraoral implantation

Extraoral implants may be established to support ocular, nasal, or auricular prostheses. In the past, such prostheses — which have a long history (Fig. 1. 11) — were mounted on a spectacle frame or secured to the skin by glues or adhesive tape. These traditional methods of fixation were not very satisfactory; prostheses were easily dislodged and skin adhesives were associated with discoloration of the prosthesis. They have been partially superseded by the techniques of osseo-integration described by Branemark (Albrektsson et al 1987). Osseo-integrated implants provide very stable prosthetic retention with minimal inconvenience. Nevertheless, older methods of prosthetic correction of a facial defect still have roles, to give temporary aesthetic cover before a definitive procedure can be done, and where individuals or societies are unable to meet the cost of osseo-integrated implants.

Implantation is routinely performed in two stages. In the first, the implant is inserted into bone and covered with soft tissue, which may need to be thinned. The second stage comes 3–6 months later, when the implant is exposed and

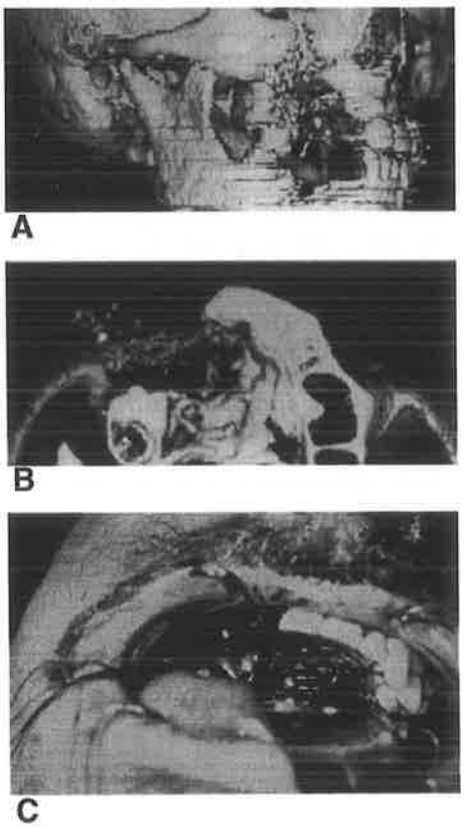


FIG. 21.87. ***Custom prosthetic fabrication from CT data.*** ***A*** *3D CT reconstruction of a 39-year-old male who had suffered a gun-shot wound to the right side of the face.* ***B*** *Reconstruction viewed from the palate. The extensive nature of the defect is demonstrated.* ***C*** *Clinical insertion of custom made titanium prosthesis. The prosthesis was constructed from a solid model produced from the patient's CT scan.*

attached to the transcutaneous abutment to which the prosthesis is attached. The bone in the usual sites for extraoral implants (orbit, midface, temporal bone) accepts implants well and the necessary duration of the subcutaneous implantation is often reduced. This apart, the techniques of implant insertion and exposure are as for intraoral implantation. Prostheses are attached by various devices, such as magnets, clips and O-rings.

The overall success rate of extraoral osseo-integration approximates to that achieved with intraoral implants. Bony integration with pure titanium and with hydroxyapatite-coated titanium implants is routinely successful, the only exception in our hands being seen when adequate soft-tissue cover was not achieved in the initial implantation (Moore & Hawker 1993). In the longer term, low grade inflammation sometimes develops around the percutaneous abutment; this is managed by regular cleaning and topical antibiotics, and has not led to loosening of the implant in the bone (McComb 1993, Moore & Hawker 1993).

Applications of this form of surgical prosthetic reconstruction are illustrated by the cases described in Figs 21.88–21.90.

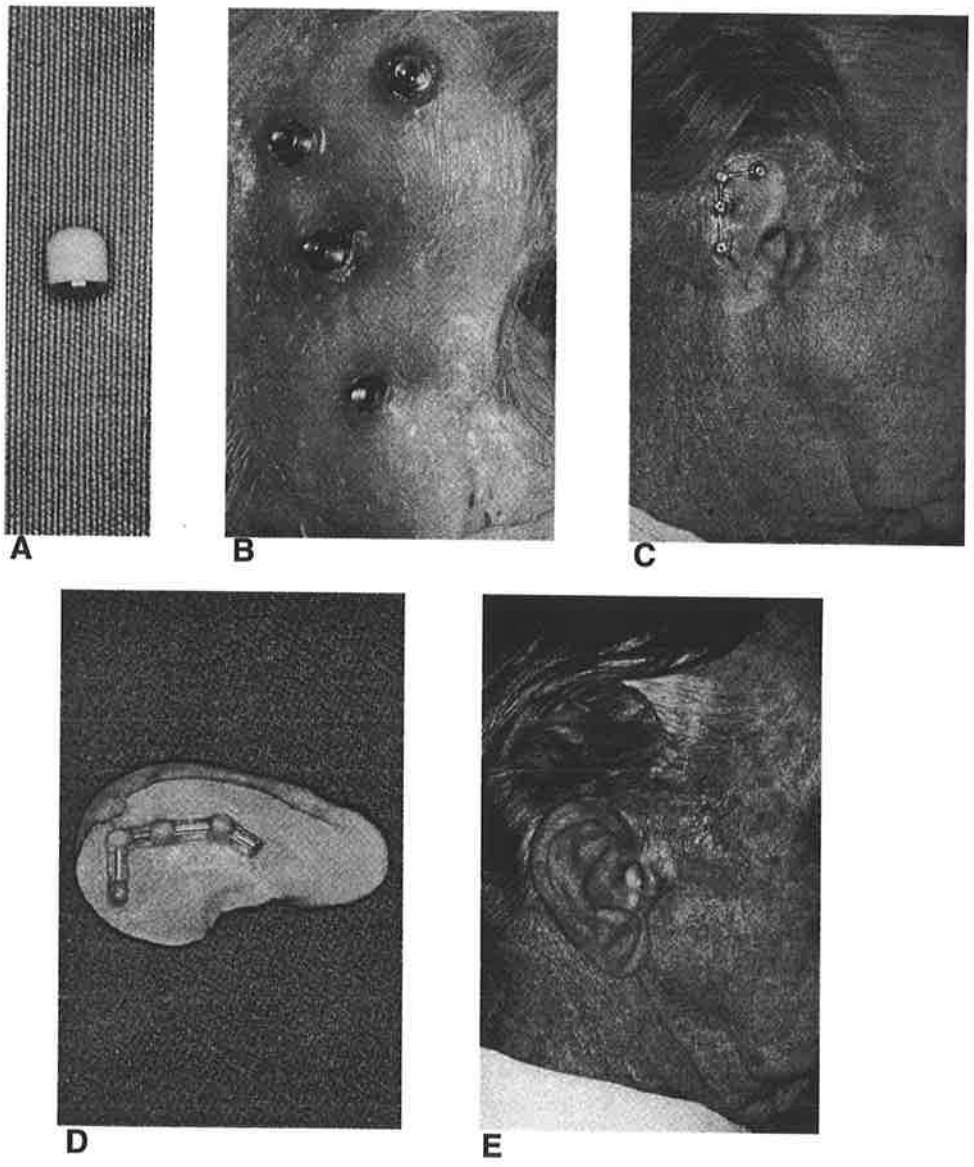


FIG. 21.88. ***Implant-borne auricular prosthesis.*** ***A*** *Hydroxyapatite-coated implant.* ***B*** *Appearance after attachment of percutaneous abutments.* ***C*** *Bar clip.* ***D*** *Medial surface of auricular prosthesis.* ***E*** *Auricular prosthesis in place.*

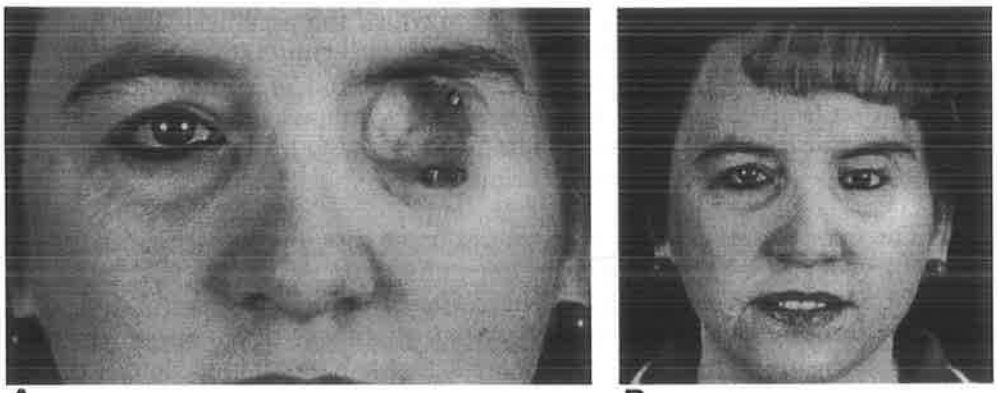


FIG. 21.89. ***Implant-borne orbital prosthesis.*** ***A*** *Implant placement in the left orbit after exenteration.* ***B*** *Orbital prosthesis in place with magnet fixation.*

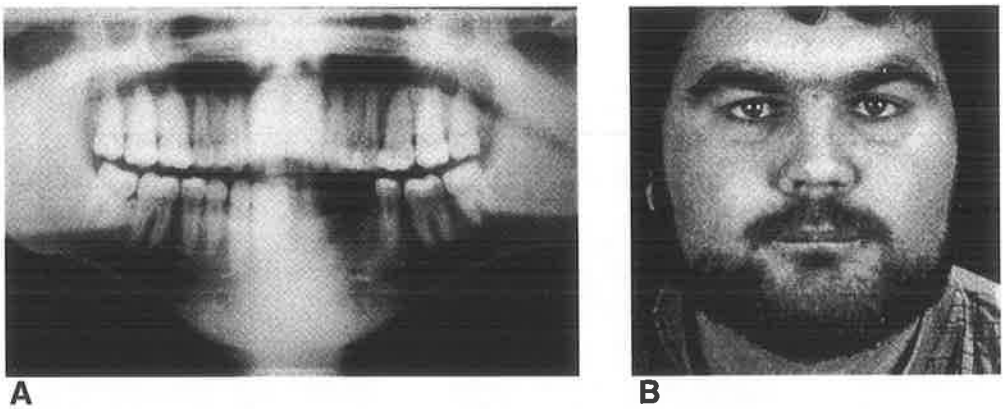


FIG. 21.90. ***Osseo-integrated implants for dental rehabilitation.*** *A young man had the anterior part of his mandible removed for a squamous carcinoma arising in odontogenic epithelium. He had a deep circumflex iliac artery replacement which was then fitted with osseo-integrated implants.* ***A*** *The mandible before excision.* ***B*** *Appearance following the mandibular excision and DCIA reconstruction.*

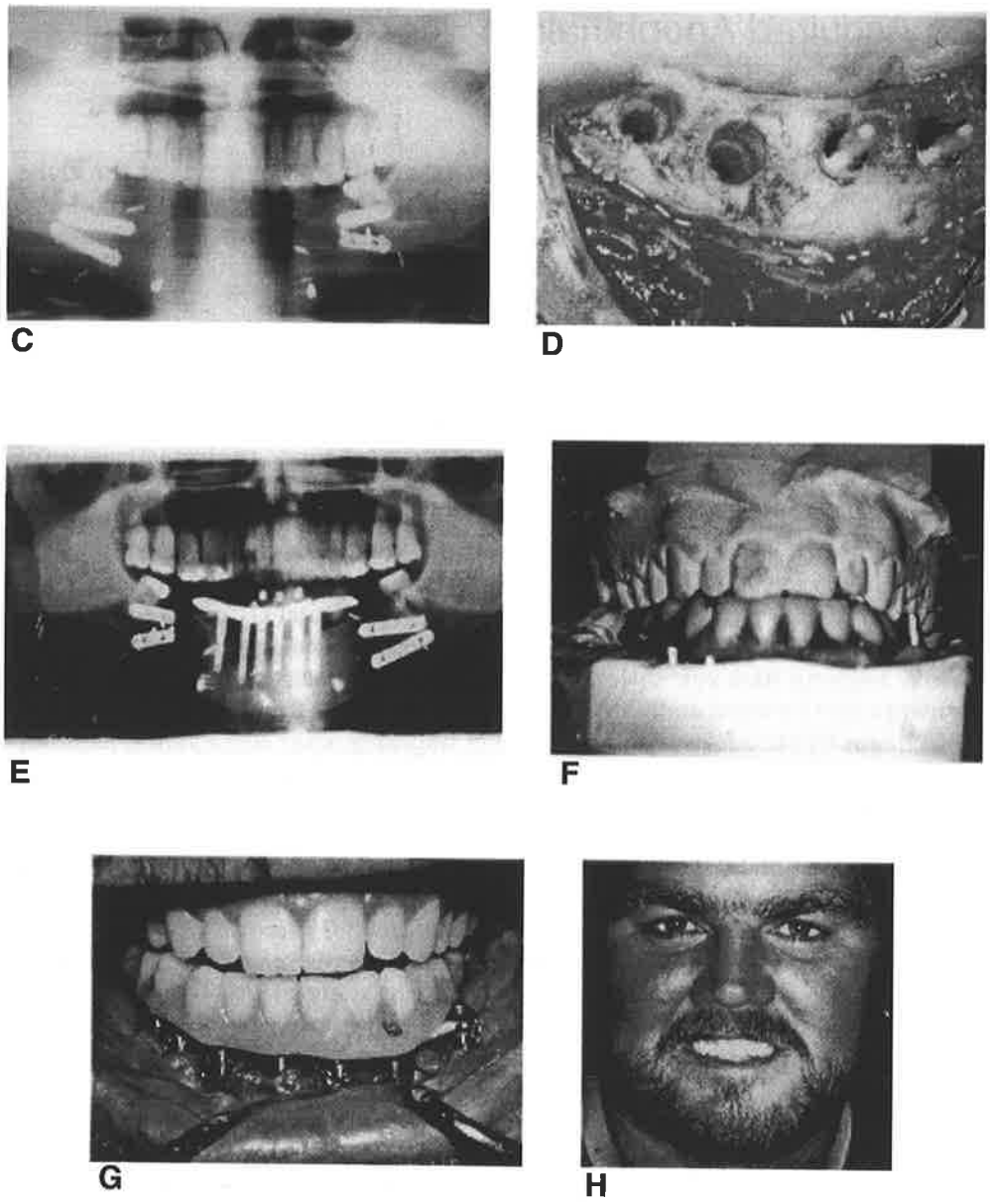


FIG. 21.90. ***Osseo-integrated implants for dental rehabilitation.*** *A young man who had the anterior part of his mandible removed for a squamous carcinoma arising in odontogenic epithelium. He had a deep circumflex iliac artery replacement which was then fitted with osseo-integrated implants.* ***C*** *Orthopantomogram after reconstruction.* ***D*** *The process of osseo-integration.* ***E*** *Osseo-integrated implants with superstructure.* ***F*** *Model planning of the prosthesis.* ***G*** *Prosthesis in place.* ***H*** *The patient fully reconstructed.*

The Acquired Anophthalmic Socket

Surgical pathology

The aesthetic and psychological problems that result from removal of the globe or destruction of orbital contents depend on the severity of the initial wound and the quality and extent of the treatment given at that time. It is not uncommon, especially in cases from remote places, for these problems to present essentially untreated many months or years after the injury.

Secondary changes occur in an anophthalmic socket, and these may lead to difficulties in fitting and retaining an ocular prosthesis and in associated problems with the motility of that prosthesis. Enophthalmos, superior sulcus deepening and lower-lid laxity are several of the most common characteristics of the anophthalmic socket (Soil 1982). Studies done using monkeys as an experimental model suggest that the contributory factors most likely to cause these complications include insufficient volume replacement by using an ocular implant that is too small, mechanical alterations and contractile scar formation (Kronish et al 1990). Smit et al (1990) used high resolution CT scans to evaluate the orbits of anophthalmic patients in whom there was no ocular implant. These showed sagging and retraction of the superior muscle complex as well as downward and forward distribution of orbital fat. These findings help to explain the frequent tipping forward of the base of implants after some years. Another problem associated with the anophthalmic orbit is chronic mucous discharge from goblet cells in remnants of the conjunctive.

Where the globe itself has been damaged and judged to be useless it may be removed by enucleation or evisceration; these operations are described in Chapter 14. This is done as soon as the indications for removal are established and accepted by the patient. A number of manoeuvres may be undertaken to ensure function of the muscles and retention of the space in which the prosthesis will be placed; increasingly, we are taking the opportunity to insert a coralline implant at the primary operation (p. 419) to mount a prosthesis. However, where there is more extensive orbital injury, with destruction of the globe and also damage or displacement of orbital contents, then there may be a significant deficiency of soft tissue within the orbit. Soft tissues are likely to be replaced by scar, which may also extend to the conjunctival sac and lids. Where there is additional damage to the bony orbit, the orbital contents may escape into the maxillary antrum, temporal fossa or into the ethmoid sinus and the orbital rim and walls may be widely displaced. Displacements affect the lids and adnexae even if these are not themselves damaged (Fig. 16.1).

Assessment

Close liaison between surgeon, ophthalmologist and prosthetist is necessary. When there has been a planned sequence from initial injury to reconstruction by the same team this is relatively easy; when the patient is referred from elsewhere with established deformity and secondary psychological problems, then a very careful multidisciplinary assessment is required. The loss of an eye from trauma can produce a lifelong psychological disability. The technical challenges of producing a satisfactory artificial eye and normal appearing eyelid structures are often not met from the patient's point of view (Perry 1991) and the initial evaluation must include a careful discussion of the patient's wishes and expectations.

Appropriate settings of the CT scan will show not only the bony orbit but also the soft-tissue remnants; from these images, the relationship between soft tissues and bone can be accurately gauged (Fig. 21.91).

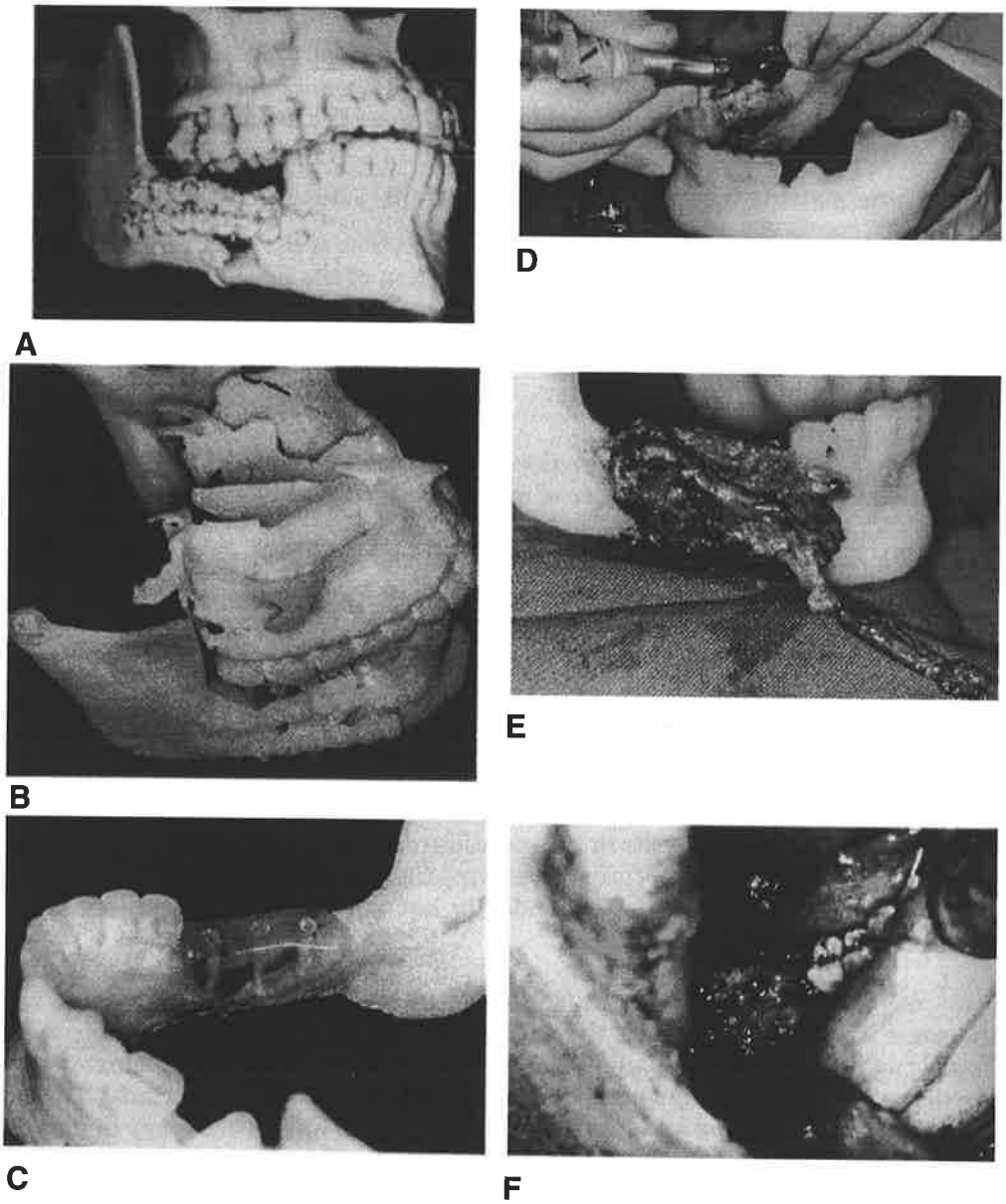


FIG. 21.91. ***Osseointegrated implants in a vascularized bone graft.*** *A fracture of the body of the mandible was complicated by bone loss both from the trauma and from subsequent infection.* ***A*** *The 3D CT scan shows the bone defect on the tight side.* ***B*** *A three dimensional model milled in nylon has been produced, here seen obliquely from above in articulation with the maxilla.* ***C*** *The planned bone graft is milled in acrylic and the precise sites for the osseointegrated implants are defined on this model. A titanium support has been produced for the lower border.* ***D*** *Vascularized ilium is cut to the exact shape and the implants placed into position according to the template.* ***E*** *The vascularized flap on the model.* ***F*** *An intraoral view of the osseointegrated implants in their correct position prior to closure.*

Management

Where one is presented with a secondary orbital deformity of large proportions, previously untreated or inadequately treated, then the surgical requirements are:

1. To reconstruct the bony orbit, rim and walls— described in Chapter 11
2. To fill the orbit adequately to ensure projection of the artificial globe and consequently to provide sufficient projection of the lids
3. To reconstruct the conjunctival sac to contain the prosthesis
4. To reconstruct the eyelids if this is necessary.

Filling the orbit
To provide adequate forward projection of a prosthesis the orbital cavity needs to be filled and there are a number of manoeuvres to do this.

The temporalis flap. The anterior portion of the temporalis muscle can be mobilized and passed through a hole burred in the lateral orbital wall. This manoeuvre is effective in the exenterated orbit, where the flap may be covered with lid skin or skin graft, and also where the orbital contents are more or less intact, when the flap is placed behind the conjunctive to bring the conjunctival sac forward. The technique is similar to that for mobilization of the temporalis muscle in TMJ ankylosis. The hole burred in the lateral orbital wall can be made with a large acrylic burr. The temporalis muscle is sutured to adjacent soft tissue or if necessary to the bone through a few burrholes passed in the orbital rim. However, there are drawbacks in this procedure. Ultimately the flap shrinks as the muscle becomes fibrotic. Moreover, moving the flap leaves a residual temporal hollowing which may be filled secondarily but still remains something of a deformity.

Bone or cartilage grafts. These can be inserted posteriorly to achieve the same effect, particularly if the walls of the orbit have been reconstructed. Bone is subject to resorption and volume changes may occur over the next year. Cartilage from the rib may be diced and packed into the orbit posteriorly. These grafts are quite effective and less likely to resorb, but have a tendency to move.

Vascularized flap. With more extensive orbital damage, the reconstruction can be achieved with a free vascularized flap from the forearm or lateral upper arm (p. 618) (Fig. 21.93).

Filling the orbit continues to be one of the most difficult problems in this area of reconstruction, particularly where there has been previous contraction resulting from fibrosis.

Reconstructing the conjunctiva
The conjunctival sac may require expansion to form adequate fornices above and below in which an appropriate prosthesis can be placed. This is done by a combination of grafting and long-term obturation. The appropriate substances are split skin, full-thickness skin or buccal mucosa. Split skin of course takes better but is subject to further fibrosis, and long-term obturation is necessary. Full-thickness skin often does not take as well and may indeed be too thick; it may lead to problems of difficulty in cleaning. The ideal substance is buccal mucosa, which is self-cleaning; unfortunately the amount available from the cheek is limited.

The operation involves wide release of the scar under the lids, down to the orbital margins, with meticulous haemostasis. The grafts are inserted and sutured with 6–0 catgut; a preformed prosthesis is then inserted. If the prosthesis is not available then an acrylic stent can be manufactured during the operation. The lids are then closed over the prosthesis or stent with tarsorraphy-type sutures

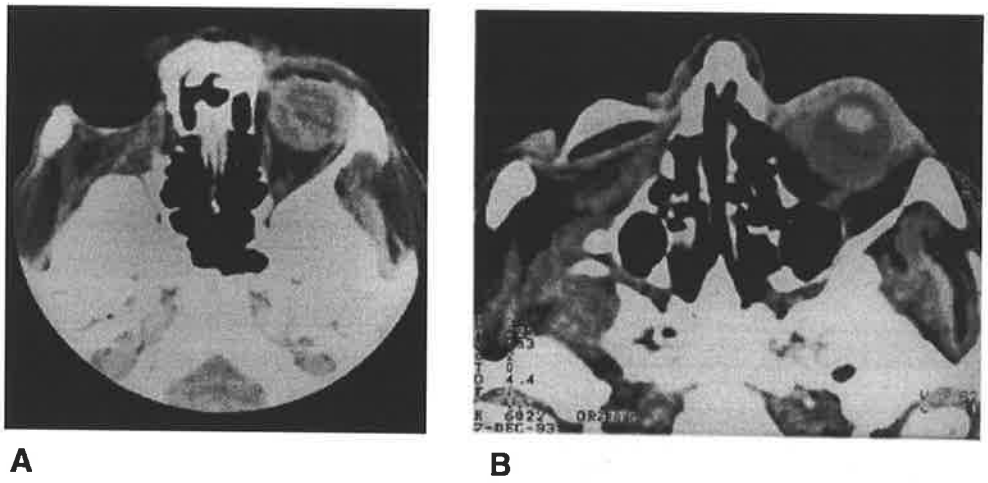
and left for as long as possible: for 3–6 months if the patient's compliance and the tarsorraphy can be maintained. When the graft has taken and the fornices are well maintained the temporary prosthesis or stent can be replaced with a permanent prosthetic eye.

Reconstructing the eyelids

The position of the lids may need to be readjusted by medial and/or lateral canthopexies. The lower lid is often too lax and sags under gravity; it may need support. This can be achieved by tightening the lid. There are several ways of doing this:

- Wedge resection of the lid
- Attachment of the lid to the lateral orbital walls (Patterson et al 1987)
- A lateral canthal sling procedure combined with a medial canthoplasty
- Insertion of ear or nasal cartilage into the lower lid to supply support.

We favour where possible the lateral sling and medial canthoplasty.



A B

FIG. 21.92. *CT evaluation of the orbit.* ***A*** *Axial CT scan of an enophthalmic orbit. The orbital rim is displaced and the residual soft-tissue contents of the orbit are shown.* ***B*** *Prosthesis in place. The deficiency in projection of the prosthetic globe is shown to be due to the relationship of the prosthesis, soft tissue, and bony wall.*

Complications and results

Except in the simplest and most straightforward cases the anophthalmic orbit remains one of the most challenging areas for secondary post-traumatic reconstruction. Almost all the tissues and substances used for forward projection of the globe are subject to resorption, fibrosis or movement. The effect of gravity on the lower lid together with the ageing process may require constant readjustment to the soft tissue and to the prosthesis. Lacrimation and lid movement may be defective, and frequent cleaning may be needed to prevent the eyelashes being stuck to the prosthesis with dried mucus; for the patient, this is a continuing nuisance. The prosthetic eye is best cleaned with the fingers under running water; chemical agents and soaps should not be used as they may be absorbed by the plastic prosthesis, giving rise to chemical or allergic conjunctivitis in the socket. It is not uncommon for anophthalmic patients to abandon the use of a prosthetic eye and either to use a patch or frosted glasses.

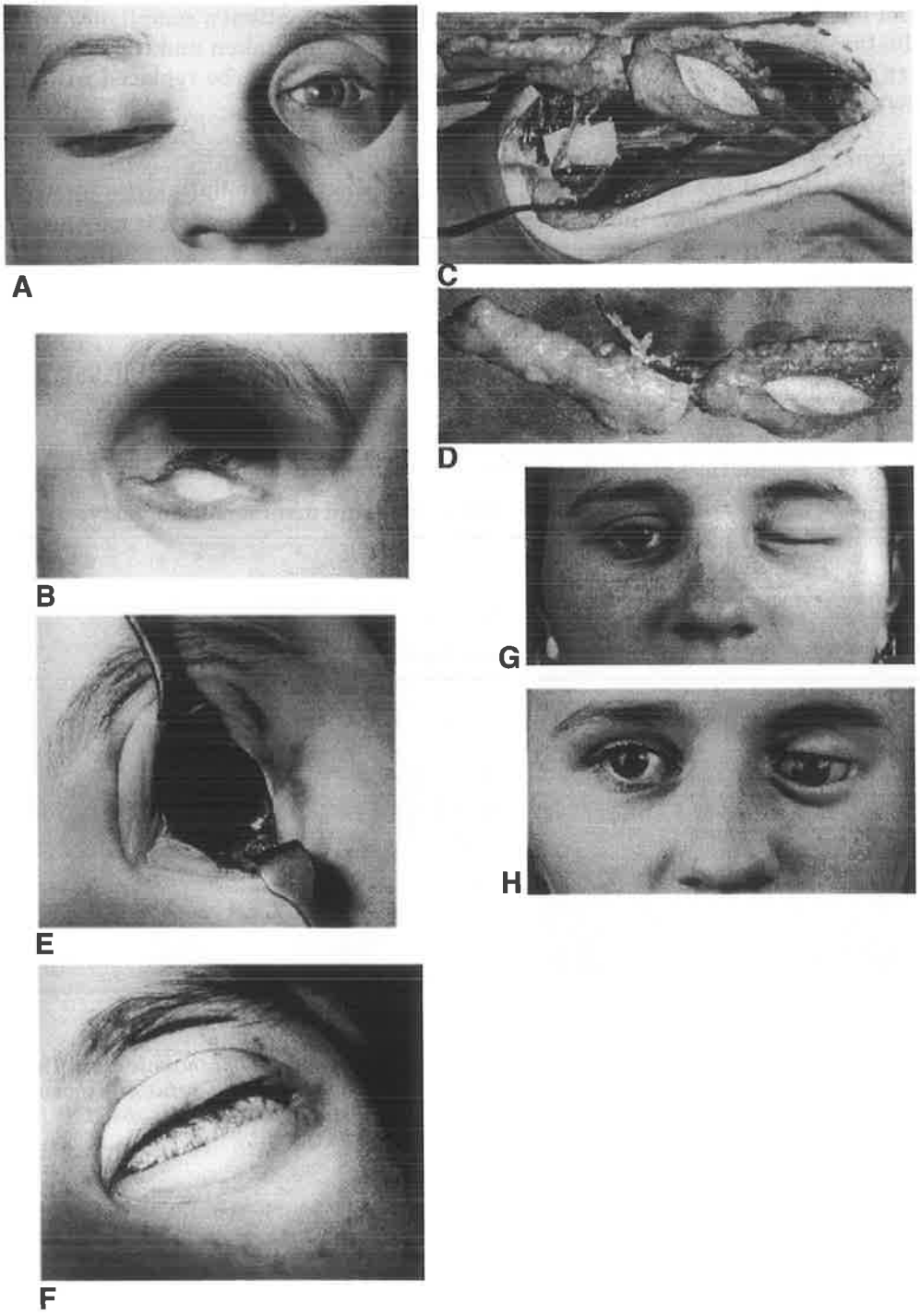


FIG. 21.93. ***Orbital reconstruction with vascularized arm flaps.*** *A 5-year old girl had a past history of retinoblastoma in the left eye at 18 months of age. She was initially treated with chemotherapy, radiotherapy, and enucleation of the left eye.* ***A*** *Prosthesis in place over the empty eye socket.* ***B*** *Contour deficit in the left temporal area.* ***C, D*** *A fasciolipomatous lateral arm flap is raised and split. A small skin ellipse is preserved for enlarging the new eye socket and as a flap monitor.* ***E*** *Left orbit prepared to receive the flap.* ***F*** *Immediately after flap transfer and revascularization* ***G, H*** *2 months postoperatively, a satisfactory orbital volume has been achieved and she was able to retain a glass eye. Since this time lateral canthoplasty has been performed, and correction of the entropion is planned.*

Conclusion

Simple problems are rare in secondary facial reconstruction after trauma; almost always, more than one organ system is involved, and therefore more than one discipline. Even the seemingly simple deformity of a displaced zygoma causing depression of the cheek prominence will by definition involve the orbit and potentially the globe; aesthetic correction must take into account any need for orbital dissection, and the possible ophthamological complications.

This chapter has dealt with the selection of surgical and prosthetic manoeuvres necessary to correct deformities; we have attempted to explain how this is done by organized team assessment. The planned sequences of reconstruction should be worked out in advance on a rational basis. But it must be accepted that the surgical intervention itself will produce some unwanted results. Complications should not be unforeseen: any treatment plan should be considered to be a working thesis and therefore subject to falsification and the re-emergence of a stronger thesis.

The seriously damaged face will never be entirely normal in spite of the huge healing forces of nature, enhanced and manipulated by good treatment. The team and the patient will have to decide when enough surgery has been done and when surgery should give way to acceptance and rehabilitative support, at first promoted by professionals but ultimately by the patient.

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Chapter 3
The Craniosynostoses

We dance around in a ring and suppose
but the secret sits in the middle and knows.

Robert Frost

3. The Craniosynostoses

The syndromal craniosynostoses of Crouzon, Apert and Pfeiffer represent the greatest of surgical challenges. Successful negotiation of these surgical ventures formed the beginning of craniofacial surgery.

By 1975 there was an obvious need to further study the aetiology, pathology, pathogenesis and natural history of these diseases in order to formulate rational treatment protocols. With the development of more sophisticated tools of investigation of craniofacial growth, such as computer tomography scanning, these protocols could be refined to include outcome measurements with respect to growth and surgical intervention.

The first two papers of this Chapter, New Perspectives in the Treatment of Craniosynostosis, 1981[1] and Severe Craniofacial Deformities and their Management, 1981[2] are the author's first published explanations of the craniofacial approach to craniosynostosis.

The monograph by David DJ, Poswillo D, Simpson DA The Craniosynostoses, Causes, Natural History and Management, 1982 include Aetiology and Pathogenesis[3]; Pathology[4] and Surgical Management[5]. It encapsulates the philosophies and treatment strategies on which the ACFU's approach to this clinical problem has been based. The author's contribution to the thought processes, concepts, philosophies and strategies were, of course, influenced by the literature but also by the fortunate association with Professor David Poswillo, Professor Donald Simpson, and Professor Tasman Brown. The author's contribution must be seen in this light.

These three articles set out the working hypotheses of the pathology and pathogenesis of the simple, complex, and syndromal

■

synostoses, relating these hypotheses to a treatment protocol which takes into account different epochs of growth.

Papers 6 and 7 on Craniosynostosis — Disorders of the Developing Nervous System: Diagnosis and Treatment, 1984[6] and Craniosynostosis in Plastic Surgery in Infancy and Childhood, 1986[7], reflect the development of the ACFU's philosophy from the neurosurgical and plastic surgical points of view.

The paper Craniofacial Infection in 10 years of Transcranial Surgery[8] describes the potentially devastating effects of this complication in craniofacial surgery and makes recommendations about new strategies to minimise this problem.

The next group of papers Hydrocephalus in Crouzon Syndrome, 1989[9]; Surgical Correction of Crouzon Syndrome, 1990[10] and Crouzon Twins with Cloverleaf Skull Malformations, 1991[11] describes the clinical outcome measurements made from a large data base, each individual work containing some small amount of information that modifies and enriches the working hypotheses.

Upper airway obstruction with all its sequelae has emerged as a major issue in syndromal craniosynostoses. Obstructive Sleep Apnea in Apert and Pfeiffer Syndromes: more than a Craniofacial Abnormality, 1990[12] and Aggressive Surgical Management of Sleep Apnea in the Syndromal Craniosynostoses, 1992[13] deal with this problem from the aspects of its recognition, surgical and non surgical management.

The Progress of Craniofacial Surgery Reflected in the Management of Craniosynostosis, 1992[14] plots the transition from the early days where surgical technique was the important issue to the present, when the disease process and its long term management demand most attention.

Phenotypic Variation in Acrocephalosyndactyly Syndromes, 1994[15] and Pfeiffer Syndrome: a Clinical Review, 1995[16] are examples of the emphasis in the craniofacial unit on analysing and collecting data to redefine syndromes phenotypically and clinically.

The genetic aspects of this advancement of knowledge with respect to the craniosynostoses and other conditions is mentioned in Chapter 7.

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New Perspectives in the Treatment of Craniosynostosis

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Summary

The history of ideas on the pathogenesis and treatment of craniosynostosis syndromes is discussed, with special reference to the emerging role of cranio-facial units. Emphasis has changed from correction of established deformities, to early surgery to prevent such deformities, as well as relief of existing pathology such as exorbitism and raised intracranial pressure. These early operations, best performed in the first three months of life, essentially involve unilateral or bilateral fronto-orbital advancements. The coronal suture is released with a craniectomy, extended to the squamosal suture, and a wide temporal decompression performed. The frontal bone and superior orbital margin are mobilized and moved to their new position, and wired in place. Intermediate and late stage operationø are also discussed, together with complications.

Key words: Craniosynostosis — Crouzon — Plagiocephaly — Craniofacial surgery.

In 1849, Rudolf Virchow [9], then already well known as an innovative pathologist and as a radical politician, left Berlin following a change of government in Prussia. He went to Wurzburg, and in that lovely and relatively liberal city he engaged in studies of the growth of the skull. He found a connection between skull deformity and premature fusion of particular cranial sutures. He formulated what is sometimes called Virchow's law: that specific skull deformities result from cessation of growth across a prematurely fused suture, and compensatory growth across normal sutures.

A century or so later, neurosurgeons tried with varying success to correct skull deformities by operations which were explicitly based on Virchow's concept [9]. Scaphocephaly, for example, where the sagittal suture fuses prematurely, was treated by creating a new suture in the form of sagittal or parasagittal craniectomies, with insertion of plastic to delay bone regeneration. Turricephaly, the towering head associated with coronal suture fusion, was similarly treated by a coronal resection. These operations had a measure of success.

The simpler deformities of the vault of the skull showed significant improvements — scaphocephaly being particularly well corrected if the operation was performed in the first few months of life. However, the operations failed completely to correct the facial deformities that so often go with premature craniosynostosis in the vault, as seen most strikingly in Crouzon Syndrome: the genetic dominant syndrome of skull deformity, proptosis, and maxillary hypoplasia.

These failures can be explained easily enough. The skull grows in part by deposition of bone in the membranous sutures, in direct response to tensions set up by the growing brain. But the driving forces for skull growth appear to be in the cartilaginous skull base, in the synchondroses, and transmitted to the vault of the skull along lines of dural tension.

Even if one does not accept Moss's [5] claim that premature sutural fusion is a symptom and not a cause of deformity, it is nevertheless clear that in many forms of craniosynostosis, the disease process affects the skull base and the facial sutures as well as the vault. Nowhere is this so evident, as in the Syndromes of Apert and Crouzon, both usually characterised by gross and crippling deformity, due to proptosis, maxillary hypoplasia, and malocclusion.

The Evolution of Cranio-Facial Surgery

In 1950, Gillies reported the first cranio-facial osteotomy on a case of Crouzon Syndrome (Gillies and Harrison [1]). It was generally considered to be an unsuccessful and somewhat frightening experience. The next significant advance in this type of surgery was delayed until Paul Tessier in 1958 corrected the deformity of Crouzon and Apert Syndromes by anterior advancement of the face using the transcranial technique. This contribution led to the foundation of the discipline of craniofacial surgery.

Tessier's [6] pioneering contributions were two fold:

1. by using a bicoronal scalp flap and dissecting at the subperiosteal level, he was able to bare the cranio-facial skeleton and dissect the orbits back under the periorbitum to within a centimetre of the optic foramen,

2. recognising the unique distortions of the cranial base in each of these cases, he saw the value of combining the techniques of plastic surgery and neurosurgery to gain access to the orbito-cranial complex. This was achieved by neurosurgical dissection of the anterior cranial fossa enabling the frontal lobes of the brain to be lifted clear of the orbital roofs.

In 1962 Tessier corrected his first case of orbital hypertelorism, and so, with the ability to move the orbital complex in three dimensions, the current tendency is to vary the pattern of cranio-facial osteotomies to suit the individual cases.

The Case for Early Operation

In 1970, Tessier applied his methods for correcting craniosynostosis to young children. The problem that provided the stimulus for this surgery was the neurosurgical need to decompress the brain and to protect the proptosed eyes in certain cases of extensive craniosynostosis. The possibility then arose of preventing the secondary facial components of the deformities resulting from craniosynostosis syndromes by doing the radical Tessier type of operation in infancy and childhood, and so giving aesthetic satisfaction as well as good cerebral and optic function. This possibility has been followed up by Hoffman and Mohr [2], Whitaker [7], Marchac [4] and McCarthy [3].

There is now a change in emphasis from the correction of established deformities to the possible interruption of a pathological process, which may permit restoration of normal growth patterns and the prevention of the ultimate deformity. This concept, of course, still remains intensely controversial.

It is a concept which is much more complex than Virchow's still widely accepted thesis. We must now postulate that the deformities of craniosynostosis represent the outcome of a conflict between normally expanding organs — the brain, the eye-balls, and the air passages — and abnormally restrictive growth especially of the base of the skull. The corollary is that if the restrictive forces are released early in life there will be normal growth not only locally but, more

remotely, in the rest of the facial skeleton. It is a concept which will remain controversial until we have sufficient properly studied clinical cases with long-term follow up, although experimental studies on a suitable model may be helpful. There are already reports of success from operations which extend into the floor of the anterior fossa, such as the procedure illustrated in Fig. 1, done for unilateral infantile craniosynostosis, which does appear to be a real advance on the simpler linear coronal craniectomy. It will indeed be an immense gain if we can prevent ultimate facial deformity by well-planned procedures done in infancy. Operations done on older children or adults for the established deformity are often brilliantly successful, but they are full of neurosurgical dangers — C.S.F. fistula, extradural bleeding, osteomyelitis, and visual damage. These dangers are enhanced by previous neurosurgical operations. There is therefore much merit in the collaborative approach of a craniofacial clinic even for severe craniosynostosis in infancy — hitherto regarded as a neurosurgical problem.

Current Practice

The original combination of plastic surgeon and neurosurgeon has been expanded into the modern cranio-facial unit. With the emerging concepts of preventative surgery, operative intervention may be considered as:

1. early operations;

2. intermediate operations;

3. late operations.

Early Operations

These may be performed:

a) for the relief of existing pathology, for example, exorbitism or raised intracranial pressure;

b) for the prevention or minimization of future facial deformity.

These operations are essentially fronto-orbital advancements which may be unilateral or bilateral, according to the pattern of the synostosis. Fronto-orbital advancement is achieved by isolating the superior orbital margin, which is advanced according to one of the following patterns:

a) with a Z-plasty (Munro, Marchac, Fig. 1),

b) with a tongue of temporal bone (Tessier, Fig. 2),

c) attached to a spur of zygoma (David and Simpson [10], Fig. 3).

During this procedure the coronal suture is released with a craniectomy, which is extended across the squamosal suture. A wide temporal decompression is performed and the cut extended across the anterior cranial fossa so that the superior orbital margin is mobilised and advanced to match the other side. The frontal bone can be either rotated or advanced to overlay the superior orbital margin. This operation has the effect of:

i) providing a roof for the exposed globe;

ii) providing a wide decompression;

iii) cutting across all the stenosed sutures in the vault and base of the skull.

It is important that these operations are done in the first three months of life because of the growth curve of the brain and skull. However, there is still some gain to be had up to about 18 months of age.

Intermediate Operations

These may be performed for:

a) developing symptoms, the most common of which result from exposure of the globes and recurrent raised intra-cranial pressure;

b) psycho-social indications.

Need for the intermediate type of operation occurs between 18 months and 8 years of age. The term intermediate is justified because the child is too old for the prophylactic aspect of the early treatment, yet too young to achieve the definitive cosmetic and functional result. There is a range of procedures performed during this time that are intermediate in the total treatment plan. The type of operation depends on the pattern of sanostosis and the degree of involvement of the fronto-orbital complex. In the younger child, the fronto-orbital advancement may be sufficient, whereas in the older child the more radical operation of fronto-orbital advancement and anterior maxillary shift may be indicated. During this stage, the operation produces the dual effect of cosmetic improvement together with cerebral decompression and significant protection of orbital contents (Fig. 4).

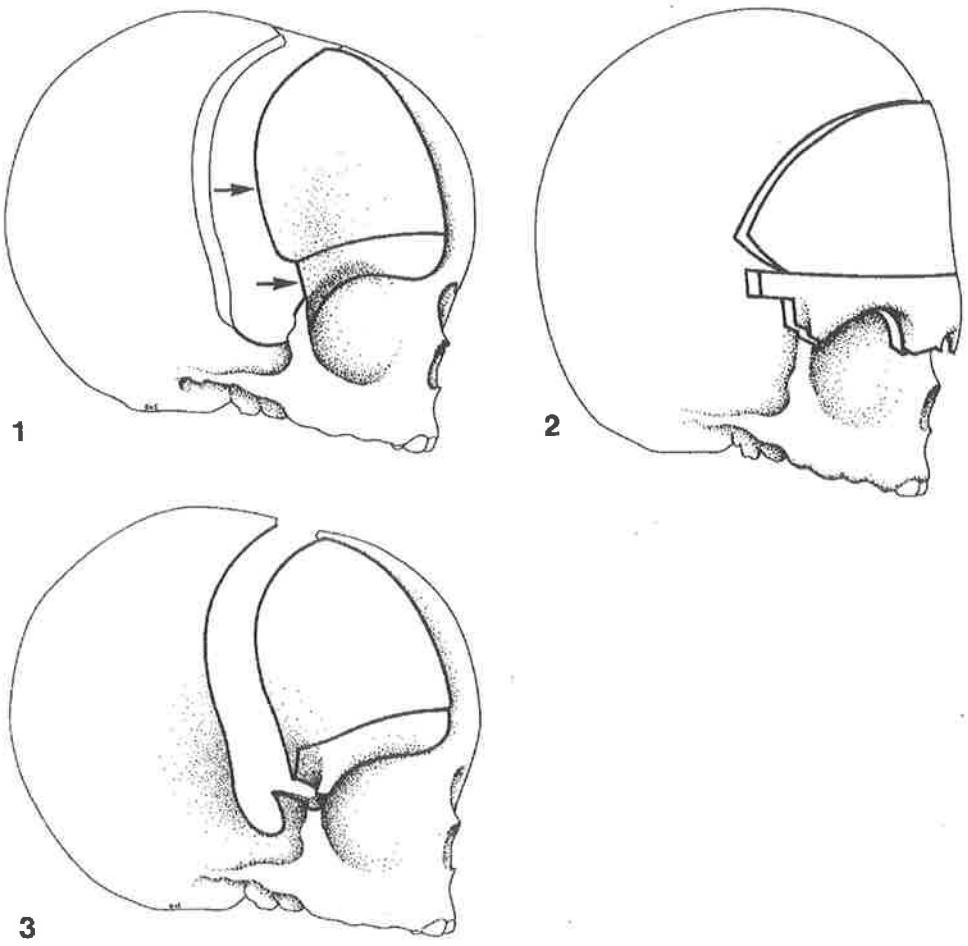

FIG. 1. *Fronto-orbital advancement (after Marchac / Munro)*

FIG. 2. *Fronto-orbital advancement (after Tessier)*

FIG. 3. *Fronto-orbital advancement (David / Simpson)*

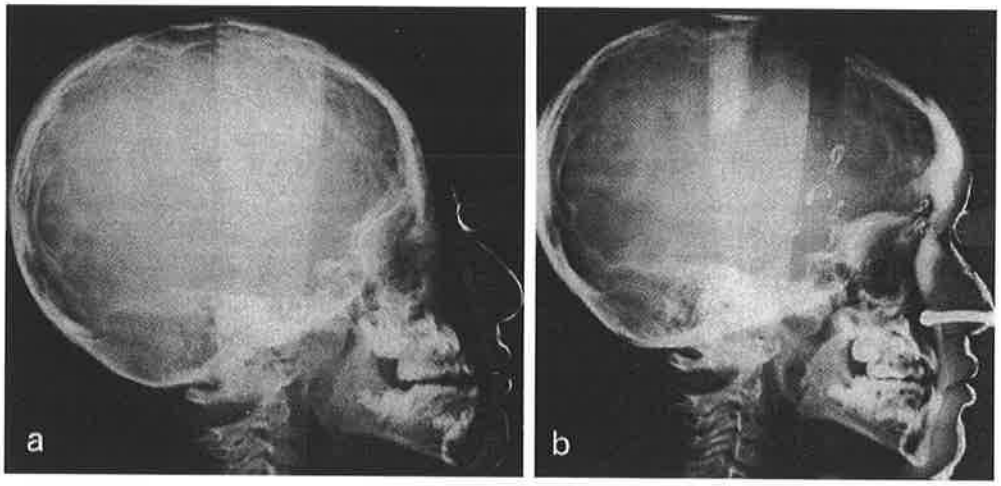

FIG. 4. a, b. *Lateral cephalometric radiographs:* **a.** *before,* **b.** *after. Bilateral Fronto-orbital advancement in a six year old with Crouzon Syndrome.*

Late Operations

These are invariably for psycho-social and cosmetic indications, but have associated functional advantages:

1. better eye protection;
2. release of upper airway stenosis;
3. improved occlusion.

The commonest symmetrical deformities are the craniofacial dysostoses of Apert and Crouzon and the other symmetrical craniosynostosis syndromes. The osteotomy pattern should be made to fit the deformity, as it is now possible to move all of the appropriate bones involved, either together or separately, and a proper cranio-facial service should be able to correct the deformity. In our practice the original Tessier type of Le Fort III osteotomy with a step advancement of the malar (Fig. 5), has given way to the technique for advancement of the middle third of the face, together with the facade of the frontal bone (Fig. 6) or fronto-orbital advancement together with advancement forwards and downwards of the middle third of the face (Fig. 7).

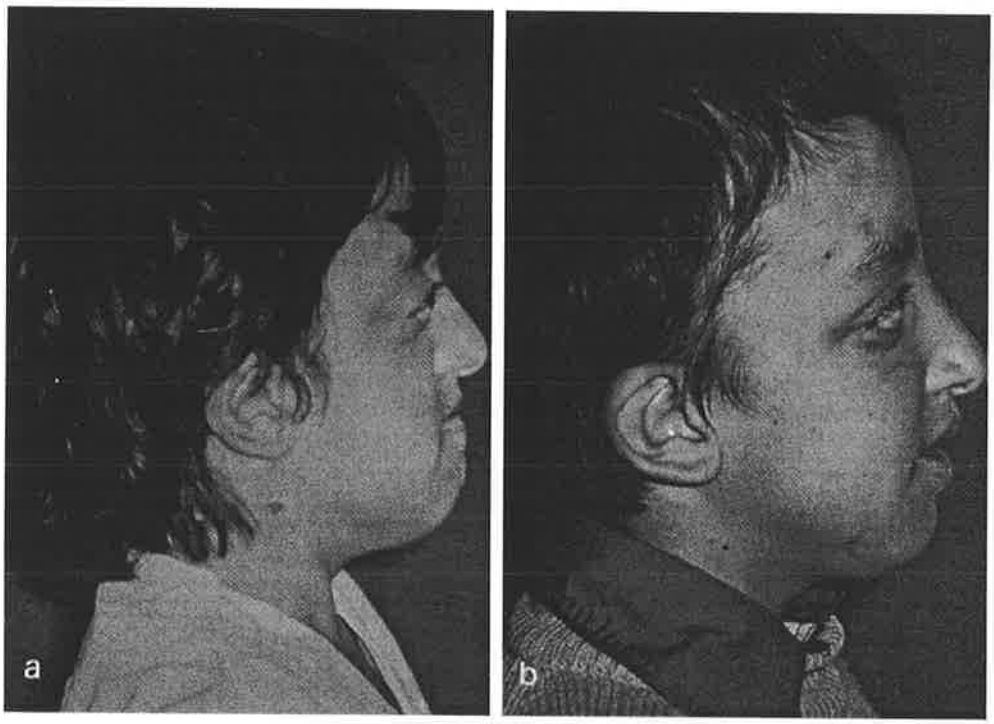

FIG. 5. a, b. *Crouzon Syndrome in a 12 year old boy.* **a.** *Before,* **b.** *after. Tessier type Le Fort III osteotomy and step advancement of the malar.*

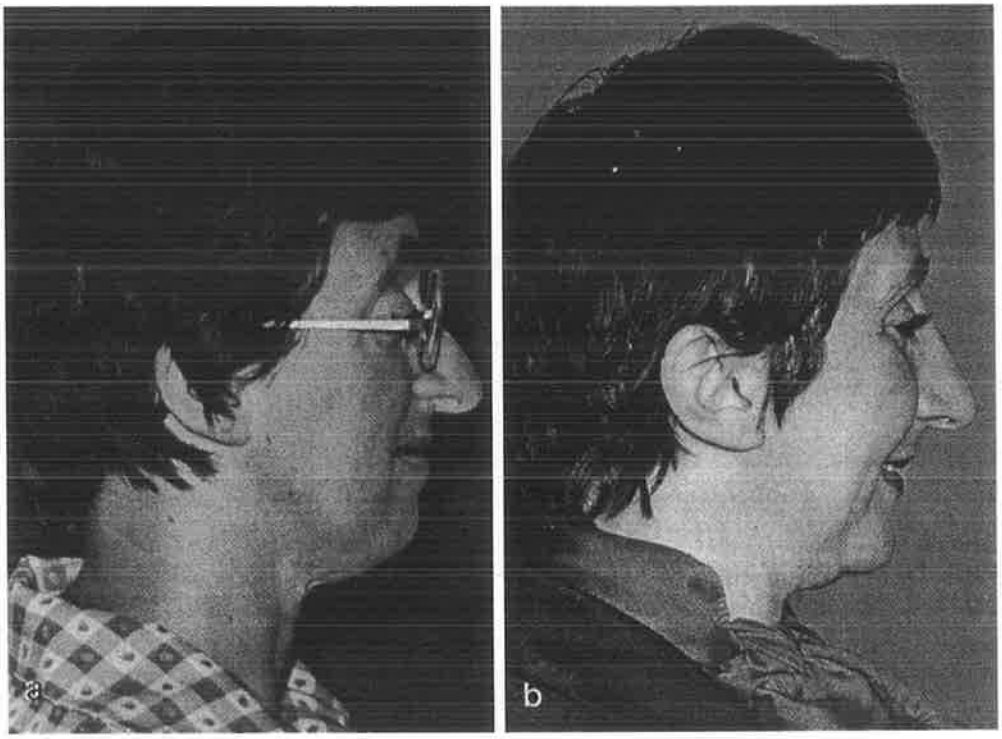

FIG. 6. a, b. *Crouzon Syndrome in a 36 year old woman.* **a.** *Before,* **b.** *after. Advancement of the middle third of the face together with a facade of the frontal bone.*

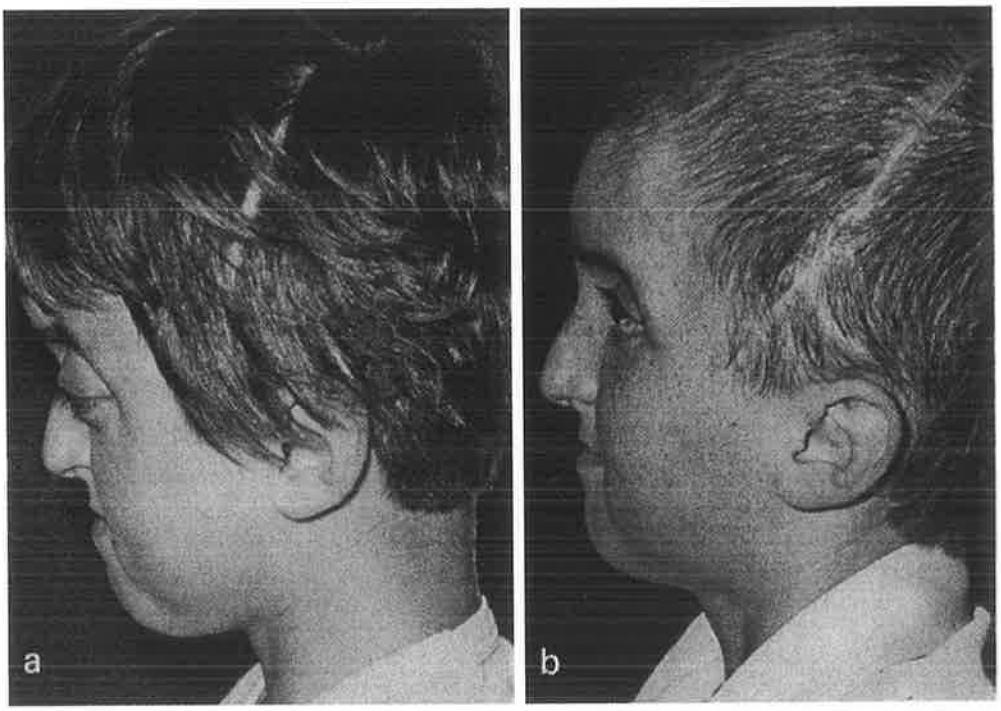

FIG. 7. a, b. *Crouzon Syndrome in a 10 year old boy.* **a.** *Before,* **b.** *after. Fronto-orbital advancement and advancement forwards and downwards of the middle third of the face.*

Asymmetrical Deformities

These are most commonly the result of uncorrected unicoronal synostosis and represent considerable tragedy because they could most probably have been prevented, certainly in part, by early surgical intervention, as advocated in the section on early operations. An established fronto-orbital deformity such as that shown in Fig. 8 can be corrected by unilateral fronto-orbital advancement of a similar pattern to that performed in infancy. The osteotomy can be extended into the root of the nose so that the twisted glabella region can be straightened. More severe cases such as that seen in Fig. 9 resulting from multiple uncorrected cranial synostoses with a severe degree of facial asymmetry affecting the orbits, maxilla and mandible present one of the most difficult problems presented to the Cranio-Facial Surgical Team. It is our current practice to treat the cases that present late in two stages, correcting the orbito-cranial aspect by transcranial operation in the first stage, and then attending to the slanting occlusal plane by operation on the maxilla and mandible at a later date.

Complications

Clearly these operations entail considerable risk, both to life and function, and can only be contemplated in the context of complete assessment of the patient by an experienced cranio-facial team. Several reports of complications in cranio-facial surgery have been published in recent years, emphasising the potential risks. Whitaker et al. [8], in reporting 793 such operations, note an overall 25% complication rate in transcranial surgery, and a 2.2% mortality rate, with reduced complication rates in sub-cranial surgery. Major problems encountered in the trans-cranial surgery correction included persistent C.S.F. leaks, often requiring surgical correction; osteomyelitis of the bone flaps, with resultant problems, including one death; blindness, bilateral or unilateral.

Our Unit, in a series of 37 trans-cranial cases has encountered a similar complication rate overall (Table 1), although we have had no deaths, or infections. We have a rigorous routine of asepsis per operation, and at the commencement of each operation intravenous Ampicillin and Methicillin, are administered, and given six hourly for the duration of the operation, and the following five days. Dosage is 25 mg/kg six hourly. The two antibiotics are alternated, such that a dose is administered every three hours.

Our two cases of persistent C.S.F. leak were both following trans-cranial Le Fort III osteotomy and advancement for Crouzon Syndrome, where frontal dura at the primary operation was abnormally thin and friable. Both cases required secondary formal dural repair.

Of the two cases of blindness, one was unilateral, possibly due to undetected intraorbital optic nerve compression, and the second, bilateral, the result of an enormous extradural haematoma which developed post-operatively, in a case with undetected clotting abnormalities. This latter case is instructive. The haematoma compressed the frontal lobes, and as the normal attachment of the dura to the floor of the anterior cranial fossa had been severed, the expanding clot caused traction on the optic nerves, resulting in blindness without the appropriate change in conscious state. This illustrates the new pitfalls for the unwary created by this surgery, where new anatomical and physiological situations exist such as an expanded cranium, a mobile dura, and free connection between the subgaleal space and the cranial cavity.

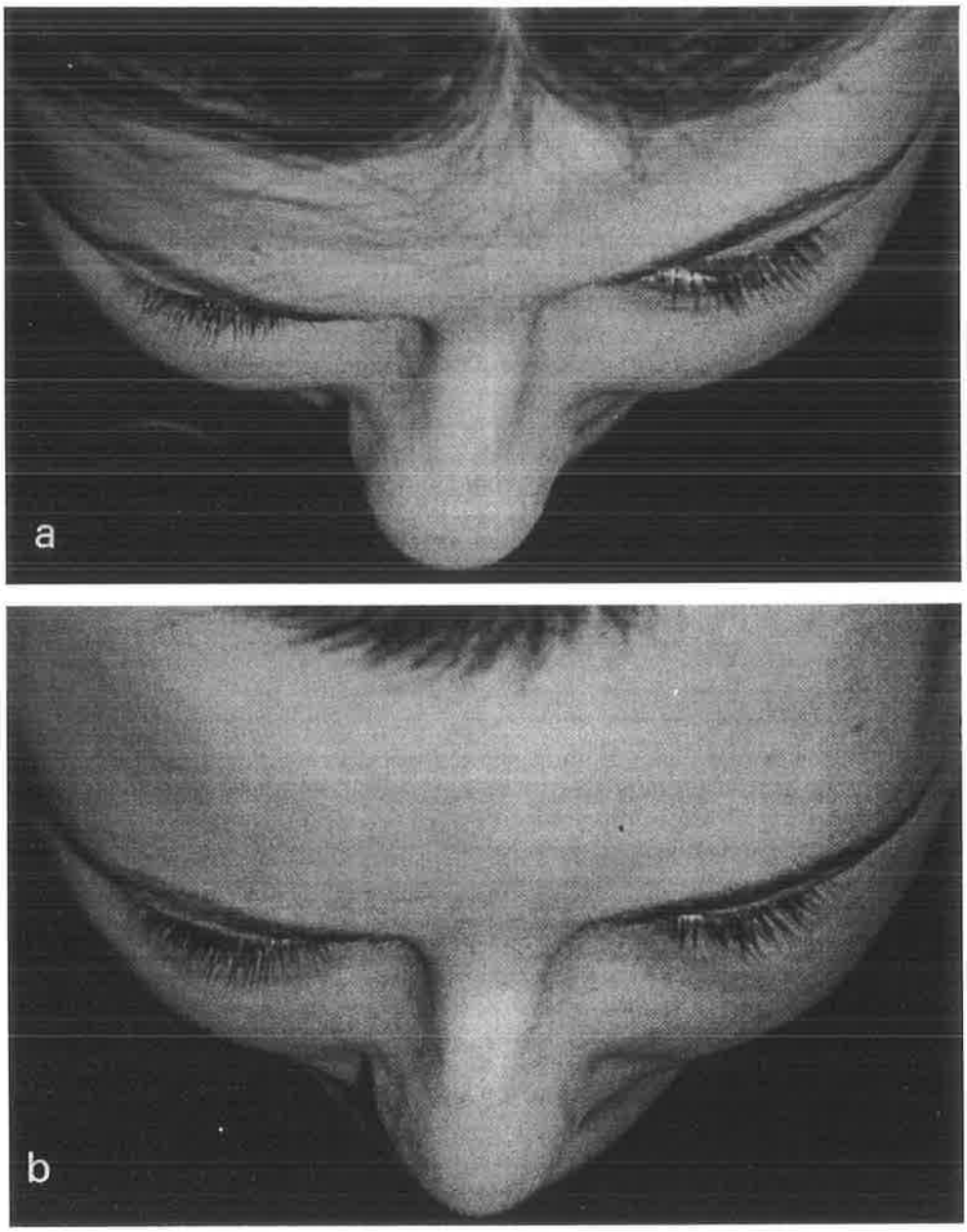

FIG. 8. a, b. *Left unicoronal synostosis with plagiocephaly in a 17 year old woman.* **a.** *Before.* **b.** *after. Unilateral fronto-orbital advancement.*

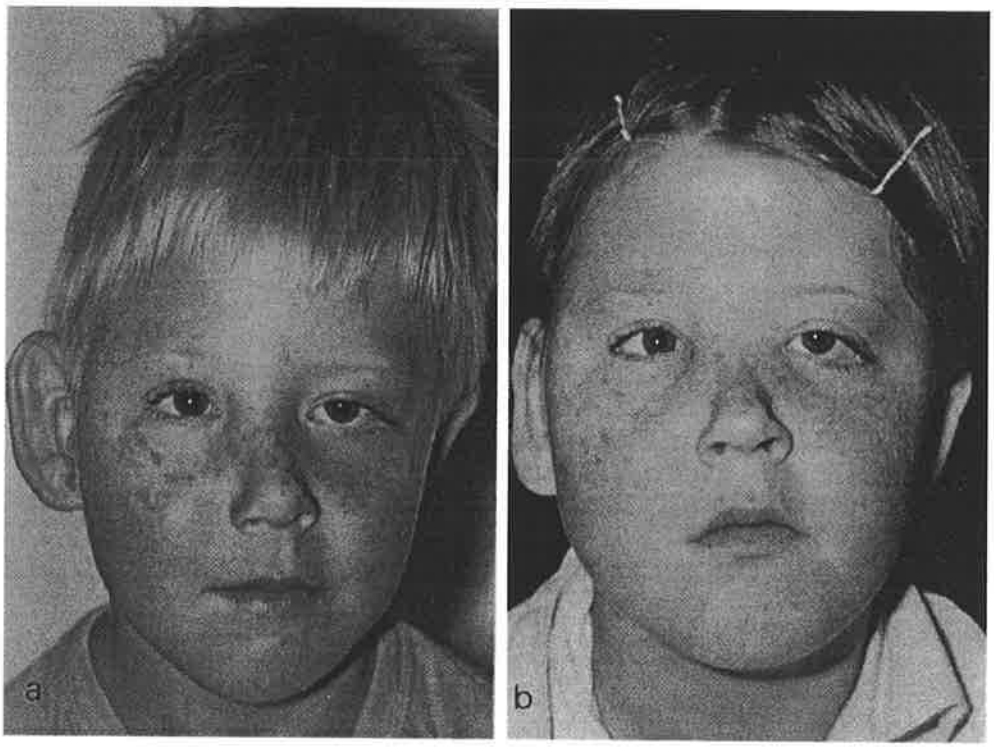

FIG. 9. a. b. *Multisutural synostosis with plagiocephaly in an 8 year old boy.* **a.** *Before,* **b.** *after. Transcranial correction with fronto-orbital advancement and maxillary onlay bone grafts.*

TABLE 1

Complications of major cranio-facial surgery

Complication	Transcranial	Extracranial + orbits	Extracranial without orbits	Total
	(*n*=33)	(*n*=37)	(*n*=10)	(*n*=80)
Death	0	0	0	0
Brain Damage	0	0	0	0
Visual loss[a]	2	0	0	0
Infection	0	0	3	3
C.S.F. leak[b]	2	0	0	2
Extradural haematoma	2	0	0	2
Overall rate	16%	0%	9%	11%

[a] One case of permanent unilateral blindness. One case of bilateral severe visual impairment

[b] Both cases required re-opening and dural repair

Conclusion

The further development of cranio-facial surgery since its inception by Tessier, has thrown new perspectives on the management of severe cranio-synostosis syndromes. It has not only enabled a more adventurous approach to the established deformities, but opened up the possibility that early operation by skilled and experienced teams can go a long way to preventing or minimising the facial deformities resulting from cranio-synostosis.

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Severe Cranio-facial Deformities and Their Management

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Summary

Historical attitudes to cranio-facial deformities are discussed. A simple nosology of cranio-facial deformities is given, the main groups being craniosynostosis syndromes, facial clefts, tumours, trauma, facial microsomias, and midface hypoplasias. Each group is briefly discussed, with emphasis on the craniosynostosis syndromes and facial clefts. Cases from these two groups are illustrated before and after surgery, showing the dramatic improvement offered by craniofacial techniques. The basic principles of cranio-facial surgery are briefly discussed, and the complications discussed. The South Australian Cranio-Facial Unit experience with complications is discussed, especially blindness and persistent CSF leaks. The necessity for only a few cranio-facial units each with a high workload is reiterated, and new directions for cranio-surgery discussed, including the use of microvascular techniques and early surgery to minimise later deformity, particularly in cases with plagiocephaly.

Key Words:
Cranio-Facial Surgery
Craniosynostosis
Crouzon
Facial Clefts
Fronto-Nasal Dysplasia
Hypertelorism

Introduction

Facial appearance has been important in most societies throughout man's history. In modern western society attractive facial appearance is actively selected for, and people with unattractive facial appearance are frequently at a disadvantage in the work place. Inevitably, therefore, individuals with cranio-facial deformities suffer greatly in our present society. It is ironical that some past and "primitive" societies not only tolerated, but in some cases revered those with cranio-facial deformities. This is particularly seen in some of the Central and South American civilisations.

Our modern ideals of beauty and acceptable appearance have ensured a demand for cosmetic procedures to improve facial appearance in the facially deformed. However, modern cranio-facial surgery has far more to offer than mere cosmetic improvement of facial appearance. There are many organic problems associated with cranio-facial deformities which can be now be cured or at least improved by the expertise of a cranio-facial team. In addition, many of those with cranio-facial deformities have normal and in some cases brilliant minds. From the humanitarian stand-point these people deserve all that modern medicine can offer them. From an economic standpoint the cost of cranio-facial treatment is small compared to the potential productivity of the patient which it may unlock. From an academic view point the concerntration of cases and expertise in a cranio-facial unit must lead to a greater understanding of these crippling deformities and their treatment. We feel that it is essential that all doctors should be aware of cranio-facial deformities and the treatment now available from cranio-facial teams so that all patients with such deformities can have a chance of assessment and treatment by a cranio-facial team.

Classification of Cranio-Facial deformities

Inevitably the wide range of deformities possible in the cranio-facial area make classification difficult. The experience the cranio-facial teams around the world is helping bring order out of chaos. Thus Paul Tessier[1] of France has introduced some order into the classification of rare cranio-facial clefts, and Cohen[2,3] is reducing the chaos of craniosynostosis syndromes. Nevertheless the nosology of cranio-facial disorders is still in flux and the classification given below is not meant to be definitive.

Craniosynostosis Syndromes

With each review the number of craniosynostosis syndromes listed has increased, but all have in common the premature fusion of one or more cranial sutures. Each syndrome has a particular pattern of anamolis which may not be limited to the cranio-facial skeleton, and may involve other parts of the body, especially limbs. Cohen[3] has recently reviewed craniosynostosis syndomes and table 1 lists the major syndromes which may present to a cranio-facial unit. Of these the best known is Crouzon Syndrome, first described by Crouzon in 1912. This syndrome, characterised by variable craniosynostosis, midface stenosis, with resultant gross proptosis and class III malocclusion is frequently seen in cranio-facial units (Fig. 1). Correction of this constellation of deformities has been one of the major challenges which has stimulated the development and growth of the field of cranio-facial surgery. As with a number of these syndromes, individuals with Crouzon syndrome frequently exhibit normal or above normal intelligence, and with appropriate treatment can fulfil a very worthwhile role in society. Without treatment the cranial vault may not keep pace with brain growth and resultant raised intra-cranial pressure can cause persistent eye irritation, and even dislocatons which may also result in blindness. The small nasal and pharyngeal airways can cause persistent upper respiratory problems and the gross mallocclusion can make it difficult for the individual to chew food which may result in dietary problems and deficiency. The overall facial appearance will often preclude successful social intercourse. Those individuals with Apert Syndrome have similar problems, the pattern of cranio-facial deformity being similar to that of Crouzon but often less severe. However, in addition Apert Syndrome includes gross soft tissue and bony syndactyly involving hands and feet and is frequently associated with mental retardaton. Our experience suggests that this latter association is less frequent than previously thought.

We believe that early surgery in these deformities may minimise or even eliminate many of the deformities and problems encounterd later in life.

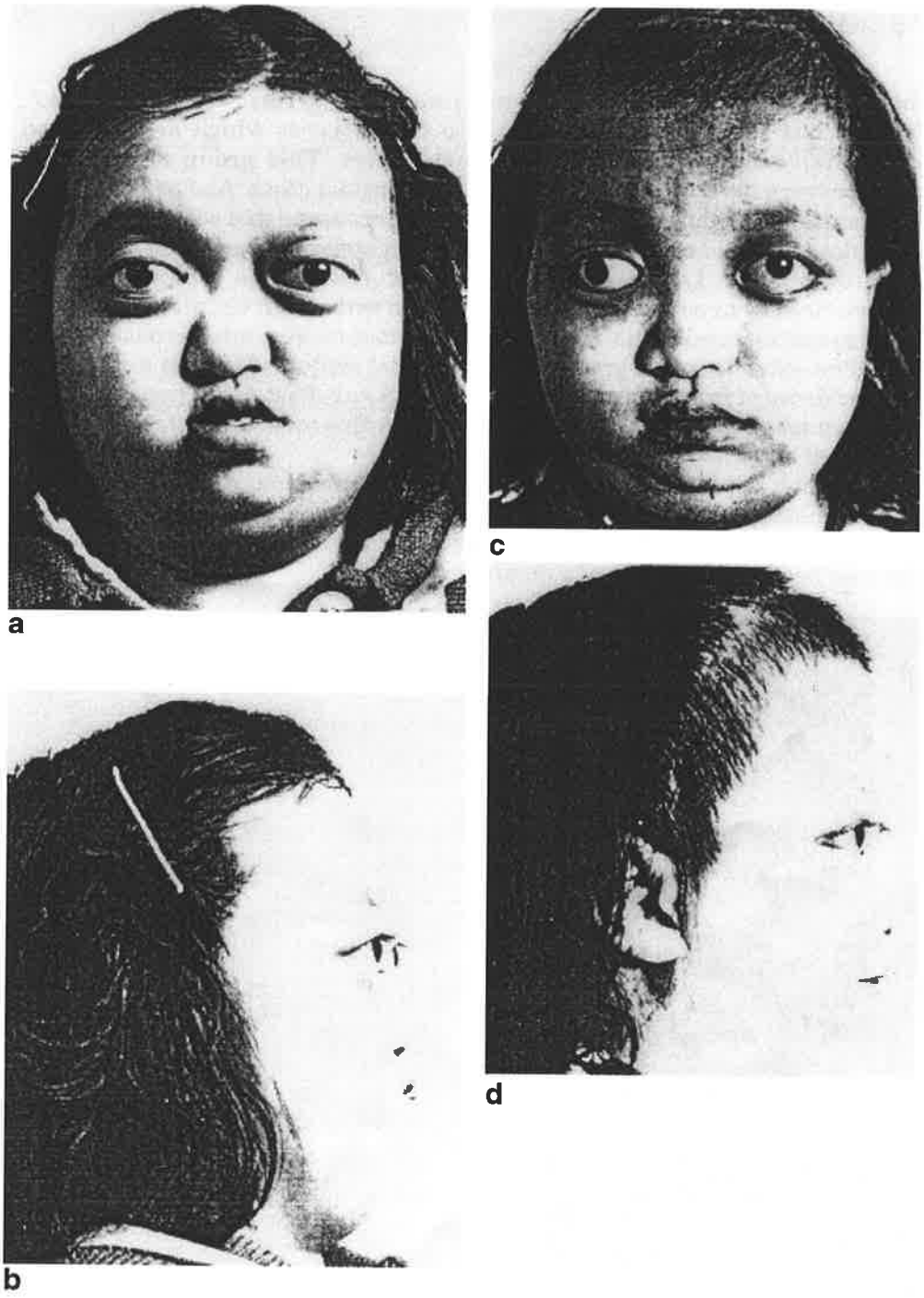

FIG 1. *Crouzon Syndrome in a 10 year old girl.*

a. *Pre-operative AP view. Note hypertelorism, exophthalmos.*

b. *Pre-operative lateral view. Note maxillary hypoplasia. Class III malocclusion.*

c. *Post operative AP view. Note midface after transcranial fronto facial advancement*

d. *Post operative lateral view. Note decreased exophthalmos, improved facial contour, normal dental odclusion.*

Facial Clefts

This comlex set of disorders has been well researched by Tessier[1] and Kowamoto[5]. Tessier[1] has classified facial clefts into fifteen types which are arranged sequentially around the orbital circumference. This group of disorders encompasses a number of distinct multiple cleft associations. Among these is the Treacher-Collins Syndrome. Many of these clefts are associated with the widening of the naso-ethmoid complex with resultant hypotelorism and nasal deformity. The fronto-nasal dysplasias may be considered in this group. They are characterised by hypotelorism, broad nose often with a cleft tip, and extension of meningo-encepalocoele usually through the foramen caecum into the nasal region. This often extends into a grossly widened nasal septum. There is a relatively high incidence of fronto-nasal dysplasia in the South-East Asian region, and we have seen many examples of this condition from this area. (Fig 2.)

Though not included by Tessier in his classification, cleft lip and palate can conveniently be considered in this group. This frequent cranio-facial deformity may be associated later in life with conditions such as maxillary hypoplasia which may also need the attention of a cranio-facial team.

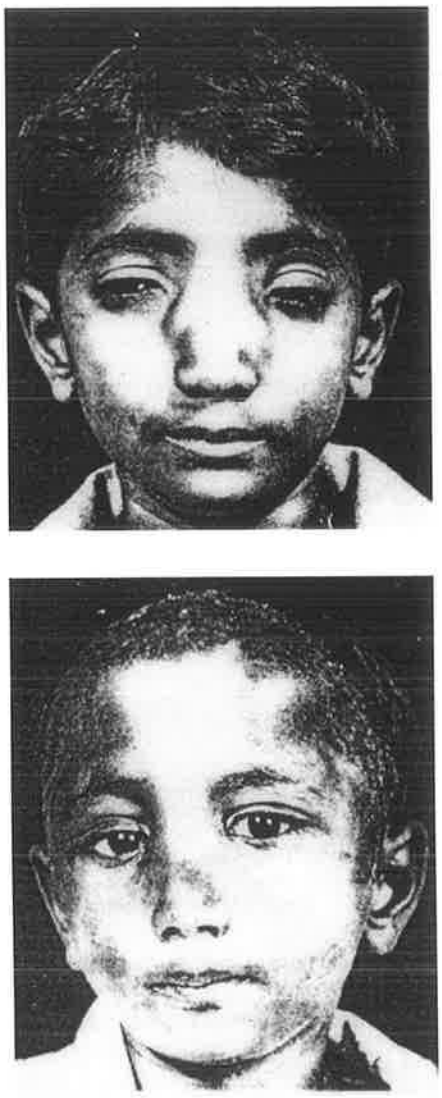

FIG. 2. *Fronto-nasal dysplasia with fronto-nasal meningoencephalocoele and hypertelorism in a 6 year-old boy.*

a. *Pre-operative AP view. Note nasal deformity, wide nasal bridge and hypertelorism.*

TABLE 1

List of principle craniosynostosis syndromes

Syndrome	Principal features
Apert Syndrome	Craniosynostosis, hypertelorism, midface hypoplasia, class III, malocclusion, complete symmetric syndactyly of hands and feet.
Carpenter Syndrome	Craniosynostosis, broad nasal bridge, preaxial polysyndactyly of the feet, variable soft tissue syndactyly and brachymesophalangy of the hands, obesity, frequent association with congenital heart defects.
Craniofrontonasal Dysplasia	Craniosynostosis, brachycephaly, hypertelorism, clefting of nasal tip, variable abnormalities of the hands and feet.
Crouzon Syndrome	Craniosynostosis, gross midface hypoplasia, exophthalmos, class III malocclussion.
Pfeiffer Syndrome	Craniosynostosis, exophthalmos, hypertelorism, midface hyperplasia, broad thumbs and great toes, variable cutaneous syndactyly
Saethre-Chotzen Syndrome	Craniosynostosis, facial asymmetry, plagiocephaly, low set frontal hairline, ptosis, variable brachydactyly and cutaneous syndactyly.

Tumour

Numerous benign and malignant lesions may use cranio-facial deformity. Of the benign tumours seen by us, fibrous dysplasia of bones of the cranio-facial skeleton is the most frequently seen. This condition is more commonly seen in females, and frequently presents in late childhood or early adolesence. Gross facial or skull assymetry may result and may require extensive cranio-facial corrective procedure.

Benign infantile teratoma may also present with cranio-facial deformity requiring extensive reconstruction. (Fig. 3)

Trauma

Advancing technology and affluence is providing more ways for individuals to traumatise the cranio-facial skeleton. Foremost amongst these in our experience are motor vehicle accidents. These may cause a wide variety of deformities depending on the position and force of the impacting object, and the resultant fracture pattern. Depression of rhe midface is frequently seen, as is unilateral orbital trauma which frequently results in orbital dystopia and enophthalmos (Fig. 4.) Cranio-facial surgery is now providing new and more effective ways of correcting these complex deformities.

Facial Deformities

This catch-all phrase covers a wide specturm of deformities which can be sub-classified.

Facial Micosomias

These deformities include a number of distinct entities such as Golden Har Syndrome. Hemifacial microsomia is frequently seen by us and is characterised by under-development of one side of the face including soft tissue and bone. The pattern of under-development is variable but always results in facial assymetry and can manifest as a very severe facial deformity. Poswillo has produced differing grades of hemifacial microsomia in animal experiments by arterial occlusion in early foetal life. In a severe case of hemifacial microsomia there can be gross malformation of the external and internal ear on that side , with maxillary hypoplasia, mandibular hypoplasia, malformed or absent temporomandibular joint and maldevelopment of overlying soft tissue.

Midface Hypoplasias

These include conditions such as Binder Syndrome or naso-maxillary hypoplasias. Also seen are isolated maxillary hypoplasias with associated maloclusions. The aetiology of these conditions is not well understood. Many other facial deformities may be encountered by a Cranio-facial Unit. Moebius Syndrome, involving maldevelopment of cranial nerves can result in unilateral or bilateral absence of the 7th nerve causing characteristic deformity of the face. Primary mandibular pathology is frequently seen.

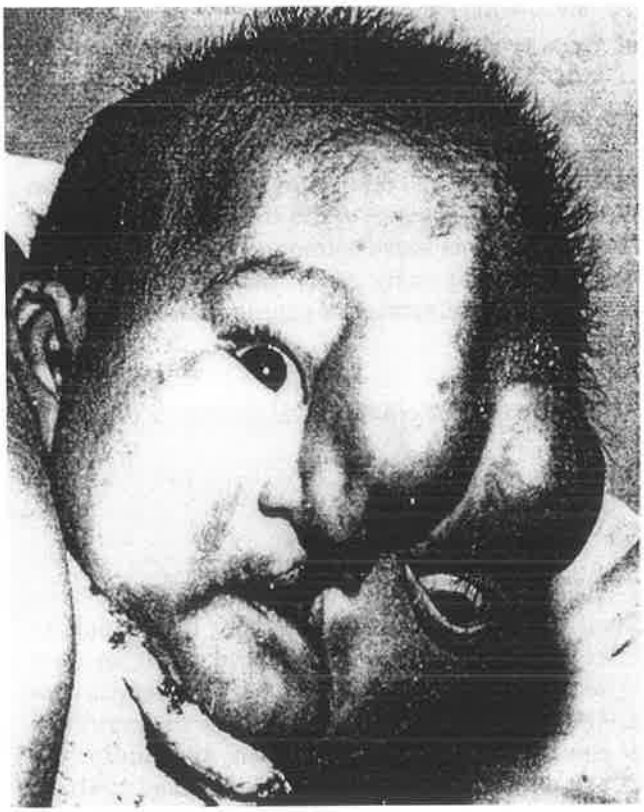

FIG 3. *Infantile teratoma in a 3-month-old boy. Separate areas of teratoma in nasal region, in left orbit displacing left eye, and several intracranial loci. Associated cleft lip and palate.*

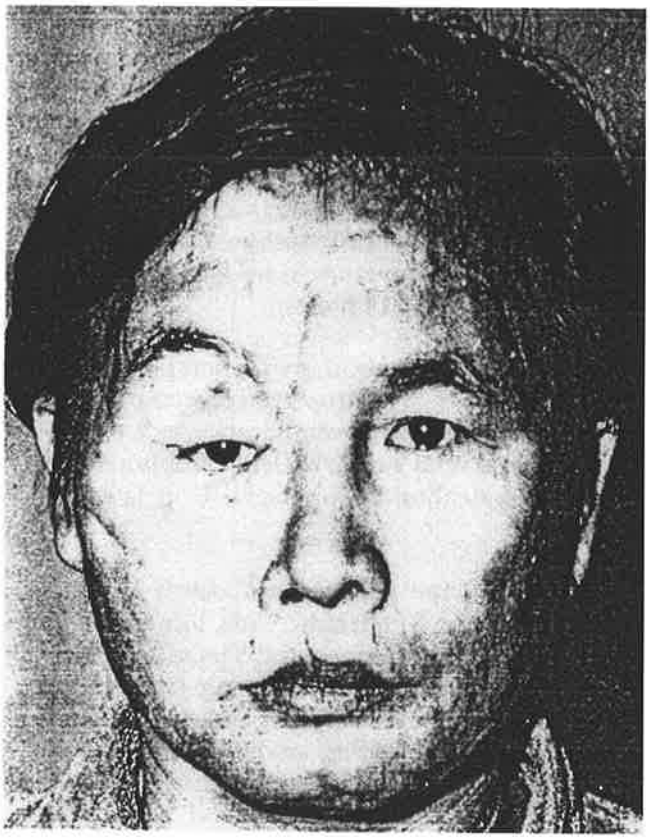

FIG 4. *Right orbital dystopia and enophthalmos following trauma in a motor vehicle accident.*

Cranio-facial surgery

Cranio-facial surgery is a recent surgical discipline whose origins can be traced back through several continents and disciplines. The major advance causing the formation or crasnio-facial surgery was undoubtably the work of Paul Tessier[6] in France, who combined the techniques of plastic and neurosurgery to develop the cranio-facial team, Table II. Numerous writers[7,8,9] have described the reasons behind the neccessity for the cranio-facial team for treatment of cranio-facial malformations. We concur that the team approach is essential to cranio-facial surgery.

Surgical Techniques

Fundamental to cranio-facial surgery is extensive access to the cranio-facial skeleton. This is achieved by a bicoronal scalp flap which can be turned down to reach the orbit, and intra-oral incisions which allow access to the mandible and maxillae for lower osteotomies. The trans-cranial approach using the skills of neurosurgeon and plastic surgeon allows access to the superior orbital region and permits complete osteotomies of the orbit, thus allowing shift of the bony orbit and its contents. In addition the neurosurgeon and plastic surgeon can resconstruct the frontal bone to fit the new contour of the face.

Amongst the most significant of recent advances in cranio-facial surgery is the extension of surgery to operations performed in infancy, particularly for correction of craniosynostosis syndromes. The release of vault and basal structures and the advancement of the fronto-orbital complex at about 3 months of age may allow a significant degree of normal facial growth, and prevent or decrease later deformity of the face.

Results and Complications

Since its inception in 1975, the South Australian Cranio-facial Unit has had a steady increase in referals each year. In the first five years of operation, 1975 – 1980, 230 patients were refered to the Unit. Of these, 27 were from overseas, mostly from the South-East Asian region, especially Malaysia, Singapore and Hong Kong. During the 5 year period 150 operations were performed by the Unit of which 45 were transcranial procedures. The average time for trans-cranial procedures was 12 hours, the longest taking over 16 hours.

There are several reports in the literature dealing with complications of major cranio-facial surgery. In 1979 Whitaker et al[10] reported on complications in 793 cranio-facial operations. They showed an overall complication rate of 16.5% with a 25.7% complication rate for trans-cranial surgery. Using similar criteria the complication rate from the South Australian Cranio-facial Unit is seen in Table III.

Of the two patients with blindness, one suffered unilateral blindness following trans-cranial surgery for Crouzon Syndrome. This blindness was complete and permanent and due to optic nerve ischaemia. The other patient with blindness suffered unibilateral blindness, again following trans-cranial surgery for Crouzon Syndrome. In this latter case there was an undetected platelet abnormality which did not show up in routine clotting studies performed pre-operatively. During surgery there was a major haemorrage requiring massive transfusion. Post-operatively a very large frontal extra-dural haematoma developed which displaced the entire cerebral tissue posteriorly and placed traction on the optic nerves. The exact nature of blindness in this case is uncertain, but the patient is regaining light perception in the lower field of vision suggesting optical tract or occipital damage rather than optic nerve pathology. Both patients with CSF leak required a second trans-cranial operation to repair the dura and subsequently made excellent recoveries. In both cases the leak of CSF was from an area of dura which appeared intact at the initial operation.

One patient developed an acute extra-dural heamatoma shortly after initial trans-cranial surgery and this was removed and the bleeding vessels coagulated without residual neurological deficit. Several patients suffered loss of bone graft, always after sub-cranial surgery. Occular problems such as ptosis and strabismus were common findings immediately post operative but all cases corrected spontaneously within 12 months.

There were several minor infections involving bone graft, all in cases of sub-cranial surgery. There were no major infections. No patient of ours died as a result of cranio-facial surgery.

The success can in part be related to the pateints feelings about the results. Nearly all the patients who underwent trans-cranial surgery were happy with the results. Most dramatic improvements were encountered in trans-cranial correction of Crouzon Syndrome and gross hypertelorism. Most patients undergoing sub-cranial surgery were happy with the results. Two patients who had sub-cranial maxillary osteotomies were unhappy with the result, partly due to continued growth of the mandible which reduced the effect of surgery.

Discussion

We believe that the results obtained from cranio-facial surgery fully justify the cost of such procedures. There are a wide range of deformities which can now be successfully corrected surgically, and new techniques may further extend the scope of this sub-specialty. In particular our experience combining cranio-facial techniques suggests that this technique will allow far more effective reconstruction

TABLE II

Cranio-Facial Team
Cranio-facial Plastic Surgeon
Neurosurgeon
Microsurgeon
Ophthalomologist
Otorhinolaryngologist
Medical Geneticist
Anaethetist
Neuroradiologist
Cardiologist
Dentist
Orthodontist
Prosthodontist
Psychiatrist
Social Worker
Speech Pathologist
Medical Illustrator

TABLE III

Complications of Cranio-Facial Surgery

Complications	Number of Cases			
	Transcranial	Subcranial with orbits	Subcranial without orbits	Total
	(n=45)	(n=20)	(n=85)	(n=150)
Death	0	0	0	0
Major Infection	0	0	3 (4%)	3
CSF Leak (requiring repair)	2 (4%)	0	0	2
Extradural Haematoma	2 (4%)	0	0	2
Blindness	2 (4%)	0	0	2
Occular Muscle Palsies (Permanent)	2 (4%)	1 (5%)	0	3
Partial Relapse of Advancement	0	0	4 (5%)	4
Total	8 (18%)	1 (5%)	7 (8%)	16 (11%)

of many facial deformities. This applies particularly to deformities such as hemifacial microsomia which do not require trans-cranial surgery.

The low incidence of cranio-facial deformities and their variable expression, combined with the immense potential hazards of surgical correction convince us that this sort of surgery should only be undertaken in a few highly specialised units who can achieve excellence through high work loads. We believe that the hinterland population for each unit should be at least 30 million people.[6,7,8,9] Our increasing experience with these cases, especially those requiring trans-cranial surgery, is allowing us to anticipate and correct problems before they become significant. We are also now able to reduce total operation time, with consequent improvement in post-operative recovery and reduction of post-operative complications.

New concepts of early surgery to prevent or minimise future deformity offer exciting new possibilities in cranio-facial deformities. many deformities associated with craniosynostosis such as plagiocephaly and midface stenosis may result as secondary effects from primary sutural problems. Once such facial deformities are established they can be extremely difficult to correct. the possibility of preventing or minimising such deformities by surgery in early life

is worth persuing. It is too early for long term clinical evaluation of these concepts. Experimental work is now being undertaken to validate or refute these theories.

Finally the physician's primary concern must be the patient's happiness and achievement of a fulfilling role in society. The cranio-facial team must act as a focal point for treatment and psycho-social support, and at the same time must play a major role in educating the public to develop a more humane attitude towards people suffering from severe cranio-facial demormity.

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Aetiology and Pathogenesis

David, Poswillo, Simpson

A cranial suture can be said to have fused prematurely when there is evidence of bony union (synostosis) occurring before the normal time of closure: the earlier the fusion, the more definite the abnormality. With certain exceptions, notably the mendosal and metopic sutures, sutures which close in childhood are abnormal. Premature sutural fusion is therefore a pathological process, but it has no aetiological specificity. It may occur in association with quite diverse abnormalities in skull growth, and at least three distinct categories of craniosynostosis can be identified.

Premature fusion of one or more cranial sutures may be found as an idiopathic developmental error. This is *primary craniosynostosis* and it may be seen in otherwise normal individuals, or as part of complex syndromes involving other developmental aberrations. The cause or causes of primary craniosynostosis are still unknown, but the condition must result from some intra-uterine growth defect and the resulting deformities are usually obvious at birth. Some of these certainly result from genetic errors. There are well recognised syndromes associated with craniosynostosis which show Mendelian inheritance: in the Crouzon and Apert syndromes, autosomal dominant inheritance seems established, while the Carpenter syndrome is clearly an autosomal recessive trait. Other types of primary craniosynostosis are also sometimes familial (Cohen 1979). In these circumstances a genetic defect is doubtless responsible, perhaps expressed through some enzymal abnormality. However, in the large majority of cases of primary craniosynostosis there is no familial incidence, and the evidence for a genetic defect is at best speculative.

Some writers have postulated antenatal mechanical causes (Doerr 19493). David Smith (Graham et al. 1979) recently suggested that the scaphocephalic deformity may result from intra-uterine cranial compression. This condition is indeed often associated with a history of obstetrical difficulties, though in our opinion these are more likely to be effect than cause: the head is usually large and the synostosis may prevent moulding. Teratogens may also deserve consideration. Aminopterin, for example, is known to cause a malformation syndrome of which craniosynostosis can be a part (Straw and Steinbach 1968; Powell and Ekert 1971). Craniosynostosis has been reported in association with various chromosomal defects (Cohen 1979; see Appendix B). At present, these appear to be rarities of little clinical importance, but this is an expanding field and more refined techniques of investigation may change our views. In the present state of knowledge, it seems wise to emphasise that in most cases of primary craniosynostosis, the cause or causes remain unknown, and an aetiological classification is impossible. Cases are best classified for clinical purposes on the basis of the morphology, and this is considered in Chap. 9.

There are also cases of *metabolic craniosynostosis:* premature sutural fusion resulting from disturbances of growth determined by obvious biochemical disorders. These are of great theoretical interest, and some practical importance; raised intracranial pressure may result and operative intervention may be needed. Table 3.1 lists the principal conditions reported to be occasionally complicated by craniosynostosis, together with our own experience. We have twice had to

The Craniosynostoses, Springer-Verlag 1982, 35–41

perform bilateral decompressive craniotomies to relieve raised intracranial pressure evidently due to this complication. In one instance, the child, a 4-year-old male cretin, presented with papilloedema and radiographic evidence of extensive symmetrical craniosynostosis as well as increased convolutional markings (Fig. 3.1); both resolved after bilateral flap craniotomies. This child had certainly received excessive thyroid hormone over a long period (Penfold and Simpson 1975). Robinson et al. (1969) report a somewhat similar case of Graves' disease also requiring operation for craniosynostosis.

Our other operated case was a 16-month-old girl with quite severe familial hypophosphatasia. At 9 months, she was found to have premature fusion of the

TABLE 3.1.

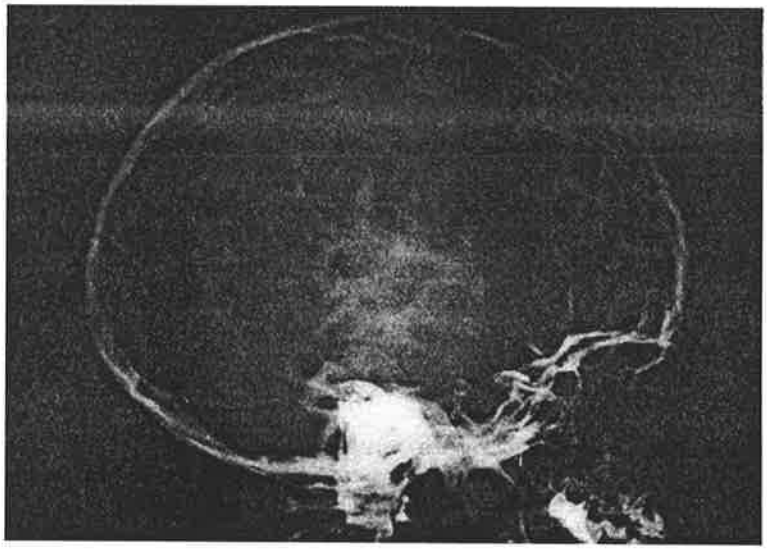
Metabolic disorders sometimes complicated by craniosynostosis

Metabolic diseases	References	South Australian Experience
Rickets		
Vitamin D-deficient	Reilly et al. 1964	–
Vitamin D-resistant	Reilly et al. 1964	–
Renal rickets	Reilly et al. 1964	–
Other rachitic states	Reilly et al. 1964	1 case
Familial hypophosphatasia	Currarino et al. 1957	1 case (operated)
Hyperthyroidism		
Spontaneous	Robinson et al. 1969	–
Iatrogenic	Menking et al. 1972; Penfold and Simpson 1975	3 cases (1 operated)
Haematological disorders	Gooding 1971	–
Polycythaemia		–
Thalassaemia major		–
Idiopathic hypocalcemia	Gooding 1971	–
Mucopolysaccharidoses	Gooding 1971	2 cases[a]

[a]Not included in series

coronal and metopic sutures. At 16 months she was found to have bilateral papilloedema. Bilateral craniotomies were performed, and at operation it was noted that the squamosal suture was also synostosed. Biopsy of the fused sutures was uninformative. The papilloedema resolved. The child's metabolic condition has become less severe with increasing years, as is usual in this condition, and she remains a healthy and intelligent child.

Metabolic craniosynostosis can thus be of clinical significance. The iatrogenic cases also provide experimental evidence to show that disproportion between skull growth and brain growth can result from arrest of growth of the vault sutures, though of course this arrest might itself be secondary to a disturbance of growth of the base.



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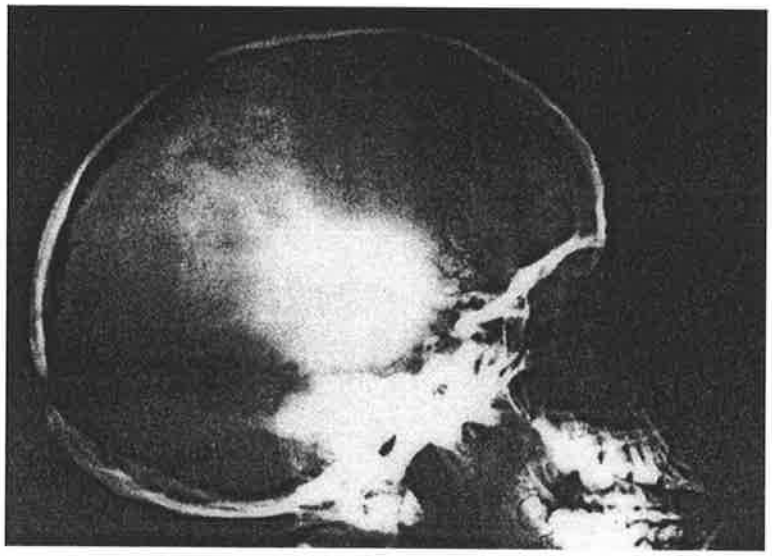

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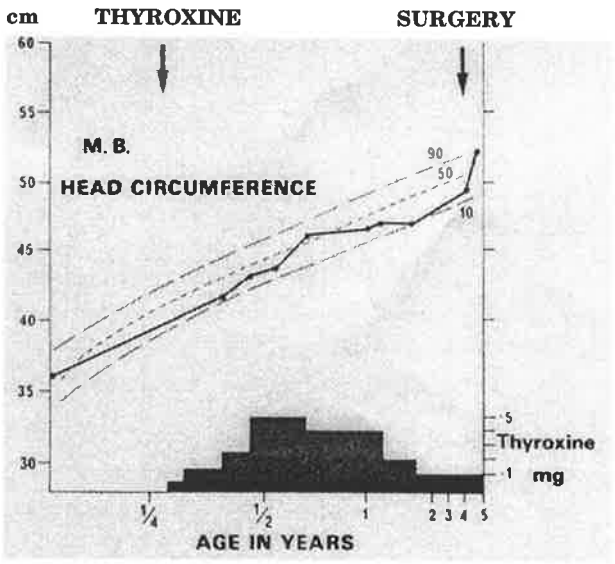


C

FIG 3.1 A. *Metabolic craniosynostosis: premature sutural diffusion in a 4–year old child with cretinism and iatrogenic hyperthyroidism.* **B.** *The same child after bilateral osteoplastic decompressions and restoration to a euthyroid state.* **C.** *Chart of head circumference in the same child: an initial acceleration of skull growth was followed by reduced growth and the appearance of papilloedema. The head enlarged again after bilateral decompression. From Penfold and Simpson (1975), by permission of* The Journal of Paediatrics.

Finally, failure of brain growth may result in premature sutural fusion. This is seen in microcephaly, and any encephaloclastic process may have the same result if it strikes during the first few years of life (Fig. 3.2). Premature sutural fusion is also often seen when severe hydrocephalus has been treated with a low-pressure shunt (Andersson 1966; Roberts and Rickham 1970; Fig. 3.3). These conditions represent *secondary craniosynostosis.* We have never seen any need to operate on this type of craniosynostosis, unless to perform subtemporal decompressions to allow dilatation of slit ventricles as part of the management of intermittent shunt malfunction. Secondary craniosynostosis is not considered in this book, except in differential diagnosis. The process is however of much theoretical interest since it illustrates the dynamic relationship between continued cerebral growth and sutural separation.

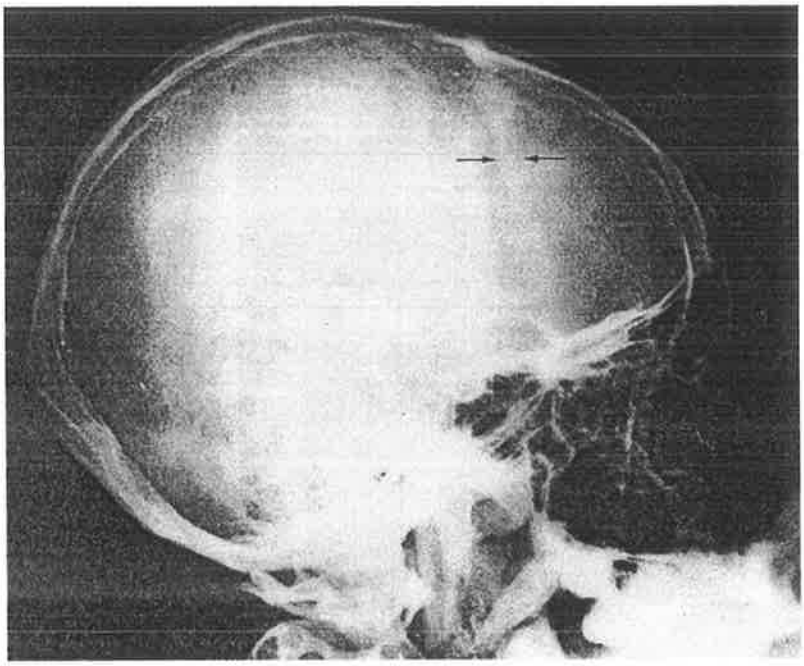


FIG. 3.2. *Premature fusion of the coronal suture* (arrows) *appearing after a severe attack of meningitis in a boy aged 5 years.*

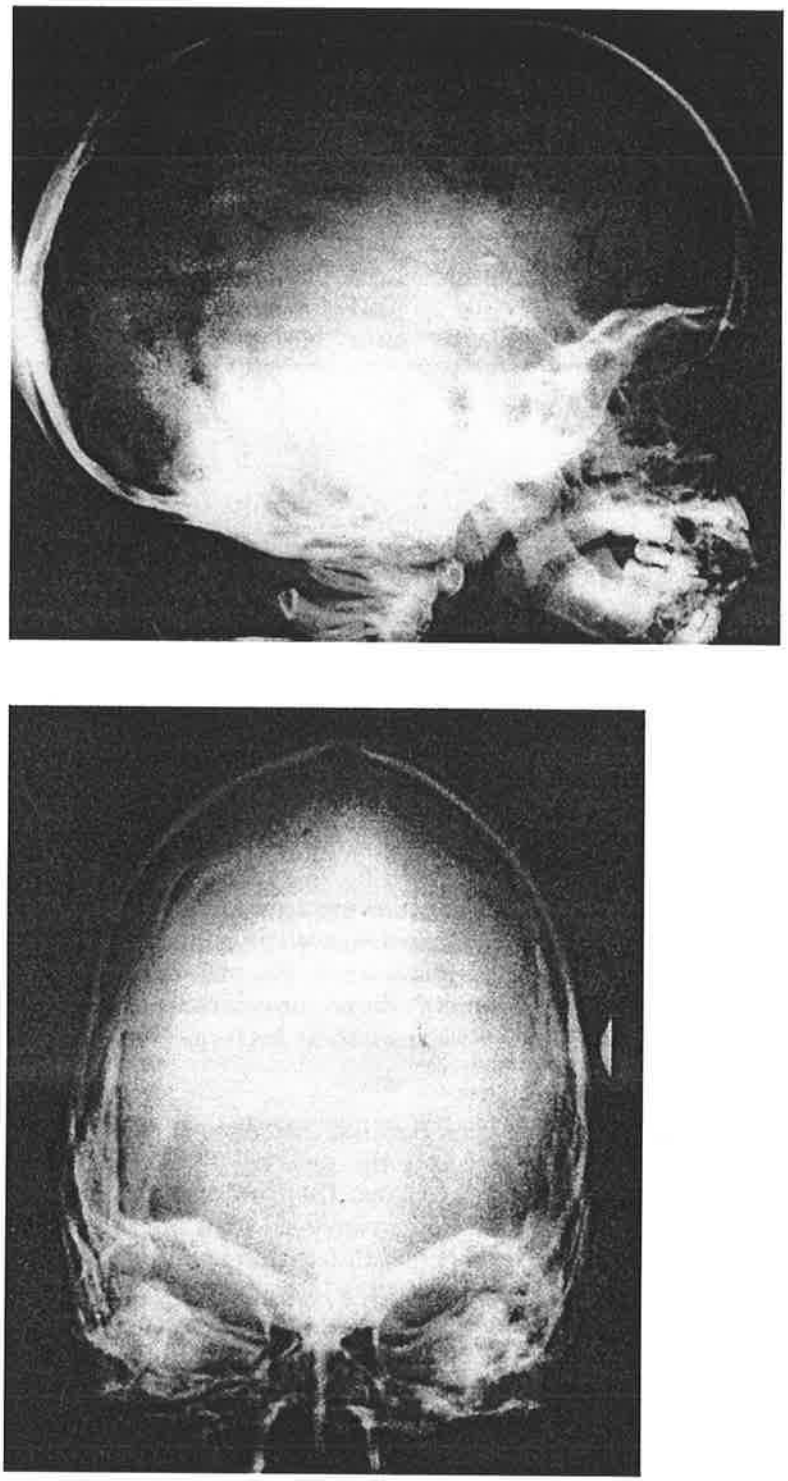


FIG. 3.3. *Premature fusion of the sagittal suture after ventriculo-atrial shunt. The head is very narrow and elongated, with a depression behind the vertex.*

Table 3.2 sets out our experience of primary craniosynostosis. The term simple craniosynostosis is taken to imply premature fusion of one or more calvarial sutures, without serious involvement of the facial skeleton or convincing evidence that the craniosynostosis is part of some recognisable malformation syndrome.

TABLE 3.2.

Craniosynostosis: Adelaide series. Cases are classified as familial if one or more first-degree relatives are affected.

			Familial	
Category of synostosis		**No. of cases**	**No. of cases**	**%**
Simple primary:	*All types*	125	11	8.8
Complex primary:				
	Crouzon etc.	25	9	36
	Saethre-Chotzen	2	–	–
	Carpenter	2	2	100
	Apert	6	–	–
	Various	3	–	–
	All types	38	11	28.9
Metabolic:	*All types*	5	1	20
Total		168	23	13.7

The classification of simple craniosynostosis is further discussed in Chap. 9. Complex craniosynostosis includes the cases with evidence of involvement of the facial skeleton as part of a defined malformation syndrome, or with evidence of a significant associated extracranial malformation, such as syndactyly. These cases have been subclassified as specific syndromes: the criteria for these diagnoses are considered in Chap. 15. The metabolic craniosynostoses are added for completeness. Cases are said to be familial when at least one first-degree relative appears to have the same condition

From the aetiological viewpoint, does familial incidence imply genetic causation? And does sporadic occurrence imply the reverse? As Cohen (1977, 1979) has said in two excellent reviews of this problem, there are many difficulties here. In our cases with familial simple craniosynostosis (two families with scaphocephaly, two with turricephaly, and one with frontal plagiocephaly) the incidence in siblings or parents could mean mendelian inheritance, and probably it does. This, however, is not to say that the remaining 96 cases are necessarily the victims of non-genetic agents: their deformities could result from new mutant genes, or incomplete penetrance, or variable expression. There is reason to believe that a gene causing turricephaly with coronal synostosis in one sib may express itself as marked brachycephaly *without* synostosis in another sib or in a parent. This observation accords well with the thesis that craniosynostosis is an extreme expression of an aberrant growth pattern rather than a localised pathological process. But from the nosological viewpoint it becomes difficult to say that a sporadic case of primary craniosynostosis is certainly non-genetic.

The complex craniosynostoses also present problems. All the conditions listed by us in this category and most of those listed in Appendix B are regarded as hereditable on a monogenic basis, but in fact there is no family history in two-thirds of our cases. Of course, very disabling conditions like Apert syndrome tend to be self-limiting, and some of our cases are too young to have reproduced yet. But in the Crouzon cases there are other possible explanations. Affection of other family members may have been overlooked. In outspoken cases of this condition, the diagnosis is easy, but the expressivity of the condition varies and mild forms may be missed, or assigned to the category of simple craniosynostosis.

It is even more likely that our interest in the Crouzon syndrome has led to over-diagnosis and inclusion in this group of other craniofacial malformations that may have quite different genetic significance. It is also conceivable that a simulation of the Crouzon phenotype, or any other craniofacial syndrome, might be produced by some unknown environmental agency. We think that this is unlikely, but in the present state of knowledge it is hard to dismiss the possibility.

There is as yet no biochemical or chromosomal laboratory test for the familial types of craniosynostosis, with the exception of those rare syndromes related to trisomy or deletion of specific chromosomes (see Cohen 1979 and also Appendix B). Diagnosis still rests on clinical and radiological recognition.

In summary, though an important minority of cases shows evidence of genetic causation, there are many which do not and which presumably represent some sporadic yet stereotyped interference in cranial growth. Even if we say that all the cases which we have classed as complex craniosynostosis do represent genetic errors. we are still left with a large majority — in our series nearly 70% — whose aetiology remains mysterious.

Pathology

David, Poswillo, Simpson

From the pathologist's viewpoint, craniosynostosis can be regarded as a normal developmental process occurring at an abnormally early age. It is true that the affected sutures may at times show hyperplastic changes not seen in sutures closing normally during adult life. There is however little or nothing in the sutural pathology of craniosynostosis to suggest that the process differs fundamentally from normal sutural closure, except in its timing. Indeed, the histopathology of the prematurely closed sutures has been disappointing as a source of information on the primary cause or causes of craniosynostosis, except in a negative way.

The cranial deformities represent the secondary pathology of craniosynostosis They can be studied in the dried skulls rightly treasured in many museums. Such specimens amplify and correct the impressions derived from radiographs and operative explorations. They are however rare, since most forms of craniosynostosis are compatible with long life, and they often lack clinical documentation. Opportunities for autopsy come rarely and often to the wrong pathologist. It is one of the responsibilities of craniofacial surgeons to ensure that autopsy material is never wasted: there is still much to be learned about the pathology of craniosynostosis This is true of the cranial pathology, as will be seen in the chapters of this book dealing with the individual calvarial deformities (Chaps. 9–14) and the craniofacial syndromes (Chap. 15). It is still more true of what can be called the tertiary pathology of craniosynostosis: the changes in the brain, the organs of the special senses, and the facial viscera.

The Sutures

The fused sutures of the cranial vault may be seen as broad ridges of solid bony overgrowth (Fig. 10.2), or may be completely obliterated and indistinguishable from the surrounding bone. As Laitinen (1956) has noted, the formation of a ridge is especially characteristic of the fused sagittal suture; a similar ridge is sometimes seen along the course of the coronal suture. We have never seen a fused squamosal or lambdoid suture as a ridge of this type; Laitinen (1956) concurs with this. Premature fusion of basal sutures also tends to be inconspicuous: a synostosis of the sphenofrontal or sphenozygomatic sutures may be hard to detect in radiographs or even at operation.

Laitinen reported at some length on 20 biopsies of clinically fused sutures. He found no histological evidence of any abnormality except early cessation of growth. Our experience of 38 biopsies has been similar. In the majority the suture was completely obliterated. In neonates, the bone in the region of the fused suture usually showed a fine cancellous structure, with trabeculae lying parallel to the external surface; in older children, there was lamellar deposition of bone under the periosteum, with evidence of remodelling and osteon formation on the inner surface. In a few cases the site of premature fusion could be deduced from an

atypical orientation of trabeculae running perpendicular to the bone surface, or more convincingly from the finding of a plate of inert fibrous tissue embedded in the bone (Fig. 4.1). Albright and Byrd (1981) have reported similar findings. They emphasised that in young subjects fusion is often confined to a localised segment of the suture.

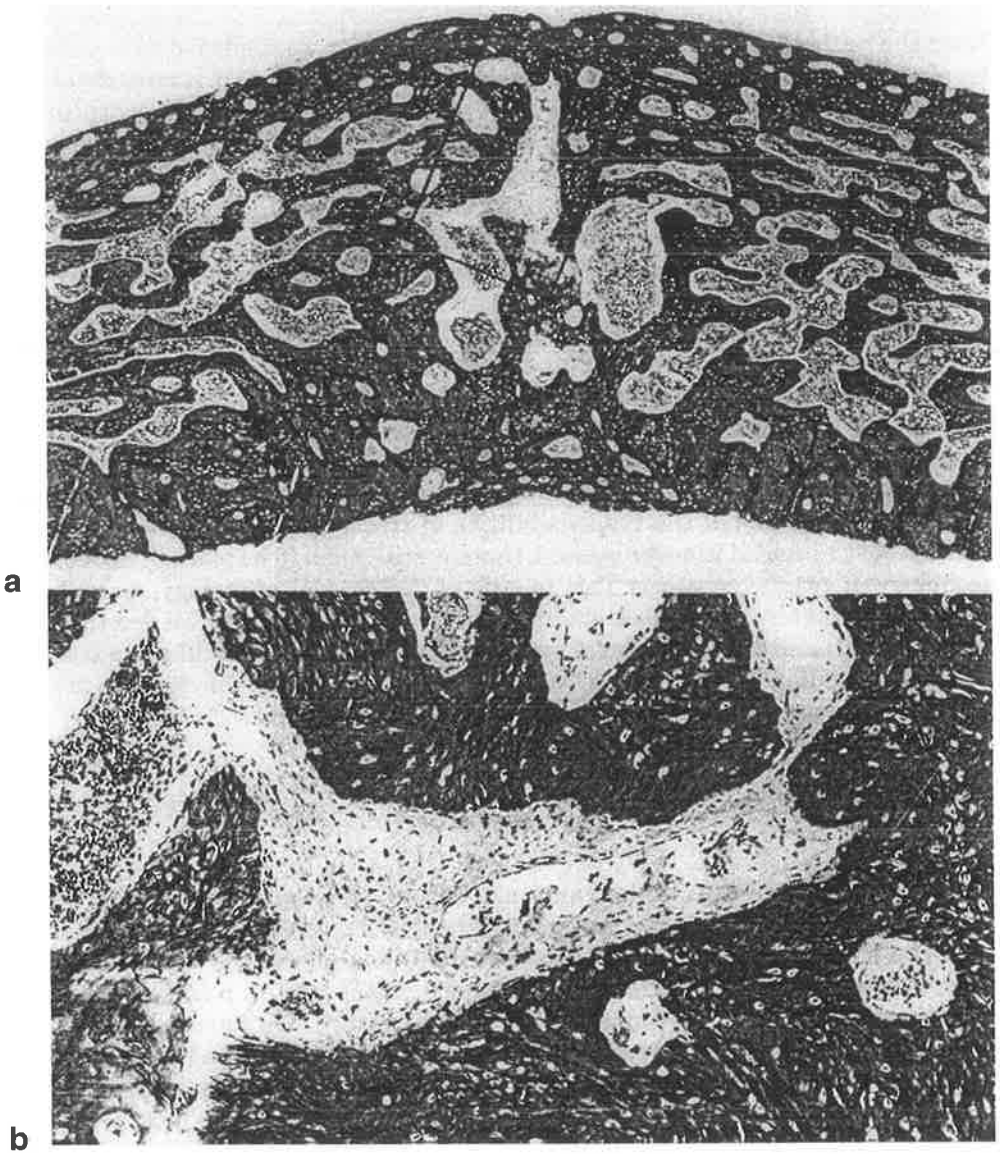


FIG. 4.1 a,b. *Fused coronal suture from infant with Crouzon syndrome; aged 8 weeks.* **a.** *The suture incompletely fused, but can be identified as a low prominence on the external surface, and by a change in trabecular pattern. In the centre there is an island of fibrous tissue, which is presumably a remnant of the suture. H & E × 25* **b.** *Higher magnification shows osteoblasts along the margins of the enclosed fibrous tissue, which is loose and sparsely cellular. It contains a small sinusoid. H & E × 100*

Particular interest attaches to biopsies taken from suture which are incompletely fused. The unfused segment usually shows a histologically recognisable suture, with however much less evidence of active osteoblastic growth than in a normal subject of the same age (Fig. 4.2; compare with Fig. 4.3). We have seen only one exception. in a biopsy from a radiologically sclerotic lambdoid suture, which showed evidence of active osteoblastic growth and a nodule of what appeared to be cartilage. This could be interpreted as the earliest stage of active sutural fusion. This interpretation is questionable; rather similar appearances

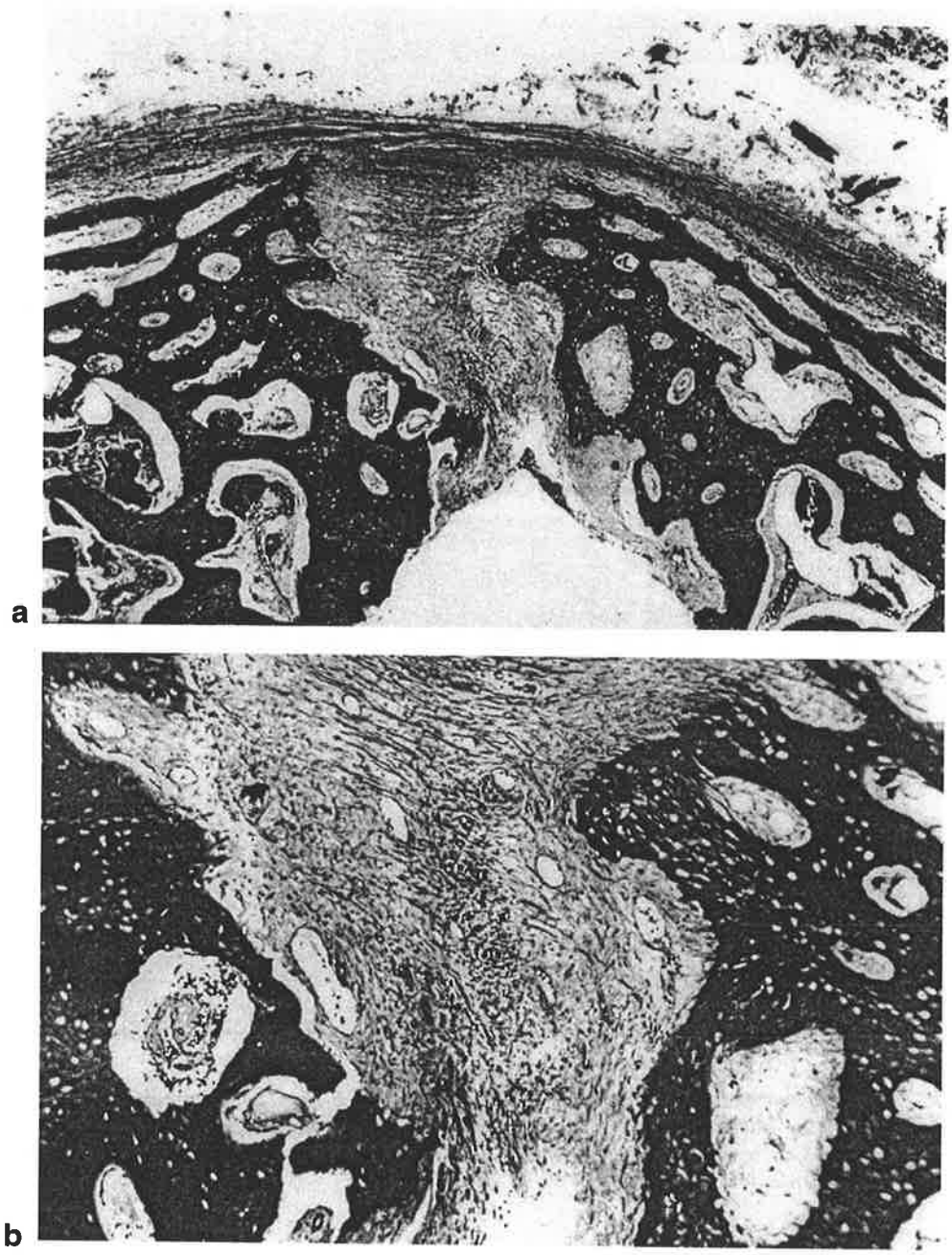


FIG. 4.2 a,b. *Fusing sagittal suture from an infant with scaphocephaly : age 10 weeks.* **a.** *The apposed bone edges are composed of woven bone without much orientation. There are many large collagenous fibres bridging the suture: they are oriented parallel to the pericranium. Masson × 40* **b.** *Higher magnification shows few osteoblasts, and many small sinusoids within the suture area. Masson × 100*

have been seen in fusing sutures of an infant who underwent shunt surgery for hydrocephalus. It is possible that the cellular dynamics of this type of secondary craniosynostosis are different from those of primary premature fusion: in another case (Fig. 4.4) we have seen strikingly osteoclastic activity, a finding not recorded in our other biopsies. The histopathology of all types of craniosynostosis deserves further study by modern histochemical methods, and consideration should be given to the facial as well as the calvarial sutures. Hitherto, neurosurgical biopsy material has been taken chiefly from the cranial vault. It has become increasingly evident that in many forms of craniosynostosis the basal sutures are also involved, but what meagre histopathological evidence we have at present indicates that craniosynostosis of such sutures as the sphenofrontal shows no special peculiarities.

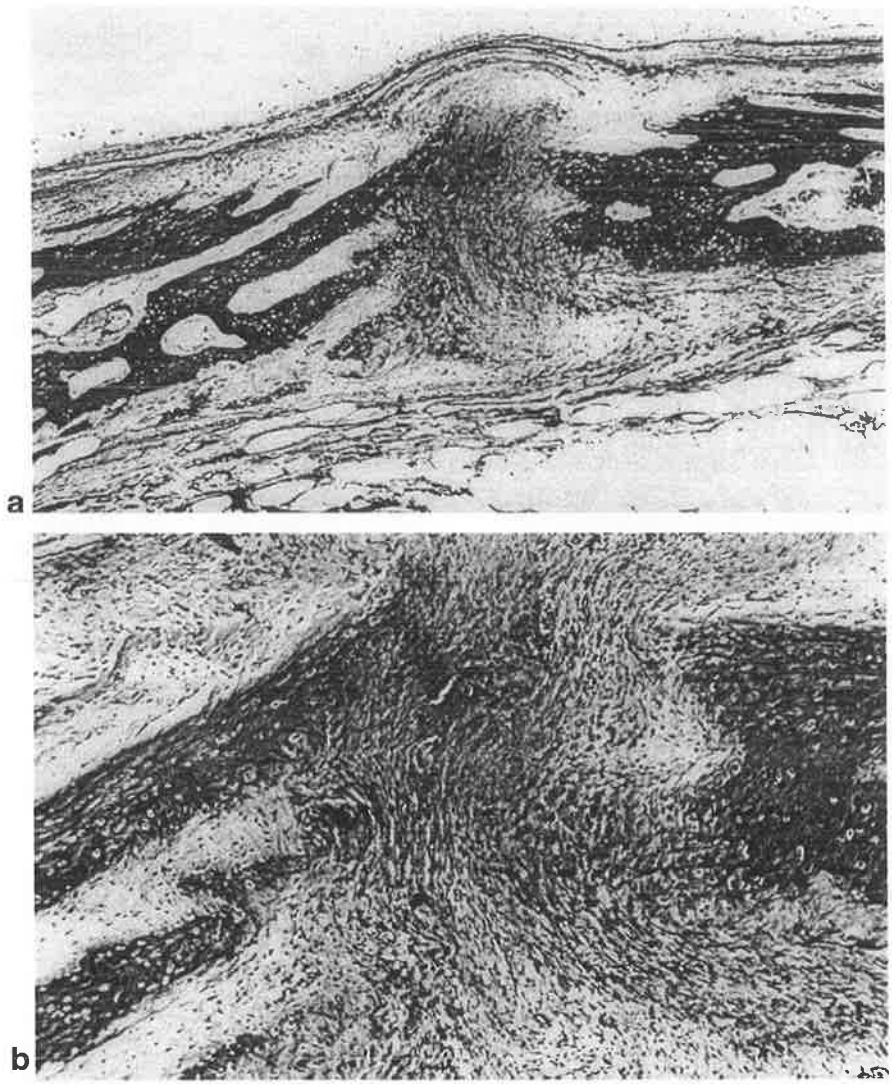


FIG. 4.3 a,b. *Normal sagittal suture from infant aged 5 months. The bone edges are composed of fine horizontal spicules with evidence of active osteoblastic activity at their tips. The capsular layers are well formed, and trans-sutural bridging fibres are not seen There are no sinusoids.* **a.** *Masson × 40* **b.** *Masson × 100*

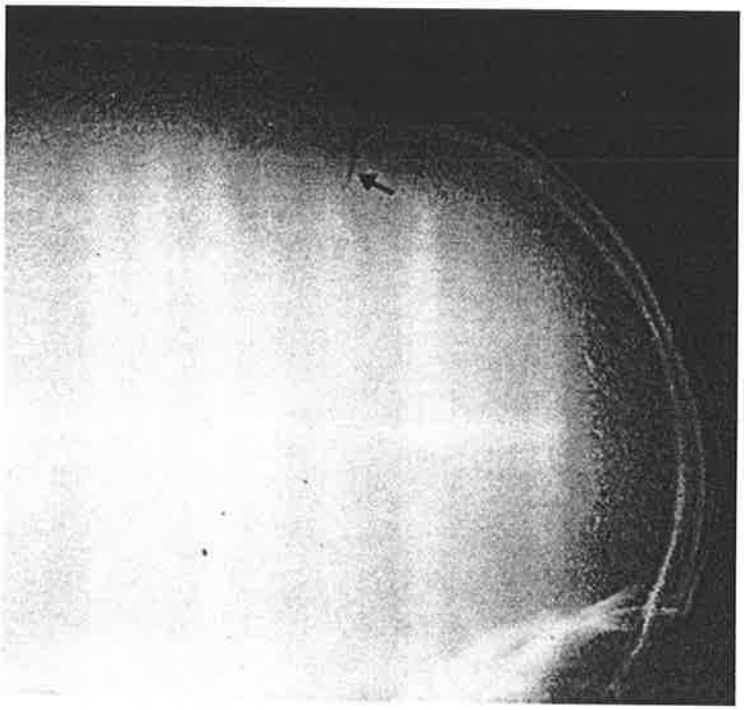


FIG. 4.4. a.

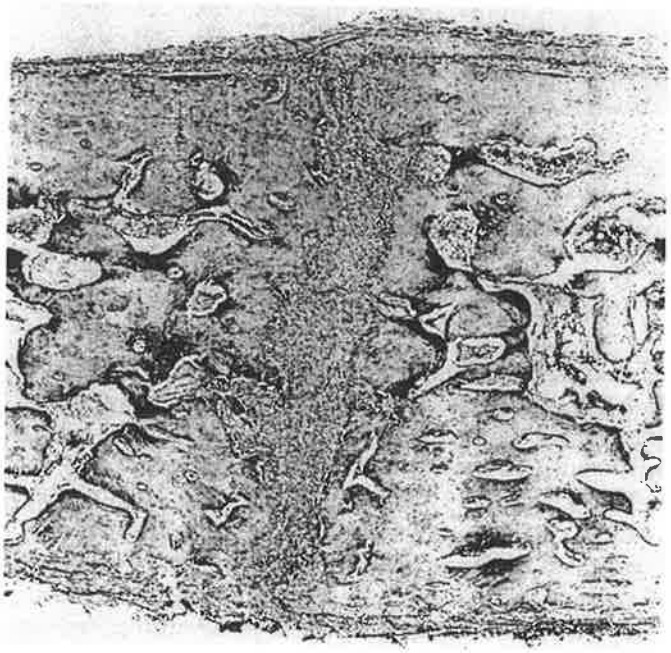


FIG. 4.4. b.

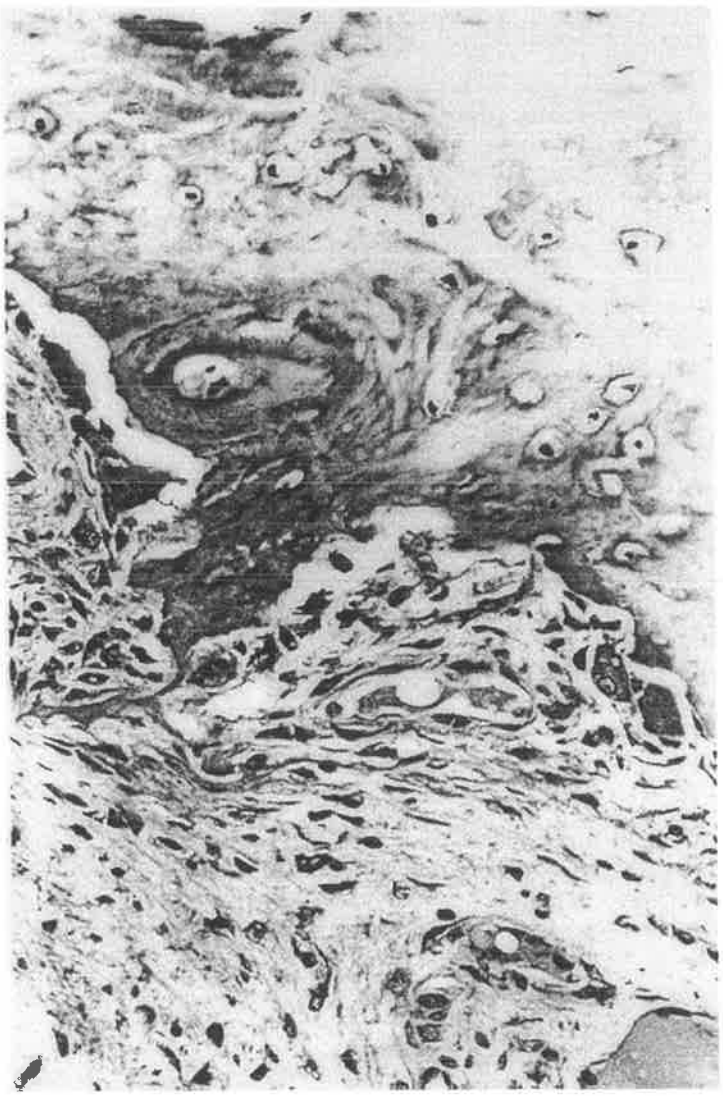


FIG 4.4. c

FIG. 4.4 a – c. *Premature sutural fusion after spino-peritoneal shunt.* **a.** *The coronal suture* (arrow) *is patent medially but fused elsewhere.* **b.** *Biopsy of coronal surface; H & E ×24.* **c.** *Higher power view shows numerous osteoclasts; this appearance has not been seen in spontaneous craniosynostosis. H & E × 300.*

The synchondroses of the skull base also deserve further consideration. As Moss (1959, 1975) has insisted, many — perhaps all — forms of vault synostosis are associated with deformities of the base; even if one does not wholly accept Moss's concepts of causation, it would be useful to know what abnormalities are seen in the chondrocranium. Unfortunately, this area is inaccessible to biopsy, and autopsy studies are few, except on cases of Apert syndrome In that syndrome, marked abnormalities have been seen. Kreiborg et al. (1976) found a very distorted skull base with a wide and highly abnormal spheno-occipital synchondrosis. Montaut and Stricker (1977) also reported abnormalities of the chondrocranium with block fusion of the petrous and spheroid bones on one side. Stewart et al. (1977), however, studying a foetus of about 21 weeks' gestation, found that the chief basal synchondroses were normally formed. Thus, even in Apert syndrome, no clear or consistent pattern to premature fusion in the synchondroses has been established; and there are many reasons (see Chap. 15) to suggest that this condition is a disease sui generis. For the Crouzon syndrome and the simple forms of craniosynostosis. one can only say that gross radiological abnormalities of the basal synchondroses have not been found.

Changes in the Skull Due to Raised Intracranial Pressure

In most cases of clinically evident deformity associated with craniosynostosis, the skull is distorted, but otherwise shows no abnormalities beyond the synostosis. There is no reason to believe that the volumetric capacity of the skull is reduced by premature fusion of one or two sutures, though as Muke (1972) has shown, the methods of determining cranial volume from radiographs are still very unsatisfactory. When multiple sutures are fused prematurely, the cranial volume is likely to be reduced, and the skull in such cases shows pathological changes indicative of raised intracranial pressure. These include convolutional impressions (circular or oval areas of thinning of the cranial vault) and the formation of small cerebral hernias in areas of even more defective cranial growth. Whether convolutional impressions—evident on radiographs as 'hammer beating' or 'digital markings'—are a specific result of raised intracranial pressure has been challenged: Crome (1961), for example, regarded them as only one manifestation of the general osseous dysplasia. We believe that severe convolutional impressions, as seen in Fig. 3.1, do indeed result from chronic raised intracranial pressure, and the same is true of the small cerebral hernias often seen in association with severe craniosynostosis. These may occupy potholes in the overlying bone, much like those described by Russell (1949) in her classic description of the effect of chronic hydrocephalus.

Skull radiographs of children with extensive craniosynostosis often show deep impressions made by the cranial venous sinuses and large venous emissary foramina. These also probably represent the pathological effects of chronic elevation in intracranial pressure in a child whose skull is unable to expand (Fig. 8.7).

Cerebral Pathology

Much of our concern over craniosynostosis, and many of the controversies over the treatment of the condition, relate to possible detrimental effects on the brain. Several writers have asserted that cranial deformities cause mental or neurological disabilities by constricting or distorting the brain, but these assertions need objective proof. After all, every neurosurgeon knows that the brain tolerates moderate intracranial hypertension very well, and every obstetrician knows that the infant's brain tolerates distortion within quite wide limits. It would be most helpful to know in detail the cerebral pathology in the different types of premature craniosynostosis. Unfortunately, relevant autopsy studies are few. Cases of Apert syndrome have often come to autopsy; Blank (1960) referred to 12 post-mortem examinations and there are many other reports of single cases. Some of these have revealed significant neuropathological abnormalities. Palacios and Schimke (1969) found pachygyria and hydrocephalus. Crome (1961) found no gross abnormalities in his case, but saw evidence of neuronal scarcity both in the neocortex and in the hippocampus. Other writers have mentioned agenesis or hypoplasia of the corpus callosum and ventricular dilatation The implications of these serious but inconstant pathological findings are discussed in relation to Apert syndrome in Chap. 15. Reports of adequate autopsies on cases of Crouzon syndrome are not numerous. Eshbaugh (1948) indeed found 24 cases in the literature, but most of these were not reported in sufficient detail. Her own case exhibited pathological changes, notably ventricular dilatation, a deep cerebral constriction due to bony deformity and hyperplasia of the leptomeninges: this was a very severe example of the Crouzon syndrome, however, and therefore of less relevance in the evaluation of the average case. Neuropathological studies on the cloverleaf skull deformity (see Chap. 14) have chiefly shown the effects of the coexisting hydrocephalus which is usually

associated with this form of multiple craniosynostosis. Gross (1957) reported careful autopsies on three cases of Apert syndrome, one case of Crouzon syndrome, and one case of turricephaly. These also were examples of very severe malformations leading to early death. Herzog's (1914) case of so-called *Turmschädel* is of more interest, since the patient was mentally normal and lived to the age of 5 years. He then became blind and died after operative treatment. Autopsy showed no neuropathological abnormalities attributable to the skull deformity, except evidence of atrophy of the visual pathways. The skull showed premature fusion of a number of sutures in the vault and also in the base. This appears to have been a very careful autopsy and the histopathological methods of the period were probably sufficient to exclude diffuse cortical damage. Reports of autopsies on examples of the much commoner synostoses of single sutures are much harder to find. We have records of autopsies of two cases of scaphocephaly, and these did not disclose any significant cerebral abnormalities. Other autopsy data will be discussed in relation to the various categories of craniosynostosis. It can be said that there are no striking or constant neuropathological changes associated with premature sutural fusion.

The paucity of autopsy evidence can be supplemented by contrast neuroradiology and computerised topography (CT). Many writers have reported that ventricular dilatation is often associated with craniosynostosis. Bertelsen (1958) found 19 examples in 48 air studies, but thought that in some of these the dilatation represented cerebral atrophy. Fishman et al. (1971) studied 14 cases, eight being examples of complex syndromes. There was no uniformity in the severity or nature of the hydrocephalus; one very severe case (with gross cerebral deformity) showed aqueduct stenosis, but others had communicating hydrocephalus. Montaut and Stricker (1977) performed pneumoencephalography in 36 of their 161 cases. They found ventricular dilatation due to aqueduct stenosis in five; only one study showed diffuse cerebral atrophy—a case of trigonocephaly.

Our own experience has been much smaller; like Foltz and Loeser (1975), we have not wished to perform pneumoencephalography unless there were compelling reasons.

Even CT has been used only when major craniofacial surgery was contemplated. In seven patients we found moderate to severe degrees of hydrocephalus in association with Apert, Carpenter, and Crouzon syndromes (see Chap. 15). In 17 other cases of synostosis in which the ventricular system has been delineated, no major abnormalities have been seen. However, CT scanning has occasionally suggested constriction or obliteration of subarachnoid cisterns, which could be taken to result from a relative reduction in the cranial capacity.

Putting together the evidence from autopsies and the neuroradiological findings, one can say that serious cerebral anomalies are most often found in association with the genetically determined craniosynostoses and especially with Apert syndrome. Such anomalies are much rarer as part of the Crouzon syndrome, but have been recorded. The chief secondary neuropathological complication is hydrocephalus, which may be severe enough to demand treatment. This is especially seen in cases of the cloverleaf skull deformity. It is not at present clear whether this hydrocephalus usually represents a primary aqueduct stenosis, or whether it results from the constrictive effect of the cranial malformation. Gross (1957) suggested that a severe kyphotic deformity of the skull base may distort or constrict the brainstem and produce hydrocephalus by compressing the basal cisterns or the cerebral aqueduct. A parallel could be drawn with achondroplasia, where hydrocephalus certainly seems to result from the distortion of the skull base. Hoffman and Hendrick (1979) have postulated chronic increased venous pressure as a cause of hydrocephalus. This is not a very convincing explanation. There is certainly evidence of raised venous pressure in many cases of severe craniosynostosis, but analogy with other conditions would suggest that this is

likely to promote chronic cerebral swelling rather then ventricular dilatation. Meningeal fibrosis (Eshbaugh 1948) is another possible cause of hydrocephalus complicating Crouzon syndrome. Evidence of cerebral pathology in other forms of craniosynostosis has been hard to find, and certainly there are few convincing reports of cerebral damage directly related to the distorted shape of the cranium. It is however true to say that minor degrees of cerebral damage would escape detection in routine post-mortem examination.

Significance of Craniosynostosis as a Pathological Process

It was shown in Chap. 2 that modern concepts of skull growth do not give the sutures a driving role in the expansion of the cerebral capsule. Rather they are zones where marginal bone deposition proceeds in response to the demands of the expanding brain and eyeball, probably transmitted along lines of dural (or periorbital?) tension. Accepting this, Moss (1959, 1975) has argued that premature sutural fusion is secondary to more fundamental dysplasias of the skull base. In this view, synostosis is a symptom, not a cause of deformity.

We do not wholly accept this. Certainly our studies of the histology of the sutures do not reveal any local pathology likely to play a primary role. It seems likely that the individual deformities associated with craniosynostosis represent dyscephalies due to disturbed growth of the entire cerebral capsule, the cranial base as well as the vault, the pericranium and dura as well as the bone (Fig. 4.5). There is however some evidence, both experimental and clinical, to suggest that premature sutural fusion does perpetuate or even exaggerate the general disturbance of growth. For surgeons the most compelling evidence is the response to adequate resections of fused sutures. Removal of bone is, often quite predictably, followed by varying degrees of correction of a calvarial deformity. Bone grows again from the dura mater and from the margins of the removal, but often this regrowth does not amount to complete synostosis and there may be radiological evidence of a new suture in the regenerated bone (Shillito 1973, Fig. 4.6). The reappearance of a previously obliterated suture surely suggests that regional cranial growth has been released by operation, allowing the brain to expand in a more normal way. If so, it seems logical to regard the premature sutural fusion, however caused, as an important pathological process, since it can lead to local distortion or restriction of the expansion of the brain. When craniosynostosis is extensive, it may impair the normal relation between cerebral growth and enlargement of the cerebral capsule: the skull may be too small for the growing brain. Intracranial pressure may then rise, either permanently or as an episodic consequence of some kind of cerebral swelling. Vision may be affected, and perhaps mentality, though this is much less certain. When the cranial capacity is reduced like this, the traditional term *craniostenosis* seems appropriate (Müke 1972).

In the same way, one can speak of *orbitostenosis*. The orbit is the bony capsule of the expanding eyeball; unlike the skull it is an incomplete capsule, being open anteriorly. In certain craniofacial syndromes, above all the Crouzon syndrome, the dysplasia affects the walls of the orbit. It is not wholly clear what part in this is played by premature fusion of the various orbital sutures, but they are probably affected and orbital craniosynostosis has been recorded (Greig 1926; Kreiborg, personal communication). In consequence the orbital cavity is distorted: it is shallow and wide, and the eyeball is extruded forwards (exorbitism).

Delaire et al. (1963) used the analogous term *faciostenosis* to describe the midfacial hypoplasia of Crouzon syndrome. Delaire considered this to be a stenotic process in the bones surrounding the upper airway. He also postulated that faciostenosis may occur as an isolated condition. It has been suggested that the facial sutures may undergo premature synostosis, and Tessier (1971c) has

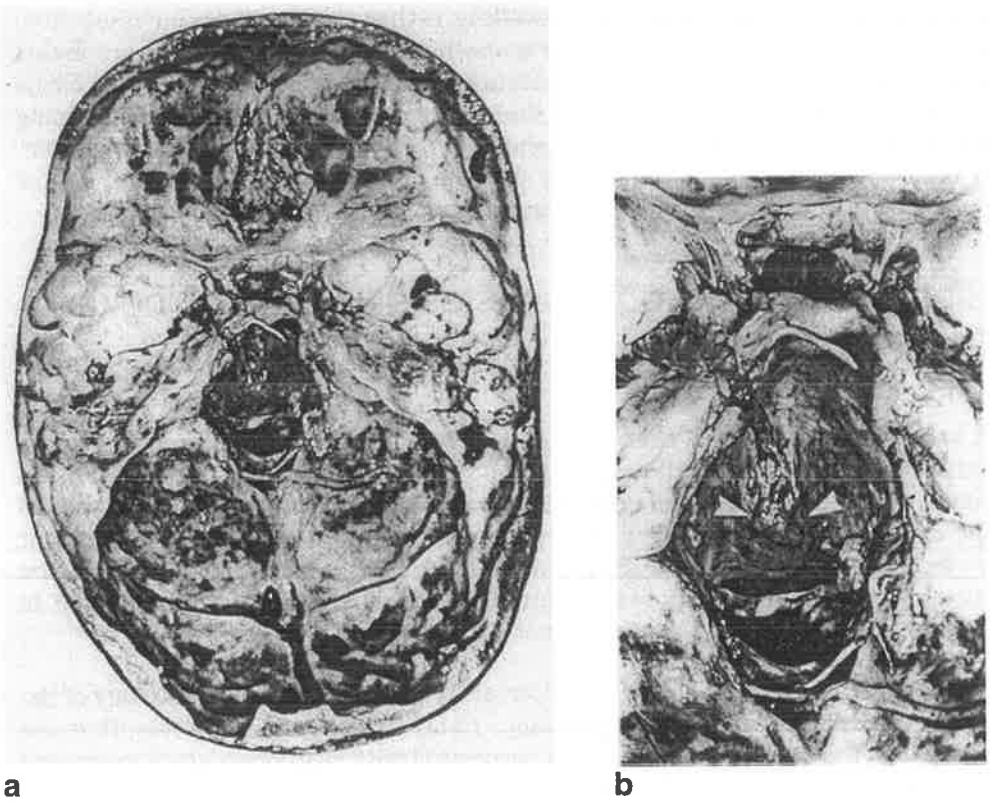


FIG. 4.5. a. *The skull base in Crouzon syndrome. There are deep convolutional impressions, especially in the middle fossae, and the grooves of the sagittal and transverse sinuses are also unduly deep.* **b.** *The basioccipital bone is distorted; there is a dorsal protruberance* (arrows) *and the foramen magnum is somewhat constricted. By courtesy of Mr. R. W. Hiles, Frenchay Hospital, Bristol.*

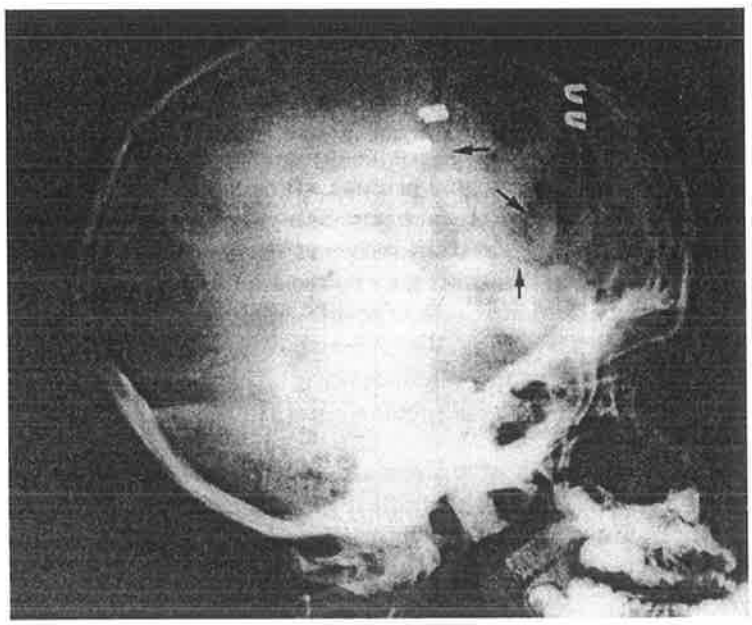


FIG. 4.6. *New suture lines* (arrows) *have been formed in the new bone regenerated from the dura after coronal linear craniectomy.*

observed complete fusion of the maxillary and spheroid bones. The pathological evidence is however meagre. It is still uncertain whether the maxillary hypoplasia of Crouzon and Apert syndromes represent an intrinsic local growth failure, associated with premature sutural fusion, or whether it is secondary to a primary dysplasia of the skull base, acting in varying permutations on growth of the vault and facial skeleton. Begging this very important question, we use the term faciostenosis to describe the state of narrowing or constriction of the facial skeleton, causing secondary functional disorders in breathing, eating, and speech.

In summary, we see premature sutural fusion as an important local manifestation of an underlying defect in the growth of the skull. This defect may be regional, as in the simple calvarial deformities, or it may be generalised: examples of generalised disorders of skull growth are seen in the metabolic craniosynostoses and in the more severe types of complex craniofacial deformity. Thus far, our concept is in accord with Moss's (1959) argument that craniosynostosis is not a primary disease process. However, we believe that premature calvarial sutural fusion has very real significance in the dynamics of abnormal craniocerebral growth. It results in a relatively unyielding cerebral capsule which fails to respond normally to the forces exerted by the expanding brain. There is now experimental (Persson et al. 1979) as well as surgical evidence to support the classical concept of the role of the sutures in determining to some extent the nature and severity of deformities of the skull vault. It is not yet possible to say whether premature fusion of facial sutures has similar autonomous importance in determining the character of facial deformities. Even if future research shows that this is not so, we believe that these deformities also represent the outcome of regional skeletal growth failure.

Surgical Management

David, Poswillo, Simpson

Introduction

In the preceding chapter we have given our views on the natural history of the complex craniofacial deformities associated with craniosynostosis. Except in the mildest forms, we believe that operative correction is needed. Originally the responsibility of neurosurgeons, this has become the work of the craniofacial team—a partnership of plastic surgeon and neurosurgeon with specialist colleagues capable of dealing with all the technical necessities likely to arise in the treatment of the patient as a whole. For the patient and his family the operative intervention, however dramatic, may be only a grand episode in a long programme of rehabilitation. But it is the technical ability to operate that makes the programme possible. This ability, pioneered by Tessier, is based on a thorough knowledge of the pathological anatomy associated with the craniosynostoses, and with all the related problems. It is this as much as anything that justifies the centralisation of craniofacial surgery in selected units, which enables the operating surgeons to build up an experience of pathological anatomy in this exacting field. In craniofacial surgery one deals with an infinite variety of three-dimensional distortions and with a multiplicity of organ systems packed into a small space. Experience is indispensable: yet each patient has unique problems and as Tessier (1971a) has said, 'each surgical case becomes an experimental case' (Fig. 16.1).

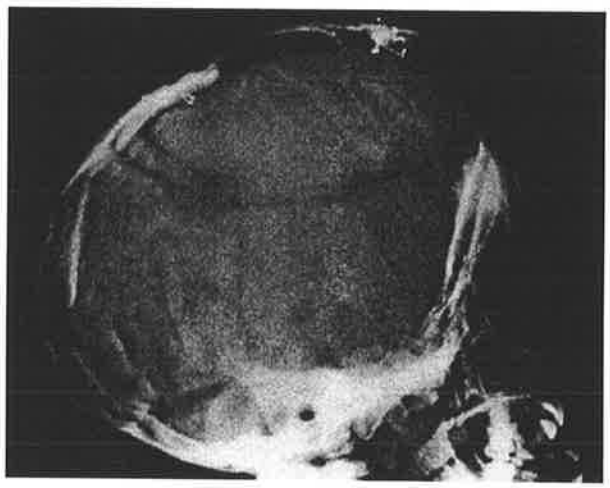


FIG. 16.1. *Lateral skull radiograph of a case of Carpenter syndrome demonstrating the bizarre cranial base, and almost complete absence of the orbits, together with the evidences of previous surgery done elsewhere.*

Primary Skeletal Pathology

As Converse et al. (1974) pointed out, two bones are key structures in the distorted anatomy of the complex craniofacial deformities: the sphenoid and the ethmoid. The sphenoid (Fig. 16.2) is a single bone, situated in the base of the skull but reaching the lateral walls. It consists of a centrally placed body, which is surgically almost inaccessible, the paired greater and lesser wings, and the paired pterygoid processes. The greater wing of the sphenoid forms the anterior part of the middle cranial fossa. the posterior part of the lateral wall of the orbit, and the anterior

medial wall of the temporal fossa. It has sutural articulations with many bones. The frontal, the zygomatic, the parietal and the squamous temporal bones all have sutural relationships with the greater wing and these may be fused prematurely, though this is hard to show radiologically. The lesser wing of the sphenoid separates the anterior and middle cranial fossae. It borders the superior orbital fissure and it has a sutural articulation with the frontal bone, the sphenofrontal suture, which is often fused prematurely in craniosynostosis. The lesser wing is continued laterally as the sphenoidal ridge. It is of great neurosurgical importance that this ridge is exaggerated in many forms of complex craniosynostosis (as well as in simple turricephaly): it may sweep like a sabre blade high around the lateral convexity of the calvaria, with an associated dural fold that indents the brain deeply in the line of the sylvian fissure. The pterygoid processes project down from the body of the sphenoid, and articulate by sutures with the palatine bones. They form the lateral walls of the nasopharynx, the medial walls of the infratemporal fossae, and the posterior walls of the pterygopalatine fossae. Their posterior surfaces front the pterygoid fossae. The pterygoid processes have surgically important vascular relationships. They are surrounded on three sides by a plexus of veins, draining back into the maxillary veins. They also have a close relation with the greater palatine arteries which arise in the pterygopalatine fossae and run down in front of the pterygoid processes to reach the palate through the greater palatine foramen. These large arteries, and the many smaller branches of the internal maxillary artery, should

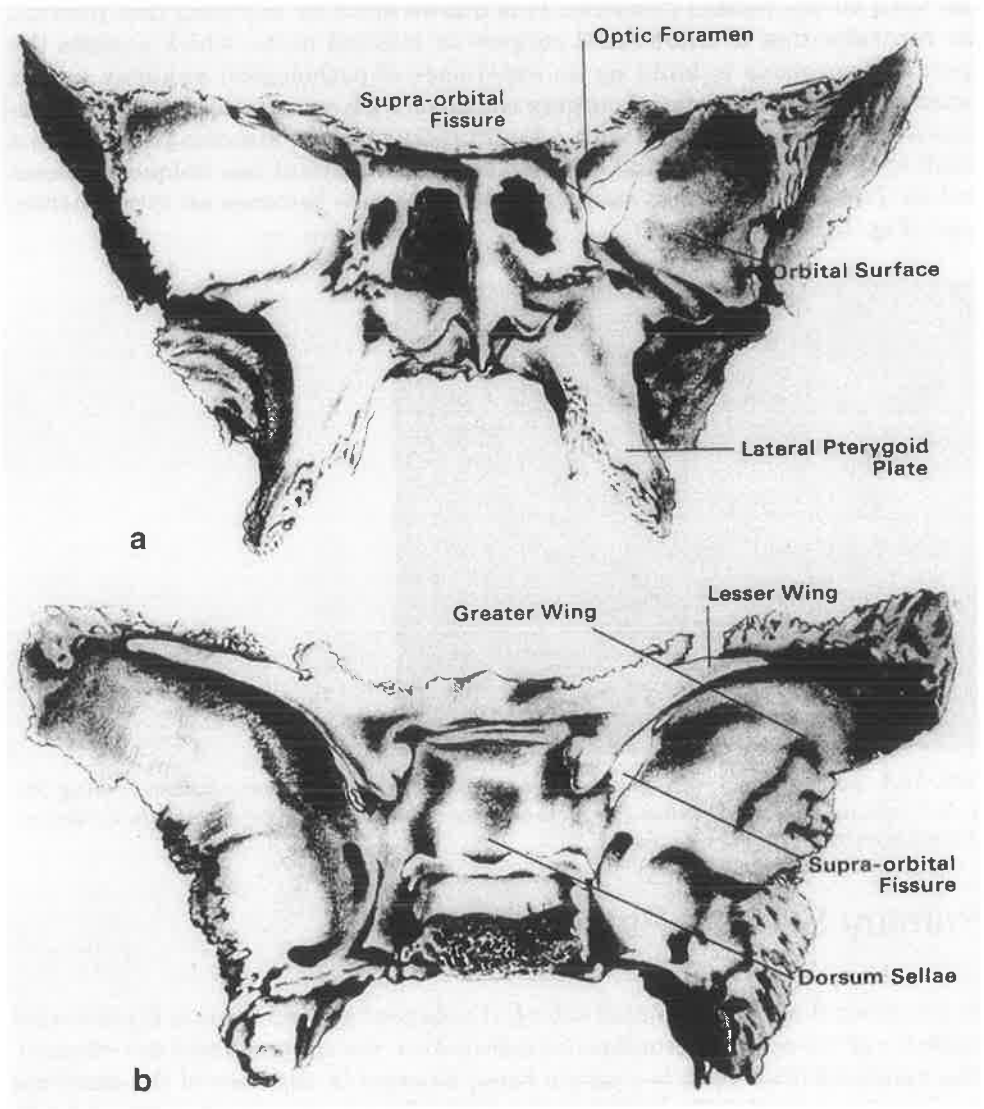
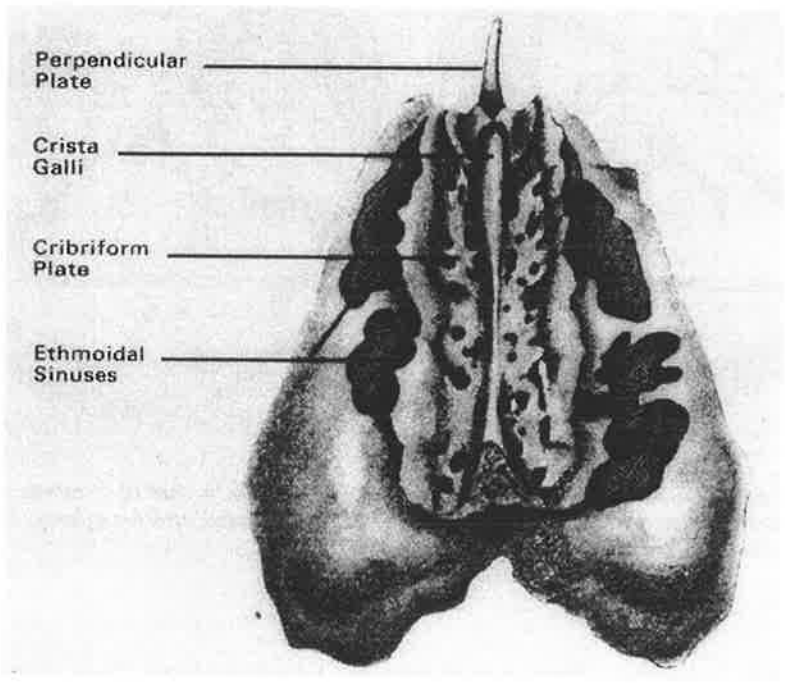


FIG. 16.2a, b. *Sphenoid bone* **a.** *Anterior view* **b.** *Posterosuperior view*

be remembered when maxillary osteotomies are carried out.

The ethmoid bone (Fig. 16.3) lies below and behind the ethmoidal notch in the frontal bone, which is closed by its upper surface. It is applied to the anterior aspect of the sphenoid body and rests below and between the two maxillae. It forms the lateral and medial walls of the upper portion of the nasal cavity, and the medial walls of the orbits. Although the ethmoid bone is wholly formed in cartilage, it articulates with the maxillae and with the frontal, sphenoid, lacrimal and nasal bones by typical sutures. The ethmoid bone consists of three distinct components. The median perpendicular plate forms part of the bony septum of the nose and articulates with the other elements of the septum, the vomer and the septal cartilage. The upper part of the ethmoid is the cribriform plate, forming the cribriform fossae and pierced by the filaments of the olfactory nerves. It is composed of extremely thin bone—often only 100μm thick. The upper part of the perpendicular plate extends above the paired cribriform fossae as the strong process of the crista galli. Anterior to this, in the groove between the ethmoid and frontal bones, is the midline foramen caecum, usually blind but occasionally containing a nasal emissary vein. The dura of the falx cerebri is firmly anchored to the crista galli, and more loosely by a process entering the foramen caecum: this process is a key landmark in anterior fossa extradural dissection. The lateral components of the ethmoid are the paired labyrinths, an intricate series of thin-walled air sinuses bounded laterally by the bony orbital plates. It is the ethmoidal labyrinths that articulate by sutures with the maxillae. The postnatal growth of the ethmoidal air sinuses and the other extensions of the nasal cavity is of considerable importance in craniofacial surgery; studies carried out in our department were reported by Caldicott et al. (1973). Figure 16.4 shows the relationships of these two key bones in the anterior and the middle cranial fossae, and their sutural articulations. Figure 16.5 shows their orbital anatomy.

Pathological distortions in the sphenoid, ethmoid, and frontal bones complicate the surgical anatomy of craniosynostosis. The relationships of the cranial fossae, the orbits, the maxillae, and the nasal cavity may be bizarre. We have encountered, in all possible permutations, the following important skeletal abnormalities in the course of our operations for craniosynostosis.



a

FIG. 16.3a. *Ethmoid bone. Superior view*

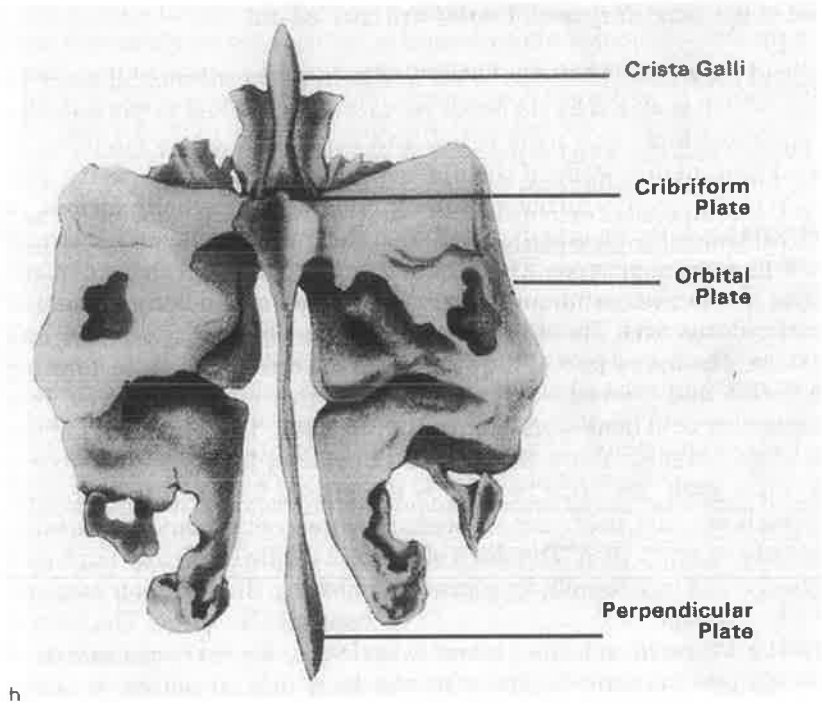


FIG. 16.3 b. *Ethmoid bone. Posterior view.*

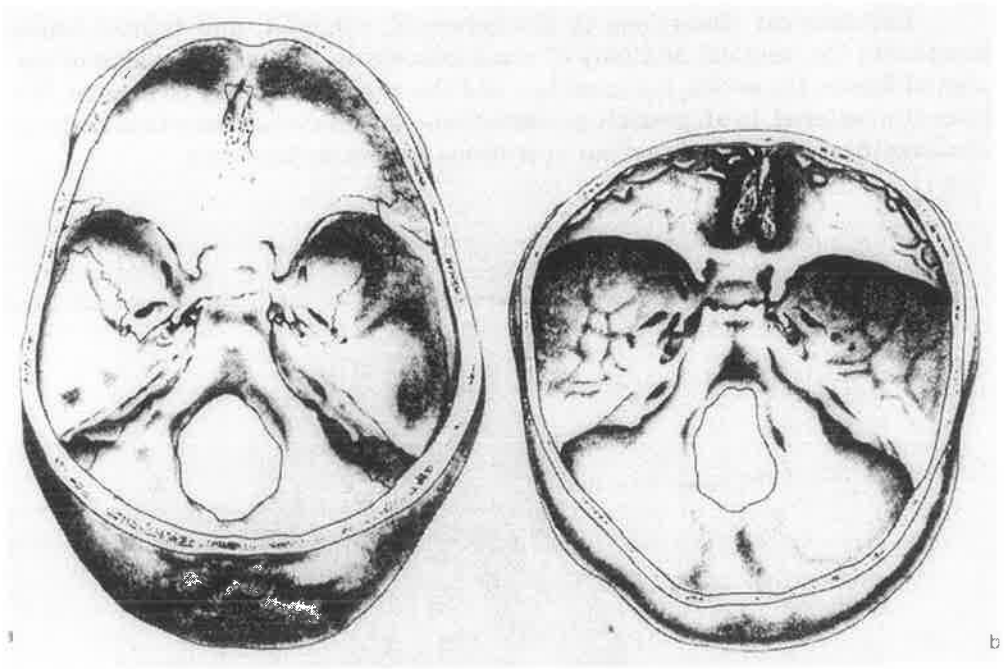


FIG. 16.4 a,b. *The skull base: superior view.* **a.** *Normal.* **b.** *A case of Crouzon syndrome showing foreshortened anterior cranial fossa, with depressed and broadened cribriform plate and bony projections from the orbital roofs.*

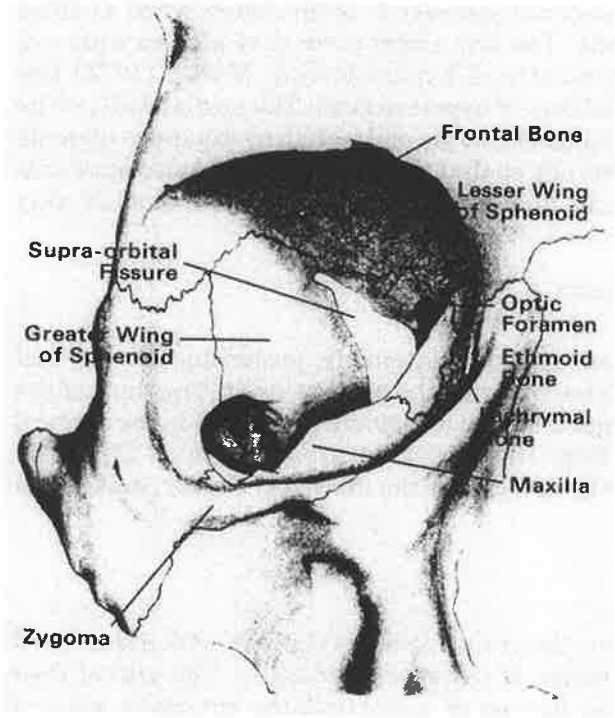


FIG. 16.5. *Orbital anatomy: anterior view.*

Calvarial Deformities

The various calvarial deformities (oxycephaly, turricephaly, and plagiocephaly) may present unexpected distortions and concavities especially in the frontal bone and the supra-orbital margin.

Thin Bone

Raised intracranial pressure thins or even erodes the bone, and the dura may be vulnerable, or even naked, especially in the orbits, where dura may fuse with the periorbita (orbital periosteum). There may be small meningeal hernias embedded in potholes in the bone.

Abnormal Venous Channels

The various emissary veins may be enlarged, also as a consequence of chronic raised intracranial pressure. Normal or abnormal venous drainage related to the sagittal sinus may need careful attention, and we have met formidable bleeding from veins related to the lateral end of the sphenoparietal sinus.

Bony Spikes

One often finds projections of abnormal bone from the roofs of the orbits, invaginating and even eroding the dura.

Swept-up Orbits

The orbital roofs often sweep up laterally in relation to the hypertrophied sphenoidal ridges; medially they are likely to slope steeply down to the cribriform fossae.

Depressed Cribriform Plate

The cribriform plate may be depressed into the nasal cavity, and this depression

is a strong argument for the transcranial approach to ensure safety when a LeFort III osteotomy (see p. 261) is done. The cribriform plate may also be widened, producing symmetrical or asymmetrical hypertelorism. Munro (1976) has emphasised the anatomical variability of hypertelorism. The medial walls of the orbits may be parallel or may be ballooned in the midportion by expanded ethmoid air cells. Or they may diverge laterally so that the dacryon is displaced outwards relative to the optic foramen; in another variant, the optic foramina may themselves be abnormally separated (Fig. 16.6).

Expanded Middle Cranial Fossa

The middle cranial fossa is often extended anteriorly, projecting forward and establishing abnormally close relations with the shallow orbit. The dura of the middle fossa may be in danger during orbital osteotomies and even in the surgical disconnection of the maxilla from the pterygoid process (see p. 258). The hypertrophied sphenoid ridge may invaginate the dura very deeply, making its dissection hazardous.

Distorted Orbital Cavities

In many types of craniosynostosis, the orbital cavity is shallow, with a shortened lateral wall in relation to depression of the zygomatic bones. The orbital floor may also be hypoplastic, and may flow on to merge with the anterior surface of the maxilla with little evidence of an inferior orbital margin. In orbital hypertelorism the position of the optic canal is usually normal, but this is not always so (see above).

Nasal Obstruction

The nasal airway is usually reduced, often asymmetrically, as there is almost invariably some deviation of the bony and cartilaginous nasal septum. Nasal intubation may be difficult especially after medial translocation of the orbits.

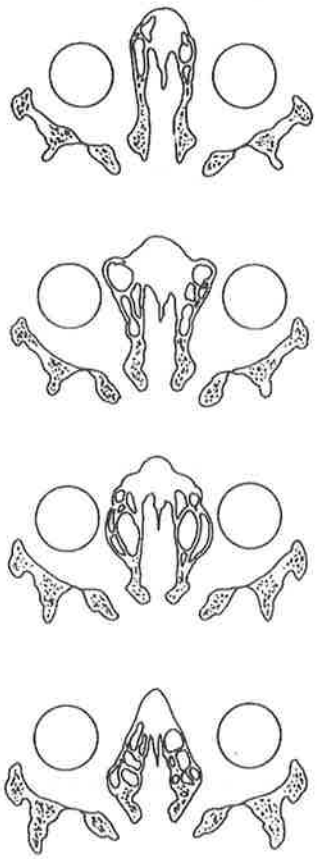


FIG. 16.6. *Horizontal sections through the orbits showing the variable patterns of the medial orbital wall deformity (by courtesy of Dr. IR Munro).*

Maxillary Hypoplasia

The maxilla is usually small in all three dimensions, and it is crowded with teeth. There is little bone, and what is there breaks easily. Maxillary hypoplasia contributes to the reduction of the nasal airway and of course to the dental malocclusion.

All these anatomical abnormalities complicate surgical management. Some of them can be anticipated by careful preoperative study of the radiographic findings. Others are only found during operation.

Secondary Surgical Pathology

Many patients who come to the craniofacial unit have undergone cranial and/or facial surgery already. These earlier operations may bring additional problems, which can be termed the secondary pathology of craniofacial deformities. It is important to have full details of previous surgical interventions. We have encountered the following problems attributable to earlier surgery, sometimes carried out by ourselves and sometimes done elsewhere.

Bone Defects and Cerebral Hernias

Earlier craniectomies may leave unexpected bone defects, where the dura may be attached to the scalp, or may even be deficient. Postoperative meningoceles or meningoencephaloceles are especially dangerous when situated in the orbital roof.

Silastic

Silastic or other plastic strips may have been inserted to delay bone regrowth. With the passage of time the Silastic becomes embedded in new bone and this can make dissection very difficult.

Scars

Earlier neurosurgical incisions may have been placed too far anteriorly, cutting across the blood supply of the standard bicoronal incision, favoured by us (Fig. 8.5b). Previous facial scars may also cause cosmetic problems and may make dissection difficult. This is especially so if a previous attempted craniofacial operation has involved a wide subperiosteal dissection.

Orbitotomies

Previous exploration of the orbits may make it hard to establish a safe plane of dissection. This is especially so when the exophthalmos of Crouzon syndrome has been 'treated' by transfrontal orbital decompression: this procedure is completely ineffective, and may make definitive treatment dangerous by leaving no safe plane of cleavage between the basal dura and the orbital periosteum.

Shunts

It may have been necessary to treat coexisting hydrocephalus by a ventriculo-atrial or ventriculoperitoneal shunt. Subsequent cranio-facial surgery is then unavoidably complicated by uncertainty about the control of raised intracranial pressure during and after operation; preliminary isotope studies of shunt function are usually needed. Findler et al. (1980) have reported the development of a tension aerocele in the extradural space in association with a low-pressure shunt.

Operative Principles

Gillies and Harrison (1950 – 1951) reported the first high maxillary osteotomy done for the maxillary hypoplasia of Crouzon syndrome. The orbital cuts in this procedure (Fig. 16.7) were made quite far forward and the maxilla was divided through the pterygomaxillary fissure and the hard palate. Uncertainty about the anatomy of the distorted cranial base delayed the acceptance of this type of correction. Tessier (1971 a) reported operations differing from Gillies' procedure in that osteotomies were made posteriorly in the orbit behind the lacrimal groove and across the orbital floor into the lateral orbital wall which was split sagittally. The line of section ran through the body of the zygoma as a stepped osteotomy. and the maxilla was separated posteriorly through the pterygomaxillary fissure (Fig. 16.8).

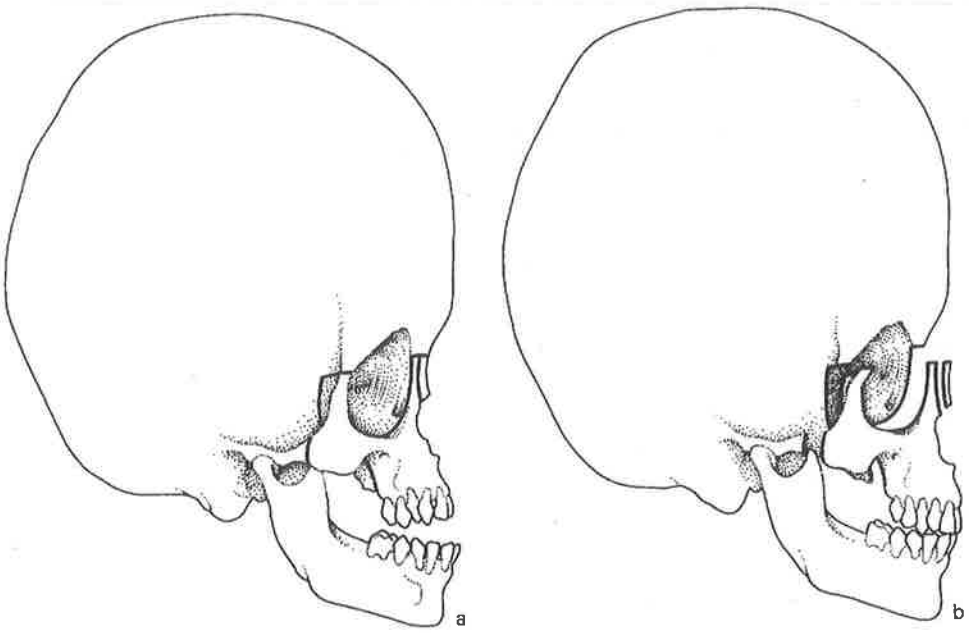


FIG. 16.7 a,b. *The maxillary osteotomy of Gillies and Harrison.* **a.** *The osteotomies pass through the nasal bones, orbital floor, lateral wall of the orbit, zygomatic arch, pterygomaxillary fossa, and across the posterior hard palate.* **b.** *After separation and advancement.*

Murray and Swanson (1968) performed a midfacial osteotomy for craniosynostosis with nasal and orbital osteotomies as in the Gillies procedure. Jabaley and Edgerton 1969) used medial orbital osteotomies like those of Tessier but dissected the whole of the zygoma.

In 1968 Tessier began to treat the calvarial deformity of plagiocephaly by rotating a large frontal bone flap. In 1970 he moved from the correction of established craniofacial deformities in older patients to operations of young children. These were seen as likely to release facial as well as calvarial growth from the restraints imposed by craniosynostosis, a hope that is now widely entertained (Hoffman and Mohr 1977; Whitaker et al. 1977; Marchac 1978) but still unproven (see p. 95).

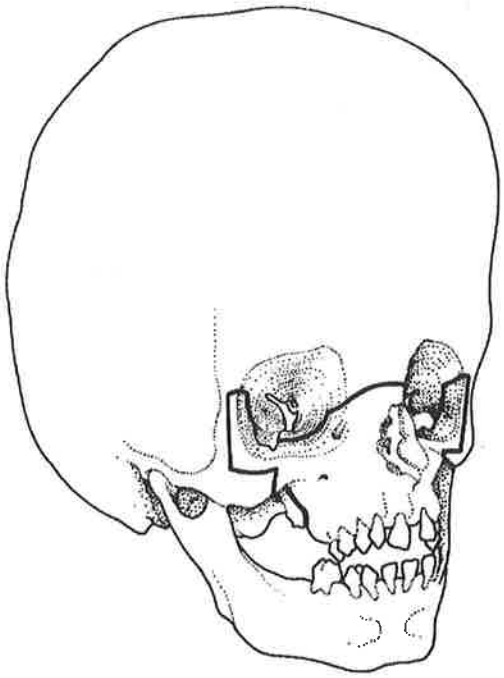


FIG. 16.8. *LeFort III osteotomy according to Tessier with osteotomies behind the lacrimal groove and a step in the zygomatic bone. The osteotomy line passes between the maxillary tuberosity and the pterygoid plates.*

In varying combinations, these operations have interwoven the techniques of plastic surgery and neurosurgery, and ambitious craniofacial operations have become possible. We now possess the ability to move all the components of the cranial skeleton except the deep central block: the skull base between the orbital apices and the posterior fossa. These technical achievements make it possible in a single operation to decompress the brain and to free the fronto-orbital complex by osteotomies extending across the anterior cranial fossa. The growth of the brain and of the orbital contents can continue unrestricted and the skeletal deformities will be ameliorated. It is also now possible to move the midfacial complex and the orbits (except their apices), together or separately, into new positions.

The principles enunciated by Tessier a decade ago are still valid. Dissections should be subperiosteal, to allow the bones to be cut and moved without soft tissue damage. Orbitocranial and faciomaxillary deformities resulting from single pathological process should be considered as one entity with respect to treatment. The transcranial route should be used to correct deformities of the skull base, since it is ultimately safer and simpler. When a complex deformity has multiple components, as many as possible should be corrected in one sitting.

Indications for Operation

Our experience is based on 25 cases of complex craniofacial deformities associated with craniosynostosis treated operatively by the South Australian Craniofacial Unit in the period 1975–1981. Also relevant is experience gained in the same period in 11 cases of simple craniosynostosis treated by similar techniques (Table 16.1).

The indications for intervention may be functional or psychosocial. As has been said in Chap. 6, the functional indications relate to the brain and to the eyes. Raised intracranial pressure from craniostenosis was evident in five of these patients, and demanded operative relief by decompression either early in infancy

or during what we have termed the intermediate period (1 to 9 years), when a previously tolerable degree of craniostenosis often exhibits decompensation. Functional problems relating to the eyes were evident in varying degrees in most of the cases of Crouzon syndrome: the chief was exophthalmos, with the risk of exposure keratitis. The aim of operative correction of orbitostenosis has been to enlarge the orbit forwards by anterior movement of the frontal and midfacial bones. Airway insufficiency due to faciostenosis is rarely in itself an indication for operation, but it is virtually a constant symptom in the Crouzon and Apert syndromes and may benefit greatly from facial advancement. Dental malocclusion may also be corrected, at least in part, by advancing the maxillae. The open bite can be corrected by lengthening the face.

Psychosocial problems emerge as the chief indication in older children. Cosmetic improvement is usually easier to give when facial growth is completed, but to wait so long may be harmful both to the patient and to his family. The introduction of a deformed infant into a family causes great stress. The introduction of a deformed child to school, usually at the age of 5 years, brings another period of crisis. Adolescence, with its psychosexual stresses, brings new needs and demands. Fine surgical judgement based on detailed assessment by the craniofacial team is required before a decision to operate or not to operate is taken on purely psychosocial indications. This is further discussed in Chap. 18.

TABLE 16.1

Craniofacial operations performed for deformities associated with craniosynostosis by South Australian Craniofacial Unit, 1975–1981. Operations for simple scaphocephaly and trigonocephaly are excluded.

Diagnoses	No of Patients
Complex syndromes	
Crouzon	18
Apert	1
Carpenter	2
Saethre-Chotzen	2
Cohen	1
Other	1
Simple	
Turricephaly	1
Plagiocephaly (all types)	10
Total	**36**

Timing of Surgery

The problems of timing have been discussed in principle in Chap. 7. The dilemmas outlined there come into very sharp focus when the complex craniofacial syndromes are under consideration. Children born with these syndromes may have the potential for normal intellectual and social development. They may benefit very greatly from extensive craniofacial surgery, and the psychosocial advantages of early correction are obvious. But these advantages may be offset by detrimental effects on cranial growth. Tessier (1971a) demonstrated continued nasomaxillary growth after a maxillary osteotomy done in childhood. The experiences of Hogeman and Willmar (1974) in Crouzon syndrome have been mentioned (p. 103); these authors expressed the view that early facial advancement will improve the appearance at the time, but the basic dysplasia will continue to affect facial growth until adolescence, so that the eventual deformity may be only slightly less than it would have been without operation. This view is still debatable and the debate must be resolved before the optimum timing of maxillary osteotomies can be determined. It is easier to pronounce on

the timing of fronto–orbital advancements. These are now being carried out safely in most craniofacial units. They undoubtedly allow the brain and the orbital contents to expand during their periods of most rapid growth (Fig. 6.3) and this may lead to improved growth in the midfacial region. Such operations are therefore done in the first year of life, and preferably in the first 3 months.

In our surgical philosophy the concept of the three chronological periods described in Chap. 8. is especially helpful when one is considering the timing of craniofacial operations.

Early Period (First 12 Months)

Operations are undertaken to facilitate normal growth, to release skeletal constraints on the growing brain and eyeballs. In most of the complex craniosynostoses these operations amount to fronto-orbital advancement with excision of the chief prematurely fused calvarial sutures, in one or two stages. Maxillary osteotomies are *never* done by us in this period.

Intermediate Period (1 to 9 Years)

Raised intracranial pressure may dictate operation in this period, and the relief of craniostenosis can be achieved by a fronto–orbital advancement combined with subtemporal decompression. It will usually be necessary to complete the mobilisation of bilateral osteoplastic flaps in a second stage (p. 100). Severe orbitostenosis may also demand operation during the intermediate period, to deal with recurrent ocular dislocation. One may also have to operate in this period to correct deformities when these are causing extreme psychosocial distress. Hypertelorism, orbital dystopia, and plagiocephaly can be corrected in the intermediate period without great fear of relapse. We believe that it is undesirable to carry out maxillary advancements in this period, but we have done so when correction seemed an imperative psychosocial necessity. Airway obstruction from very severe faciostenosis may also dictate early operative intervention (Pruzansky, personal communication). In these cases there may need to be a further LeFort I maxillary osteotomy or a mandibular setback procedure if continued disproportionate mandibular growth causes the deformity to recur.

Late Period (From 10 Years)

This is the time for definitive correction of the established deformity. Operations are done for psychosocial reasons, supported by the hope of benefit for such functional problems as airway obstruction, dental malocclusion and impaired speech. After 11 or 12 years, maxillary advancement can be done with reasonable confidence: most of the maxillary growth is completed and the permanent dentition is sufficient for secure intermaxillary fixation. Where possible, total correction is done in a single operative session. In advancing the maxilla, some over-correction is desirable to compensate for future mandibular growth.

Pre-operative Patient Assessment and Preparation

With Munro (1975), we believe that the multidisciplinary team approach is mandatory if mortality and morbidity are to be kept at an acceptable level. This concept has been fully accepted by all members of the South Australian Craniofacial Unit. The particular functions of each person in our team are set out in Appendix A. Each member collects relevant data within his area of interest, and these data are collated at Craniofacial Unit meeting, at which the patient is present. At this meeting decisions are made on the need for surgery, the timing, and the general nature of the proposed operations. These meetings may take place many months before the patient is actually admitted to hospital for surgery. There is therefore need for a detailed final assessment, and for quite elaborate

preparations of various types, before craniofacial operations are carried out. Especially for patients who come from other parts of the world, adequate time must be allowed: local residents can be sent home if reconsideration is necessary, but this is a real hardship for patients who have come long distances at much expense. Thus, before each major craniofacial operation, a week or more must be devoted to a planned programme of reassessments and preparations.

Psychosocial (see Chap. 18)

The patient and the family need support during the stressful preoperative period. The hospital routine and the surgical procedures must be fully explained. The staff who do this pass the patient's fears and doubts back to the surgeon so that he can more easily solve them. Serious emotional problems may come to the surface during the preoperative period, and these often need much insight and delicacy; our system of counselling and support is outlined in Chap. 18.

Surgical

The surgeons who will operate must make a final complete re-examination, including up-to-date measurements relating to the dimensions of the proposed changes in cranial and facial appearances. When an intracranial operation is to be done, the neuro-surgeon must make a final neurological examination. He must look once again at the optic fundi and he must check olfaction. In young patients, he should also review the neurological development.

Ophthalmological

The ophthalmologist also reviews the patient, repeating such of the measurements listed on p. 77 as are relevant to the proposed operation. These usually include the inner and outer intercanthal distances, the interpupillary distance, the length and width of the palpebral fissures, and the degree of exophthalmos. The cortical visual evoked responses may be recorded.

Dental

An up-to-date dental check is important to exclude such problems as carious teeth likely to interfere with intermaxillary fixation. In our unit, fixation is usually done with cast metal cap splints. A bite wafer may be needed. The cap splints are cast in moulds from final dental models made a week before operation. They are fitted by the orthodontist one or two days preoperatively, and are made so as to fit together exactly in their new positions. If necessary the upper cap splint is fitted to hold an anterior rod for craniomaxillary fixation.

Anaesthetic

The anaesthetist is responsible for the final assessment of general health and condition of the respiratory tract. He reviews recent chest radiographs. He notes the condition of the patient's veins and orders an appropriate number of units of blood; for an adult undergoing major craniofacial surgery six units (3 l) are usually ordered and the blood bank is warned that much more may be needed. The patient's serum biochemistry is checked. The anaesthetist also reviews the airway. It is not always easy to forecast what operative and postoperative difficulties will be encountered. Attention is given to the size of the mandible relative to the maxilla and to the size of the tongue relative to the oral capacity. The mobility of the temporomandibular joints and of the neck are important in intubation.

Cardiovascular and Pulmonary

In our practice the cardiologist routinely checks the cardiac state. Cardiac anomalies are often associated with craniofacial syndromes (see pp. 198, 208, and 221), and these may affect the circulatory efficiency. An implanted pacemaker (present in one of our Crouzon cases) may prohibit the use of monopolar

diathermy. Pulmonary function is checked only if there is suspicion of lung disease.

Haematological

This needs special mention. Several patients referred to our unit were found to have defects in blood coagulability or bleeding time. A very serious bleeding tendency appeared during operation in one patient (see p. 285). Another was denied operation because of a coagulopathy. A full haematological assessment including bleeding and coagulation studies is always carried out.

Bacteriological

Cultures are taken from the nose, throat, and conjunctivae. Significant pathogens include *Staphylococcus aureus, Streptococcus pneumoniae* and other haemolytic streptococci. Colonisation of the nose or conjunctival sac by one of these organisms is treated by local antibiotic ointment or drops. Colonisation of the throat may need systemic chemotherapy.

Planning the Operation

In our unit, surgical planning takes place at a special meeting of the surgical team together with the radiologist and the orthodontist. At these meetings the radiographic data to be reviewed include standard skull views, tomograms in at least the sagittal and coronal planes, CT scans, and cephalometric views. Photocephalometric films are also important, especially in planning the new profile. Dental models are indispensable when there will be a midfacial shift. Moulages of the face and frontal skull are useful when there will be a difficult frontal reconstruction, as in plagiocephaly. The various measurements made directly on the patient's face and head are also used in the final stages of operative planning, especially in orbital shifts.

Each set of data has its special field of usefulness. Standard skull radiographs present the general pattern of the craniosynostosis and are informative on the nature of previous operations, which are sometimes poorly documented. Tomograms show best the pathology in the skull base, especially the relationship of the deformed cribriform plate to the nasal cavity and the orbits (Fig. 16.9). Tomograms also demonstrate the anterior extent of the middle cranial fossae, which may be in danger during lateral orbital osteotomies. Coronal tomograms delineate the shape of the orbital walls and show the degree of bony hypertelorism (Munro 1976). CT scans are needed to determine whether there is coexisting hydrocephalus, always a possibility in Crouzon and Apert syndromes, and to exclude cerebral malformations. CT scans will also show the orbits and orbital contents in different planes of section. The newest scanners cut thinner slices with better resolution and make it possible to reconstruct the findings in any desired plane. These will doubtless be used increasingly in planning orbital surgery.

Cephalometry is useful in showing the facial deformity and in determining the true relationships of the maxilla and mandible to each other and to the skull base. In the complex forms of craniosynostosis, the skull base is likely to be deformed, and this has to be taken into account if the data obtained are used in operative planning (Firmin et al. 1974). For the plastic surgeon the most important cephalometric measurements are the SNA and SNB angles, which quantitate the anteroposterior relationships of the maxilla, mandible, and cranial base (Fig. 16.10). The SNA angle is constructed by lines joining three points identified in lateral cephalometric views: the midsellar point *S*. the nasion (*N*: the most anterior point on the nasofrontal suture), and the point *A* which is the deepest (most posterior) midline point on the premaxilla. Converse et al. (1977) give the normal range as 79°–85°. To construct the SNB angle, the point *N* is joined to a point *B*

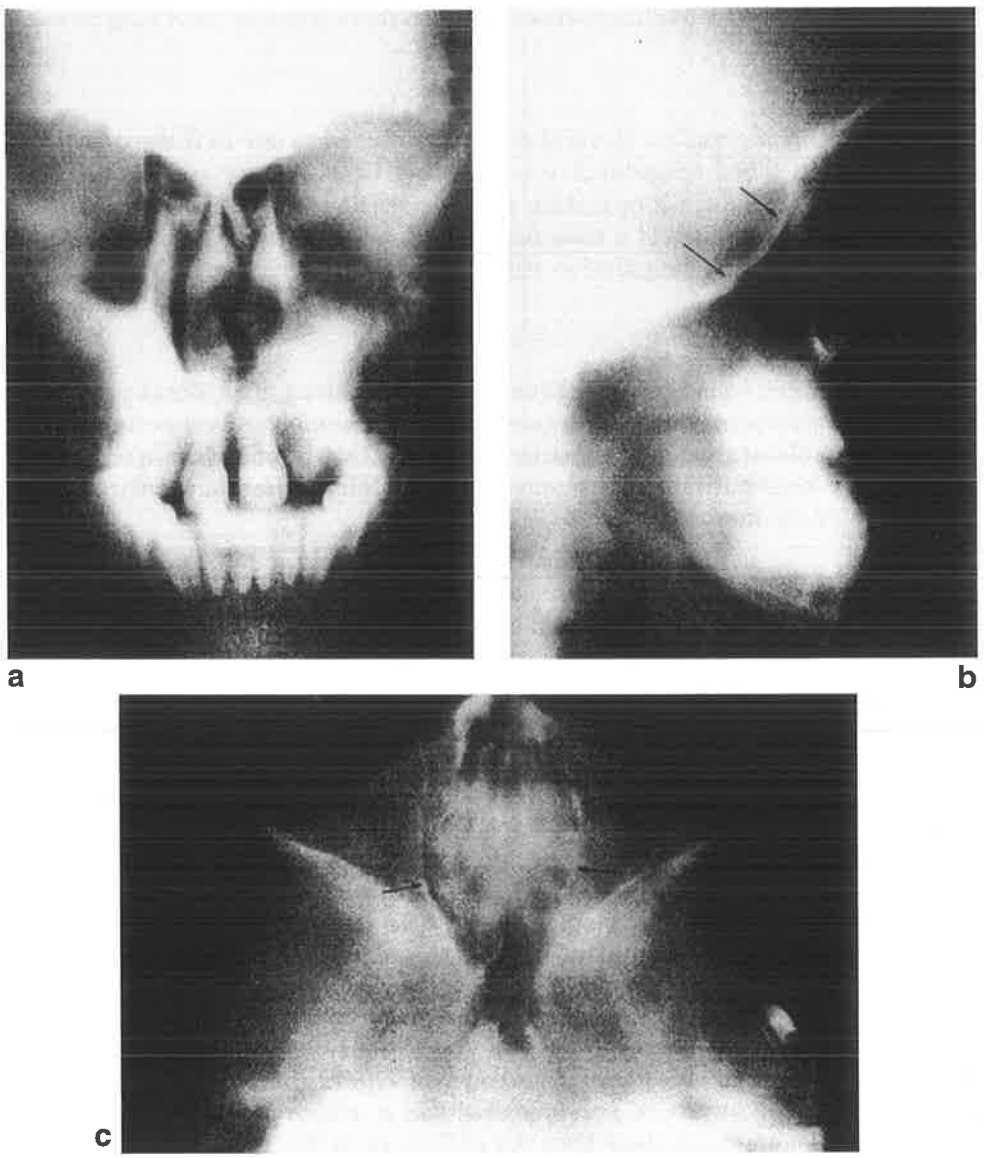


FIG. 16.9a–c. *Anatomical distortions in Crouzon syndrome.* **a.** *Anteroposterior tomogram demonstrating the prolapsed cribriform plate and its relationship to the nasal cavity and the steeply sloping orbital roofs.* **b.** *Lateral tomogram showing the temporal ballooning of the middle cranial fossa* (arrows) *and its relationship to the orbit.* **c.** *Basal tomogram showing the hypertelorism and the shape of the medial orbital wall* (arrows).

which is the deepest (most posterior) point on the concavity of the anterior mandible. The normal range is 76°–84°. Therefore SNA is normally 2°–4° greater than SNB; in maxillary retrognathia it is smaller. In cases of Crouzon and Apert syndrome the mandible may appear prognathic, but this is often not so: extreme retrusion of the maxilla may make the mandible relatively prominent when it is truly retrognathic in relation to the skull base. In such cases, the SNA and SNB angles are both abnormally small, but the SNA angle will be markedly less than the SNB angle. In planning correction of the maxillary retrusion of Crouzon syndrome it should be remembered that co-existing hypoplasia of the anterior cranial fossa sets the point *N* abnormally far posteriorly; if it is intended to perform a fronto-orbital advancement, the new position of the point should be plotted on the cephalometric tracing.

Photocephalometry (Fig. 16.11) is done by a method based on that reported by Henderson (1974). A lateral cephalogram is first obtained, the soft tissue profile being outlined with radio-opaque paste. The relevant cephalometric points are marked on the film and the frontal bone, maxilla and mandible are delineated. A

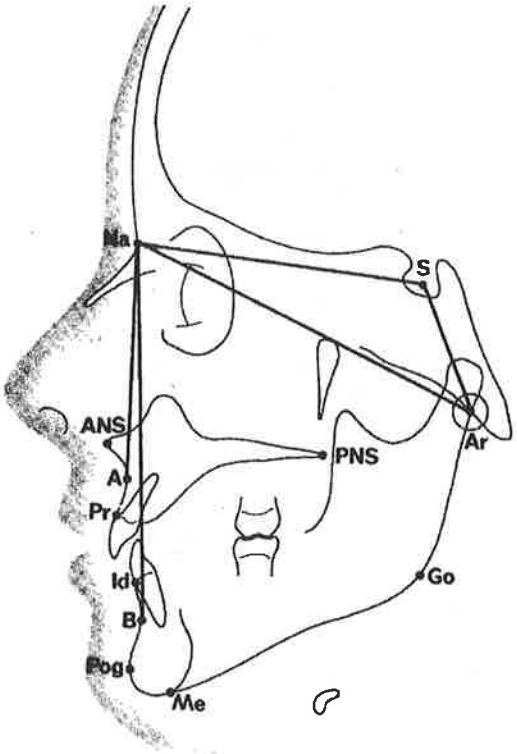


FIG. 16.10. *Normal cephalometric tracing. Lines from the midsellar point S to the nasion* Na *(N) and from the nasion to a point A (most posterior point on the surface of the pre-maxilla) form the angle SNA and relate the maxilla to the cranial base. A line from the nasion to the point B (most posterior point on the anterior surface of the mandible) forms the angle SNB and relates the mandible to the cranial base. Other points useful in cephalometry are:*

ANS: *anterior nasal spine*

Pr: *prosthion—most anterior inferior point on maxilla*

Id: *infradentale—most anterior superior point on mandible*

Pog: *pogonion—most anterior point on contour of mandible*

Me: *mention—lowest point on mandibular symphysis*

Go: *gonion—most posterior inferior point on body-ramus junction of the mandible*

Ar: *articulare—intersection of cranial base and posterior surface of mandibular condyle*

PNS: *posterior nasal spine*

transparent photographic pint of the face in lateral view is then produced at exactly the same magnification as the cephalogram.

This print is then superimposed on the cephalogram and the markings made on the X-ray film are traced onto the transparent print. This gives a photocephalogram, which can be cut along the proposed osteotomy lines; the segments are then moved into what are regarded as ideal positions from the aesthetic viewpoint. Correction of dental malocclusion can also be simulated in this way. One must take into account the ratio of soft tissue to hard tissue movement. Thus, the soft tissue of the chin follows the advancement of the bone in a 1 to 1 ratio and the lower lip in a 2 to 3 ratio. while in advancement of the upper jaw the lip follows the bone in a 2 to 3 ratio. This method is particularly applicable in planning lower facial corrections (Steinhauser 1975; Freihofer 1977), but as Henderson (1974) has pointed out, it is less reliable in predicting the outcome of high facial osteotomies of the LeFort III type. We have also used this technique in planning the correction of hypertelorism and orbital dystopia; a postero–anterior X-ray cephalogram is used with a transparent full-face

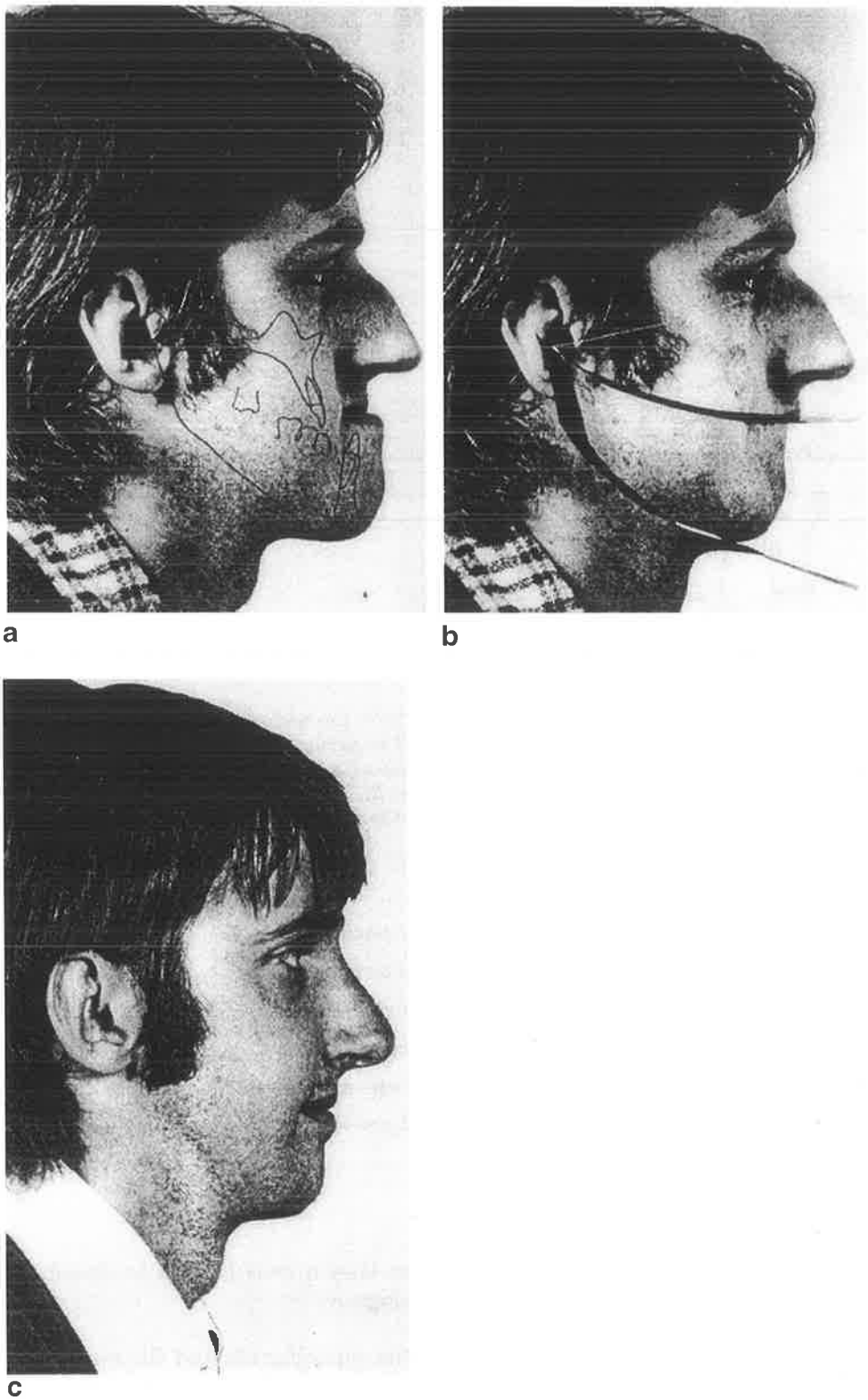


FIG. 16.11a–c. *Profile assessment in a man with deformity of the mid and lower face using the technique of Henderson (1974).* **a.** *Transparent photography with superimposed jaw relationships taken from the cephalometric radiographs.* **b.** *Profile plan showing proposed advancement.* **c.** *Postoperative profile (actual result).*

photograph superimposed on it. This two-dimensional analysis of a three-dimensional problem has been found interesting but somewhat difficult, and the results are not always predictable.

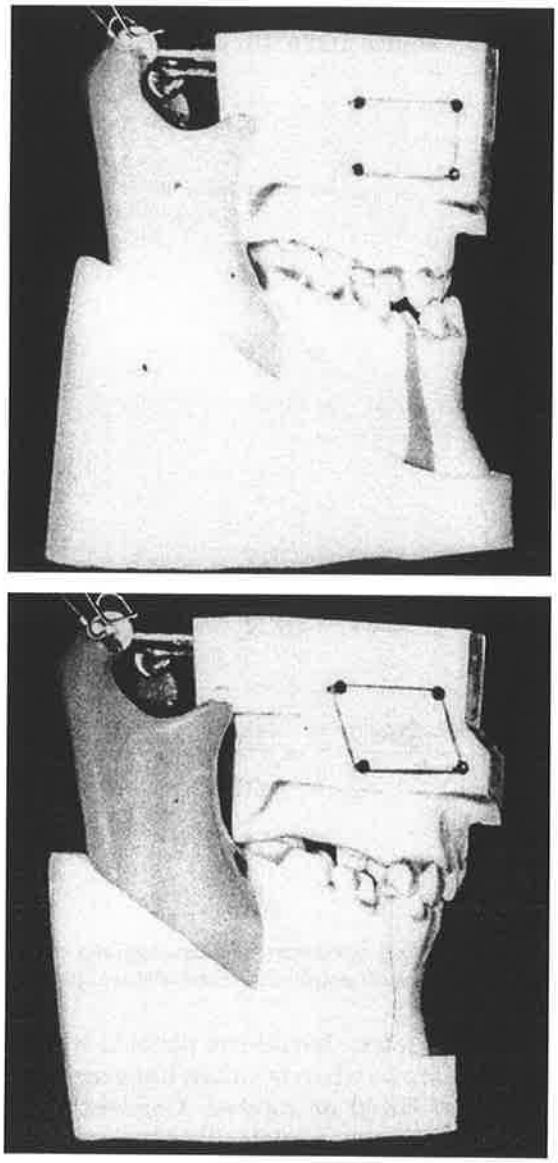


FIG. 16.12 a,b. *Model planning showing the results of maxillary advancement and bilateral mandibular body osteotomy:* **a.** *before and* **b.** *after simulated surgery.*

Dental models (Fig. 16.12) are used in conjunction with cephalometry and profile planning to assess the occlusion to be expected after a proposed face shift. They are also needed in planning pre- or postoperative orthodontic treatment, should this be necessary.

Direct measurements made on the patient are especially important in planning the correction of retrusions of the face and/or forehead. In these deformities one must know the relationships of the superior, inferior, and lateral margins of the orbit to the cornea (see p. 78). Normally the superior margin is 10 mm in advance of the apex of the cornea. The inferior margin may be in the same coronal plane as the apex whereas the lateral margin is at least 7 mm behind it (Bertelsen 1954). After midfacial and fronto-orbital advancements, the orbital correction may not exactly match the occlusal correction. Preoperative planning should take account of this, to forecast the need for onlay grafts or even for a

double maxillary osteotomy of LeFort I plus III type. Direct measurements are also of great importance in planning the correction of hypertelorism and orbital dystopia: in these deformities the most relevant measurements are the interpupillary and intercanthal distances and the vertical separation of the two eyes (Fig. 7.1). The asymmetrical craniofacial deformities pose very difficult planning problems. They may combine calvarial plagiocephaly, orbital dystopia, hypertelorism, deviation of the nasomaxillary angles and a tilted occlusion plane

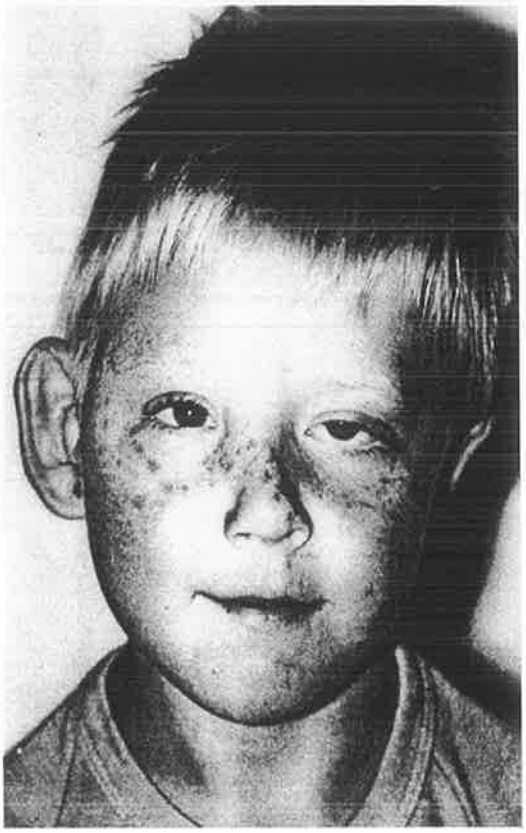


FIG. 16.13. *A complex craniofacial deformity exhibiting hemicranial plagiocephaly with orbital dystopia, hypertelorism, deviation of the nasofrontal angle and tilted occlusal plane.*

(Fig. 16.13). There is great difficulty in finding a true horizontal plane to which proposed shifts can be related. This is even more so when (as often happens) the patient's normal head posture is somewhat tilted or rotated. Conventional cephalograms and photocephalograms may then give a misleading impression. For these conditions we have therefore tended to operate in two stages, first correcting the fronto-orbital component of the deformity and then the occlusion.

Preoperative planning is thus a synthesis of many impressions and measurements and objectivity is always desirable. We can hope in the future for further objective help from newer techniques; computer-simulated models based on the findings of advanced CT scanning are already being used and have much promise. But subjective assessment by the experienced craniofacial surgeon remains the cornerstone of our work. Valuable as they are, measurements rarely negate the impressions formed after facial inspection and discussion with the patient, who is the unique factor in every planning process. and whose aesthetic demands should always be taken into account. Measurements are no substitute for clinical assessment and aesthetic judgement.

Choice of Surgical Procedure

This depends on the age of the patient, the indications for surgery, and the characteristics of the deformity. The diagnostic category, in terms of the

syndromes listed in Chap. 15, is much less important than the pathological anatomy, and different syndromes may present identical surgical problems.

In the early age period, as has been indicated, the emphasis is on wide excision of stenosed calvarial sutures, in conjunction with unilateral and bilateral fronto-orbital advancements when the craniosynostosis syndrome entails plagiocephaly or a brachycephalic deformity, respectively. In some cases of Crouzon and Carpenter syndromes the calvarial deformity is scaphocephalic, and then the operative choice may be some kind of lateral craniectomy planned to release potential for temporal and parietal expansion and so to broaden the head (Fig. 6.6).

In the late age period, the options are more complex. It is now possible to move the bony components of most of the cranium, including the facial skeleton, in three dimensions to correct all the deformities associated with craniosynostosis. The principles of the late calvarial resections have been discussed (Chap. 8) and categorised in relation to the individual calvarial deformities (Part III). The osteotomies of the midfacial skeleton are conveniently categorised in terms of LeFort's classification of facial fractures (Rowe and Killey 1968):

Type I: a low section through the maxillae and nasal septum just above the alveolar bone and the palate

Type II: an intermediate section through the nasal bones and into the orbit but below the zygomatic buttresses

Type III: a high section through the nasal and ethmoid bones detaching the maxillae and the zygomatic bones from the base of the skull

All three sections end in the region of the pterygomaxillary fissure, where the maxilla and palatine bone must be detached from the pterygoid process. Of course the osteotomy lines do not exactly duplicate LeFort's fractures, but the comparison gives a useful shorthand identification of these complex midfacial procedures.

In the late age period, we have used the following procedures:

Subcranial LeFort III osteotomy and facial advancement, done when the anatomy of the cranial base is relatively normal and no calvarial reconstruction is needed.

Transcranial LeFort III osteotomy and facial advancement, done when the anatomy of the cranial base endangers the temporal lobes or the contents of the cribriform fossae (see p. 234). The procedure may be combined with a *fronto-orbital advancement.*

Combined facial osteotomies (Tessier 1976) may be appropriate. Thus we have combined LeFort III and I sections in a case with exophthalmos and nasomaxillary retrusion but normal dental occlusion: the upper maxillary complex can then be advanced independently of the palate and dentition. This is termed a LeFort III minus LeFort I section. LeFort III and I sections can be combined (Obwegeser 1969) when the upper jaw has to be moved independently of the orbitonasal complex and it is expected that correction by onlay bone grafts would be unsatisfactory. When there is hypertelorism as well as maxillary hypoplasia, LeFort III and LeFort I sections can be combined with transcranial paramedian nasoethmoid resections and orbital shifts.

Transcranial orbital translocations are now standard procedures for the correction of hypertelorism and orbital dystopia. They may be combined with frontal reshaping. Orbital translocation has been done in the intermediate period for such problems as the hypertelorism of Cohen syndrome (see p. 223) or for severe frontal plagiocephaly (see p. 164).

Mandibular osteotomies are necessary when for any reason there is significant mandibular disproportion.

Theatre Logistics

In the South Australian Craniofacial Unit two days a week are set aside for craniofacial surgery, one for children and one for adults. Wherever possible the same anaesthetists and theatre nursing teams are employed. If the transcranial approach is to be used, the neurosurgeon has his own nursing team. A protocol is issued to the theatre staff well in advance, outlining the sequence of surgical steps in each case. This enables the theatre nurses to set up their instrument trays and to organise their routine throughout the day. Craniofacial operations are often long and staff planning then includes provision for relay relief both for surgical assistants and for nurses; the plastic surgeon and the neurosurgeon relieve each other. The Unit Registrar is responsible for bringing to the theatre all radiographs and models, and also the treatment plan. The radiographs and the chief photographs are displayed on a large viewing screen.

Every effort has been made to speed up routine theatre procedures and to cut operating time to a minimum. Unnecessary transit of visitors through the area is prohibited during transcranial surgery. If teaching or demonstration is required, closed circuit television is used. It is our belief that our hitherto low incidence of infection is in part due to these precautions.

Anaesthesia

Patients are given oral premedication with diazepam or intramuscular Omnopon-Scopolamine, according to their preferences. Anaesthesia is usually induced with thiopentone and suxamethonium and maintained with nitrous oxide, curare, and controlled ventilation. Intravenous narcotics (e.g. morphine 0.5–1 mg/kg) are given liberally to depress the autonomic vascular responses during pain-producing facial dissections.

Intubation may be difficult. We have sometimes found it useful to employ the combination of intravenous diazepam (adult dose 10mg) and transcricoid local anaesthesia (4 ml 4 % lignocaine); the patient may be awake or lightly anaesthetised with halothane. This has been done in adults and in children as young as 18 months. Adults given diazepam have not recalled the intubation. If nasal intubation is required, the nose is anaesthetised with 1% cocaine paste.

Tracheostomy may be needed, especially when intermaxillary fixation is planned. In our practice the procedure is done by a senior member of the team, usually the plastic surgeon or the neurosurgeon. We think it important to make the stoma just below the first tracheal ring. Over the age of 11 years, a small flap of the trachea is sutured to the deep fascia to maintain stomal patency; it is based caudally. In younger children a simple slit stoma is made, the aperture being held open by lateral sutures. Care is taken to use the largest tracheostomy tube which will fit comfortably in the trachea, to avoid over-inflation of the cuff, and to ensure that the tip of the tube does not touch the carina.

Certain anaesthetic problems are inherent in the nature of craniofacial surgery. Dissection in the orbits may produce reflex bradycardia. This is transient and harmless if the patient is deeply anaesthetised, and we do not advocate suppression by atropine as this may cause tachycardia and hypertension. We avoid hypertension at all stages and control it when it occurs, but do not routinely use deliberate hypotension (see p. 286). We believe that the maintenance of normal vascular reflexes makes it easier to assess the blood volume.

Circulatory control is of course a major problem in craniofacial surgery. The need for transfusion is best monitored by venous and arterial lines to a manometric recorder giving continuous readings throughout the operation. We

routinely insert a central venous catheter and usually also a catheter in the radial artery. These, together with a urethral catheter, are inserted at the beginning of the procedure and are maintained in the postoperative period. Actual calculation of blood loss has been found impracticable, by reason of the many swabs and packs and the numerous wounds, some of which are concealed for better asepsis by additional drapes.

The other major challenge to the anaesthetist is the control of intracranial pressure. This has been surprisingly easy. Dexamethazone (approximately 0.1 mg/kg every 6 h) is routinely given, and negative-phase ventilation is always employed during the dissection of the anterior cranial fossa. These measures, combined with surgical gentleness, have usually made it unnecessary to give dehydrating agents (mannitol, glycerol, or frusemide). We no longer routinely employ spinal catheterisation and we avoid ventricular drainage except in emergency. It is of course an essential requisite in transcranial surgery to avoid hypoxia and hypercarbia and to restrict the fluid and sodium intake. Normal estimates of the fluid requirements should usually be halved during and immediately after craniofacial surgery, since there is likely to be a strong metabolic stress response and this will include an antidiuretic reaction. Mild dehydration helps the neurosurgeon, though prolonged dehydration is of course undesirable.

Operative Exposures

The *bicoronal scalp flap* (Fig. 8.5b) extends behind the hairline from one pre-auricular region to the other, and the incision is taken down to the level of each zygomatic arch. The line of the incision is infiltrated with a 1:200 000 solution of adrenaline in normal saline or in lignocaine (0.25 % in infants, 0.5 % over the age of 1 year). This reduces bleeding and helps to separate the tissues; if there is an area of bone deficiency, the dura may be subcutaneous and easily opened by a hasty cut.

The scalp flap is at first elevated by subaponeurotic dissection, but when the pericranium and the temporalis fascia are exposed sufficiently, the operator leaves the subaponeurotic plane and continues the dissection solely in the subperiosteal plane. A frontal flap of pericranium is turned down to the orbital margin and the orbit is entered, still in the subperiosteal plane. The supra-orbital nerves are extricated intact from their notches; when the notch is a foramen, a tiny osteotome is used to open it. Laterally the temporalis muscles are reflected as separate flaps, again by subperiosteal dissection. There is now an excellent exposure of the frontal bones and temporal fossae. Further sharp subperiosteal dissection exposes the malar region, the roofs and lateral walls of the orbits, and the frontonasal angle.

In recent years, encouraged by Munro (personal communication), we have been able to expose also the orbital floor and the anterior surface of the maxilla and so to carry out all the osteotomies needed for fronto-orbital and midfacial advancement through this single incision (Fig. 16.21). Elevation of the temporalis muscle also gives access to the pterygomaxillary fissure, and we now prefer this approach for the posterior mobilisation of the maxillae when transcranial operations are being done, to avoid opening into the unsterile mouth. The scalp incision is closed in two layers with continuous sutures (3/0 Dexon for the aponeurosis and 5/0 Prolene for the skin).

The *upper buccal sulcus incision* remains ideal for low facial osteotomies or for onlay bone grafting of the maxilla. It may be done bilaterally in the posterior part of the sulcus or anteriorly across the midline. The dissection is subperiosteal and the mucosal wounds are closed with a few catgut sutures.

The *conjunctival approach* (Tessier 1973) offers a direct view of the orbital floor, anterior surface of the maxilla, and medial malar region without any facial scar. We (David 1974) have been using this approach for the last 10 years and have found it wholly satisfactory, though it is now used less because adequate access is being obtained from above through the bicoronal incision. In this approach the lower eyelid is drawn out with two skin hooks and the conjunctive is incised 2–3 mm below the tarsal plate (Fig. 16.14). A stay suture retracts the inner edge of the incision, exposing the plane of dissection and protecting the cornea. This plane is developed with curved scissors between the orbital septum and the orbicularis oculi muscle; as the dissection approaches the inferior orbital margin a malleable retractor can be used to support the orbital contents. Two small curved retractors now replace the skin hooks to hold back the lower eyelid. The inferior orbital rim is exposed. The periosteum is incised on the maxillary surface of the rim, not on the actual crest, to prevent injury to the orbital septum and periorbita, which would lead to escape of orbital fat. Further subperiosteal dissection exposes the orbital floor. After the osteotomies have been completed, the wound is closed with a few fine catgut sutures, not buried, through the conjunctive alone.

Bilateral Fronto-orbital Advancement

This operation has been used for cases of Crouzon and Apert syndromes with turricephalic or oxycephalic deformities as well as for simple turricephaly (Chap. 12). It should be done during the first year of life and ideally during the first 3 months. The procedure has also been used for cases of Crouzon syndrome presenting with craniostenosis in the intermediate period, when a fronto-orbital advancement can be the first stage in a full bilateral decompression (p. 100).

The patient is anaesthetised by the oral endotracheal route and the various lines detailed on p. 251 are established. The standard bicoronal scalp incision is used. The frontal bone is exposed down to the glabella and orbits by subperiosteal dissection and the temporal fossae are bared of muscle. The state of the sutures and of the anterior fontanelle is noted. The proposed osteotomies are then marked on the skull (Fig. 16.15). A channel 1 cm wide is then cut in the area of the fused coronal suture and this is extended across the squamosal suture into the temporal bone. A temporal craniectomy is carried out and the bone removal is taken into the orbit in the region of the sphenofrontal and sphenozygomatic sutures

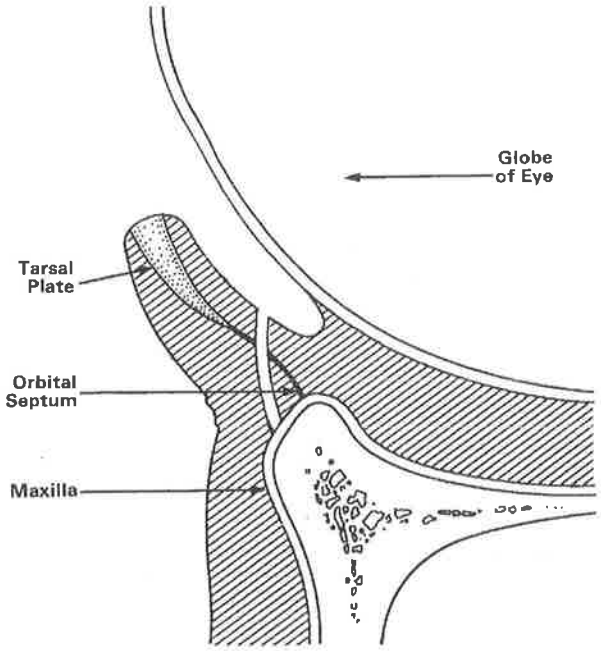


FIG. 16.14. *Conjunctival approach to the orbital floor and the upper maxilla and zygoma.*

(McCarthy et al. 1978). A horizontal frontal cut is then made and the frontal bone is temporarily removed. Extradural dissection exposes the anterior fossa as far back as the foramen caecum and the lesser wings of the sphenoid bone. The hypertrophied sphenoidal ridge is now rongeured away if it was not resected earlier. Osteotomies are then taken across the anterior fossa through the roofs of the orbits and the glabella, along the lines shown in Fig. 16. 15. The fronto-orbital crown is now demarcated. When cuts have been made in the lateral orbital walls as shown in Fig. 16. 15, the crown is wholly mobile and can be advanced as a free bone block. The complete procedure represents a resection of the whole coronal suture system including its anterior fossa extensions (Scott 1967) and should release frontal growth.

The fronto-orbital crown is moved to be at least 1 cm in advance of the cornea. (This must be checked by inspection during the operation.) The crown is wired at three points, to spurs of the lateral orbital walls on each side and to the glabella by wires passed around or through an interposed bone graft. The spurs (Fig. 16.15) are formed from the zygomatic and perhaps the sphenoid bones. The bone grafts are the excised craniectomy strips or are taken from the temporal bone. The free frontal bone can be wired to the advanced fronto-orbital crown,

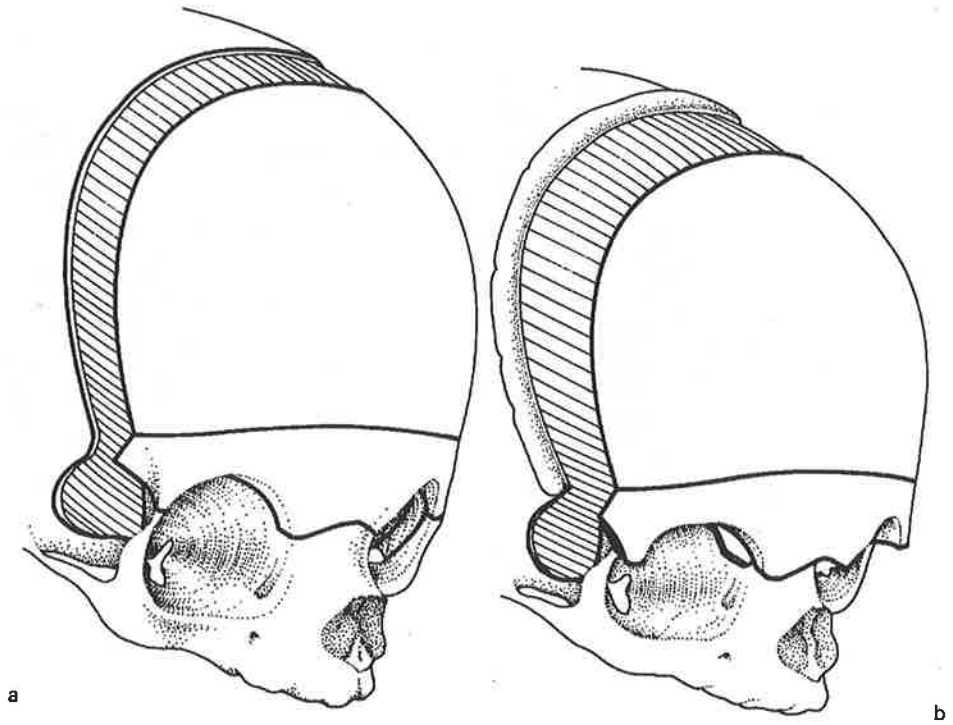


FIG. 16.15 a,b. *Bilateral fronto-orbital advancement: the osteotomy lines include the fused coronal sutures and extend across the squamosal suture. The temporal craniectomy is taken into the orbit to involve the sphenofrontal and sphenozygomatic sutures.* **a.** *Before and* **b.** *after advancement*

leaving a gap posteriorly, or it can be first divided and transposed as advised by Marchac (1978) and by Stricker (Montaut and Stricker 1977). We believe that if the released frontal bone and the fronto-orbital crown are to be carried forward by the growing brain, then there should be no bone grafts or struts to bridge the newly created zone of skeletal freedom. McCarthy's account of Tessier's method (McCarthy et al. 1978) and the procedure illustrated by Hoffman and Hendrick (1979) both entail a strut crossing the craniectomy and seemingly constraining growth. At the completion of the advancement, the posterior margin of the craniectomy is wrapped with Silastic. Pericranium and temporalis muscles are sutured over the fronto-orbital crown; care is taken to bring the temporalis muscles forward to avoid an unsightly temporal hollow. Scalp closure is along standard lines. The effect is shown in Fig. 16.16.

Unilateral Fronto-orbital Advancement

This operation has been recommended for cases of simple frontal plagiocephaly presenting in the early period (Chap. 13). It is also appropriate in cases of Saethre-Chotzen syndrome and other complex craniosynostosis syndromes associated with severe plagiocephaly.

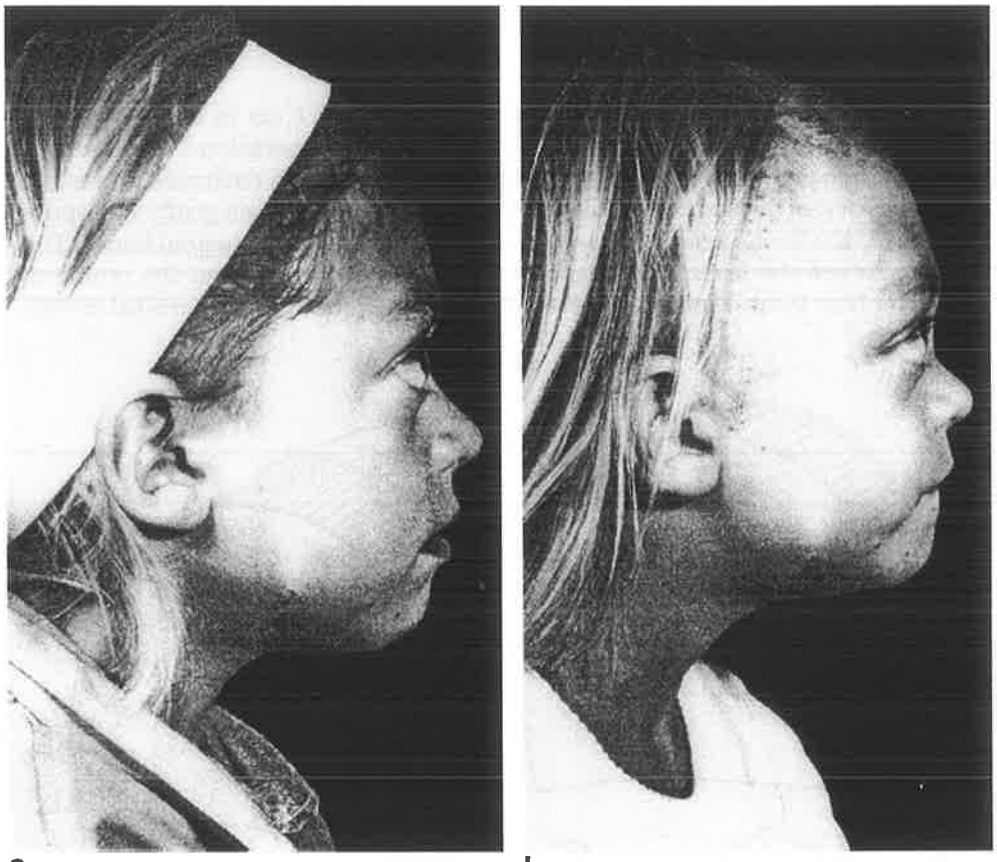


FIG 16.16a,b. *A patient with Crouzon syndrome.* **a.** *Before and* **b.** *after fronto-orbital advancement*

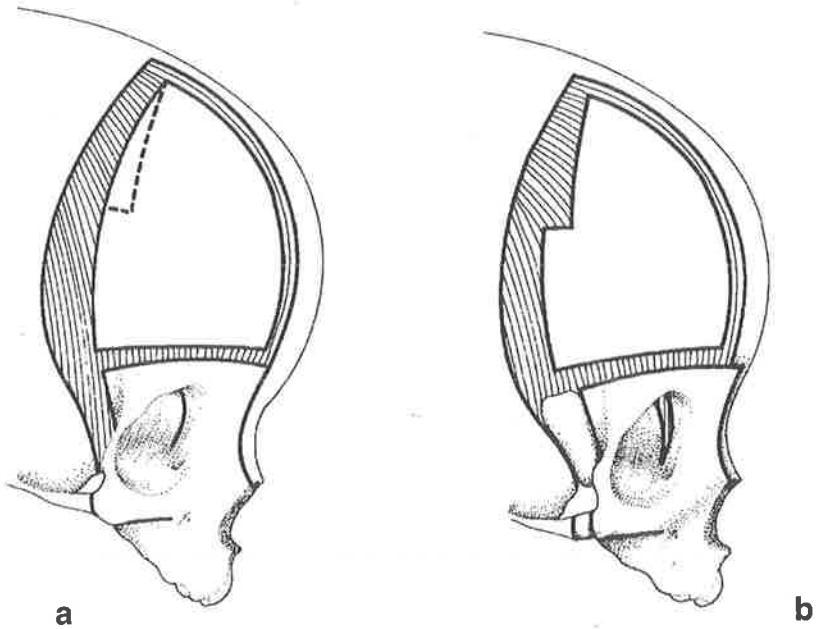


FIG. 16.17 a,b. *Fronto-orbital advancement, Whitaker's technique.* **a.** *Before and* **b.** *after insertion of grafts cut from the frontal bone flap.*

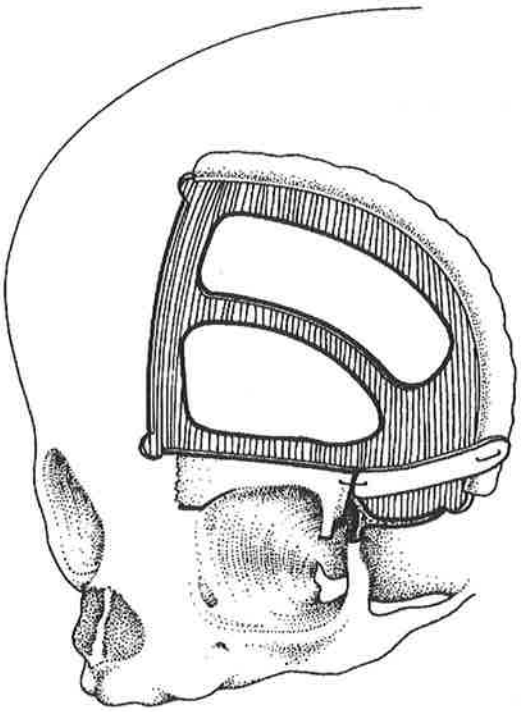


FIG. 16.18. *Fronto-orbital advancement after Hoffman and Mohr. The advancement is supported by a strut of bone anchored to the posterior margin of the craniectomy.*

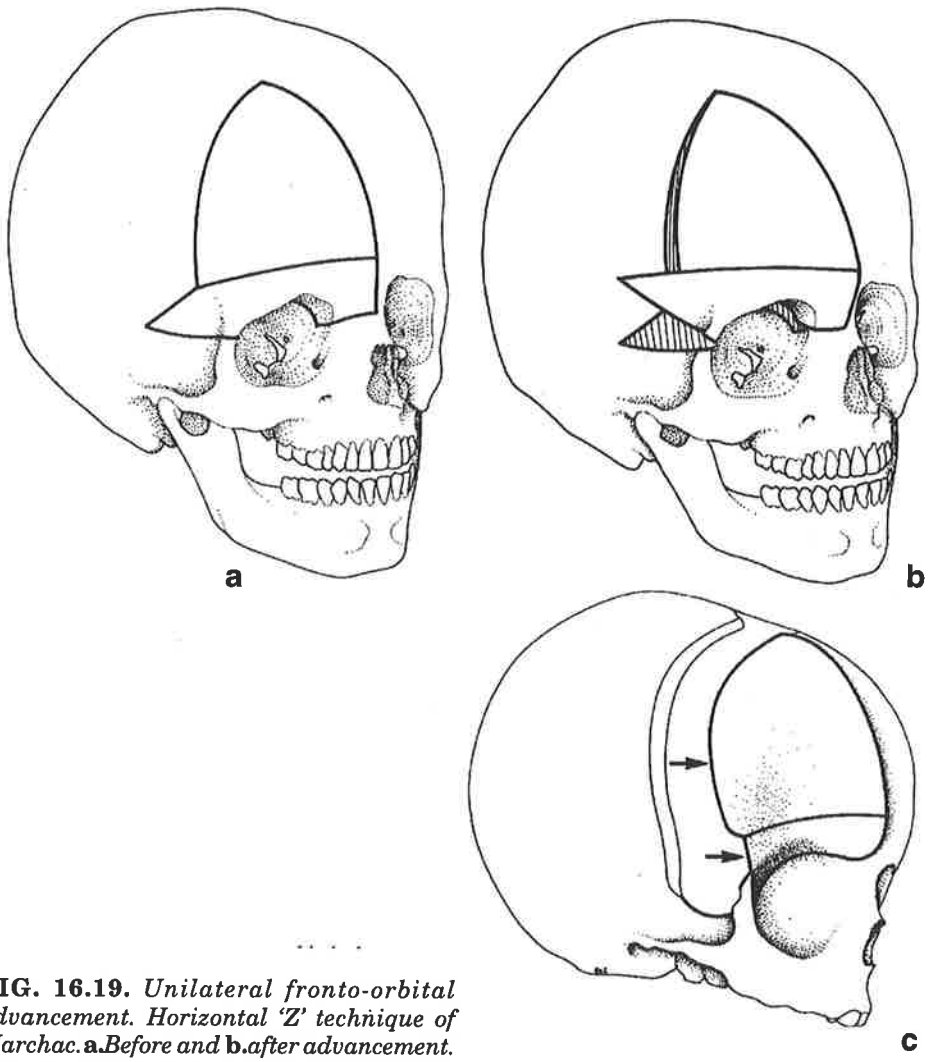


FIG. 16.19. *Unilateral fronto-orbital advancement. Horizontal 'Z' technique of Marchac.* **a.***Before and* **b.***after advancement.* **c.***Vertical 'Z' technique of Munro.*

Our method of unilateral frontal advancement in the early period has already been described (p. 163). To recapitulate, it is a unilateral version of the bilateral procedure. It entails a unilateral coronal craniectomy, a wide temporal decompression, and removal of the sphenoidal ridge. The fused anterior basal sutures are resected as far as possible. Whitaker et al. (1977) preferred an even more extensive craniectomy, taking the cut to the anterior maxilla below the orbital rim. This is shown in Fig. 16.17. The unilateral frontal bone flap and mobilised segment of the supra-orbital margin are advanced and anchored in place by wires passed into a spur of zygomatic bone behind the orbital rim. Hoffman and Mohr (1976) anchored their advancement with a strut of bone (Fig. 16.18) to the posterior margin of the craniectomy. Marchac (1978) used a horizontal Z-plasty (Fig. 16.19a, b) and Munro (personal communication; Fig. 16.19c) a vertical Z-plasty in the region of the frontozygomatic suture.

In the early period, experience summarised in Chap. 13 has shown that it is not obligatory to secure a perfect orbital contour, as continued cerebral growth is likely to improve the appearance. In neglected cases presenting in the intermediate or late periods more meticulous reconstructive procedures are needed to give a good fronto-orbital contour. We have used three different methods, selected at the time of the operation after appraisal of the degree and nature of the deformity.

1) Onlay bone grafts may be used to correct the deformed orbital contour.
2) The free frontal bone flap can be rotated to give a better forehead contour.
3) In addition to this rotation, the frontal bone flap can be laid in front of the supra-orbital margin and firmly wired to it; the contour can then be smoothed out by removing surface bone with a large powered burr.

In these intermediate and late unilateral fronto-orbital advancements no Silastic is used. If bone defects are created by the advancement, they are closed as much as possible with small grafts and with bone dust.

Subcranial LeFort III Osteotomy and Facial Advancement

This procedure has been used in selected cases of Crouzon syndrome and in isolated faciostenosis (p. 52).

Anaesthesia is initially established by oral endotracheal intubation and a tracheostomy is then performed. The various catheters for monitoring and for fluid replacement are inserted and the patient's occiput is then supported in a padded neurosurgical rest. This support is important in all long craniofacial operations: prolonged pressure on the occiput can cause baldness or even scalp necrosis. The head should be lifted from the rest at least every 4 h.

Bone grafts are taken from the right side of the rib cage and the right iliac crest, and stored in antibiotic solution (Methicillin 0.2 %). When this has been done the surgeon further secures the previously cemented cap splints with transpalatal and circummandibular wires. The members of the surgical team then scrub again and while they do so the anterior scalp is shaved.

The scalp is sterilised with the sequence of antiseptic solutions detailed on p. 97. Betadine in half strength is used to sterilise the conjunctive and to clean the face, including the anterior nares and buccal cavity. The head is draped to allow access to the eyes and mouth as well as to the scalp; however, the face is temporarily covered with a plastic sheet (Op-site). Throughout the operation intravenous chemotherapy is given and this is continued for 5 days after operation; the choice depends on the bacterial flora of the nose and mouth, but as a rule we now use concurrent flucloxacillin and amoxycillin.

After all the planned incisions have been infiltrated with a 1:200 000 solution of adrenaline, a bicoronal scalp flap is turned down. In our earlier operations we used also the buccal sulcus and conjunctival incisions, but latterly we have been able to carry out all the necessary manoeuvres through the scalp flap exposure. A wide subperiosteal dissection is done exposing the orbits from above, the nasolacrimal apparatus being carefully preserved. The orbital contents are guarded with malleable retractors and subperiosteal dissection is carried around the medial and lateral walls to the orbital floor. The zygomatic bones on both sides are widely stripped, the masseter muscles being detached from the zygomatic arches. We agree with Jackson (1978) that periosteum must be incised at all proposed osteotomy sites.

The osteotomies are then marked with indelible pencil (Fig. 16.20a). The cuts begin at the nasal bridge and are extended back and down above the lacrimal fossa. A cut is then made through each medial orbital wall behind the posterior lacrimal crest and across the orbital floor. Laterally the osteotomies are made through the frontozygomatic sutures and on to the lateral orbital walls. Each cut extends to the temporal fossa and care is taken to avoid injury to the temporal

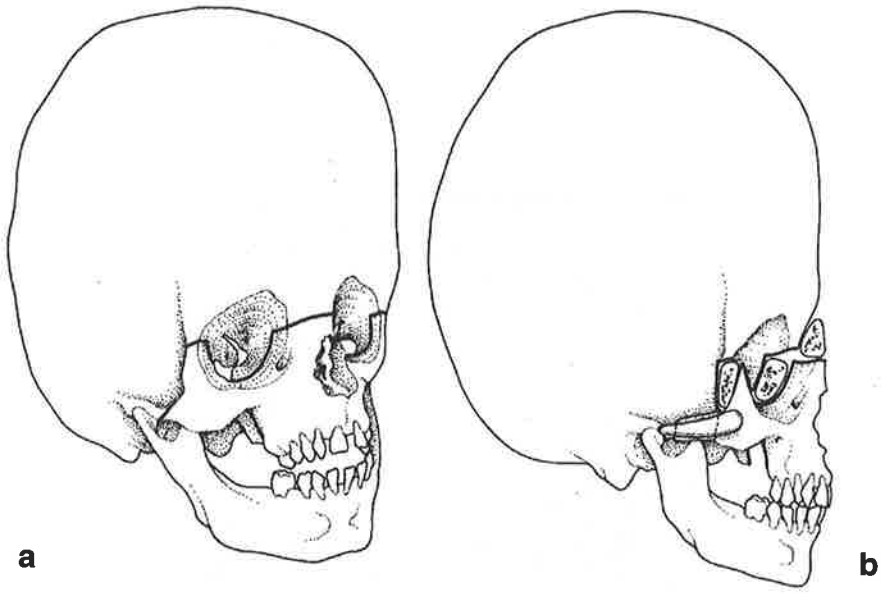


FIG. 16.20 a,b. *Subcranial LeFort III osteotomy.* **a.** *The line of the subcranial osteotomy extends across the glabella beneath the level of the cribriform plate, down the medial orbital wall behind the posterior lacrimal crest, across the orbital floor to the inferior orbital fissure and upwards through the lateral orbital wall to the frontozygomatic suture. The zygomatic arch is cut obliquely and the dysfunction is completed by separation of the maxilla from the pterygoid plates.* **b.** *Movement of the midface is forward and downward. The defects are filled with bone grafts.*

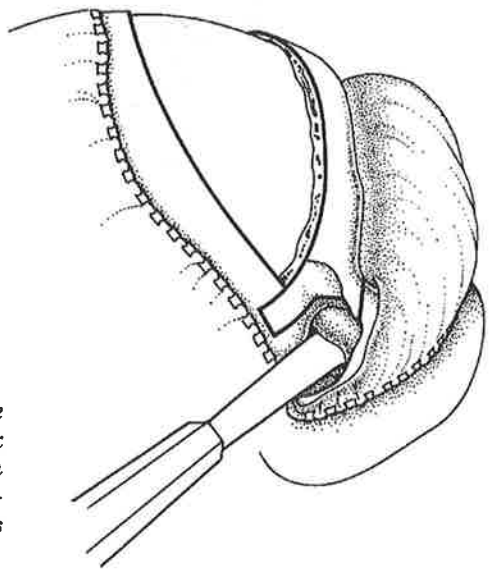


FIG. 16.21. *A bicoronal scalp flap gives wide exposure to the orbitocranio-facial complex (see Fig. 8.5b for skin incision). This diagram shows how the osteotomy at the pterygo-maxillary fissure can be made via this exposure from above.*

lobes which may lie far forward if there is orbital hypoplasia (see p. 234). Each lateral wall cut is extended to the infraorbital fissure with the powered saw (Stryker or Zimmer) and this cut is then completed across the orbital floor with a fine osteotome to join the medial osteotomy. The zygomatic arches are then divided obliquely. A curved osteotome is then inserted into the pterygomaxillary fissure from above via the temporal fossa (Fig. 16.21). Its position is checked with a finger in the mouth and the osteotome can be felt when it has separated the maxillary tuberosity from the pterygoid process. This is done bilaterally. There may be bleeding from the many blood vessels around the pterygoid process (p. 231); this is controlled by packing. It then remains to separate the nasal septum from the base of the skull with a curved osteotome inserted through the glabellar osteotomy.

The maxilla can now be mobilised with the Rowe disimpaction forceps and with Tessier's special 'decrouzoniser' maxillary mobilising levers. The correctness of the final position is confirmed when the dental splints lock into place. Intermaxillary fixation is then effected by wiring the locked splints together.

The advancement of the maxilla leaves areas of bone deficiency and these are filled with bone grafts. If the face is to be lengthened in the vertical plane the interlocked maxilla and mandible are swung down and bone grafts are wired

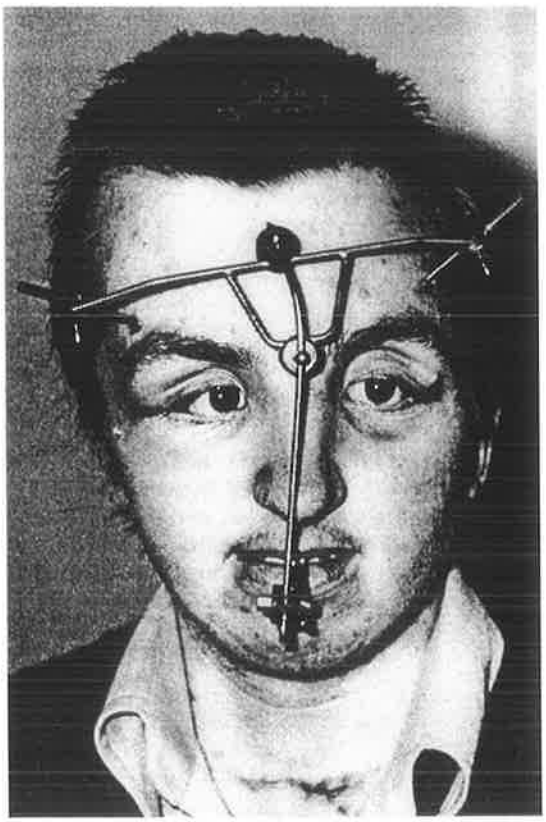


FIG. 16.22. *Craniomaxillary fixation using the Levant frame, after subcranial LeFort III osteotomy.*

into the resulting vertical defects in the glabella and in the lateral orbital walls. The advancement results in a horizontal defect in the zygomatic arches. Each arch is therefore lengthened by interposing a piece of rib. The ends of the rib graft are cored out to form sockets into which the sharp obliquely cut ends of the arch fit tightly (Jackson 1978). Additional support is given by a split rib graft wired over the arch and extending onto the anterior maxilla. We have never inserted bone grafts into the site of the pterygomaxillary osteotomy as Tessier (1971a) originally advised. The medial orbital walls and the orbital floors are

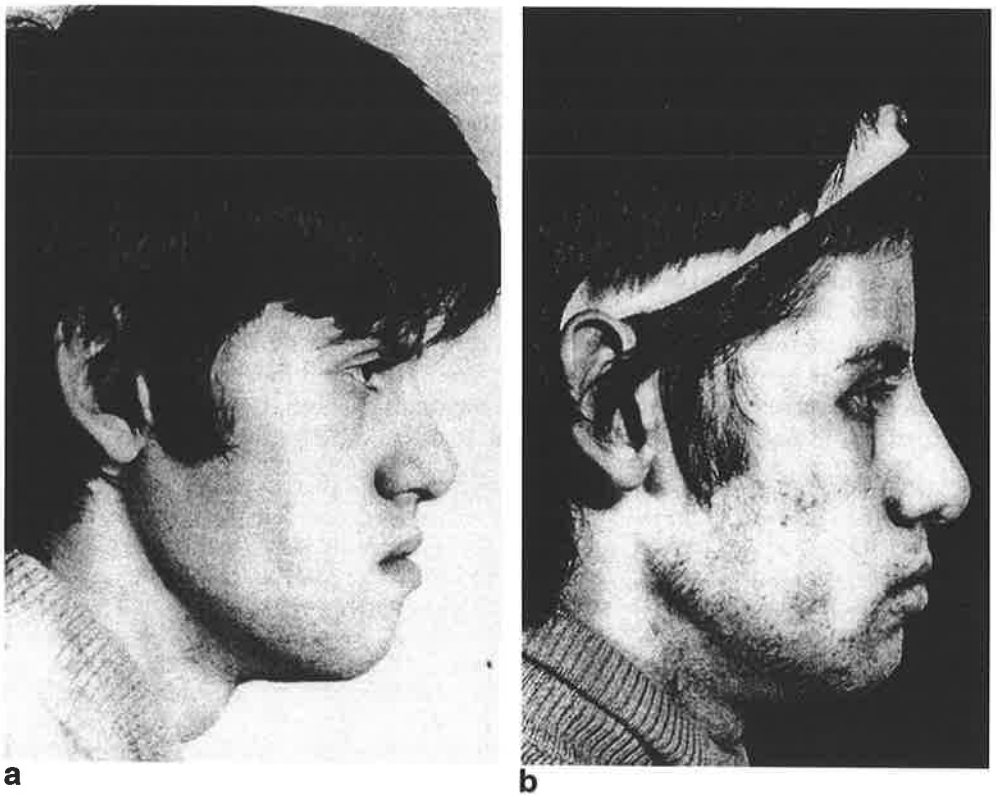


FIG. 16.23 a,b. *Results of subcranial LeFort III osteotomy in an 18-year-old boy* **a.** *Before and* **b.** *after surgery.*

grafted with thin moulded pieces of rib. Further onlay bone grafting to the glabella and the anterior maxillae may be needed. Closure is along standard lines: the temporalis muscle flap is sutured to the lateral orbital wall and the scalp is closed in two layers. Drainage is rarely used: ordinary gravity drains block, while suction drains may aspirate air through holes in the nasal mucosa at the osteotomy site.

Craniomaxillary fixation is effected with a Levant frame (Fig. 16.22). After a subcranial osteotomy the frontal pins of the frame can be inserted through the scalp flap into the bone just above the eyebrows. Care has to be taken in these cases of craniosynostosis as the calvarial bones are often thinned in an unpredictable way. Lastly, a nasogastric tube is inserted for postoperative feeding. This advancement usually gives an acceptable correction of the facial deformity (Fig. 16.23).

Transcranial LeFort III Osteotomy and Facial Advancement

The basic indications for this have been set out on p. 249. Initially all our cases of Crouzon and Apert syndromes were treated by this combined approach. As we gained more experience in interpreting the relations of the facial skeleton and the skull base, we became prepared to make more use of the subcranial route when there is no need for a fronto-orbital advancement and no special hazards in the region of the cribriform plate or the middle fossae.

Preparation and anaesthesia are as for the subcranial LeFort III operation, with the addition of measures to facilitate retraction of the brain.

As these combined operations bring together plastic surgical and neurosurgical teams, the sequence of events needs to be well planned and understood. After the harvesting of bone grafts and the securing of the dental splints, the neurosurgeon raises the scalp flap and dissects down to the orbits.

He is responsible for scalp haemostasis now and later, and must take care that haemostatic clips are temporarily removed every 4 h. He is then joined by the plastic surgeon and together they dissect the orbits and temporal fossae. In all these combined operations the concept of equal collaboration is stressed, and this facilitates exchange of ideas and techniques. The plastic surgeon then bares the zygomas and the rest of the orbital walls and floor. He next performs as many of the osteotomies described in the previous account of the LeFort III procedure as can be done without risk in the areas of dural vulnerability. He then marks the frontal osteotomy lines, which will demarcate a standard bifrontal bone flap (Fig. 16.24). The neurosurgeon cuts out and temporarily removes the flap; we have not found it necessary to retain it on a pedicle of temporalis muscle though some surgeons prefer to do so. An extradural dissection of the anterior fossa is then carried out to expose the foramen caecum, the anterior ends of the cribriform fossae, and the orbital roofs. If the middle fossae are ballooned, small

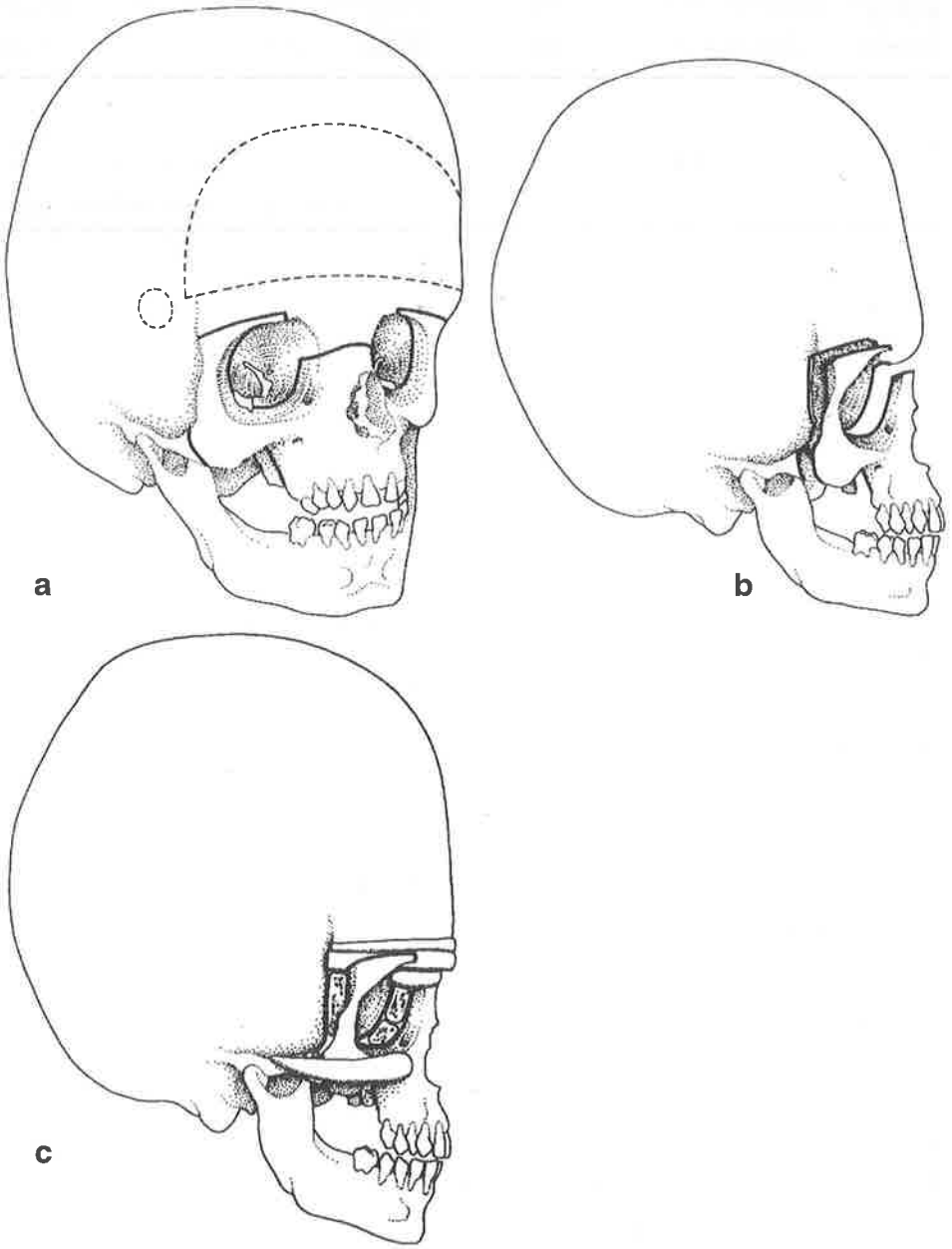


FIG. 16.24a–c. *Transcranial LeFort III osteotomy, using Tessier's frontal facade technique.* **a.** *The dotted lines indicate the frontal bone flap and the burr-hole which can be enlarged downwards to give access to the middle cranial fossa.* **b.** *Lateral view after advancement of the midface.* **c.** *Bone grafts are placed in the gaps: rib grafts are interposed in the frontal region and onlaid along the zygomatic arch.*

anterior temporal craniectomies are also made through which the middle fossa dura can be dissected away from the sphenoid bones in the vicinity of the proposed cuts in the lateral orbital walls, which are often shallow, especially in Apert syndrome. It is during the neurosurgical exposure of the anterior and middle fossae that many of the dangers associated with craniofacial surgery may be met. The dura is easily torn because of scars left by earlier surgery (p. 236) or because there are pathological hazards: bony spikes arising from the orbital roofs, potholes, adhesions between dura and periorbita in areas of bone erosion (p. 234). Dural tears must be meticulously repaired. Direct suture under tension is bad: when the brain swells, as it usually does after prolonged retraction, the dural suture line will dehisce. We have twice seen a persistent cerebrospinal fluid rhinorrhea from this accident. It is better to insert a generous graft of temporalis fascia or pericranium into the subdural space and suture the margins of the tear to this graft.

When the anterior fossa is exposed, the orbital and glabellar osteotomies can be completed in safety, the dura being covered with Telfa strips and protected from the powered saw by retractors.

The pattern of the LeFort III osteotomy cuts has been varied somewhat in our practice during the last 6 years. We began initially with Tessier's (1971c) original plan of splitting the lateral orbital wall and cutting a step in the zygomatic bone to allow advancement of the midfacial segment in the horizontal plane. In several patients with flattened supra-orbital margins we have used Tessier's (1976) more recent technique of advancing a facade of the lateral part of the supra-orbital ridge in continuity with the main facial mass (Fig. 16.24). This frontal facade is cut almost transversely, the contents of the anterior fossa and the orbit being protected in the usual way. Split rib grafts are placed behind the advanced facade.

Neurosurgical haemostasis must be as good as possible. Since no fronto-orbital advancement has been done there is relatively little dead space in the extradural plane. The bone flap is replaced with wire fixation and the scalp is sutured as usual.

Craniomaxillary fixation is again effected with the Levant frame, but it is undesirable to insert the fixating pins in or near the bone flap and they are therefore placed behind the bicoronal skin incision. Again care must be taken because the bone in this region may be thin. The effect of this operation is shown in Fig. 16.25.

Transcranial LeFort III Osteotomy, Facial Advancement, and Fronto-orbital Advancement

This represents an extension of the previous operation and is appropriate principally in severe forms of the Crouzon syndrome. It is a massive surgical procedure and needs to be accomplished with speed and efficiency if complications are to be avoided.

The scalp flap is raised and the periorbital dissection is done as in the previous procedure. Again, as many of the lower osteotomies as possible are done before the anterior fossa dissection is begun. When the frontal bone is exposed the plastic surgeon marks out the lines of a standard bifrontal bone flap and also delineates a fronto-orbital crown. This crown is given lateral flanges in each temporal region, in the vicinity of the sphenoidal ridge, for fixation at the end of the operation. The neurosurgeon then cuts and removes the frontal bone flap and strips the dura back from the floor of the anterior fossa even further than in the previous procedure. It is often helpful to avulse the dural anchorage from the floor of the foramen caecum to gain additional exposure. Small temporal

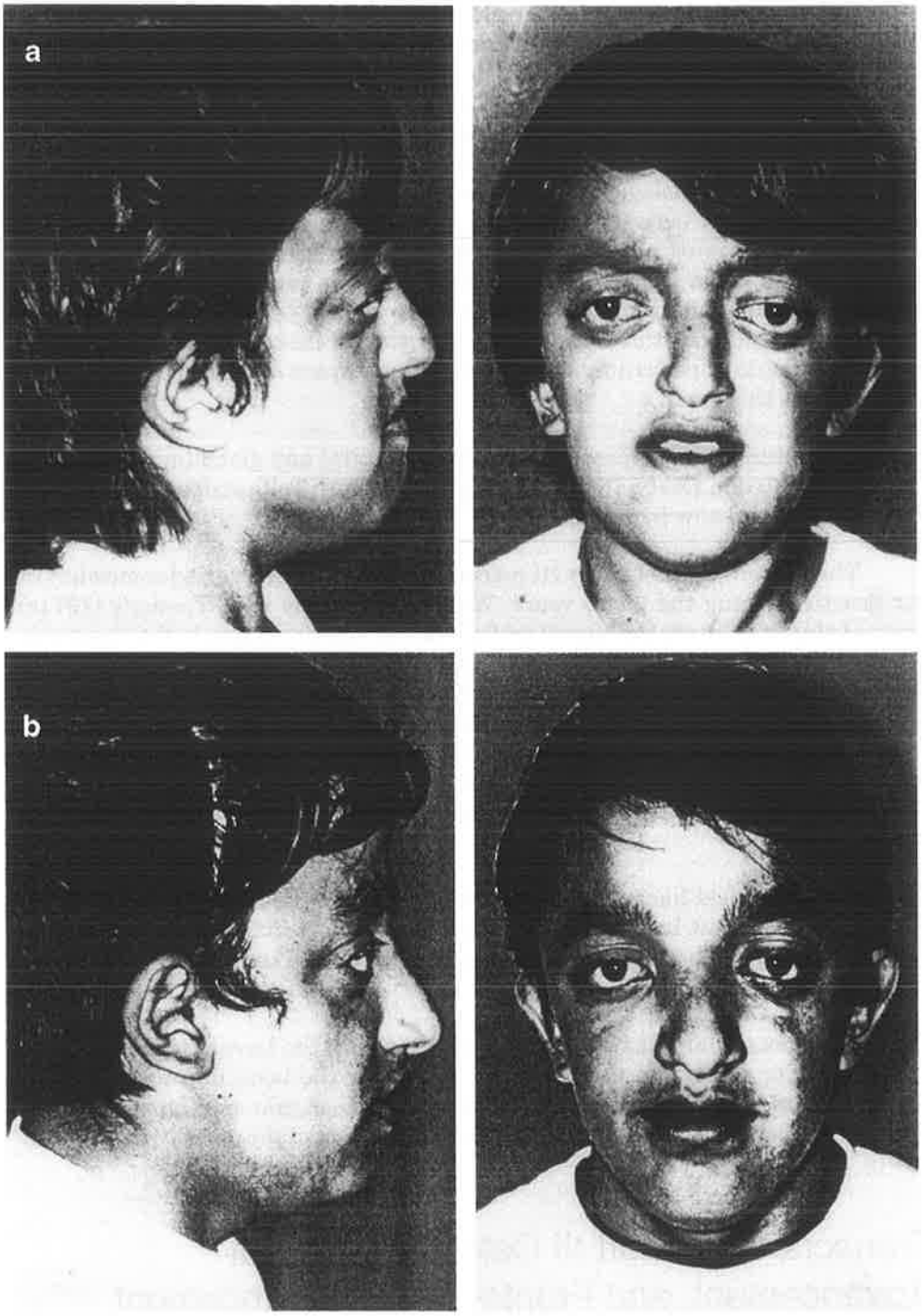


FIG 16.25a,b. *Twelve-year old boy with Crouzon syndrome corrected by transcranial Lefort III advancement osteotomy* **a.** *Before and* **b.** *after.*

craniectomies are usually needed to protect the dura in the vicinity of the planned temporosphenoidal flanges. The plastic surgeon then makes the orbital and glabellar cuts that demarcate the fronto-orbital crown, which is finally freed laterally in the region of the sphenoid ridges (Fig. 16.26). The crown is then temporarily removed. If the dura was torn, there is now an ideal opportunity for dural repair with grafting, as the frontal exposure is superb.

The plastic surgeon then completes the mobilisation of the midfacial segment, and the stage is set for the combined facial and fronto-orbital advancement. The desired advance of the fronto-orbital crown is measured on the temporosphenoidal flanges, which are the lateral fixation points of the crown. The desired distance of facial advance is verified by the exact occlusion of the performed cap splints and occasionally by a bite wafer. The various bone gaps

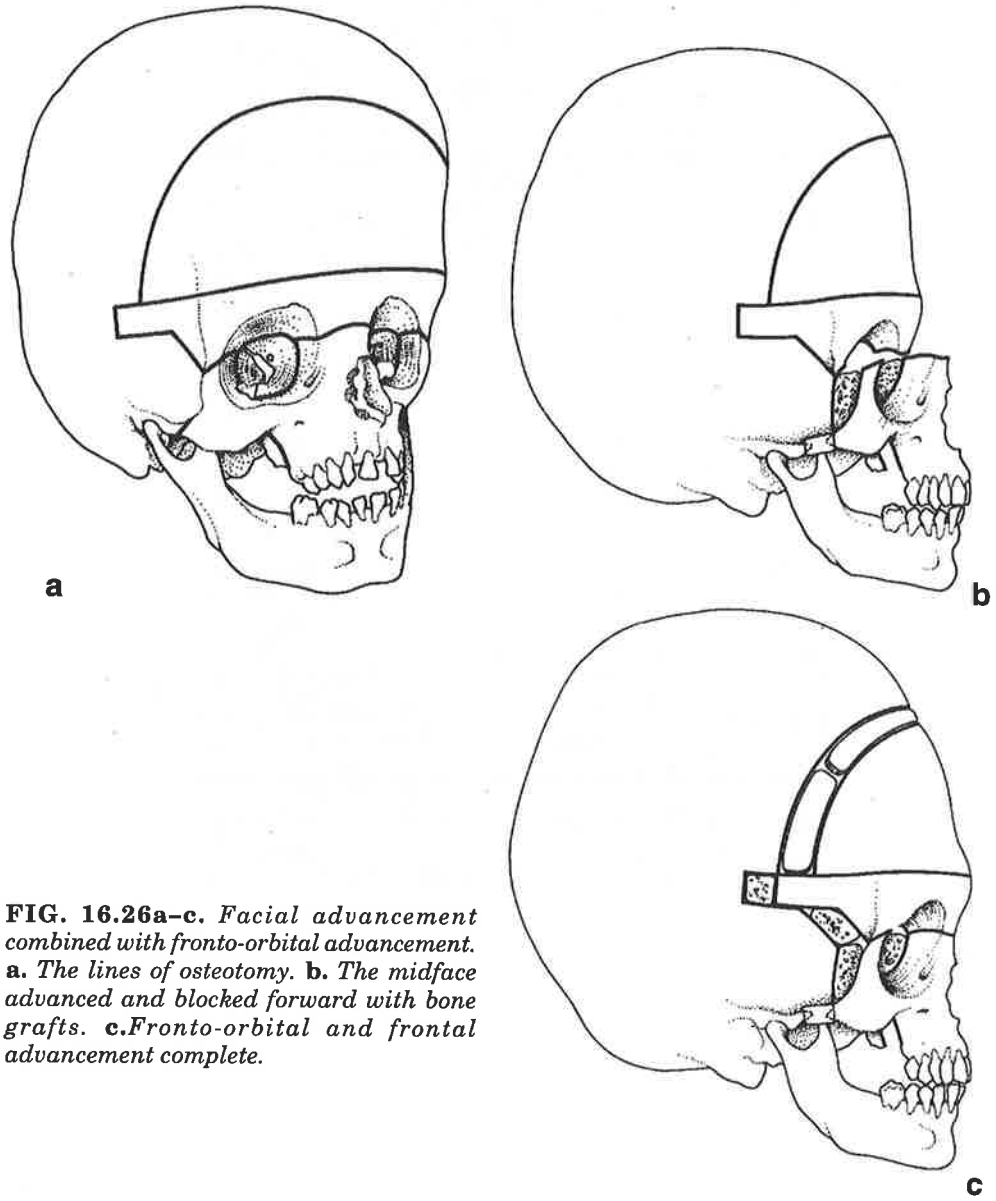


FIG. 16.26a–c. *Facial advancement combined with fronto-orbital advancement.* **a.** *The lines of osteotomy.* **b.** *The midface advanced and blocked forward with bone grafts.* **c.***Fronto-orbital and frontal advancement complete.*

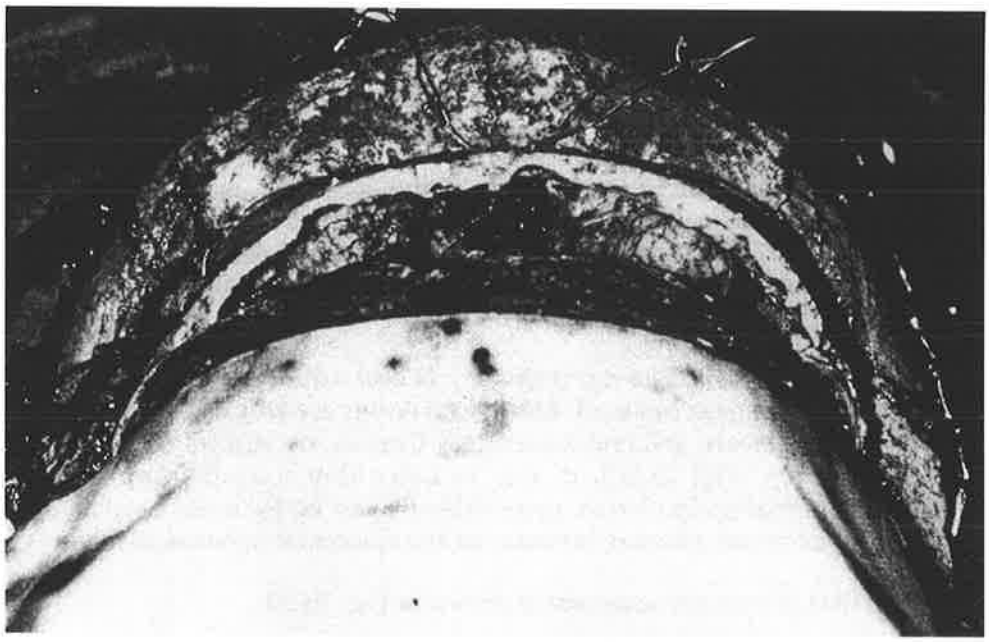


FIG. 16.27. *The large frontal extradural space after craniofacial advancement.*

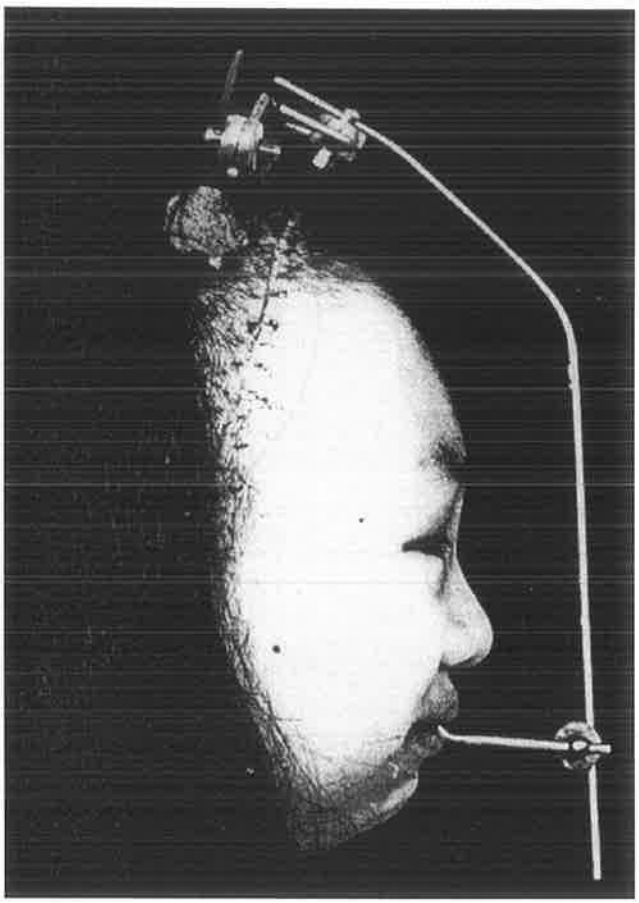


FIG. 16.28. *Craniomaxillary fixation after transcranial fronto-orbital and midface advancement.*

are then closed with bone grafts and the frontal flap is wired to the fronto-orbital crown, now securely wired in its advanced position. This usually leaves some exposed dura, which is covered with bone grafts. The bone dust formed during neurosurgical drilling is helpful as a supplementary graft.

This operation leaves a large frontal extradural space, usually in free communication with the paranasal air sinuses. This dead space (Fig. 16.27) is a potential site of infection, and invites continued bleeding. We know of no safe way to obliterate this obviously dangerous cavity. Our worst postoperative extradural haematoma developed in such a cavity despite two suction drains. It has to be accepted as one of the hazards of the operation, and the risks are minimised if dural haemostasis is good. The usual neurosurgical manoeuvre of suturing the dura to the bone edges has some value; if the dural attachment to the foramen caecum was avulsed then it should be hitched to the fronto-orbital crown.

Dural haemostasis, like dural closure, is best achieved before the crown and frontal flap have been replaced. Extradural drains are only used in desperate situations. Scalp closure and craniomaxillary fixation are carried out as in the previous operation (Fig. 16.28). It may be noted that a large fronto-orbital advancement makes scalp closure quite difficult and under some tension. We have not yet had to use relaxing incisions in the epicranial aponeurosis.

The effect of this advancement is shown in Fig. 16.29.

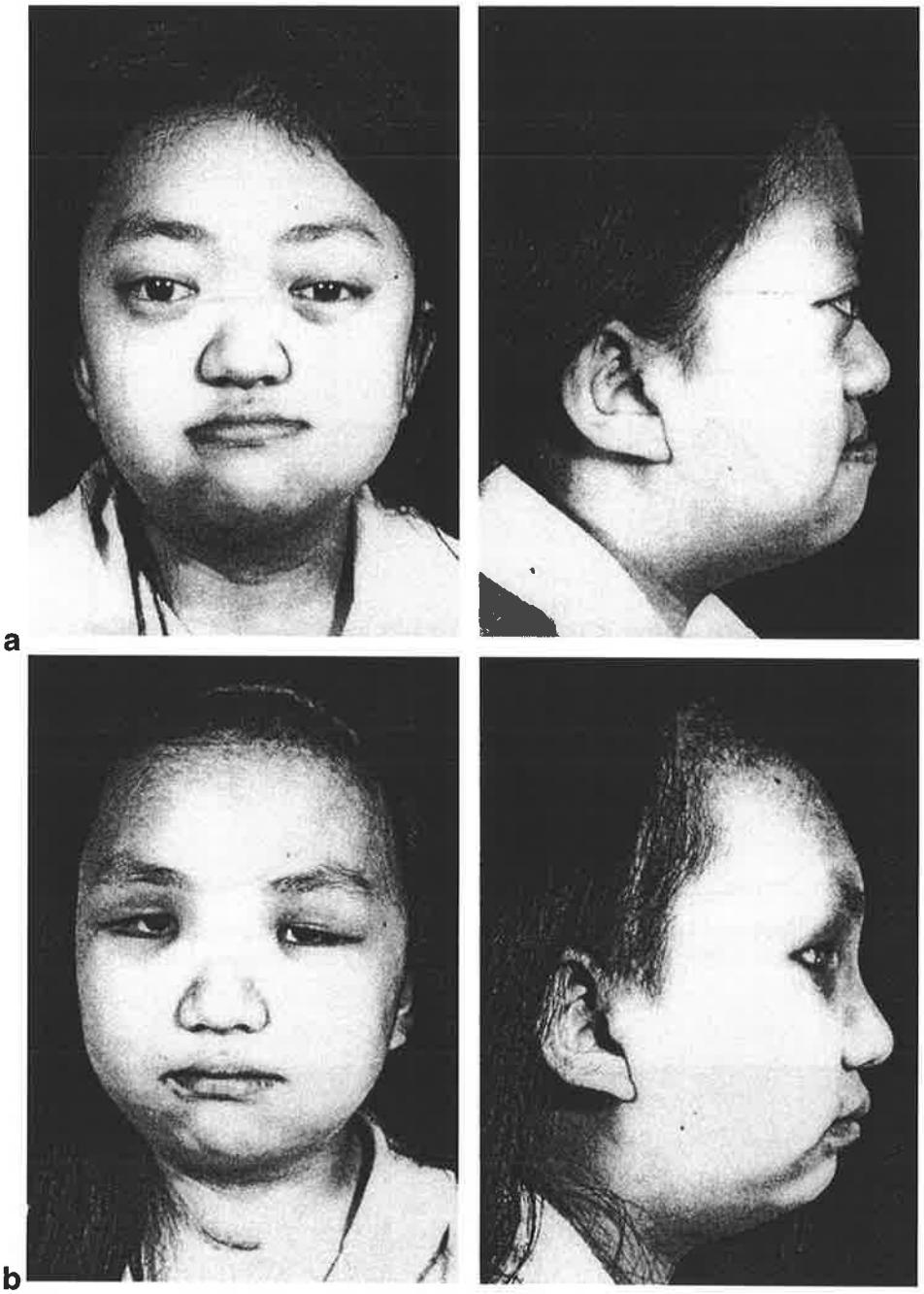


FIG.16.29 a,b. *Thirteen-year-old Chinese girl with Crouzon syndrome, treated by fronto-orbital and midface advancement.* **a.** *Before.* **b.** *After.*

LeFort III Osteotomy Combined with LeFort I Osteotomy

This combination is appropriate when the upper jaw has to be moved a greater or a lesser distance than the orbitonasal complex. The upper components of the LeFort III osteotomy are made through the exposure given by the bicoronal scalp flap, as described above. The buccal sulcus is then incised and the anterior surfaces of the maxillae are bared by subperiosteal dissection. The LeFort I osteotomy is made along lines running on each side above the apices of the teeth and below the infra-orbital foramina (Fig. 16.30). The cuts enter the piriform aperture medially and run back along the lateral walls of the nose, which are divided with an osteotome. The septum is separated from the floor of the nose, again subperiosteally. Laterally each cut runs into the pterygomaxillary fissure, which is finally opened with Kawamoto's curved osteotome. The midfacial skeleton is

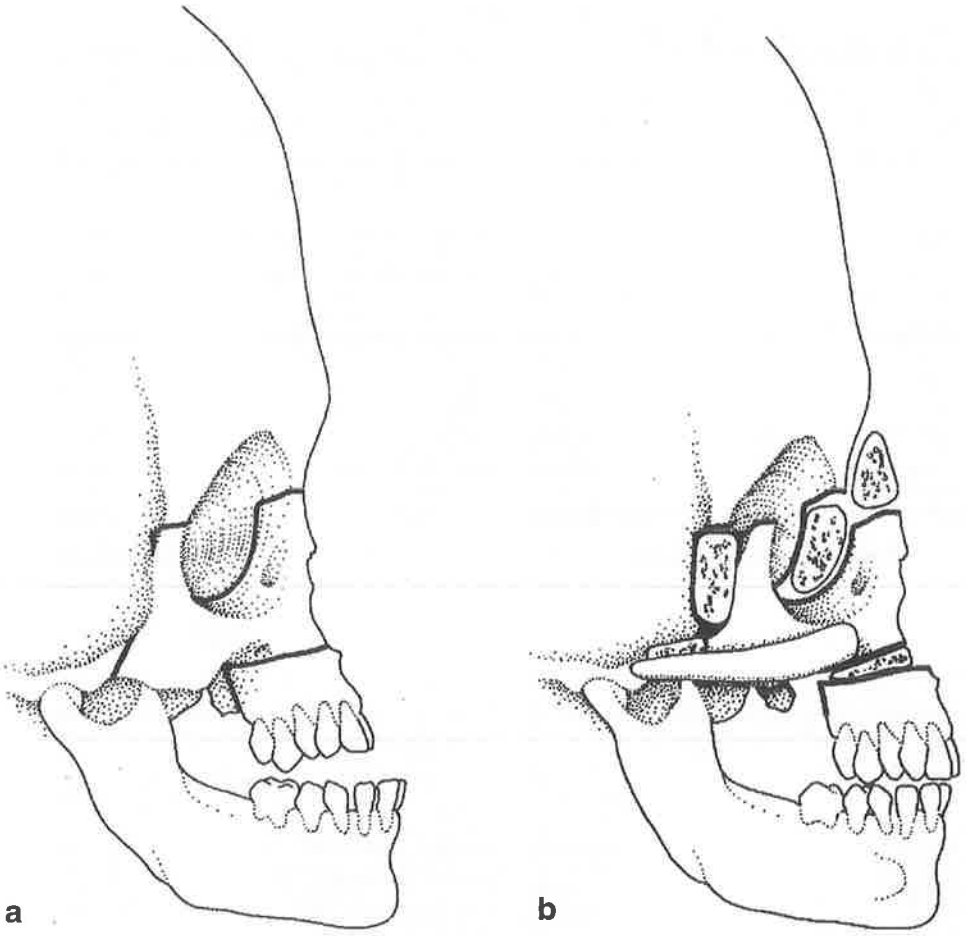


FIG. 16.30 a,b. *LeFort [11 plus LeFort I osteotomy.* **a.** *Lateral view' of the lines of osteotomy.* **b.** *The combination allows advancement of both blocks by differing amounts. The defects are bone-grafted.*

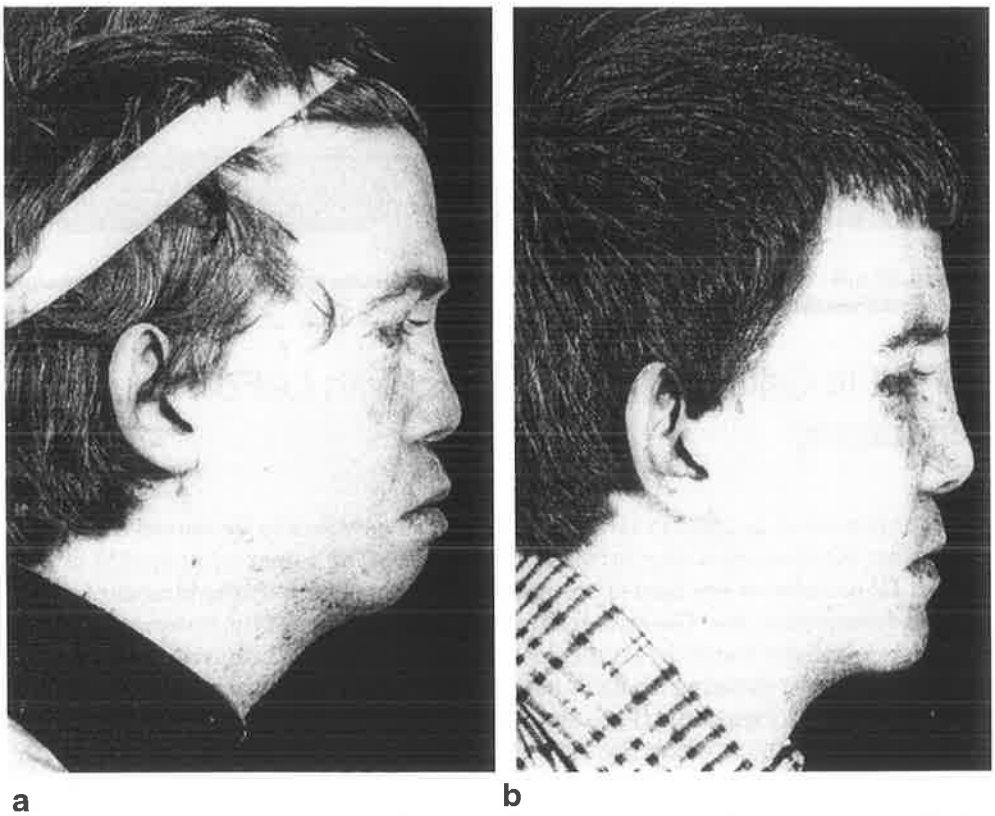


FIG. 16.31 a,b. *Young man with unidentified craniosynostosis syndrome: midfacial hypoplasia treated LeFort III minus LeFort I osteotomy.* **a.** *Before.* **b.** *Alter.*

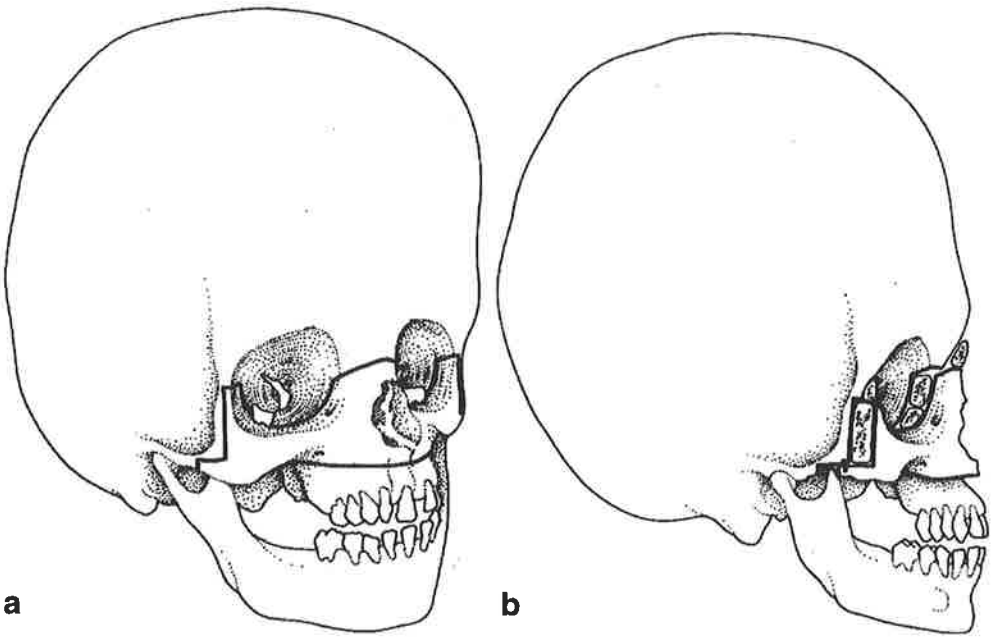


FIG. 16.32 a,b. *LeFort III minus LeFort I osteotomy.* **a.** *The lines of osteotomy.* **b.** *The advanced portion of the midface leaving the tooth-bearing maxilla posteriorly. Bone grafts and the zygomatic step stabilise the advanced segment.*

now divided horizontally into two segments, each independently mobile, which can be fixed in the desired positions. The lower segment is secured with cap splints and craniomaxillary fixation, the upper segment with bone grafts wired into the orbital and glabellar defects.

LeFort III Osteotomy Minus LeFort I Osteotomy

This combination was described by Tessier (1971d, 1976). We have used the procedure in a case of craniosynostosis believed to be an unreported syndrome (Fig. 16.31) and in cases of faciostenosis with exophthalmos and nasal retrusion but normal occlusion. Again the bicoronal and transbuccal exposures are used, as in the previous procedure. Osteotomies are performed which separate the malar and orbitonasal complex en bloc, leaving the lower maxillae attached to the pterygoid processes. Careful leverage is needed to mobilise the isolated facial segment without breaking the component bones. Rib grafts can be fitted into the divided zygomatic arches as in the complete LeFort III procedure (Fig. 16.32), and good fixation is obtained by direct wiring across the line of section in the floor of the piriform aperture. The orbital and maxillary bone defects are filled with bone grafts. No external fixation is needed.

Transcranial Orbital Translocation

Hypertelorism is corrected by medial orbital translocation. Orbital dystopia is corrected by vertical orbital translocation. In our series of cases of craniosynostosis, hypertelorism and orbital dystopia have been seen in various combinations in Saethre-Chotzen syndrome, Cohen syndrome, Crouzon syndrome, and simple frontal plagiocephaly. In all cases treated, the transcranial route has been used. In principle. corrections have been achieved by the method of Converse et al. (1970), in which the orbital contents are shifted without injury to the cribriform plates and nasal septum.

The bicoronal scalp flap is used. The subperiosteal dissection can be taken almost to the nasal tip and there is no need for the midline nasal skin incision

necessary in the correction of the hypertelorism associated with frontonasal encephaloceles. Originally we approached the orbital floor and anterior maxilla through the conjunctival incision but now the entire procedure is usually done from above. When the frontal bone, orbits, and upper facial skeleton have been exposed the proposed osteotomy lines are drawn and the plastic surgeon makes as many of the cuts as possible without entering the cranial cavity This reduces the time in which the brain is exposed and lessens the risk of inadvertent cerebral damage from hand-held powered tools.

The lateral orbital wall is split sagittally, the cut being taken into the body of the zygomatic bone (Fig. 16.33a). The cut then goes medially across the anterior maxilla below the infra-orbital nerve to the margin of the piriform aperture. The medial cuts define the paramedian blocks of bone to be removed. The orbital osteotomies are made well back in the orbit, posterior to the transverse equator of the eyeball: this is essential as otherwise the eye will not move when the orbital box is translocated. The cut in the medial orbital wall extends inferiorly across the orbital floor to the inferior orbital fissure and up the lateral wall. The upper orbital osteotomies are not done until the anterior fossa is exposed.

This is done by the neurosurgeon, who now cuts a standard bifrontal bone flap and dissects the anterior fossa dura back to the foramen caecum, the cribriform fossae, and the orbital roofs. Again added exposure is obtained by avulsing the dural anchorage from the foramen caecum. The upper orbital osteotomies are then marked out by the plastic surgeon, and the planned cuts leave intact a transverse frontal bar of bone corresponding in position with the fronto-orbital bar of other operations. This bar will support the orbits when they are relocated. The exposed dura is protected by Telfa strips and by malleable retractors, and the upper osteotomies are then made as shown in Fig. 16.33b. The sphenolateral corner is often hard to cut as the bone is thick and one needs a good three-dimensional understanding of the direction of the cut. Sometimes the lateral orbital wall is too thin to be split and then it is mobilised in its full thickness, leaving only the orbital rim attached laterally to the zygomatic arch.

Now the demarcated paramedian blocks of bone are excised. These blocks are strips of the nasal, frontal, and ethmoid bones, being cut out from in front. The ethmoid air cells are exenterated on each side to give room for the medial orbital translocation (Fig. 16.33b). The inferomedial edge of the mobilised orbital box is excised to prevent narrowing of the piriform aperture. If necessary one can also do a submucous resection of the septum or a septoplasty together with reduction of the turbinates to prevent nasal obstruction.

The orbits should now be mobile and they are shifted a calculated distance medially along the frontal bar, to which they are wired. Solid inlay bone grafts are placed laterally in the orbital wall. Anterior maxillary bone grafts are placed over the osteotomy sites to prevent hollowing of the cheeks.

The canthal angles must also be translocated. Medial canthopexies are done by catching the medial canthal ligaments with 3/0 stainless steel sutures which are left double. The ends of each suture are passed transnasally with an awl and tied on top of the nasal bones, on which a bone graft is usually laid. Dexon sutures are used to pull the periosteum against the bone of the nasal pyramid to prevent formation of a subperiosteal haematoma that might organise and give rise to fibrosis and intercanthal thickening. After dural haemostasis is secured the frontal bone flap is replaced and the scalp is closed.

This describes the correction of hypertelorism associated with craniosynostosis as done in a case of Cohen syndrome (Fig. 16.34). It is less extensive than the correction originally described by Tessier (1972), in which the cribriform plate and nasal septum are sacrificed. This more extensive procedure is occasionally needed in correcting the hypertelorism associated with central facial clefts and encephaloceles, with which this book is not concerned.

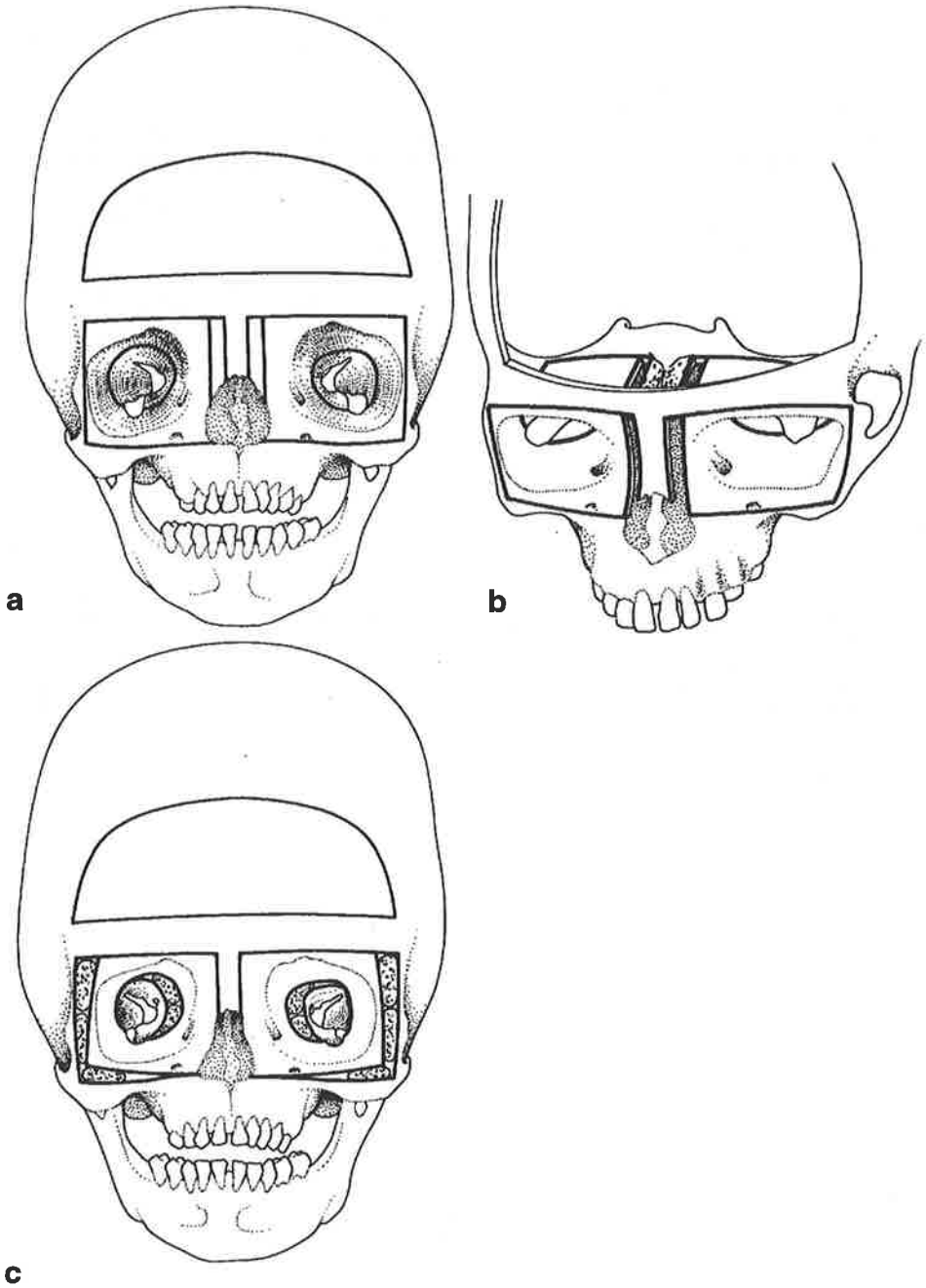
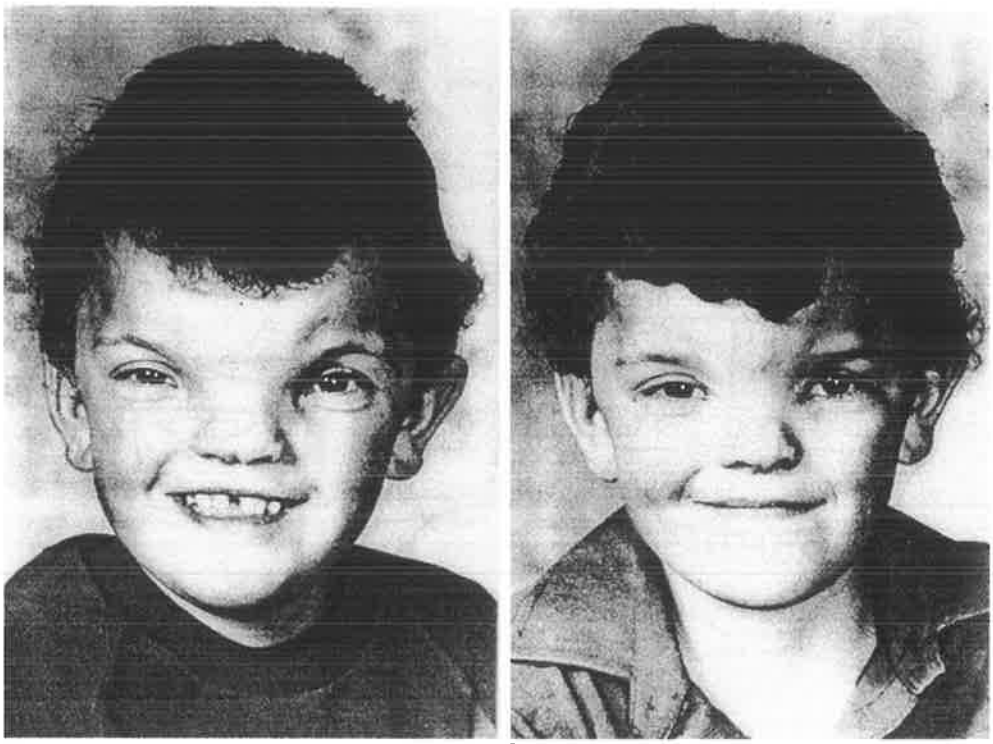


FIG. 16.33a–c. *Surgical technique used in the correction of orbital hypertelorism, after Converse. The cribriform plate and midline nasal structures are preserved.* **a.** *Osteotomy lines.* **b.** *The bone resection consists of paramedian segments on the face extending into the anterior cranial fossa on each side of the cribriform plate.* **c.** *After translocation of the orbits the gaps are filled with bone grafts.*



a b

FIG. 16.34. *Orbital translocation for hypertelorism associated with Cohen syndrome.* **a.** *Before operation.* **b.** *After operation.*

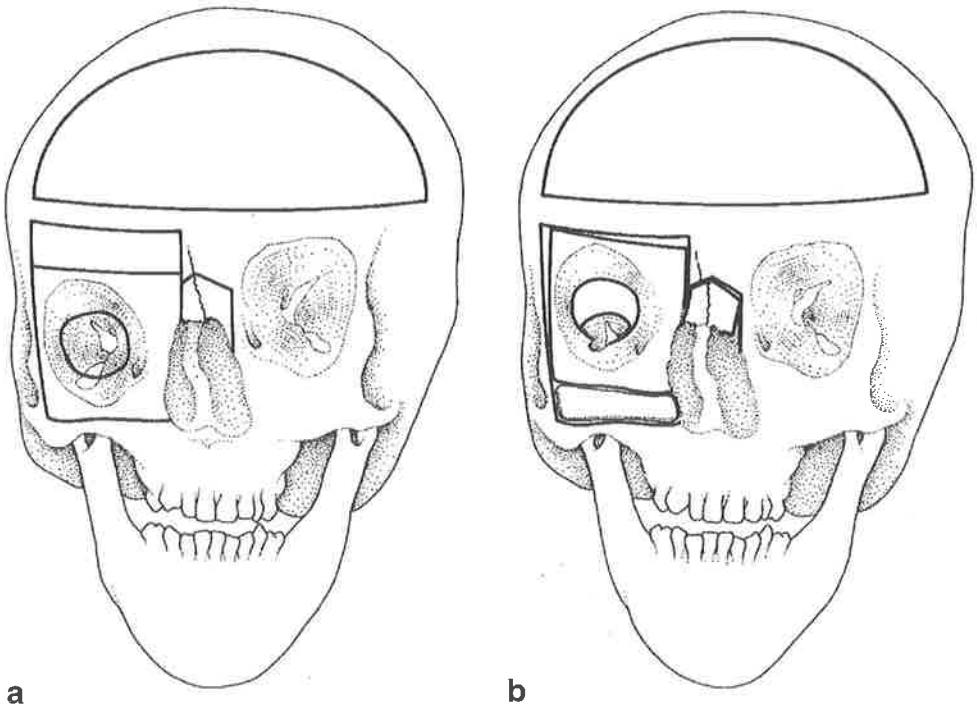


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FIG. 16.35 a,b. *The osteotomies for correction of the forehead and upper face in plagiocephaly, showing vertical translocation of the orbit. A LeFort I type osteotomy, with a mandibular osteotomy, can be performed at a later stage to straighten the occlusal plane.* **a.** *Before.* **b.** *After.*

Orbital dystopia is corrected by moving one orbital box in the vertical direction. The orbital osteotomies are similar (Fig. 16.35). The frontal bar is again preserved and a planned strip of bone is cut from its lower surface to receive the translocated orbit. This strip is a wedge if an element of rotation is being corrected, a rectangle if a rectilinear shift is desired. The excised strip of frontal bone makes an elegantly exact bone graft to fill the inferior bone defect in the anterior maxilla.

Combined Procedures for Severe Plagiocephaly with Orbital Dystopia, Nasal Deformity, and Tilted Occlusion

This complex of asymmetrical deformities can be corrected in part through the transcranial approach: this permits a fronto-orbital advancement together with correction of orbital dystopia (Fig. 16.35). A perinasal osteotomy can be done to straighten the nasal pyramid; with care the nasal mucosa can be preserved intact. In the past we have also corrected the tilted occlusion at the same time by a LeFort I osteotomy. However, reports from other centres (Munro, personal communication) suggest that this combination carries an increased risk of infection and we now favour doing the LeFort I osteotomy at a second stage.

Complementary Techniques and Secondary Revisions

Bone Grafting

This is an essential procedure in the operations of facial and fronto-orbital advancement and in orbital translocation. Onlay bone grafts may be needed to augment the infra-orbital rim in conjunction with advancement of the maxilla. The rim, which is often thin and sharp, may curve abruptly down to the hypoplastic maxilla; the resulting indentation is filled with split rib or cancellous bone moulded with Tessier's contouring forceps. If possible, the grafts are wired into place.

Bone grafts may also be needed to give a good nasal profile. When using the bicoronal scalp flap we have not needed L-shaped nasal bone grafts: we have preferred to build up the nose with a cantilever graft held in position by the wires used in the transnasal canthopexy (see p. 271). Rib is used for the cantilever graft, either as a flat elongated ellipse with a transverse dorsal groove to secure the wire or on edge to give a well-contoured naso-frontal angle.

Bone grafts may also be used to fill secondary calvarial defects, and we have so far not had to employ alloplastic materials such as acrylic or titanium (Simpson 1965, 1979) in cases of craniosynostosis. A massive loss of calvarial substance might well require alloplastic cranioplasty, but if possible this should be avoided.

Bone grafts are harvested from three sites: the calvaria, the rib cage, and the iliac bone. Small pieces of calvarial bone can often be taken en passant during a cranial reconstruction. The drilling of burr-holes yields useful bone dust. In adults the outer table of the skull can sometimes be separated, leaving the inner table for protection, but usually the bone is too thin to allow this to be done. Rib grafts are more frequently used. They are harvested in pairs through a single skin incision, one rib being left intact between each pair of resected ribs to prevent thoracic collapse. If the periosteum is reconstructed the ribs will regenerate very well (Longacre 1968). Care should be taken to avoid injury to the pleura. Iliac bone is cut, usually as a rectangular block, from the inner surface of the ilium;

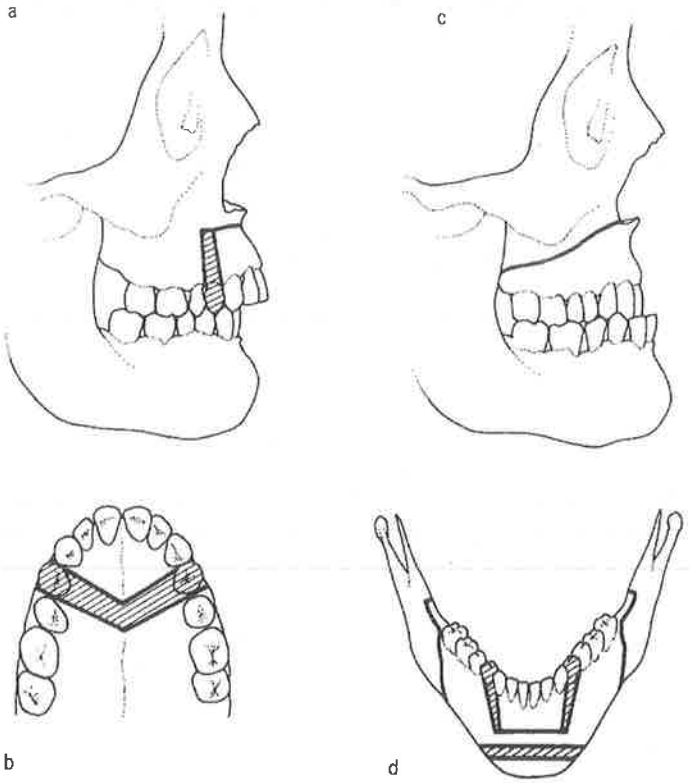


FIG. 16.36a-d. *Secondary surgical procedures on the jaws.* **a.** *Anterior maxillary osteotomy.* **b.** *Anterior maxillary osteotomy.* **c.** *LeFort I osteotomy.* **d.** *Three types of mandibular osteotomy — the bilateral sagittal split, the anterior segmental osteotomy of Köle, and the chin slide (sliding advancement genioplasty of Osbwegeser).*

the iliac crest (cartilaginous in children) is elevated and preserved, still attached to the lateral iliac periosteum.

Rhinoplasty and Septoplasty

Rhinoplasty may be necessary in cases of Crouzon and Apert syndromes. It is usually done as a secondary procedure. The shape of the nose varies greatly in these syndromes (see Fig. 15.7) and it is often the nasal cartilage's that determine the deformity. Care should be taken to avoid an excessive resection of the bony hump when the main component of the nasal prominence is cartilaginous. Usually the operation amounts to a small reduction of the bony hump, a larger resection of cartilage, infracturing of the lateral walls, and elevation of the nasal tip.

Nasal obstruction is virtually a constant concomitant of those syndromes and part of this obstruction results from the distorted 'cockled' septum. Septal resection is therefore needed, usually as a separate procedure, though occasionally the septoplasty can be done during the course of surgery for hypertelorism through the bicoronal flap.

Mandibular Reconstruction

As has been emphasised, the apparent mandibular prognathism often seen in the Crouzon syndrome is only relative: the mandible may actually be small. In one of our cases this was accentuated by previous surgery: the mandibular condyles had been excised in childhood to give a good dental occlusion.

Most often the chin appears flattened and may need augmentation even when good occlusion has been achieved. We have used Silastic implants when only a small soft tissue advancement is needed; we have more usually preferred sliding advancement genioplasty (Fig. 16.36d).

Mandibular reconstruction may also be needed for prognathism, either because the mandible has continued to grow relative to the maxilla or because there has been maxillary retrogression after an initially successful midfacial advancement. In our experience mandibular overgrowth is the commoner problem. As a result there may be a class III malocclusion. Most of these cases have been successfully treated by orthodontic methods, but occasionally the mandible must be set back either by an anterior segmental alveolar osteotomy (Kole 1959) or by a sagittal split mandibular osteotomy (Trauner and Obwegeser 1957): Fig. 16.36d. If there has been a serious relapse of the midfacial advancement it is best corrected by a LeFort I osteotomy (see p. 267), delayed until eruption of the permanent teeth.

Correction of Velopharyngeal Incompetence

Advancement of the midface changes the speech pattern and occasionally produces nasal escape. All patients undergoing midface advancement are examined before and after operation by nasendoscopy, with lateral cine fluoroscopy where necessary, and these investigations may demonstrate velopharyngeal incompetence. This was shown in three of our cases of Crouzon syndrome. It is important to wait at least 3 months before trying to correct this, as the elongated velum may with time make a functional recovery in its new position. The size of the residual deficiency must be estimated as accurately as possible. Correction is achieved with a pharyngeal flap based superiorly; this must not be too large or it will obstruct the normal nasal flow and perpetuate the hyponasal speech disorder so characteristic of Crouzon and Apert syndromes and of faciostenosis generally.

Canthopexy

Medial canthopexies are routinely done in the course of major craniofacial operations (see p. 271) but further surgery is often necessary. Unsightly epicanthic folds may develop and these are corrected by Mustarde's (1971) 'jumping man' type of Z-plasty. Lateral canthopexy may be needed, chiefly to correct an antimongoloid slant. We try to achieve this at the primary operation by hitching the lateral orbital periosteum in the vertical direction to one of the wires used in the frontozygomatic area or to the advanced temporalis aponeurosis, using 4/0 nylon. This may be unsuccessful and then secondary correction is needed. We have done this by a Z-plasty, transposing the lateral canthus as an arm of the Z and taking it upwards to the lateral orbital margin where it is fixed to a hole drilled in the bone. We have not been able to define the anatomy of the lateral canthal ligament as described by Couly et al. (1976).

Correction of Ptosis

Ptosis which was present before operation may need to be corrected. If ptosis is associated with lateral canthal dystopia, then it may be improved when the lateral canthus is fixed in a better position. Ptosis may also appear after operation as a result of the subperiosteal orbital dissection. We wait at least 9 months before considering whether to reoperate. In all our cases the ptosis has returned to the preoperative state after this period. When correction is needed it is done by shortening the levator palpebrae superioris.

Squint Surgery

In Crouzon and Apert syndromes there is usually some degree of imbalance in the external ocular muscles. After surgery this imbalance is still present but its character may have been altered by the subperiosteal dissection of the orbits, which detaches the anchorages of the superior and inferior oblique muscles. It is our policy to delay any definitive surgery for squint or diplopia until 9 months after operation. By then any residual muscle imbalance is likely to be permanent and can be treated on its merits.

Syndactyly

Apert syndrome (acrocephalosyndactyly type I) is invariably expressed as a severe fusion of all four fingers although the little finger may be only partly syndactylised to the main mass. The index, middle, and ring fingers have a common nail and there is always distal bony union between these fingers. The thumb is usually partly syndactylised. It is shorter than normal, and its function is severely limited because of its poor range of joint movement. There may or may not be an interphalangeal joint present in the thumb but the metacarpophalangeal joint has a fair range of movement, at least in early life. The fingers have poorly functioning interphalangeal joints and in adults they are invariably stiff. The toes also show severe syndactyly and as a rule all five are fused together.

In the other types of acrocephalosyndactyly and in acrocephal–opolysyndactyly, fusion is much less severe and usually function is adequate. The appearances may however cause distress.

Hands

The thumb should be released in the first few months of life. This can occasionally be done by using a Z-plasty, making the flaps as large as possible. It has been found however, that when a Z-plasty only is used, further release will be required at about 5 years of age using a full thickness skin graft to obtain completely free movement of the thumb and to deepen the web to allow for grasping of larger objects. It appears that best results are obtained by the use of a Z-plasty combined with a full thickness skin graft at the first operation, the graft being placed between the two flaps.

Partially syndactylised fingers (usually only the little) should be freed as early as possible and this procedure should be combined with thumb release.

Division of index, middle, and ring fingers when interdigital bony fusion is present is often unrewarding and results are disappointing because interphalangeal joint function is poor. Nevertheless the index finger should be released early and allowed to grow normally. On cessation of longitudinal growth the stiff joints can be fused in a position of function to allow thumb-index opposition.

If it is thought desirable to separate all four fingers and thumb, this procedure can be reduced to two stages by separating the thumb and the middle-ring fusion at the first stage. About 1 year later the index-middle and ring-little fusions can be separated.

Flatt (Hoover et al. 1970) has proposed that a three finger hand be produced by amputating the middle finger and thereby obtaining sufficient good sensitive skin to cover the index and ring fingers.

Only three of our cases of Apert syndrome have undergone hand surgery in Adelaide, carried out by our colleague Mr. DN Robinson. This experience is too small to allow us to formulate a confident plan of management; Flatt's strategy appears to have functional advantages (Fig. 16.37), but older patients may nevertheless prefer to have four fingers.

Feet

There is usually complete fusion of all five toes but this does not appear to affect foot function. There is seldom any indication to interfere surgically with this condition, although occasionally great difficulty may be found in providing acceptable footwear for these children. Rarely it may be necessary to narrow the forefoot by removing the fifth or fourth and fifth digits, taking care to produce a

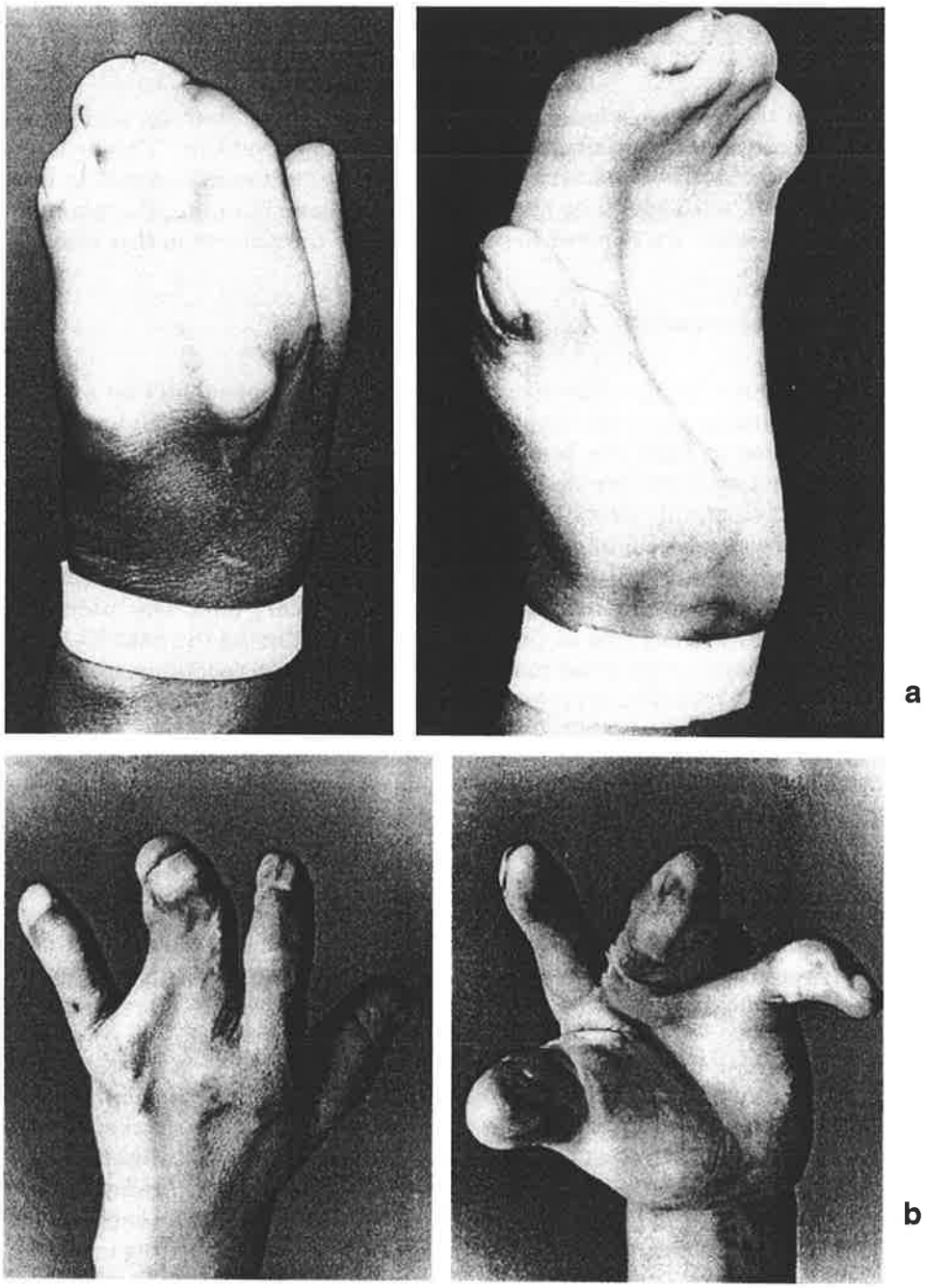


FIG. 16.37 a,b. *Hand reconstruction in Apert syndrome.* **a.** *Before operation: aged 15.* **b.** *After operation: a three-finger hand has been achieved by separating the index and little fingers. The patient can oppose his thumb and index finger quite effectively. If the patient wishes, a fourth finger can still be fashioned.*

dorsal scar.

In Apert syndrome, the disadvantages far outweigh the benefits of individual toe separation. In the less severe forms of acrocephalalosyndactyly, surgery may have something to offer; in Pfeiffer syndrome, for example, a wedge section osteotomy may make the grotesque medial deviation of the hallux less unsightly.

Postoperative Care

After operation the surgeon's immediate concern is to verify that the patient is awake, able to see, and in satisfactory cardiorespiratory condition. This is done as soon as the effects of anaesthesia have worn off. The patient then goes to the intensive care unit, with which he has already been made familiar. The parents or other close relatives are allowed to see and talk to the patient in this unit as soon as possible.

Neurological Observations

Sight is tested immediately after operation and retested repeatedly by asking the patient to count fingers with each eye and to read or identify small toys. The pupillary reactions to light are tested; there is usually little value in testing ocular movements as these are disturbed by the orbital dissections. Conscious state is assessed chiefly in terms of motor response to commands; concurrently the muscular power in all four limbs is checked. The findings are noted on a large chart embodying the well-known Glasgow coma scale (Teasdale and Jennett 1974) and retested serially by the staff of the intensive care unit. The intervals between serial tests are reduced as soon as possible, but during the first 12–24 h it is usually necessary to verify the conscious state, pupillary reactions, and limb movements every 30 min. Vision is checked several times during the same period.

Such serial observations are part of the routine work of any neurosurgical unit and they sound simple and easy. But in practice the nature of major craniofacial surgery makes neurological assessment extremely difficult. The patients are often young, they are likely to be exhausted by long operations and in pain from facial dissections and from bone-harvesting operations on the chest and hip–all much more painful than most neurosurgical operations. The eyes may soon be obscured by orbital oedema. Tracheostomy and intermaxillary fixation may reduce the patient's ability to communicate, he may only be able to mime what he sees, and even his gestures may be burdened by intravascular lines.

Some help comes from the new techniques of physiological monitoring. The light responses in the visual cortices are now being routinely checked using computer-averaged recording of occipital potentials, and this investigation can be done through closed eyelids. Intracranial pressure can be monitored either in the extradural or subdural spaces; we have not so far used this after surgery for craniosynostosis, but it has obvious utility in cases where brain swelling is severe or where conscious state is slow to recover. But whatever monitoring procedures may be used there is no substitute for alertness and the insight that comes from knowledge of the potential neurological dangers. It is thanks especially to good clinical observations by colleagues in the nursing staff that our two postoperative extradural haemorrhages were diagnosed.

Airway Management

This is the forte of the staff of the intensive care unit. Nasal intubation, tracheostomy, and all forms of airway stenosis require special attention. In older patients who have had post-operative nasal intubation, it should be possible to remove the tube quite early, when the patient is co-operative and can cough and swallow. The management is more difficult when facial advancement has necessitated tracheostomy. A cuffed tracheostomy tube is used initially; when the patient is conscious and can swallow his own saliva the cuffed tube is changed and a fenestrated tube is inserted for a further 24 h. It can then be removed; unless there is much pharyngeal swelling or a depressed cough reflex, tracheostomy is usually discontinued 3 or 4 days after operation.

The lungs also need attention. Very rarely a pneumothorax may result from pleural damage during rib resection; one of our patients needed underwater

seal drainage because of this. A more common sequel of rib resection is chest pain with consequent difficulty in coughing; pain relief and physiotherapy are needed to prevent secondary pneumonia.

Fluid Replacement

Transcranial operations entail retraction of the frontal lobes and therefore a risk of cerebral oedema. It is important to avoid fluid overload. As a rule we give half the calculated fluid requirements during the first 48 h, and thereafter progressively increase hydration, being guided by urine output, serum electrolytes, and central venous pressure. Blood loss is however replaced when it occurs.

Nutrition

This is maintained by a nasogastric tube, feeding being started 24–48 h after operation. At first the patient is given small quantities of buffered dextrose in water. When it is clear that this is being absorbed, a full diet is given by the tube, which remains in place until the patient can eat. This may take a week or more, especially when the jaws are wired together. The staff help the patient and his family to experiment with different foods, consistencies of food, and methods of feeding. Some will do best with a straw, others with a long spouted cup or spoon, or even a syringe.

Pain Relief

This is essential, but must not interfere with neurological monitoring. A slow intravenous infusion of pethidine (0.2%) solution) gives good analgesia and is safe under close observation; an initial dose of 0.25 mg/kg is given, and repeated when necessary. Usually this is not needed for more than 3 days.

Antibiotics

The chosen antibiotics (see p. 258) are given at the beginning of the operation and continued for 5 days, unless some special problem demands a change in therapy or a longer course. A postoperative cerebrospinal fluid leak poses a choice of dangers. Meningitis is an obvious risk, but prolonged administration of broad-spectrum antibiotics may lead to opportunistic infections or pseudomembranous colitis. Unless there is clinical evidence of infection we give Septrim (trimethoprim-sulphamethoxazole) until the leakage ceases.

Anticonvulsants

We do not routinely give anticonvulsants. Converse et al. (1975) mention postoperative seizures as an occasional complication of craniofacial surgery, but it appears that patients with craniosynostosis are not especially liable to epilepsy (see p. 67), and a properly conducted transcranial operation should not greatly increase the risk. Of course those patients with a history of previous epilepsy should stay on anticonvulsant medication. Such patients should undergo particularly careful haematological assessment before surgery as several of these drugs (notably phenytoin and sodium valproate) may cause blood dyscrasias.

Wound Care

The scalp wound is covered with a simple strip of dressing. Suction drains are rarely used (see p. 260) except in the hip wound after harvesting the bone from the iliac crest. Scalp sutures are removed after 10–14 days. Special attention is given to the cranial pins used for external maxillary fixation: the entry wounds are cleaned daily and wrapped in gauze soaked in Betadine. Mouth toilet is also important when there is intermaxillary fixation and especially when there are oral wounds. A weak solution of hydrogen peroxide is used, at first by the staff

and then by the patient and/or parents when they have learned how to maintain oral hygiene.

Management of Intermaxillary Fixation

Fixation must be maintained until the osteotomised facial skeleton is stable. After a LeFort III osteotomy done for Crouzon syndrome in an adult, it may be necessary to maintain intermaxillary fixation for 12 weeks; in an 11-year-old child, 6 weeks may be enough. With good external craniomaxillary fixation, the intermaxillary fixation may be released several weeks earlier, before the final removal, allowing first mandibular exercises and then normal eating. Craniomaxillary and intermaxillary fixation both cause a good deal of distress and social embarrassment. Patients from other parts of the world stay in hospital until fixation is removed; local patients may go home but need weekly examination. Removal of the cranial pins is easy and painless but removal of the cap splints occasionally needs general anaesthesia.

Duration of Stay in Hospital

Patients who have undergone fronto-orbital advancement or orbital translocation usually make quick recoveries and spend only 10-14 days in hospital. When a midfacial advancement is done, and particularly when a transcranial exposure is needed, the time in the intensive care unit is longer, as is the total time in hospital. These patients are usually unwell for several weeks after operation.

Craniosynostosis

Donald A. Simpson, David John David

Development of the Cranium

In embryonic life, the developing brain is surrounded by a membrane of condensed mesenchyme. In the second month after conception, skeletal tissues form in this mesenchymal capsule. Cartilage is laid down as the future skull base; after the sixth week this process of chondrification is supplemented by endochondral bone formation, and at birth most of the skull base is ossified. The chief bones of the skull base are thus cartilaginous bones, growing by ossification in synchondroses. The synchondroses that separate the exoccipital and supraoccipital bones normally fuse soon after birth, but the important sphenooccipital synchondrosis remains open until adolescence (12–16 years).

The remainder of the cerebral capsule ossifies directly, by deposition of bone in the mesenchymal membrane. After the sixth week of intrauterine life, renters of membranous ossification appear over the cerebral hemispheres, and these are the primordia of the parietal, frontal, squamous temporal, and squamous occipital bones. Osteoid is formed first and mineralised into spicules of bone, radiating outward in the membrane, which differentiates into an outer layer, the pericranium, and an inner layer, the dura mater. Both are osteogenic. By the fifth month, the flat bones of the skull vault are well formed, and they are separated by zones of unossified membrane, the fontanelles and sutures.

The fetal brain increases in volume very rapidly (Fig. 20-1), and this volumetric expansion continues after birth; the cranial capsule must grow to accommodate the brain, and much of this capsular growth takes place at the margins of the bones, in the sutures. It is now generally agreed that the cranial sutures are not primary growth centers. Sutural bone is laid down as a secondary response to capsular tensions, and these tensions represent the sum of the dynamic effects of the expanding brain and the independent growth of the skull base, transmitted through the dura mater and its septa. The developing eyes and the facial viscera also modify cranial growth. Sutural bone deposition is not the only process involved in the growth of the skull vault: surface deposition of bone by the pericranium and removal of bone internally are also important, especially in later childhood. Nevertheless, the sutures are of crucial importance as growth zones in infancy, and premature sutural fusion, or craniosynostosis, is often associated with striking cranial deformities. There is now convincing experimental proof that sutural growth failure alone can deform the growing skull (Persing et al. 1981; Alberius and Selvik 1984).

Occurrence

Premature sutural fusion may be secondary to an arrest of cerebral growth, as in microcephaly. Craniosynostosis is also seen as an effect of metabolic disease, such as rickets or hyperthyroidism (David, Poswillo, and Simpson 1982); however, for the neonatologist and the pediatrician, craniosynostosis is most often seen as a primary congenital disorder of skull growth, associated with various types of skull deformity, and occasionally with serious neurological impairment.

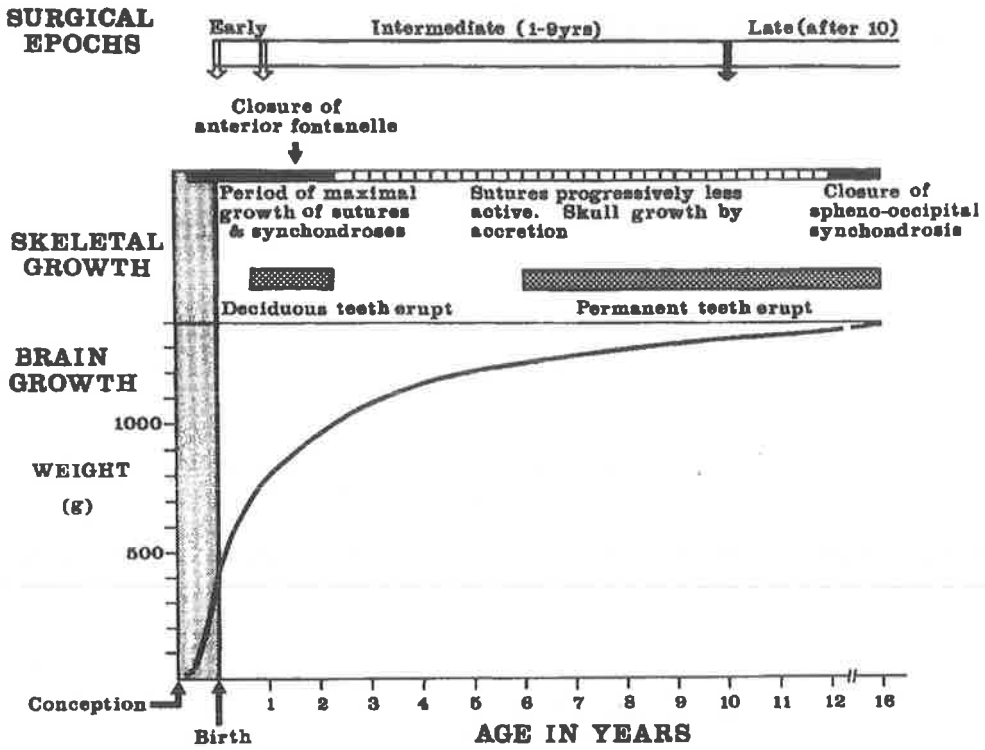


FIG. 20-1. *The graph shows the increase in brain weight from the tenth gestational week onward (data from Lemire et al. 1975); the figure relates this schematically to the stages of cranial and dental growth, and to the timing of operative treatment.*

Craniosynostosis is a common condition; clinically detectable craniosynostosis has been reported in up to 1 in 1900 births (Myrianthopoulos 1977). It is clearly not a simple disease entity. In perhaps 10–20 percent of cases, there is evidence of Mendelian inheritance, and there may be associated deformities, such as osseous syndactyly (Apert syndrome) or maxillary hypoplasia (Crouzon syndrome); many more or less well-defined craniosynostosis syndromes have been described (David, Poswillo, and Simpson 1982). In most cases, however, there is nothing to suggest genetic causation: the craniosynostosis is seen as an isolated congenital anomaly, without a family history of similar deformities, and in these cases the cause or causes are unknown. Some authors postulate intrauterine compression (Graham, De Saxe, and Smith 1979; Graham, Badura, and Smith 1980).

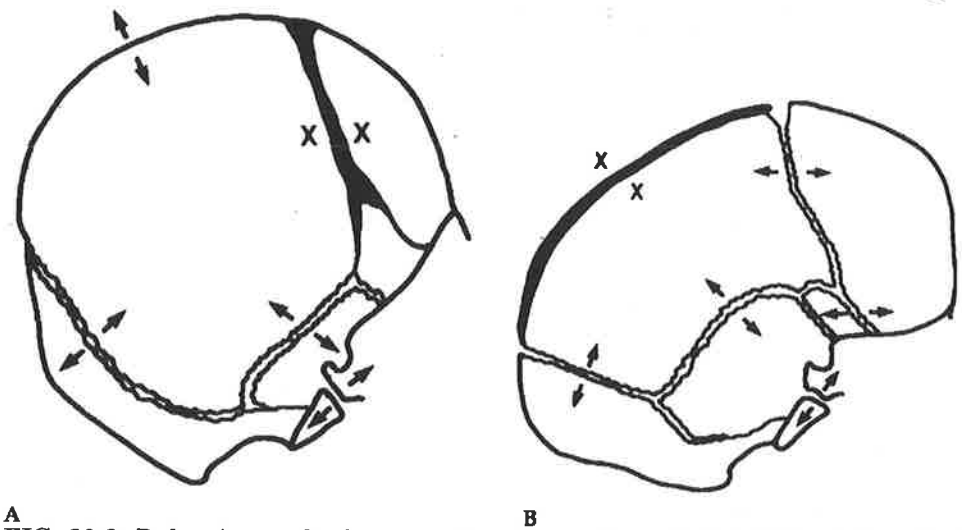
At present it seems wise to regard the craniosynostoses as primary idiopathic disorders of skeletal growth, affecting different parts of the cerebral capsule. In this view, the premature sutural fusion is an important and treatable element in the growth disorder, which, however, also affects the dura mater, the skull base, and sometimes the facial skeleton.

Symptomatology

The clinical significance of the craniosynostosis varies greatly, but some general features can be identificd.

Skull Deformity

The skull vault is usually deformed. The deformity is anatomically related to the sutural fusion: delay of growth across one suture reduces the dimension of the skull in the plane perpendicular to the fused suture, while compensatory overgrowth gives increased dimensions in planes perpendicular to the unaffected sutures (Fig. 20–2). When craniosynostosis affects multiple suture systems, the



A B

FIG. 20-2. *Deformity results from cessation of growth across fused suture lines (X—X) and compensatory overgrowth across normal suture lines* (arrows). **A.** *Turricephaly: fusion of coronal sutures and their basal extension.* **B.** *Scaphocephaly: fusion of sagittal suture.*

geometry of the deformity is more complex and probably relates to the time sequence of the underlying growth failure. The abnormal head shape can be categorised by anthropometric measurements; in practice, the most useful is the horizontal cephalic index: (maximum breadth/maximum length) × 100.

The fused sutures may be palpable as bony ridges, with loss of normal mobility. There is often premature fontanelle closure. The sutural fusion can be shown by x-ray (Fig. 20-3) as a line of sclerosis or as complete absence of a normally patent suture. In planning surgical treatment, it is necessary to know which sutures are involved, and some clinicians use this as the basis for classification (Matson 1969). However the correlation of sutural fusion and deformity is not always in accordance with expectations, and many authors (Montaut and Stricker 1977; Marchac and Renier 1982) prefer to classify the craniosynostoses on their external appearances, using morphological terminology. Our descriptive system is given in Table 20–1.

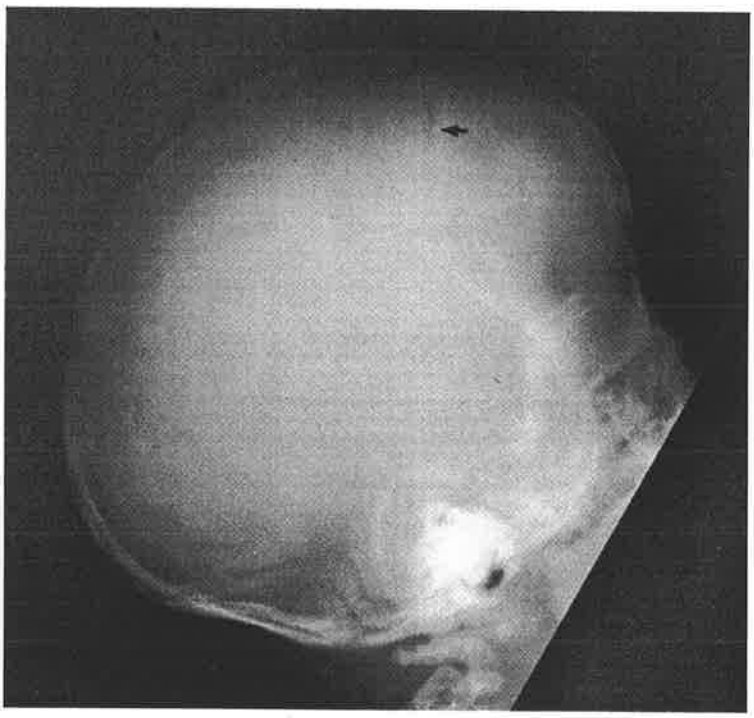


FIG. 20-3. *Turricephaly. The coronal suture is fused in the region of the sphenoid ridge; medially it is still patent though hyperostosed* (arrow).

The skull deformities are usually evident at birth or soon after; some forms become more striking in early childhood. In this period, they may cause much emotional distress (Barritt, Brooksbank, and Simpson 1981). Spontaneous improvement is unusual, though hair growth and increased occipital muscle bulk make some deformities less noticeable.

Raised Intracranial Pressure

In severe forms of craniosynostosis, the cranial volume becomes too small for the growing brain (craniostenosis), and intracranial pressure may rise. This is associated with high intracranial venous pressure and sometimes with obstructive hydrocephalus. There is debate over the cause of the hydrocephalus. Constriction of subarachnoid cisterns has seemed to us the likeliest explanation, but Sainte-Rose et al (1984) suggest that raised venous pressure may be responsible when associated with an open fontanelle. Both craniosynostosis and hydrocephalus are especially often seen in Crouzon syndrome, but also occur in other types of craniosynostosis. Raised intracranial pressure is rare when only a single sutural system (e.g., sagittal, metopic, or unicoronal) is affected, though examples have been reported (Renier et al. 1982).

TABLE 20–1

Skull Deformities Associated with Premature Sutural Fusion

Deformity	Horizontal Cephalic Index	Chief Sutures Affected*	Frequency	
			Simple: Isolated Deformity	Complex: Part of a Craniofacial Syndrome
Scaphocephaly	55–70	Sagittal	45%–50%	Often in Crouzon syndrome (30%–40%)
Trigonocephaly	60–80	Metopic	5%	Seen in Carpenter syndrome
Frontal Plagiocephaly	Varies	Unilateral coronal and sphenofrontal	20%–25%	Often in Saethre-Chotzen syndrome
Occipital Plagiocephaly	Varies	Unilateral lambdoid?	?/10%	Very rare
Turricephaly	80–110	Bilateral coronal and sphenoid	5%–10%	Usually in Apert 100%) Crouzon (50%–60%) syndromes
Oxycephaly	Varies	Multiple	5%–10%	
Triphyllocephaly (Kleeblattschädel)	90–100	Multiple	Rare	Crouzon syndrome, usually with associated hydrocephalus
Pachycephaly	80–100	Bilateral lambdoid	Rare	Rare

Incidences of diagnostic categories vary according to case selection. Our material (1956–1983) consisted of 221 cases and included a high proportion (28%) of complex craniofacial syndromes referred for specialist treatment—the percentages given for simple (nonsyndromal) craniosynostosis are therefore estimated from our experience before the inception of the present South Australian Craniofacial Unit.

*In the craniosynostosis syndromes, multiple fusion is almost always seen, but the skull shape relates to the sequence of fusion and the chief sutural system affected is the first to fuse.

Raised intracranial pressure usually becomes evident within the first five years of life, though later presentation is occasionally seen. Papilledema is the most important clinical sign and should be looked for in all cases; failure to do so may result in blindness from optic atrophy. In established cases, plain skull radiographs usually show increased convolutional markings (Fig. 20–4). Hydrocephalus is detected by ultrasonography in infants (Fig. 20-5) or CT scanning (Fig. 20-6), and these investigations also serve to exclude coincidental cerebral malformations such as holoprosencephaly. They should be used routinely.

The finding of raised intracranial pressure needs careful evaluation. Papilledema unquestionably demands treatment to save vision. Increased convolutional markings are also, in our opinion, a strong indication for surgical treatment, if detected during the period of rapid brain growth (0–2 years). In doubtful cases, intracranial pressure manometry by a subdural bolt is useful, and this may unexpectedly disclose high pressure in an otherwise well child. It is not easy to know in an individual case what significance should be given to this finding. There is an understandable fear that chronic intracranial hypertension may impair intelligence. At present, the evidence that this happens is questionable: Many patients with severe untreated craniostenosis retain normal intelligence, whereas some patients with craniosynostosis show intellectual retardation in the absence of raised pressure (David, Poswillo, and Simpson 1982). But uncertainty and fear of irreversible cerebral changes justify operation as a preventive measure in infants with multiple sutural involvement.

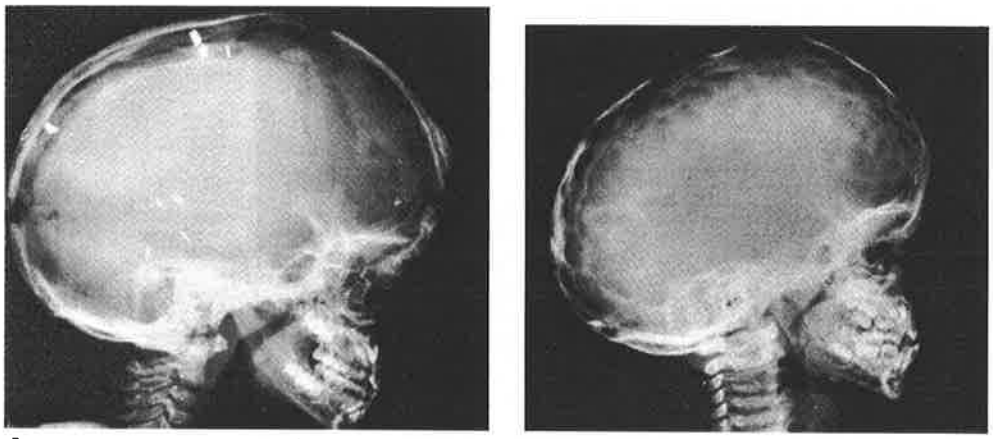


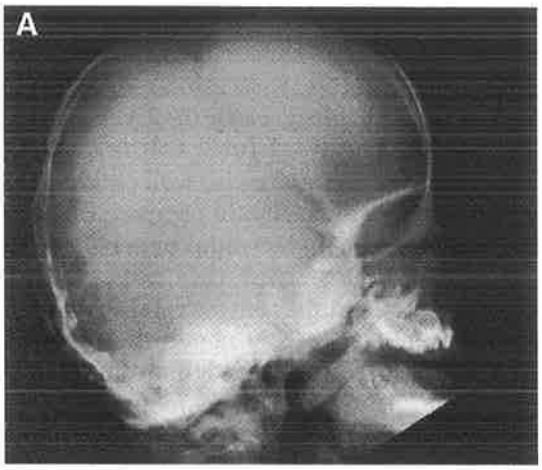
FIG. 20-4. *Scaphocephalic skull deformity (Crouzon syndrome) with multiple sutural fusion and increased convolutional marking:* **A.** *before operation;* **B.** *one year after frontoorbital advancement and decompressive lateral craniotomies; convolutional markings have disappeared, and new sutures are visible.*

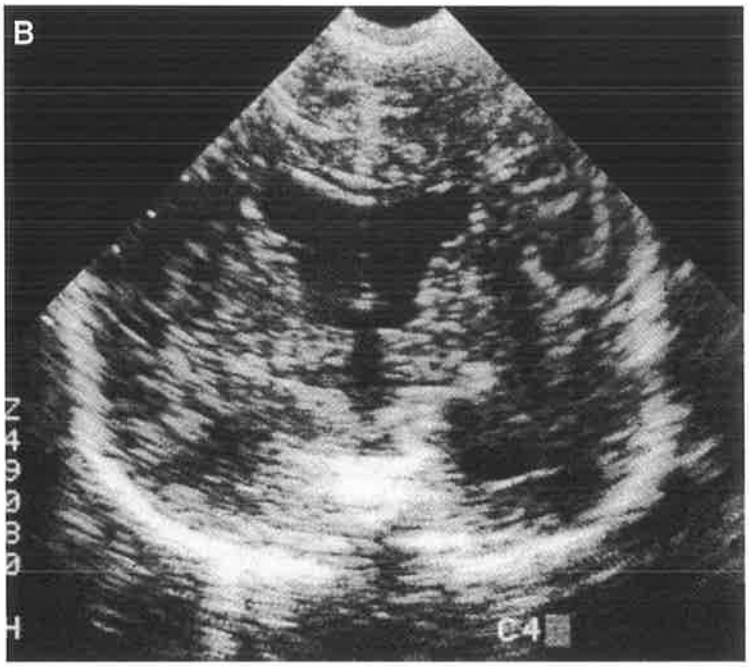
Facial Abnormalities

Premature fusion of the chief vault sutures is usually associated with distortion of the cranial base, and this in turn may modify the growth of the facial skeleton. This is very striking in cases of untreated frontal plagiocephaly, where growth delay in the coronal and sphenofrontal sutures is associated with a distorted face (Fig. 20–7). In many forms of syndromal craniosynostosis, notably Crouzon syndrome, there may be severe maxillary hypoplasia, causing proptosis (*orbitostenosis*) and nasal obstruction (*faciostenosis*); this is associated with premature fusion of the maxillary sutures (Kreiborg 1981), and appears to be a primary failure in facial skeletal growth.

Extracranial Abnormalities

The commoner nonsyndromal types of craniosynostosis are not as a rule associated with other congenital defects. The syndromal craniosynostoses are discussed in Chapter 21 (Classification of Craniofacial Dysmorphism); they are characteristically associated with important skeletal and visceral defects. In Apert syndrome, severe syndactyly is an obligatory accompaniment; in the less severe forms of acrocephalosyndactyly, notably the Saethre–Chotzen syndrome, syndactyly is variable and often absent. Serious cardiac anomalies are often found in Carpenter syndrome. The clinician who sees a new case of craniosynostosis should keep these possible associations in mind; a full clinical examination is essential, as is also a genetic history.





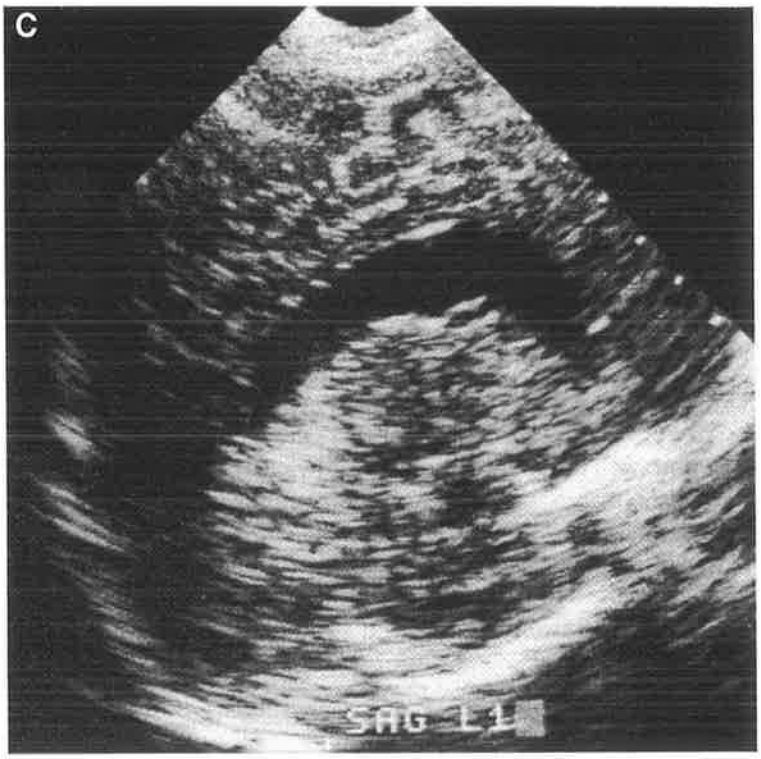


FIG. 20-5. *Radiographic studies in an infant with bilateral lambdoid craniosynostosis and probable Crouzon syndrome.* **A.** *Lateral skull radiograph showing absence of lambdoid sutures.* **B.** *Coronal sonogram, showing mild hydrocephalus.* **C.** *Parasagittal sonogram, showing distortion of the body of the lateral ventricle.*

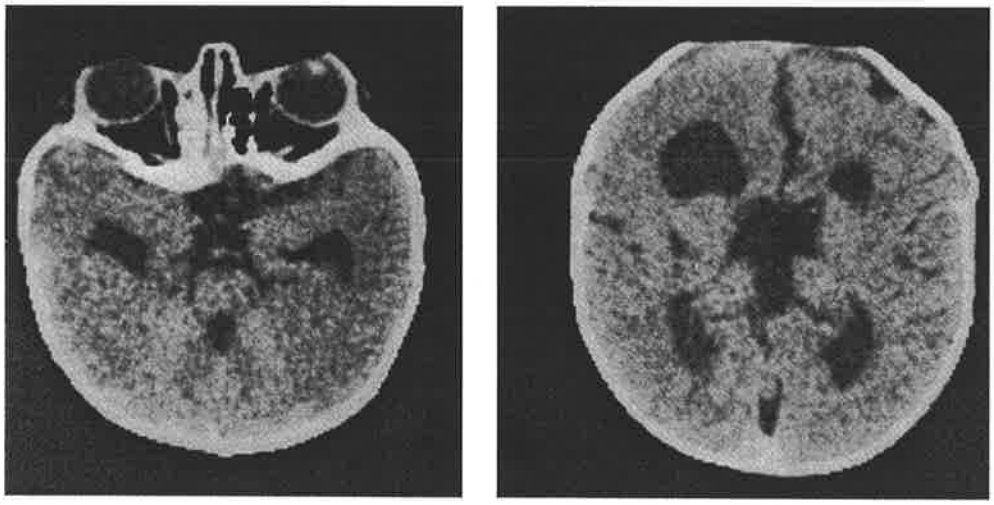


FIG. 20–6. *Computerized tomography in infant with Apert syndrome. There is mild dilatation of the lateral ventricles and deformity of the grossly enlarged third ventricle, due to associated agenesis of the corpus callosum.*

Principles of Treatment

When craniosynostosis affects only one suture, and the associated cranial deformity is not severe, then expectant treatment may be appropriate. This entails periodic review because occasionally the growth disorder is more severe than is evident from the neonatal skull radiographs; this is especially so in Crouzon syndrome (David, Poswillo, and Simpson 1982). Operative treatment may, however, be indicated to correct unacceptably severe cranial or facial deformities; the decision on what constitutes an unacceptable deformity is not easy and must take into consideration the feelings of the parents, the society in which the family lives, and the natural history of the particular type of craniosynostosis. Operative treatment is unquestionably indicated when there is evidence of raised intracranial pressure, or a likelihood that this will develop.

In planning operative treatment, one must take into account the phases of craniocerebral and facial growth.Three surgical epochs can be identified (Fig.20–1).

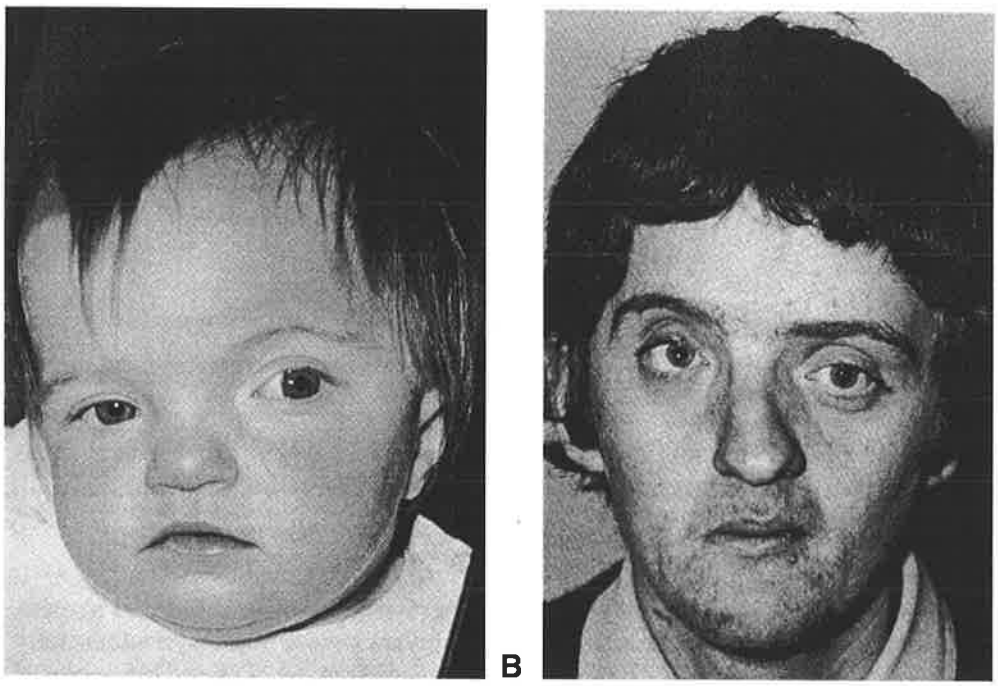


FIG. 20–7. *Untreated frontal plagiocephaly with facial distortion in* **A.** *an infant;* **B.** *a young man.*

Early Period: Up to 12 Months

In this period, the velocity of cerebral growth is greatest, and craniosynostosis is most detrimental. The chief principle of operation is wide excision of the fused suture(s): The growing brain can then expand, stretching the dura mater at the site of the new artificial suture and so reshaping the brain. The validity of this concept is well attested (Matson 1969; Shillito 1973). However, failure or incomplete success has often been experienced, and there are several reasons for this. First the artificial suture may be fused by regrowth of bone before the reshaping process is complete. Refusion may be delayed by wrapping the bone edges in plastic film (Fig. 20–8). In most cases, this procedure gives sufficient time for remolding to take place. More radical ways of preventing reossification by the dura, such as chemical coagulation or excision of the outer dural layer, have been proposed; we prefer to accept the occasional need to reoperate when there is recurrent craniostenosis or deformity. An inadequate sutural excision is a second cause of disappointment. It was not at first realized that coronal synostosis represents failure of a sutural system that includes the sutures of the skull base, especially the sphenofrontal and perhaps the frontoethmoidal sutures. It has also become evident that the dynamic effect of cerebral growth may be insufficient, especially when the growth disorder resulting in craniosynostosis is severe. More effective surgical procedures have now been devised (Hoffman and Mohr 1976; Marchac and Renier 1982) which extend the craniectomy into the skull base, and also remodel the deformity as a primary procedure, allowing the brain to reexpand without any osseous constraint.

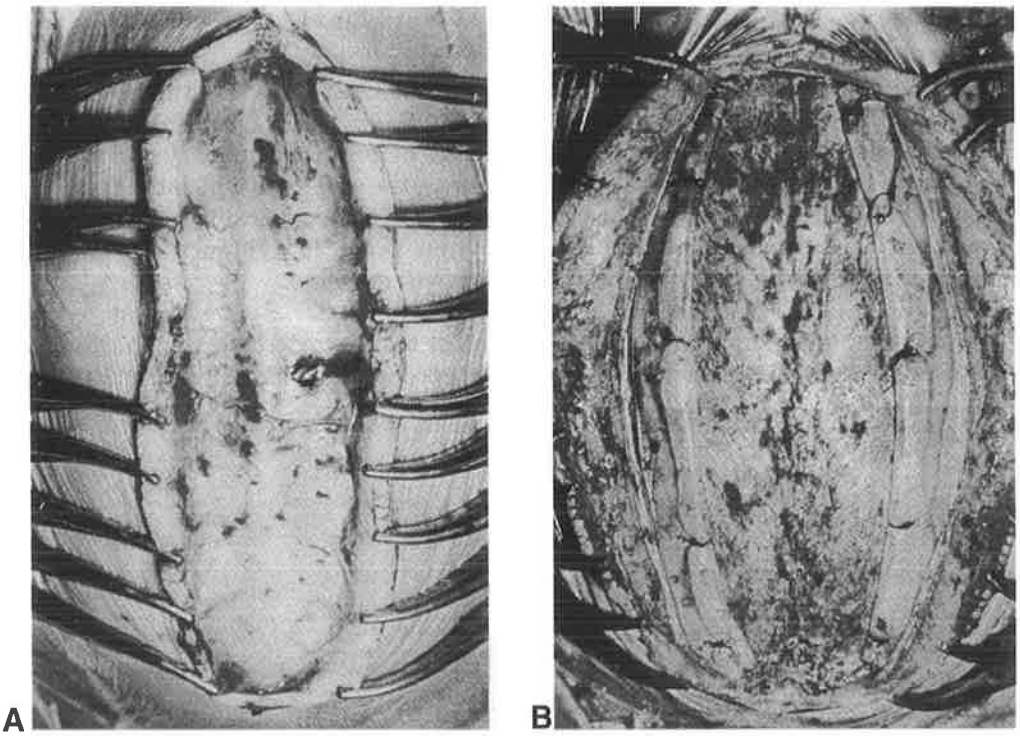


FIG. 20–8. *Operative treatment of scaphocephaly* **A.** *exposure of the fused sagittal suture;* **B.** *wide median craniectomy and insertion of silastic strips.*

These operations should be done early, and preferably within the first three months. After the first year, they are ineffective in correcting deformity.

Intermediate Period: 1–9 Years

In the second year, the velocity of cerebral growth slows, and sutural growth becomes progressively less significant. Nevertheless severe craniosynostosis may still lead to craniostenosis, with papilledema and other signs of raised intracranial pressure. Simple sutural excision is ineffective, and extensive bilateral decompressions may be needed; such operations can be planned to correct

associated deformities of the frontal region and orbits (Figs. 20–4A and 20–4B). Deformities of the middle and lower face sometimes cause distress in this epoch, but if possible, operative correction is deferred. The facial skeleton is still growing actively: maxillary deformities corrected too early may relapse (Hogeman and Willmar 1974), and growth of normal skeletal components, notably the mandible, may lead to facial skeleton imbalance and dental malocclusion.

Late Period: From the Tenth Year

Cerebral complications are now unlikely, but facial deformities may need correction. In Crouzon syndrome and other craniofacial syndromes, proptosis and maxillary hypoplasia are especially likely to need operative treatment (Tessier 1971). The operative procedures (orbital relocation, maxillary osteotomy, etc.) are very demanding, but older children can appreciate the need to correct deformity, and from the psychological viewpoint, this epoch is a favorable time. The complexity of these craniofacial reconstructions, along with the considerable operative risks, has justified the formation of specialized multidisciplinary craniofacial units.

Hydrocephalus

The hydrocephalus often associated with severe craniosynostosis may arrest spontaneously. It is therefore usually appropriate to treat the craniosynostosis first, and to perform an extracranial shunt only if there is evidence of progressive hydrocephalus after the cranial operation.

Scaphocephaly

This is the commonest form of craniosynostosis. Boys are much more often affected; this high (80%) male predominance is unexplained. The scaphocephalic head is long and narrow, with frontal bossing and a prominent occiput (Figs. 20–2B, 20–9). The sagittal suture fuses prematurely, and in babies can be felt as a midline bony keel; the anterior fontanelle is usually small. In the great majority of cases, the craniosynostosis affects only the sagittal suture; occasionally there is also involvement of the coronal or metopic sutures. These additional synostoses may not at first be evident.

Scaphocephaly is often associated with birth difficulties, perhaps because the head is large and may not mold readily. The deformity can usually be diagnosed in the neonatal period, though the synostosis may still be incomplete, or even undetectable by x-ray. As a rule there are no associated extracranial abnormalities and no familial history; however, some patients with syndromal craniosynostosis, especially Crouzon and Carpenter syndromes (David, Poswillo, and Simpson 1982) may have scaphocephalic head shapes, and these conditions should be kept in mind. Craniostenosis is rarely seen except where there is associated fusion of the coronal sutures. Most cases, whether treated or not, show no mental or neurological abnormalities. In a minority there is developmental delay, sometimes severe, but we have not been able to relate this causally to the cranial deformity.

Operative treatment may be indicated on aesthetic grounds. A midline scalp incision exposes the fused sagittal suture, which is widely excised. Care is taken not to injure the sagittal sinus. The resulting craniectomy is 4–5 cm broad and extends well across both lambdoid and coronal sutures. Silastic strips are inserted on each side (Fig. 20–10A). This simple procedure has in our experience been satisfactory. There is objective improvement in the horizontal cephalic index over the next six months, and parents have usually been happy with the result, except in those few cases where there is developmental delay; improvement in this should not be predicted. The operation is done no later than the sixth month

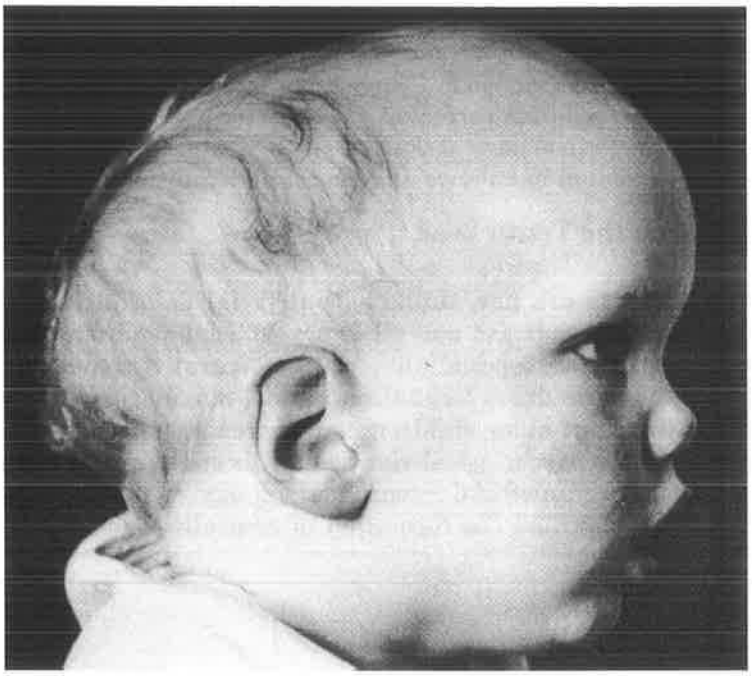


FIG. 20–9. *Scaphocephaly associated with premature fusion of sagittal suture.*

of life, and preferably sooner. In neglected cases, if operation is really needed, more elaborate reconstructions of the cranial vault have been advised (Marchac and Renier 1982; refer to Chapter 22, Management of Craniofacial Dysmorphism).

Trigonocephaly

In this less common deformity, the frontal region, seen from the vertex, is wedge-shaped, with a median ridge representing the fused metopic suture (Fig. 20-11). The forehead is narrow. The eyes often have a Mongolian slant; they are set close together, and there is true hypotelorism. Boys are more often affected.

A somewhat similar cranial deformity may be secondary to dysplasia of the frontal lobes, and this important association should be excluded by ultrasound or CT scanning. In the absence of such a dysplasia, the prognosis is quite good, though occasional cases of trigonocephaly do show developmental retardation. We have never seen raised intracranial pressure in primary trigonocephaly.

Operative treatment may be indicated on aesthetic grounds. A coronal scalp incision is used. Simple excision of the fused metopic suture has not been satisfactory. Better results are obtained if the sutural excision is supplemented by bilateral frontal craniotomies; the free frontal bones are bent, rotated, and fixed to the orbital margins to broaden the forehead (Fig. 20–10B). We also insert silastic film along the margins of the metopic craniectomy, though the need for this is questionable. The procedure should be done at the latest by the sixth month, and preferably earlier. In neglected cases, a more extensive frontal remodeling may be undertaken (Montaut and Stricker 1977), though experience of untreated trigonocephaly has shown that the facial appearance tends to improve with age.

Frontal Plagiocephaly

This deformity is second in frequency to scaphocephaly. Girls are somewhat more often affected than boys. There is premature fusion of one coronal suture and usually also of the ipsilateral basal sutures, especially the sphenofrontal

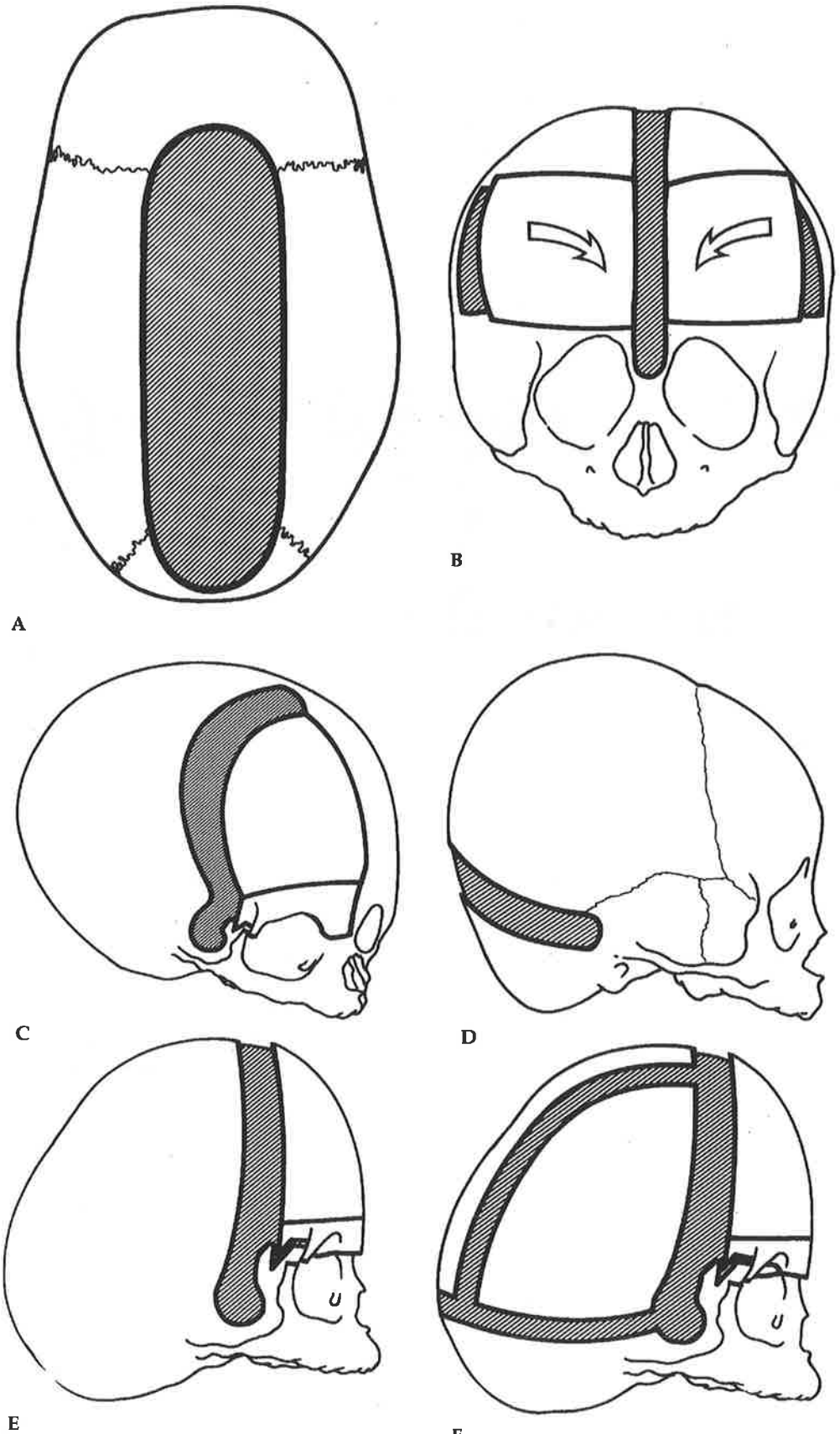


FIG. 20–10. *Operative techniques for different forms of craniosynostosis.* **A.** *Scaphocephaly; median sagittal synostectomy.* **B.** *Trigonocephaly; metopic synostectomy, mobilization, and rotation of frontal bones.* **C.** *Frontal plagiocephaly; coronal and sphenofrontal synostectomy and unilateral frontoorbital advancement. The advanced frontoorbital bone is fixed by a tongue of bone set in a slot cut in the lateral orbital wall.* **D.** *Occipital plagiocephaly; lambdoid synostectomy.* **E.***Turricephaly; coronal and sphenofrontal synostectomy and frontoorbital advancement (tongue-in-slot fixation).* **F.** *Oxycephaly; bilateral osteoplastic craniotomies combined with frontoorbital advancement, usually done in two stages. These diagrams show the craniectomies in black; mobilized structures are shown in the final position. Silastic inserts are not shown.*

suture; rarely there is a fusion of other ipsilateral sutures, giving a hemicranial craniosynostosis. In the average case, there is flattening of one frontal region, with elevation and recession of the eyebrow and orbital margin on the same side; the ipsilateral ear is often prominent, and there is a bony bulge in the contralateral parietal vault. Facial asymmetry is often striking, especially in neglected cases (Fig. 20–7). The deformity of the skull base displaces the mandibular condylar fossa, distorting mandibular growth and tilting the dental occlusal plane of the lower jaw. The root of the nose and the vertical plate of the ethmoid tilt toward the affected side. Radiographs show the coronal synostosis, and also the orbital deformity and curvature of the skull base. Ultrasound or CT scanning will often show ventricular asymmetry, but serious cerebral anomalies are rare, and we have not seen clinically evident raised intracranial pressure. In occasional cases, there are signs of localized craniostenosis in the vicinity of the synostosis.

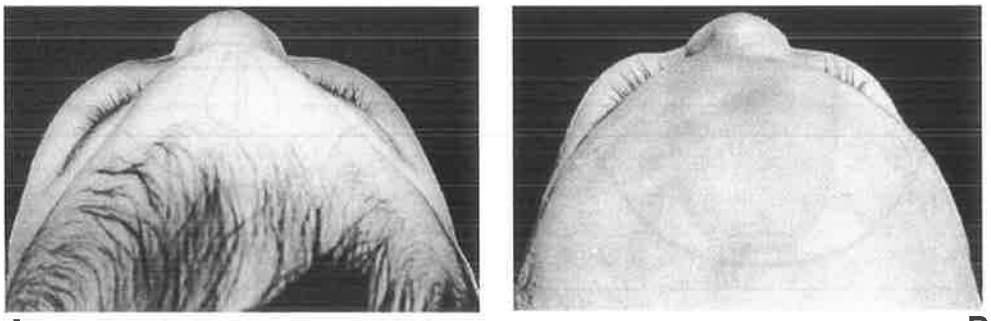


FIG. 20-11. *Trigonocephaly, with premature fusion of metopic suture:* **A.** *before operation;* **B.** *after operation.*

The deformity can usually be recognized in infancy, and may be associated with extracranial anomalies (e.g., minor syndactyly or anal ectopia). The Saethre-Chotzen syndrome (David, Poswillo, and Simpson 1982) may be suspected, especially if there is a family history of birth defects. The untreated condition is often quite disfiguring, and in our opinion the case for operative treatment is strong.

A coronal scalp incision is used. The fused coronal suture is excised from across the midline into the skull base; great care is needed in resecting the saber-shaped sphenoidal ridge, which is swept up on to the cranial convexity. Simple excision of the fused coronal and sphenofrontal sutures is sometimes disappointing, and this procedure is usefully supplemented (Hoffman and Mohr, 1976) by a frontal craniotomy and advancement of the ipsilateral orbital margin. To do this, the supraorbital ridge and the anterior part of the orbital roof are dissected extradurally and through the orbit; they are then mobilized by osteotomies as a single frontoorbital block. This is advanced and secured in the advanced position by two vertical spurs of bone, one cut from the lateral orbital wall below and the other from the inferior margin of the frontoorbital block (Fig. 20–10C). The free frontal bone flap is bent, rotated, and replaced to correct the frontal deformity. Silastic is inserted from across the midline to a point well below the squamosal suture; it is our practice to do this only on the posterior margin of the craniectomy.

This procedure is best done in early infancy. However, quite satisfactory results can be obtained up to three years of age (Hoffman, Hendrick, and Munro 1982). In neglected cases, the deformity may include nasal deviation and an ugly asymmetry of the orbits; these may require major craniofacial correction (Chapter 22).

Occipital Plagiocephaly

This common and puzzling deformity is characterized by unilateral occipital flattening on one side, with prominence of the ipsilateral frontal region; seen

from above, the skull is deformed into a parallelogram, sometimes with prominence of the ipsilateral ear. There is very little facial deformity.

The place of this deformity among the craniosynostoses is uncertain. In some cases, there is radiological evidence of lambdoid sclerosis which may go on to fusion; in such cases, lambdoid craniectomy may achieve a more normal skull contour. However, very similar plagiocephalic skull deformities have been seen as a consequence of postural pressure in infancy, or in association with cervicooccipital abnormalities, spinal tumors, and torticollis. Indeed, occipital plagiocephaly may be found in the absence of any detectable cause, whether in the sutures, the skull base, or the neck. Fortunately, this type of plagiocephaly is rarely associated with cerebral involvement, and tends to be less noticeable in older children. If there is strong parental concern, and if the lambdoid suture is radiologically abnormal, the condition may be treated by excising the entire suture (Fig. 20–10D) with silastic inserts on each side of the resulting craniectomy (Hoffman, Hendrick, and Munro 1982).

Turricephaly

A broad towering head (Fig. 20–2A) may be associated with premature fusion of both coronal sutures; in the skull base, the sphenofrontal and frontoethmoidal sutures are often also involved. As an isolated anomaly, turricephaly is one of the less common forms of craniosynostosis. However, turricephaly is the commonest pattern of skull deformity in Crouzon syndrome (Kreiborg 1981) and is invariably found in Apert syndrome, where, however, there are often additional anomalies in the cartilaginous skull base. It is not unusual to see a delayed onset of sagittal synostosis in older infants in whom, in the neonatal period, there was involvement of the coronal sutures only; this widespread craniosynostosis is characteristic of oxycephaly (see below), and the two deformities are so closely allied that some writers do not distinguish between them. Turricephaly is not the only deformity associated with extreme brachycephaly: premature fusion of both lambdoid sutures is also associated with a very brachycephalic skull, the occipital region being quite flat. This has been termed pachycephaly (Fig. 20–5).

Turricephaly is usually evident at birth, though it may be missed, especially when there is a family tendency to brachycephaly. Absence of the anterior fontanelle may arouse suspicion. There is female preponderance. Associated anomalies are often found, and evidences of genetic causation should be looked for because the condition is often familial, and may be part of a syndrome. When Crouzon syndrome presents in infancy with turricephaly, the diagnosis may be missed, as the maxillary hypoplasia is often at first inconspicuous. The syndactyly of Apert syndrome is of course unmistakable, and in this condition the turricephaly has peculiar features, notably a wide metopic bone defect.

Untreated turricephaly is compatible with normal intelligence (Jensch 1941–2). However, there is a high incidence of craniostenosis, and a significant aesthetic deformity; in our view, therefore, there are strong indications for early operative treatment. This is done through a coronal scalp incision. The fused coronal sutures and the hypertrophied sphenoidal ridges are excised, and subtemporal craniectomies are performed (Fig. 2010E). The frontal bones are elevated. The operation can then be completed by inserting silastic strips along the posterior margin of the new coronal suture, and replacing the frontal bones as onlay grafts in front of the orbital margins: This simple procedure has given excellent results when the involvement of the skull base is not severe. In the past few years, however, we have been impressed by the benefits of the more extensive frontal mobilization advised by Marchac (1978). By extradural and intraorbital dissection, the orbital margins and glabellar area are freed and resected as a single block, advanced up to 2 cm, and wired to spurs cut on each side from the lateral orbital walls, with an interposed bone graft holding it to the

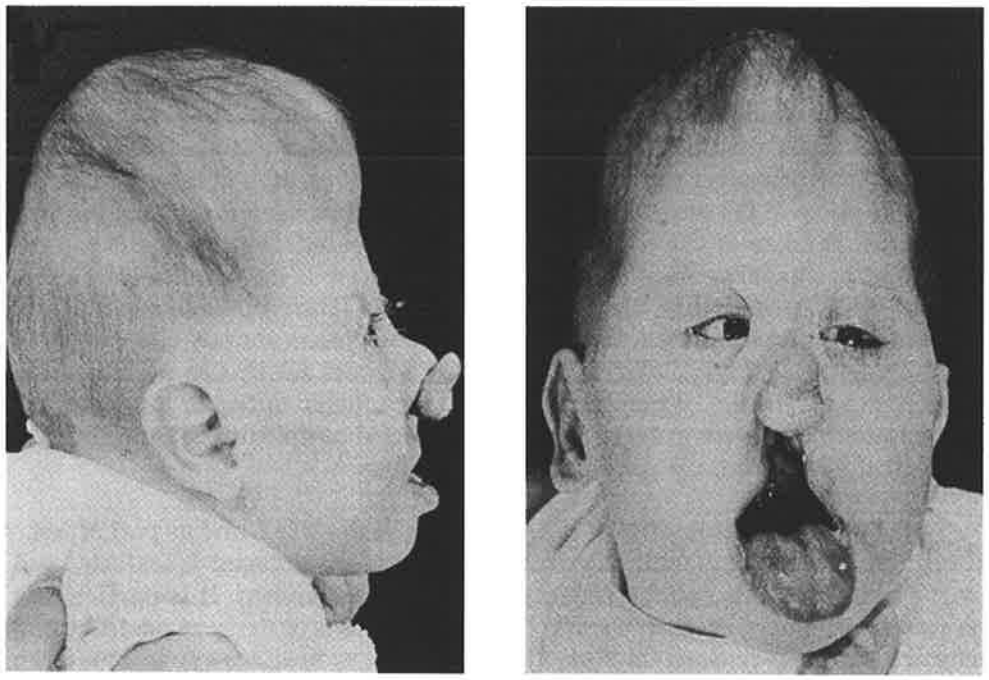
root of the nose. Although best done before three months of age, this procedure has been satisfactory even in childhood.

Oxycephaly

More or less simultaneous fusion of multiple sutural systems can produce diverse deformities. When the outcome is a conical head, with the forehead sloping back to a bony boss at or behind the bregmatic area, then the term oxycephaly (sharp head) seems appropriate. The external appearances may be striking, or relatively normal, and there may or may not be associated facial abnormalities, notably the absence of the frontonasal angle (Marchac and Renier 1982) (Fig. 20–12A). Whatever the skull configuration, premature fusion of multiple sutures is usually an imperative indication for operative treatment, aimed at decompressing the constricted brain and correcting the skull deformity. In most cases, we have effected this by two-stage procedures, excising the prematurely fused sutures bilaterally and advancing or broadening the frontal region (Fig. 20–12B). If such procedures are done in infancy, silastic is inserted and reoperation is often needed; in cases presenting after the first year, silastic is omitted. It is for very extensive craniosynostosis diagnosed in infancy that Hanson et al (1977) advised total or subtotal excision of the calvarial vault; reported results of this procedure have not always been happy, and we think it more logical to use the older strategy of remolding by selective craniectomy and frontal reconstruction (Fig. 20–10F).

Triphyllocephaly (Kleeblattschädel)

This dramatic deformity is also a manifestation of very extensive craniosynostosis developing in utero. We have seen four cases, three with obvious Crouzon syndrome. As the name indicates, the infant's skull, from the front, looks like a cloverleaf. There are large temporal bulges and a median bregmatic bulge, separated by constriction bands of bone and dura, running more or less along the line of the Sylvian fissure (Fig. 20–13A). There is usually a severe communicating hydrocephalus. Triphyllocephaly requires early operative treatment. We have employed staged frontal and occipital craniectomies, with frontoorbital advancement, and the remodeling has been dramatic (Fig. 20–13B). Two less severe cases have done well; unhappily two other cases later died of respiratory difficulties related to associated severe faciostenosis. When there is associated hydrocephalus, one may defer insertion of a shunt until the completion of the planned cranial surgery, in the hope that the hydrocephalus will arrest spontaneously. However, shunting should not be delayed too long.



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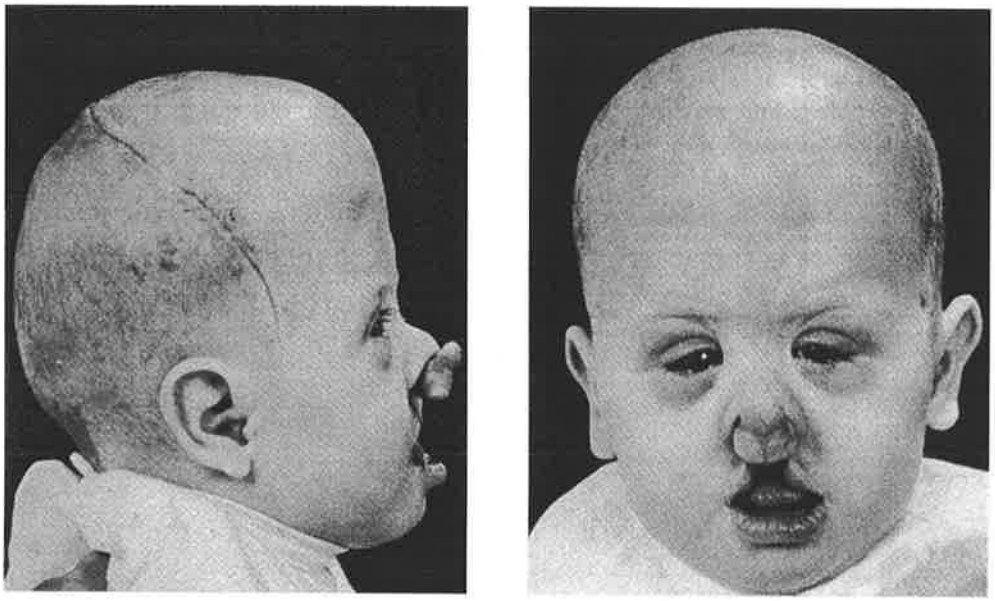

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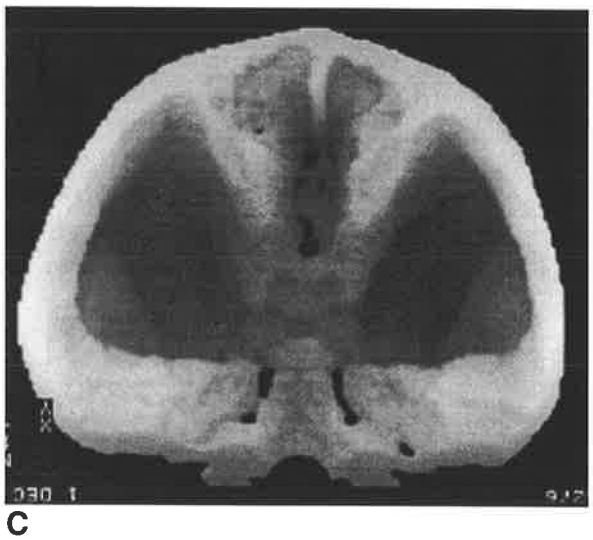


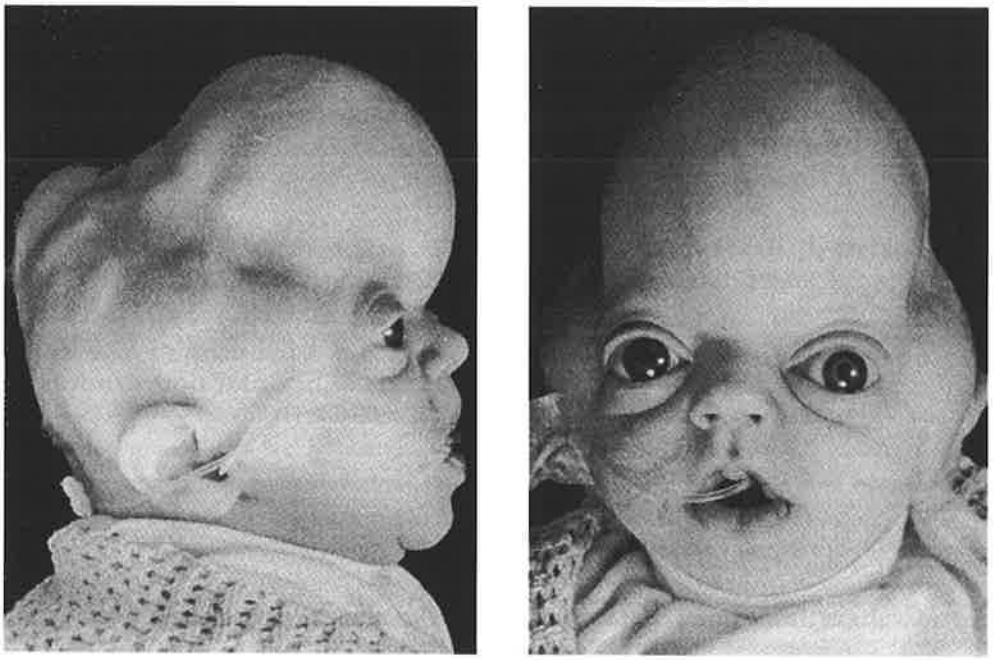
C

FIG. 20–12. *Oxycephaly associated with extensive premature sutural fusion and bilateral cleft lip and palate:* **A.** *before operation;* **B.** *after operation.* **C.** *Preoperative three–dimensional CT scan in the same case, showing distortion of orbital roofs.*

Conclusion

Most types of craniosynostosis can be correctly diagnosed in the first month of life. Pediatricians should know the significance of the various skull deformities associated with premature synostosis because surgical treatment is most effective when done early. It is true that the need for surgery has been much exaggerated in the past. Studies of the natural history of untreated craniosynostosis have shown that premature fusion of a single suture system is most unlikely to affect cerebral growth adversely. But the same studies (Barritt, Brooksbank, and Simpson 1981) have shown that some abnormal head shapes can result in considerable psychological distress, and many parents see this as an indication for operative correction. When there is premature fusion of two or more sutural systems, there is a much greater risk of neurological complications, and the case for surgical intervention is proportionately stronger.

In an important minority of cases, craniosynostosis is found as part of some inheritable syndrome, of which Crouzon syndrome (craniofacial dysostosis) and Apert syndrome (acrocephalosyndactyly) are the best known. It is particularly desirable to diagnose these syndromal craniosynostoses as early as possible, and there are two good reasons for this. First, the craniosynostosis syndromes often show Mendelian inheritance (Cohen 1979), and genetic counseling can be most helpful. Second, the craniosynostosis is likely to be severe and extensive, and there are often grotesque associated facial deformities. It is these unfortunate infants who will benefit particularly from early referral to a specialised craniofacial unit: Many of them will require repeated operative procedures as they grow older, and continuity in management will avoid much trouble and suffering (see Chapter 22).



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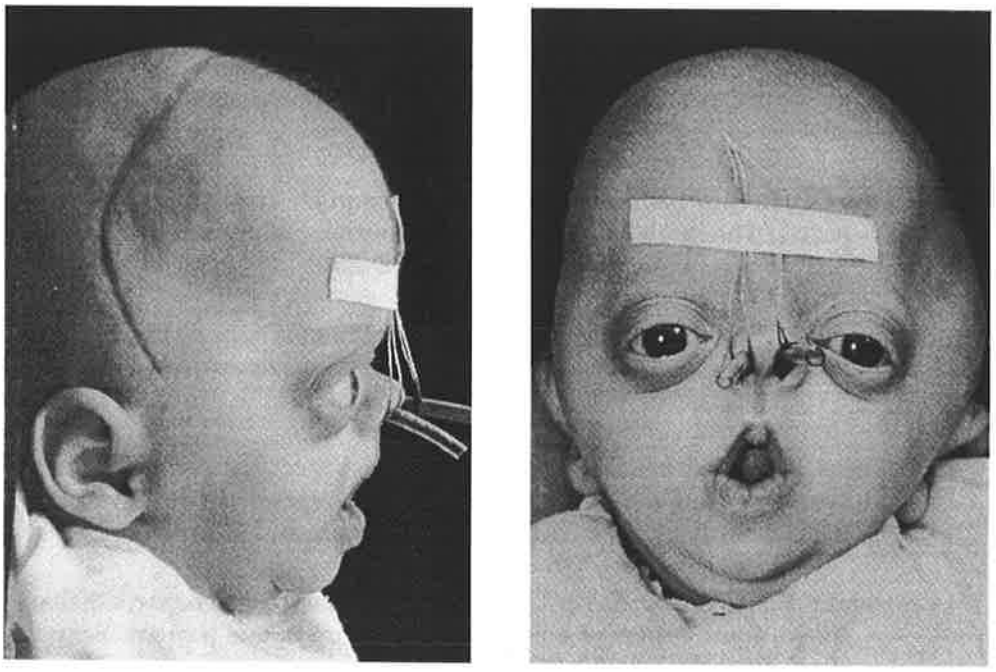

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FIG. 20-13. *Triphyllocephaly (Kleeblattschädel) in a case of Crouzon syndrome:* **A.** *before operation;* **B.** *after operation.*

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Craniosynostosis

D. J. David and D. A. Simpson

Premature fusion of cranial sutures represents a striking disturbance of normal skull growth often associated with gross deformities of the head and face, less often with functional impairments of the brain, eyes, air passages and jaws. The deformities of the cranial vault are well recognized and can be treated by standardised operative procedures. Deformities of the facial skeleton are less well understood and less easily treated; however, the evolution of craniofacial surgery has made available operative techniques which can be applied in most cases with reasonable prospects of success. The timing of intervention is of crucial importance and must be related to the processes of normal and abnormal craniofacial growth and to a knowledge of the pathological process.

Pathophysiology

Craniosynostosis, seemingly a very simple process, is in fact a complex and poorly understood condition with many causes. To understand the pathophysiology of sutural closure, a knowledge of current ideas on normal craniofacial growth is needed. These concepts have been set out in a monograph from which the present account is largely drawn (David et al 1982).

Development of the cranium

In embryonic life, the developing brain is surrounded by a membrane of condensed mesenchyme. Skeletal tissues form in this mesenchymal capsule: cartilage is laid down as future skull base, and after the 6th week this process of chondrification is supplemented by endochondral bone formation. At birth most of the skull is ossified. The chief bones of the skull base are thus cartilaginous bones, growing by ossification in synchondroses which resemble the epiphyses of long bones (Fig. 10.1). The remainder of the cerebral capsule ossifies directly, by deposition of bone in the mesenchymal membrane which differentiates into an outer layer, the pericranium, and an inner layer, the dura mater.

Both are osteogenic. After the 6th week of intra-uterine life, centres of membranous ossification appear over the cerebral hemispheres. By the 5th month, the flat bones of the skull vault are well formed. They are separated by zones of unossified membrane, the fontanelles and the sutures (Fig. 10.2).

Histologically the sutures in early life show active osteoblastic bone deposition (Fig. 10.3), with many mitotic figures; with increasing age, the appearances suggest more indolent cellular activity, and finally the suture is composed of fibroblasts, collagen fibres and venous sinusoids (Fig. 10.4 shows these appearances in a pathological specimen).

The fetal brain increases in volume very rapidly (Fig. 10.5), and the cranial capsule expands to accommodate the brain. Much of this capsular growth takes place at the margins of the bones, in the sutures. It is now generally agreed that the cranial sutures are not primary growth centres; sutural bone is laid down as a secondary response to capsular tensions. These tensions represent the sum of

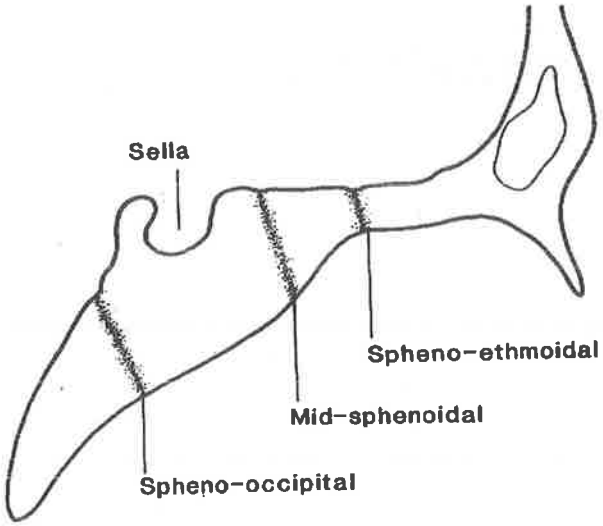
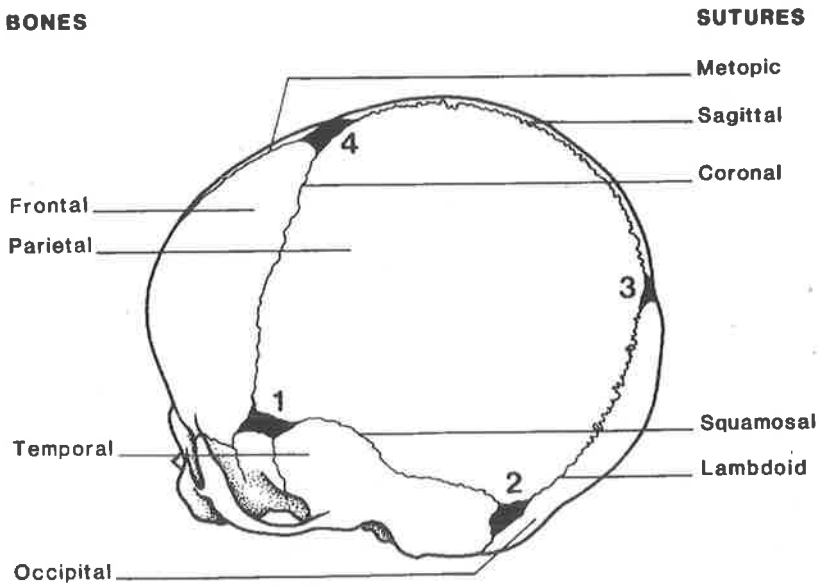
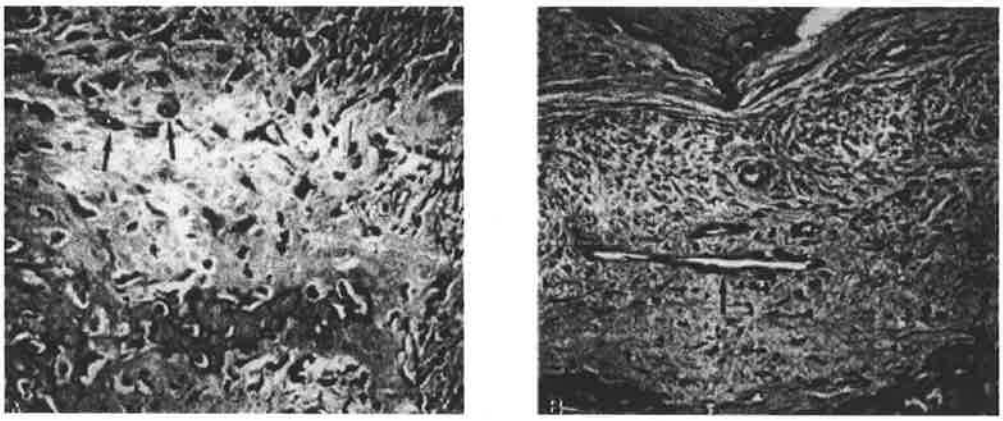


FIG. 10.1. *Structure of the cranial base. The frontal sinus usually develops by the 7th year. The spheno-occipital synchondrosis remains open until adolescence; the other synchondroses close in infancy (after David et al 1982)*



FONTANELLES in order of closure
1 Sphenoidal
2 Mastoid
3 Occipital
4 Frontal

FIG. 10.2. *Schematic arrangement of the bones, sutures and fontanelles. In order of closure the fontanelles are:* **1.** *spheroidal,* **2.** *mastoid,* **3.** *occipital and* **4.** *frontal*



A B

FIG. 10.3. A. *Normal osteoblastic activity in partially fused coronal suture of 6-week-old infant with plagiocephaly (arrows indicate two osteoblasts).* **B.** *Early fusion with cessation of active growth in lateral end of same suture (arrow indicates venous sinusoid). (H & E: by courtesy of Department of Histopathology, Adelaide Children's Hospital)*

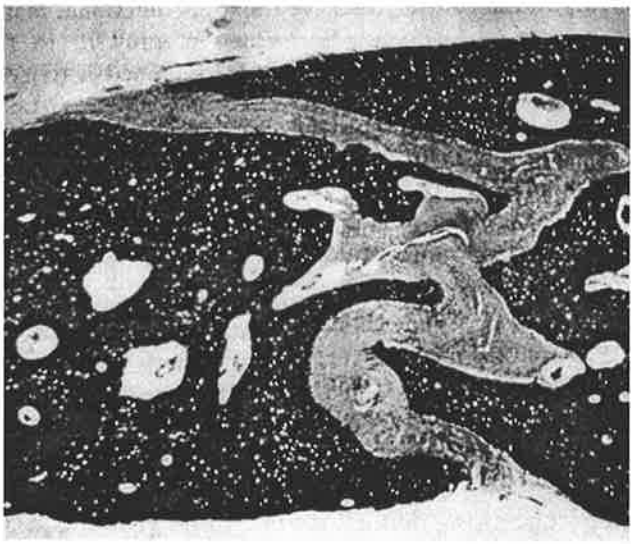


FIG. 10.4. *Indolent growth in coronal suture of child with plagiocephaly, aged 2 years; elsewhere the suture was obliterated. (Masson; by courtesy of Department of Histopathology, Adelaide Children's Hospital)*

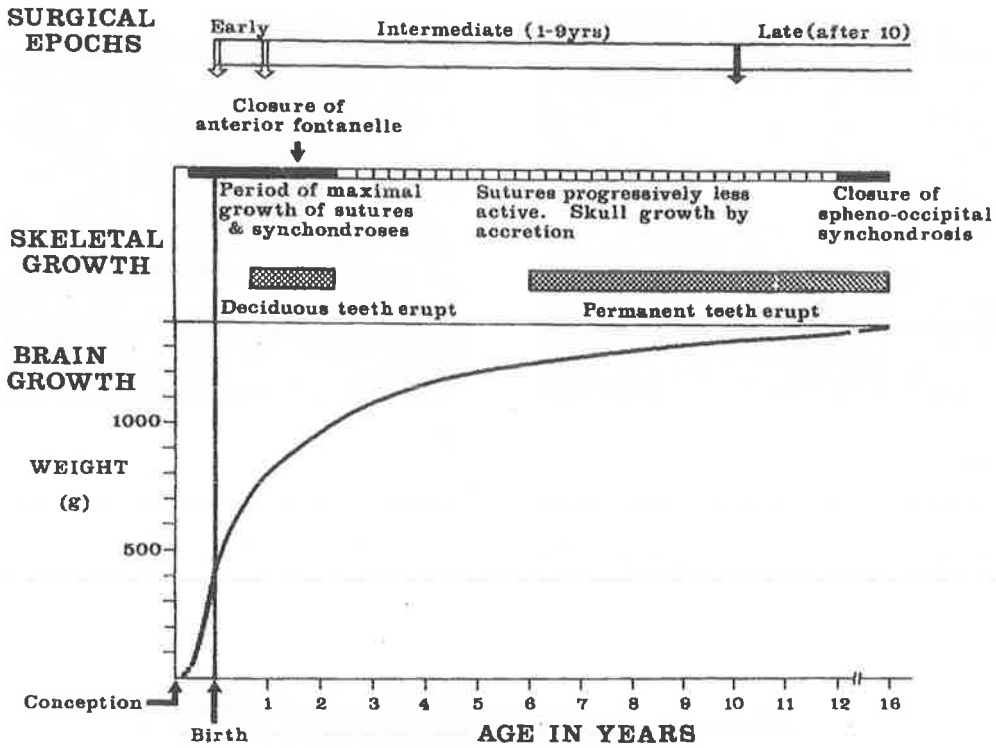


FIG. 10.5. *The three phases of surgical intervention in relation to the growth of the skull, teeth and the brain (from Simpson and David 1986)*

the dynamic effects of the expanding brain and the independent growth of the skull base, transmitted through the dura mater and its septa. The developing eyes and the facial viscera also modify cranial growth. Sutural bone deposition is not the only process involved in the growth of the skull vault: surface deposition of bone by the pericranium and removal of bone internally are also important, especially in later childhood. Nevertheless, the sutures are of crucial importance as growth zones in infancy, and premature sutural fusion (craniosynostosis) is often associated with striking distortions of cranial growth.

Development of the face

For clinical convenience, the face can be divided into upper, middle and lower thirds. These correspond to the embryonic frontonasal, maxillary and mandibular processes. Bone formation in the middle and lower thirds of the face occurs either by ossification of the cartilaginous visceral arches, or by intramembranous ossification. The concept that the tissues of the face have a dual origin, derived partly from neural crest ectomesenchymal cells and partly from the mesoderm of the visceral arches, helps to explain how defective formation of bone derived from the neural arches can be partly overcome by catch-up development in bone derived from mesoderm. The intramembranous facial bones develop from numerous ossification centres in the embryonic frontonasal and maxillary processes. The sutural connections between the individual facial bones and their connections with the bones of the cranial base above them permit antero-inferior expansion of the face relative to the neurocranium. The form of the face is influenced by at least three processes of growth. First the eyes, the ears and the nasal cavities act as functional matrices in the midface. Second, the cartilaginous nasal septum also contributes to the interstitial growth, though its dynamic role is still in dispute. Unrestricted growth of the nasal septum, seen in severe bilateral cleft lip and palate, leads to gross proboscis-like nasal deformity. Finally, the expansion of the developing muscles of the tongue and pharynx plays an important part in shaping the middle and lower thirds of the face.

The direction in which upper facial development occurs is associated with the expansion of the eyeball and with the growth of the frontal lobes of the brain and the anterior cranial fossa. Acting as an adjustable link between these two prime movers is the cranial base, the template on which the face develops. Thus any severe disturbance in the intricate topography and dimensional relationships which exist between the face and brain may greatly change the characteristic design of the human face.

The role of the sutures in facial growth is still controversial. Despite many attempts, it has not been possible to demonstrate autonomous expansion of these sutures. Growth of the midface depends to a large degree on the growth of the orbits and their contents, the nasal cavities, the maxillary sinuses and the teeth. It is nevertheless likely that the facial sutures, like the calvarial sutures, are growth zones in which bone is deposited in response to the expansion of the facial viscera.

The lower third of the face develops from the paired mandibular processes. The functional mandible also constitutes a growth matrix. The mandibular bone itself enlarges by apposition and resorption of bone, while growth of non-osseous tissues, particularly the condylar cartilage and the muscles of mastication, promotes progressive change in size. While condylar growth undoubtedly has a beneficial affect on normal growth of the ramus, it is unlikely that it has a fundamental role in determining the development of the lower third of the face.

Most of the changes that occur with facial growth are thus brought about by the combination of several mechanisms. Expansion of the brain lengthens the anterior cranial base, enlargement of the globe of the eye expands the orbits, the spheno-occipital synchondrosis is an active growth centre in the skull base, the nasal cartilage advances the maxilla and there is a small contribution from surface apposition. The sutures probably act more as growth adjustors than as growth initiators. After the 7th year, there is little further growth of the orbit and midface growth by expansion greatly diminishes (Scott 1954). It is believed that about this time nasal cartilage activity is greatly diminished. The perpendicular plate of the ethmoid unites with the vomer and from now on most midface growth is achieved by surface deposition and internal resorption. The malar bones become more prominent as adolescence proceeds; this change can be ascribed to the lateral displacement and drift of these cheekbones. The palate continues to drift away from the cranial base and the nasal cavities continue to enlarge as the respiratory demands of the body are increased. However, not all the striking increase in vertical height can be ascribed to drift alone. It is likely that direct expansion of the maxilla accounts for about half the downward growth. The dynamics of this expansion have been disputed. Scott (1956) proposed that the growth of the nasal septum forces the maxilla downwards and forwards; Moss (1968), on the contrary, believed that his functional matrix theory can best explain the method of growth. In this hypothesis it was supposed that the combined activity of expanding organs, functional space and muscles, bone, cartilage and nerves operate as a harmonious unit to displace the maxilla and to initiate the internal resorption and superficial deposition which produced drift. However this may be, it is obvious that new tissue continues to be laid down in the region of the maxillary tuberosities to accommodate the developing molar teeth. The maxilla therefore does not advance forward by apposition of bone on its anterior surface; on the contrary, there is some anterior surface resorption as posterior bone deposition proceeds. Thus anterior displacement by posterior bone growth plays the predominant role in forward growth of the maxilla.

Pathology

Fused sutures in the cranial vault may appear as broad ridges of bony overgrowth, or may be completely obliterated and indistinguishable from the surrounding bone. Histologically, the appearances vary with the degree of fusion. In advanced

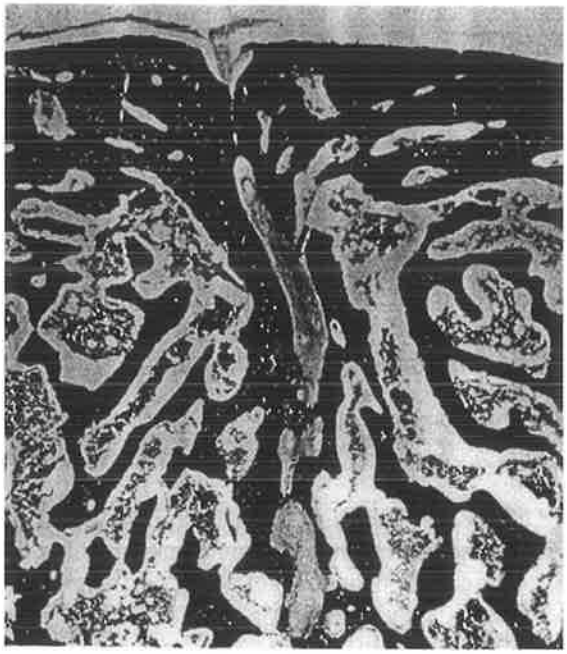


FIG. 10.6. *Obliteration of metopic suture (island of fibrous tissue indicated by arrow) in infant with trigonocephaly, aged 3 months. (Masson: by courtesy of Department of Histopathology, Adelaide Children's Hospital)*

cases, the site of fusion may be undetectable, or evident only as an atypical orientation of trabeculae. Less complete fusion may be marked by islands of fibrous tissue embedded in the bone (Fig. 10.6). In the early stages of fusion (Figs. 10.3b and 10.4), the suture may show the recognisable histological features of a mature suture, as described by Pritchard et al (1956), but without the signs of active osteoblastic growth appropriate to the age of the infant (Fig. 10.3a).

In most cases, one sees no features suggesting specific interference with normal sutural growth, though there may be evidence of hyperplastic bone growth of an abnormal type. Rarely, there may be islands of cartilage (David et al 1982, Hinton et al 1984) but this also has been reported in normal sutural growth. Thus, in the narrow sense, the pathology of craniosynostosis suggests normal sutural closure occurring at an abnormally early age. This interpretation has been challenged by Albright & Byrd (1981) who argue that sutures are not normally obliterated by fusion. It is true that normal closure of the metopic suture in infancy, or of the coronal and sagittal sutures in adult life, does not show the hyperplastic changes sometimes seen in craniosynostosis, but this may only represent the difference between a rapid and disorderly cessation of sutural growth in craniosynostosis and the slower and orderly cessation of growth in normal suture closure. The histological findings in biopsies taken from different parts of an incompletely fused suture certainly suggest a growth process in different stages of failure, ranging from complete cessation of sutural growth and replacement of bone, to virtual normality (Figs. 10.3, 10.4 and 10.6).

Fusion of facial sutures is unfortunately less well documented. Synostosis of maxillary sutures does occur in some of the craniosynostosis syndromes (Kreiborg & Björk 1982), but it is at present uncertain whether the process of facial synostosis has any regular peculiar histological features.

Experimental sutural fusion

Many attempts have been made to duplicate the process of craniosynostosis in an experimental animal. Persson et al (1979) were able to produce skull deformities in rabbits by immobilising the growing coronal suture with cyanoacrylate. We have failed to do this in the infant marmoset, and the

significance of these experimental studies in relation to natural craniosynostosis is unclear (Foley & Kokich 1980). They provide strong support for operations designed to promote more normal craniofacial growth by excising fused sutures (Alberius & Selvik 1984), but do not as yet throw light on the mechanisms and nature of spontaneous craniosynostosis.

The concept that intra-uterine cranial compression may be a cause of craniosynostosis, presumably by inducing sutural fusion through cessation of growth, still awaits convincing experimental evidence in support (Graham et al 1979, 1980).

Significance of craniosynostosis as a pathological process

It is now agreed that the cranial sutures do not have a driving role in the expansion of the cerebral capsule. Rather, they are zones where marginal bone deposition proceeds in response to the demands of the expanding brain and eyeball, probably transmitted along the lines of dural (or periorbital) tension. Moss (1959,1975) has argued that premature sutural fusion is secondary to more fundamental dysplasias of the skull base.

In his view, synostosis is a symptom, not a cause of deformity. It is hard to accept this view in total. It does indeed seem likely that the individual cranial and facial deformities associated with craniosynostosis represent dyscephalies due to disturbed growth of the entire cerebral capsule, the cranial base as well as the vault, the pericranium and dura as well as the bone. However, there is evidence to suggest that premature sutural fusion does perpetuate or even exaggerate the general disturbance of growth. To the surgeon, the most compelling evidence is the response to adequate resection of fused sutures (Fig. 10.7). Removal of bone is quite predicably followed by varying degrees of correction of calvarial deformity. Bone grows again chiefly from the dura mater, but often this regrowth does not amount to complete synostosis and there may be radiological evidence of a new suture in the regenerated bone. The reappearance of a previously obliterated suture suggests that regional cranial growth has been released by operation, allowing the brain to expand in a more normal way. If so, it seems logical to regard the premature sutural fusion, however caused, as an important pathological process, since it can lead to local distortion or restriction of the expansion of the brain. We ourselves see craniosynostosis as an important and treatable element in cranial growth disorders that may be regional or generalized; the causes of these disorders include genetic aberrations, metabolic diseases and possibly intra-uterine constriction. In many cases, the cause is unknown.

When craniosynostosis is extensive it may impair the normal relations between cerebral growth and enlargement of the cerebral capsule: the skull may be too small for the growing brain. Intracranial pressure may then rise. Papilloedema may result; vision may be affected and perhaps mentality, although this is less certain. When the cranial capacity is reduced like this, the traditional term *craniostenosis* seems appropriate (Muke 1972) (Fig. 10.8).

In the same way, one can speak of *orbitostenosis*. The orbit is the bony capsule of the expanding eyeball. Unlike the skull, it is an incomplete capsule, being open anteriorly. In certain craniofacial syndromes, notably Crouzon syndrome, the dysplasia affects the walls of the orbit. It is not wholly clear what part premature fusion of the various orbital sutures plays in this process, but they are undoubtedly affected in some cases (Kreiborg & Bjork 1982). In consequence, the orbital cavity is distorted. It is shallow and wide and the eyeball is extruded forward (exorbitism) (Fig. 10.9).

Following Delaire et al (1963) we use the analogous term *faciostenosis* to describe the midfacial hypoplasia of Crouzon syndrome and the consequent interference in nasal breathing and in mastication (Fig.10.10). Delaire considered this to be a stenotic process in the bone surrounding the upper airway and

postulated that faciostenosis may occur as a primary condition, with or without craniosynostosis of the calvarial vault. The pathological evidence is, however, still somewhat meagre. We believe that the maxillary hypoplasia of Crouzon and Apert syndromes does represent an intrinsic local growth failure often associated with premature sutural fusion, and many authors now accept this view, though there are still those who believe that it is secondary to a primary dysplasia of the skull base, acting in varying permutations on growth of the vault and facial skeleton. Our own studies of 48 cases of Crouzon syndrome and 21 cases of Apert syndrome show that early reconstruction of the anterior cranial fossa does not prevent ultimate deformity of the mid-face.

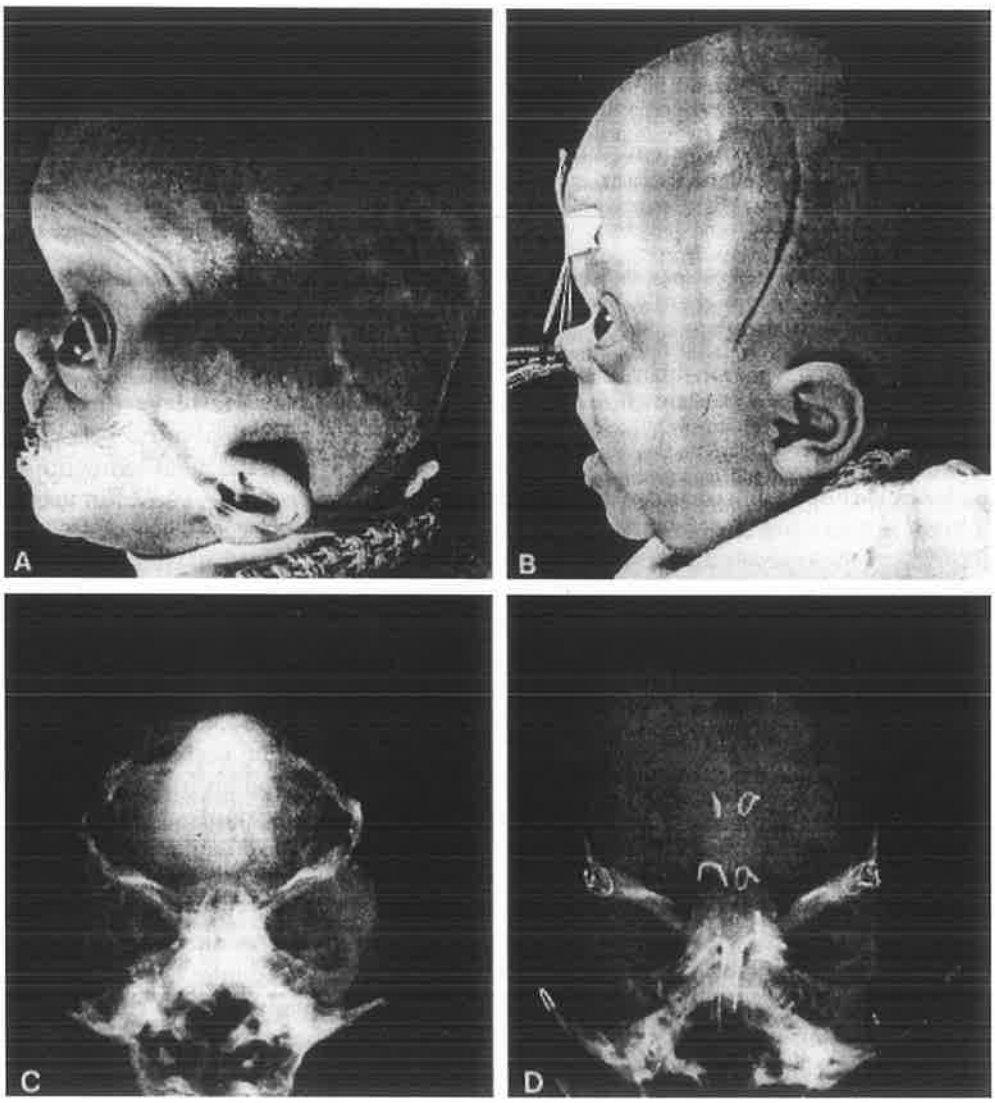


FIG. 10.7. A. *Crouzon syndrome in a 3-month-old infant. There is widespread calvarial synostosis and severe craniostenosis producing a clover-leaf deformity (triphyllocephaly).* **B.** *The same child 3 weeks after sutural release and fronto-orbital advancement, demonstrating the striking change in skull shape.* **C.** *A radiography of the same child before surgery.* **D.** *A radiograph after sutural release. The calvarial remodelling is dramatic.*

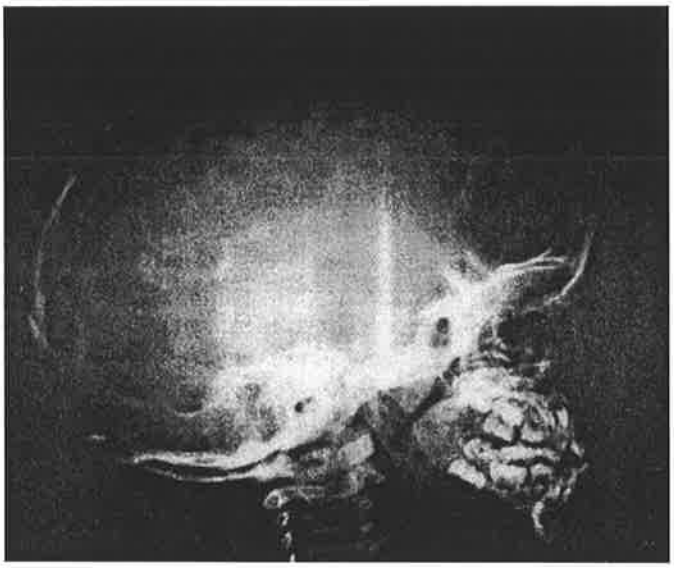


FIG. 10.8. *Increased convolutional markings in an infant aged 20 months associated with multiple premature sutural fusions (Crouzon syndrome), producing craniostenosis*

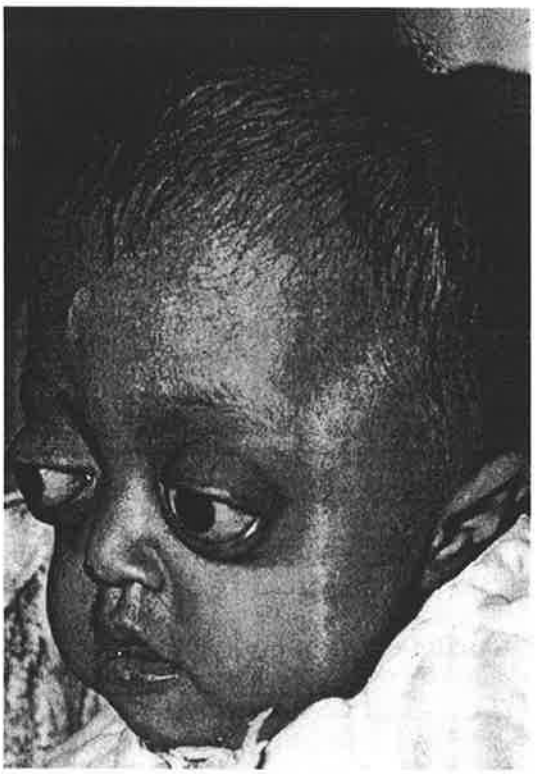


FIG. 10.9. *An infant with Crouzon syndrome demonstrating severe dysplasia of the orbital walls, resulting in extrusion of the eyeball (orbitostenosis)*

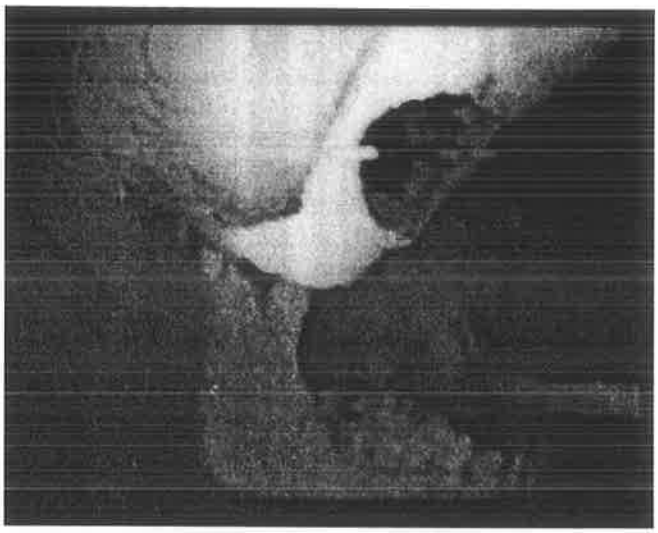


FIG. 10.10. *A three–dimensional reconstruction from CAT in an adult with Apert syndrome showing the extreme maxillary hypoplasia, producing faciostenosis about the upper airway. An endotracheal tube is shown (From Hemmy et al 1983 with permission of the Editor, Journal of Neurosurgery) (From David et al 1982)*

Classification

Morphological

1. Calvarial deformities (Fig. 10.11)

Heads vary greatly in shape and size. When these variations are extreme, they are regarded as deformities, and this judgement is based on intuitive concepts of normality. The unusual head is thought to be too long, too broad, too high, too large or small, or it is lopsided. These concepts vary in different societies and cultures. Objective measurements can be used to supplement aesthetic impressions; the various craniometric indices devised by anthropologists to quantify racial variations have some utility in defining deformities. The most relevant is the cephalic index (more strictly the horizontal cephalic index), defined as (maximum breadth/ maximum length) × 100 and measured either directly or on skull radiographs.

Cranial asymmetry is very hard to quantify. In basal radiographs one can construct a sagittal line bisecting the occiput through the foramen magnum and determine its relation to the nasal septum or the metopic suture; if the cranial asymmetry involves distortion of the skull base, the median axes of the occiput and frontal portion of the skull may not be identical. These two-dimensional analyses of radiographs have severe limitations in their ability to describe a deformity. A more sophisticated radiological investigation is given by CAT, from which a three–dimensional image is generated. This enables the observer to have a more accurate image of all features of the skull viewed from any angle. As yet, however, such images are qualitative rather than quantitative and a classification based on mathematical quantification is not yet possible. We therefore advocate a simple descriptive classification using traditional terms with supplementary help from craniometry and radiography.

Scaphocephaly (Fig. 10.11a). This is the commonest calvarial deformity associated with craniosynostosis. The head is long and narrow, with frontal bossing and a prominent occiput. The sagittal suture fuses prematurely, and in babies can be felt as a midline bony keel. The anterior fontanelle is usually small. In the great majority of cases, the craniosynostosis affects only the sagittal suture; occasionally there is also involvement of the coronal or metopic sutures.

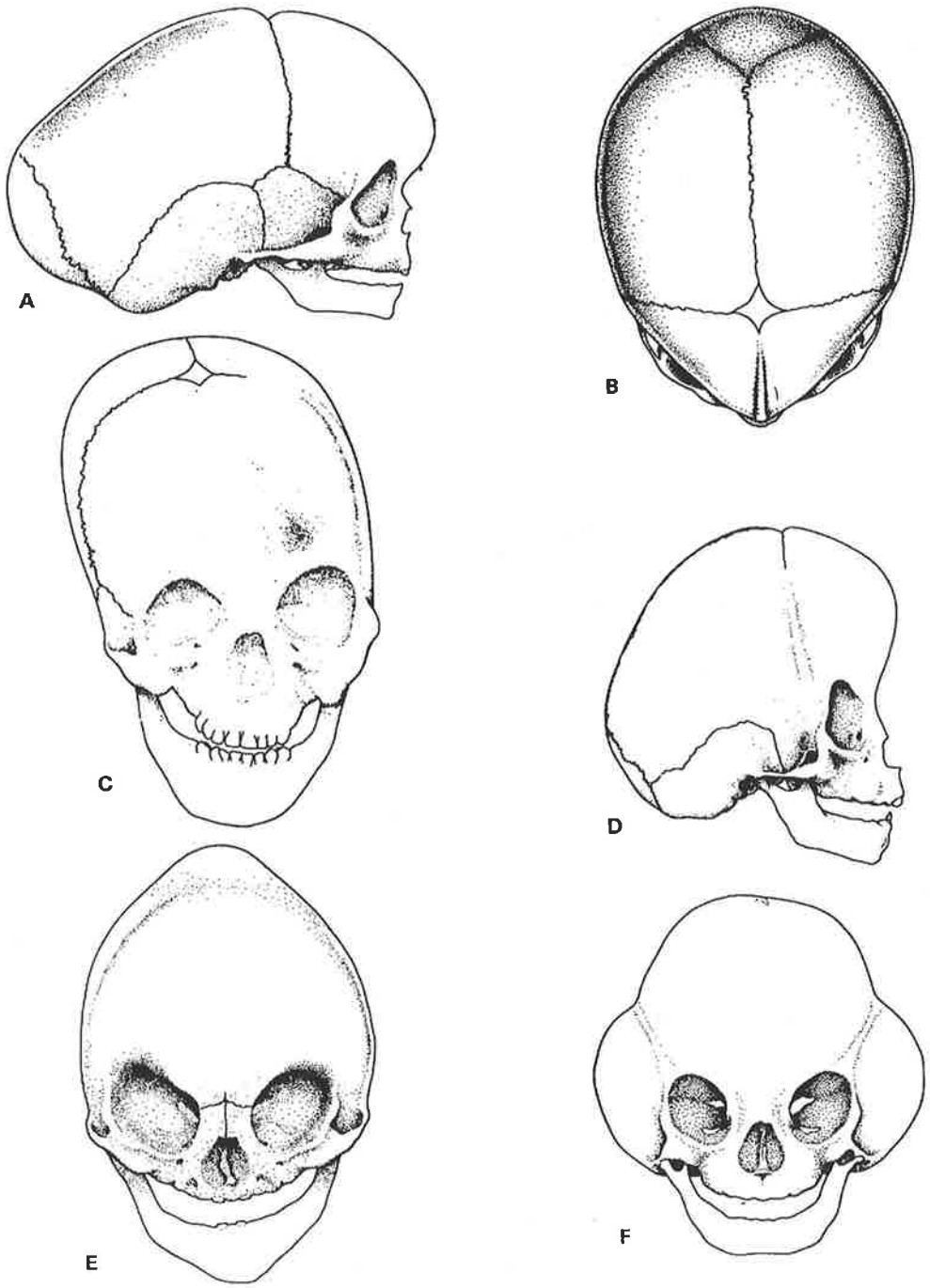


FIG. 10.11. A. *Scaphocephaly.* **B.** *Trigonocephaly.* **C.** *Frontal plagiocephaly.* **D.** *Turricephaly.* **E.** *Oxycephaly.* **F.** *Triphyllocephaly (clover-leaf deformity).*

The deformity can usually be diagnosed in the neonatal period, though the synostosis may still be incomplete, or even undetectable by X-ray. As a rule there are no associated extracranial abnormalities and no familial history; however, some cases of Crouzon and Carpenter syndromes (David et al 1982) may have scaphocephalic heads. Craniostenosis is rare. Most cases, whether treated or not, show no mental or neurological abnormalities. In a minority of cases there is developmental delay, sometimes severe.

Operative treatment may be indicated on aesthetic grounds, and in our hands the established neurosurgical procedure of linear craniectomy has been satisfactory. A midline scalp incision exposes the fused sagittal sinus. The resulting craniectomy is 4–5 cm broad and extends well across both lambdoid and coronal sutures. Silastic strips are inserted on each side. The operation is

done no later than the 6th month of life, and preferably sooner. In neglected cases, if operation is really needed, more elaborate reconstructions of the cranial vault have been advised (Montaut & Stricker 1977; Marchac & Renier 1982).

Trigonocephaly (Fig. 10.11b). In this less common deformity the frontal region is wedge shaped, with a median ridge representing the fused metopic suture. The forehead is narrow. The eyes often have a mongoloid slant; they are set close together, and there is true hypotelorism. A somewhat similar cranial deformity may be secondary to dysplasia of the frontal lobes, and this should be excluded by ultrasound or CT scanning. In the absence of a cerebral dysplasia, the prognosis is quite good, though occasional cases do show developmental retardation. We have seen craniostenosis in association with this deformity.

Experience of the untreated condition has shown that the facial appearance tends to improve with age. Nevertheless, when the deformity is pronounced, operative treatment may be indicated on aesthetic grounds. A coronal scalp incision is made. Excision of the fused metopic suture is supplemented by bilateral frontal craniotomies. The free frontal bones are bent, rotated and fixed to the orbital margins to broaden the forehead. We also insert silastic film along the margins of the metopic craniectomy, though this may be unnecessary. The procedure should be done at the latest by the 6th month, and preferably earlier. In severe cases, a more extensive frontal remodelling may be undertaken by lateral canthal advancement.

Frontal plagiocephaly (Fig. 10.11c). In this deformity, which is second in frequency to scaphocephaly, there is premature fusion of one coronal suture and usually also the ipsilateral basal sutures, especially the sphenofrontal suture. There is flattening of one frontal region, with elevation and recession of the eyebrow and orbital margin on the same side. The ipsilateral ear is often prominent, and there is a bony bulge in the contralateral parietal vault. Facial asymmetry is often striking, especially in older untreated cases. The deformity of the skull base displaces the mandibular condylar fossa, distorting mandibular growth and tilting the dental occlusal plane of the lower jaw. The root of the nose, and the vertical plate of the ethmoid, tilt towards the affected side. Radiographs show the coronal synostosis, and also the orbital deformity and curvature of the skull base (Fig. 10.12). Ultrasound or CT scanning will often show ventricular asymmetry, but serious cerebral anomalies are rare and we have not seen clinically evident raised intracranial pressure.

The deformity can usually be recognized in infancy, and may be associated with extracranial anomalies (e.g. minor syndactyly or anal ectopia). The deformity may be part of Saethre-Chotzen syndrome (David et al 1982). Untreated, the condition is often quite disfiguring and in our opinion early operative treatment is usually justified.

A coronal scalp flap is used. The fused coronal suture is excised from across the midline into the skull base; great care is needed in resecting the sabre–shaped spheroidal ridge, which is swept up onto the cranial convexity. Simple excision of the fused coronal and sphenofrontal sutures is sometimes disappointing, and this procedure should be supplemented (Hoffman & Mohr 1976) by a frontal craniotomy and advancement of the ipsilateral orbital margin. To do this, the supra–orbital ridge and the anterior part of the orbital roof are dissected extradurally and through the orbit; they are then mobilized by osteotomies as a single fronto–orbital block. This is advanced and secured in the advanced position by two vertical spurs of bone, one cut from the lateral orbital wall below and the other from the inferior margin of the fronto-orbital block (Fig. 10.13). The free frontal bone flap is bent, rotated and replaced to correct the frontal deformity. Silastic is inserted from across the midline to a point well below the squamosal suture; it is our practice to do this only on the posterior margin of the craniectomy, and we omit the insertion of silastic above the age of 6 months. This procedure is best done in early infancy. However, quite satisfactory results can be obtained

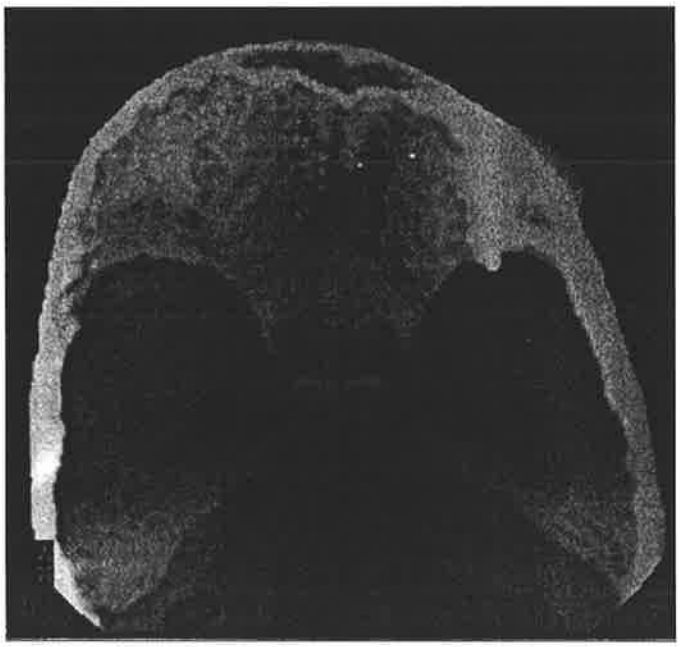


FIG. 10.12. *3D reconstruction showing the base of skull deformity associated with frontal plagiocephaly. The foreshortened anterior fossa, the midline curvature of the skull base and the distortion in the middle cranial fossa are well demonstrated.*

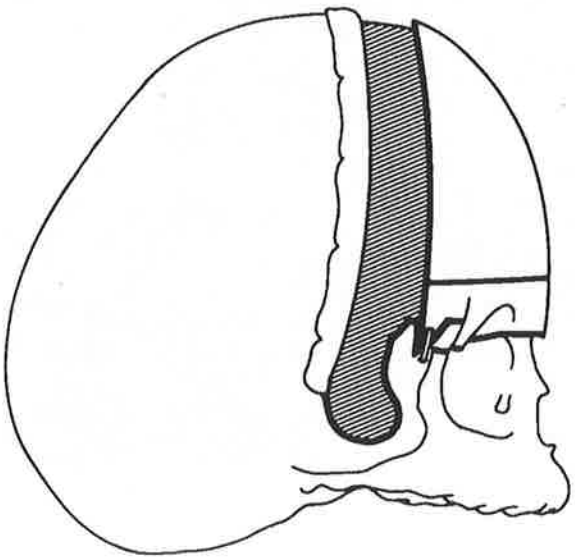


FIG. 10.13. *Coronal linear craniectomy and fronto-orbital advancement. The advanced fronto-orbital segment is secured by two vertical self-stabilizing osteotomies in the lateral orbital wall. The posterior margin of the cranicaomy is coated with silastic. Fronto-orbital advancement can be unilateral for frontal plagiocephaly, or bilateral for turricephaly, resulting from bicoronal synostosis*

up to 3 years of age (Hoffman et al 1982) (Fig. 10.14 a,b,c,d). In neglected cases, the deformity may include nasal deviation and an ugly asymmetry of the orbits; these may require major craniofacial correction (see Fig. 10.15a,b).

Occipital plagiocephaly. This common deformity is characterised by unilateral occipital flattening on one side, with prominence of the ipsilateral frontal region; seen from above the skull is deformed into a parallelogram, sometimes with prominence of the ipsilateral ear. There is very little facial deformity.

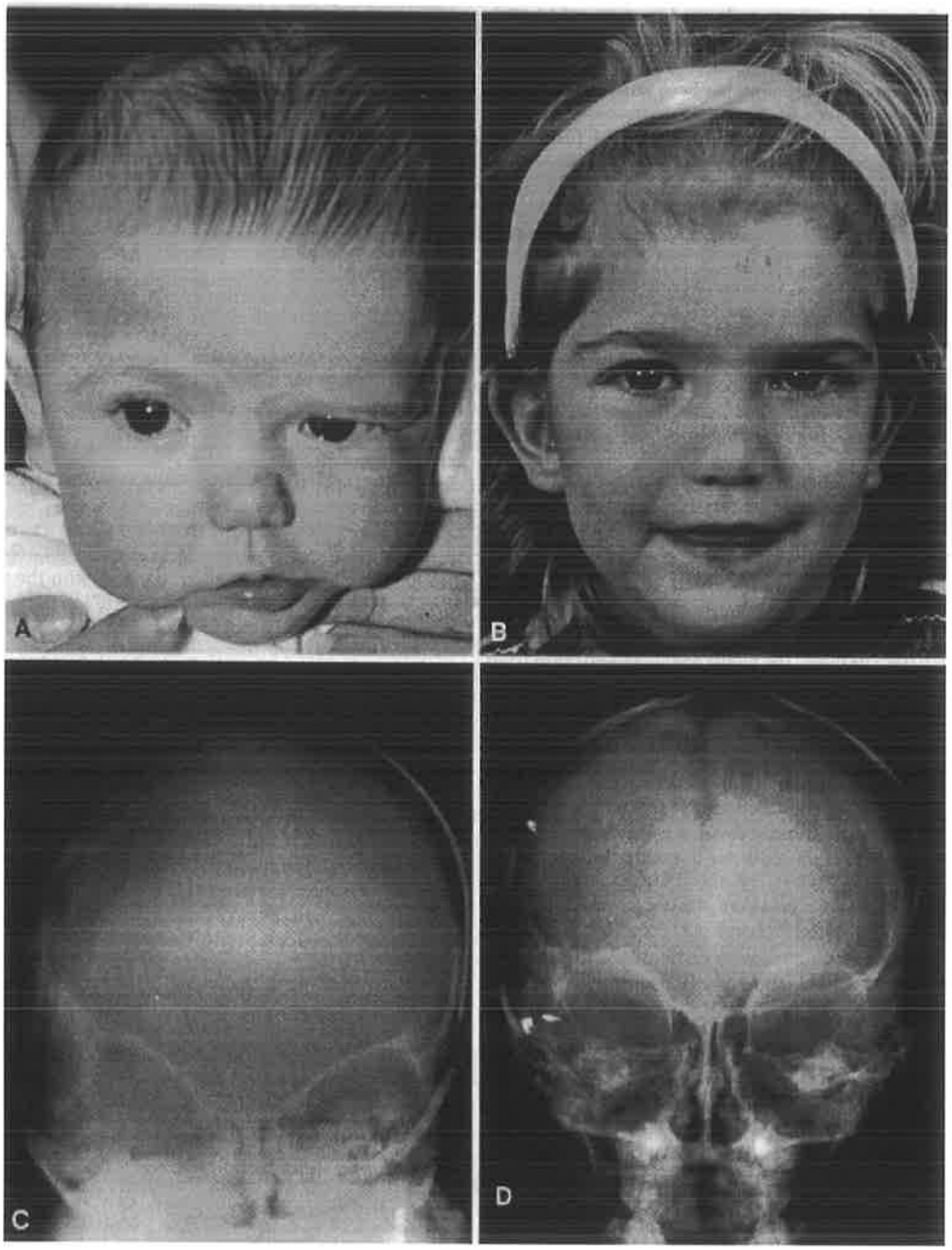


FIG. 10.14. A. *3-month old infant with frontal plagiocephaly.* **B.** *Same child aged 4 years after linear craniectomy and fronto-orbital advancement.* **C.** *A pre-operative radiography showing the swept-up sphenoidal ridge.* **D.** *A radiography at age 4½ years.*

The place of this deformity among the craniosynostoses is uncertain (Hinton et al 1984). In some cases there is radiological evidence of lambdoid sclerosis which may go on to fusion. However, very similar plagiocephalic skull deformities have been seen as a consequence of postural pressure in infancy or in the absence of any detectable cause, whether in the sutures, the skull base, or the neck. This type of plagiocephaly is rarely associated with cerebral involvement, and tends to be less noticeable in older children. If there is strong parental concern, and if the lambdoid suture is radiologically abnormal, the condition may be treated by excising the entire suture with silastic inserts on each side of the resulting craniectomy (Hoffman et al 1982). The results of this operation are not always good, perhaps because there is an associated deformity of the skull base.

Turricephaly (Fig. 10.11d). This deformity is characterised by a broad towering head and is associated with premature fusion of both coronal sutures. The sphenofrontal sutures are as a rule also involved. As an isolated anomaly,

turricephaly is the commonest pattern of skull deformity in Crouzon syndrome (Kreiborg 1981) and is invariably found in Apert syndrome. It is not unusual to see a delayed onset of sagittal synostosis in older infants in whom there was initially involvement of the coronal sutures only; this widespread craniosynostosis is characteristic of oxycephaly (see below) and the two deformities are closely allied. Turricephaly is not the only deformity associated with extreme brachycephaly: premature fusion of both lambdoid sutures is also associated with a very brachycephalic skull, the occipital region being quite flat. This deformity has been termed *pachycephaly*.

Turricephaly is usually evident at birth, though it may be missed, especially when there is a family tendency to brachycephaly. Absence of the anterior fontanelle may arouse suspicion. Associated anomalies are often found, and evidences of genetic causation should always be looked for, as the condition may be part of Crouzon syndrome. This diagnosis is easily missed, as maxillary hypoplasia is often inconspicuous in infancy. The syndactyly of Apert syndrome is of course unmistakable, and in this condition the turricephaly has peculiar features, notably a wide metopic bone defect.

Untreated turricephaly is compatible with normal intelligence (Jensch 1941–2). However, there is a high incidence of craniostenosis and significant aesthetic deformity; in our view therefore there are strong indications for early operative treatment. This is done through a coronal scalp flap incision. The fused coronal sutures and the hypertrophied spheroidal ridges are excised and subtemporal craniectomies are performed. The frontal bones are elevated by craniotomy above the orbits. The operation can then be completed by inserting silastic strips along the posterior margin of the new coronal suture, and replacing the frontal bones as onlay bone grafts in front of the orbital margins: this simple procedure has given excellent results when involvement of the skull base is not severe. In the last few years, however, we have been impressed by the benefits of the more extensive frontal mobilization advised by Marchac (1978). By extradural and intra-orbital dissection, the orbital margins and glabella area are freed and resected as a single block, advanced up to 2 cm, and wired to spurs cut on each side from the lateral orbital walls, with an interposed bone graft holding it to the root of the nose. While best done before 3 months of age, this procedure has been satisfactory even in childhood.

Oxycephaly (Fig. 10.11e). Multiple sutural fusion can result in various deformities, depending on the sequence of fusion. When the result is a cone-shaped head, with a sloping forehead rising to a bony boss at or behind the bregmatic area, then the term oxycephaly (sharp head) is appropriate (see Fig. 10.16). The external appearances may be striking, or relatively normal, and there may or may not be associated facial abnormalities, notably the absence of the frontonasal angle (Marchac & Renier 1982). Whatever the skull configuration, premature fusion of multiple sutures is usually an imperative indication for operative treatment, aimed at decompressing the constricted brain and correcting the skull deformity. In most cases we have achieved this by two-stage procedures, excising the prematurely fused sutures bilaterally and advancing or broadening the frontal region. If such procedures are done in infancy, silastic is inserted and re-operation is often needed; in cases presenting after the 1st year, silastic is omitted.

Triphyllocephaly (Kleeblattschädel) (Fig. 10.11f). This dramatic deformity is also a manisfestation of very extensive craniosynostosis developing in utero. As the Greek and German names indicate, the infant's skull looks from in front like a clover leaf: there are large temporal bulges and a median bregmatic bulge, separated by constriction bands of bone and dura, running more or less along the line of Sylvian fissure. There is usually, but not invariably, a severe communicating hydrocephalus. Triphyllocephaly requires early operative treatment. We have employed staged frontal and occipital craniectomies, with fronto-orbital advancement, and the remodelling (Fig. 10.7) has been dramatic.

When there is associated hydrocephalus, one should defer insertion of a shunt until the completion of the planned cranial surgery, in the hope that the hydrocephalus will arrest spontaneously, and because raised intracranial pressure promotes remodelling of the skull. However, shunting should not be delayed too long.

2. Facial deformities

The following facial deformities may occur in simple craniosynostosis, but are more often seen in the craniosynostosis syndromes. They are not always present and are not always of equal severity. The traditional methods of objective measurement, namely cephalometric analyses, have shown some limitations in quantifying facial deformity, particularly for the asymmetrical problems of facial scoliosis. New concepts of three–dimensional analysis are needed (e.g. photogrammetry or objective measurements from three–dimensional CT scanning techniques).

a. Orbit.

(i) Orbitostenosis (Fig. 10.9). This gives rise to exorbitism or inadequate coverage of the globe by the bony orbit. Such incomplete growth of the orbit is contributed to by the anterior cranial fossa hypoplasia, which results in lack of forward projection of the sphenoid bone, giving rise to a foreshortened anterior cranial fossa and lateral orbital wall. In diseases such as Crouzon and Apert syndromes, the orbitostenosis is significantly increased by the maxillary hypoplasia and so both roof and floor of the orbit are poorly developed. The orbital cavity is shallow and the stenosis may be so severe that the eye is extruded. In Crouzon syndrome, orbitostenosis may be extreme and the resulting exophthalmos may be very serious. The visual axes of the extruded eyes tend to diverge and in some patients there is obvious divergence; even when this does not happen, binocular vision may be impossible. Lid closure may be inadequate, resulting in chronic conjunctivitis. In the pupillary line the superior orbital margin should be 1 cm in front of the globe and the inferior orbital margin should be level with the globe. Lateral canthal dystopia results in an antimongoloid slant to the palpebral fissures. In many types of craniosynostosis, the orbital cavity is shallow with a shortened lateral wall resulting from distortion of the sphenoid bones. The orbital floor may also be hypoplastic and flow on to merge with the anterior surface of the maxilla with little evidence of an inferior orbital margin.

(ii) Orbital dystopia (Fig. 10.15). In this deformity one orbit is lower than the other. This may be measured against the occlusal plane. However, in a very twisted face it is often difficult to decide on a suitable reference point. Orbital dystopia often occurs in frontal plagiocephaly, where the pathological process of craniosynostosis affecting the ring of vault and base sutures on one side produces an orbit that is depressed, shallow and sloping away to its inferolateral margin. Orbital dystopia also occurs in other asymmetrical deformities, as in Saethre–Chotzen syndrome, or in a previously partially treated case, where cranial surgery has been more effective on one side than the other. Recent advances in three–dimensional reconstruction from CT scans have shown very well the spatial anatomy of these deformities (Hemmy et al 1983).

(iii) Hyper– and hypotelorism (Fig. 10.16). Hypertelorism is an increase in the distance between the orbits, measured as the interpupillary distance in relation to the appropriate scale for age. The intercanthal distance may be increased, in which case, the deformity is called *telecanthus*. In severe hypertelorism, there is outward rotation of the orbits. The area of the cribriform plate may be widened, producing symmetrical or asymmetrical hypertelorism. This may be due to primary dysplasia in that area, or be secondary to generalized craniosynostosis, which directs brain growth into the base of the skull, depressing the cribriform plate and widening the area between the orbits. Munro (1976) has emphasized the anatomical variability of hypertelorism. The medial walls of the

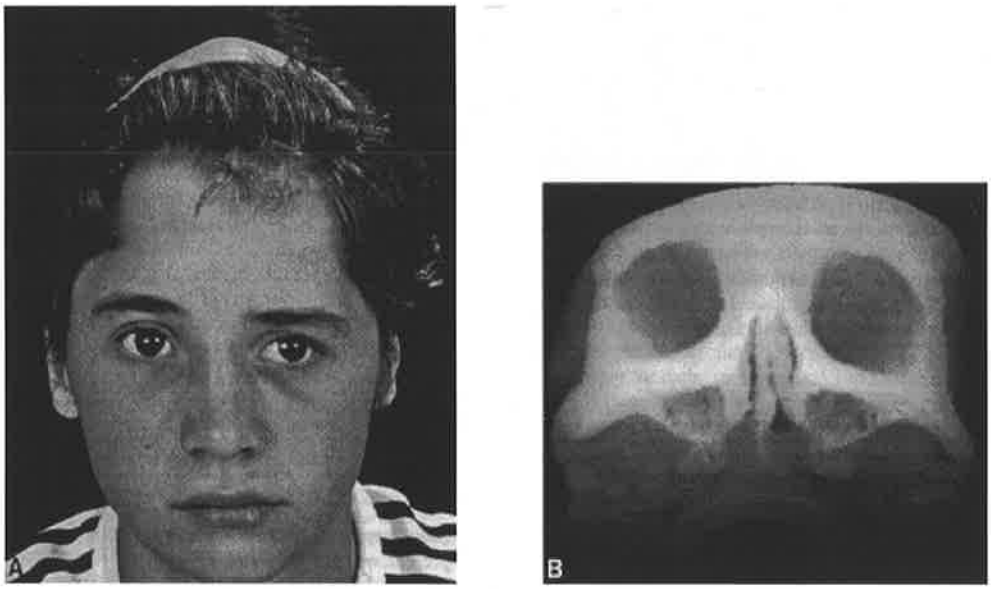


FIG. 10.15. A. *A 16-year-old boy with frontal plagiocephaly producing orbital dystopia and an symmetrical face.* **B.** *3D CT reconstruction of the same patient demonstrating the vertical discrepancy in the position of the orbits.*

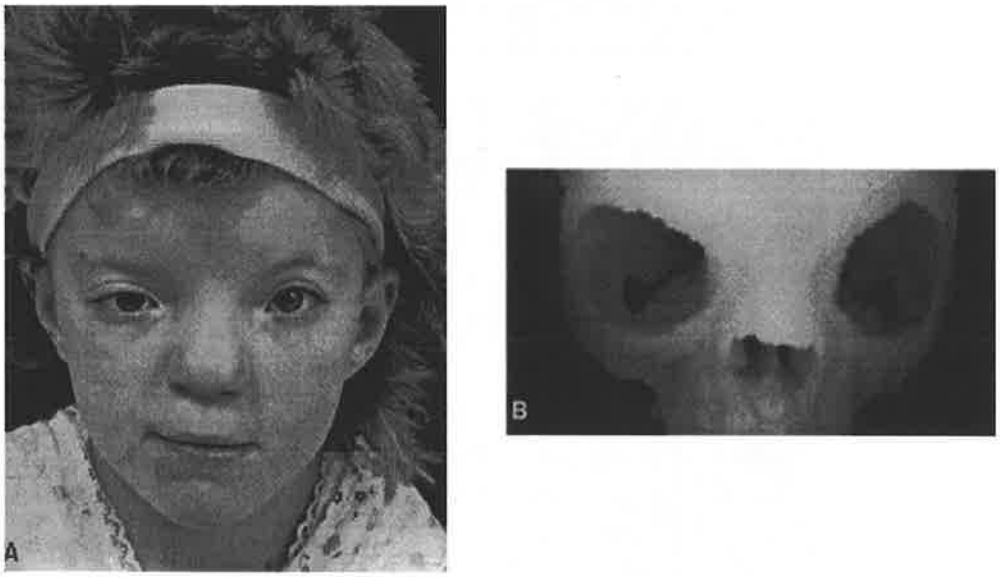


FIG. 10.16. A. *Hypertelorism in a child with Cohen syndrome.* **B.** *3D CT reconstruction of same child's skull showing the hypertelorism and the difference in orbital shape, reflecting the asymmetry resulting from the degree of craniosynostosis affecting the ring of frontal sutures.*

orbit may be parallel, or ballooned in the midportion by expanded ethmoidal air cells, or they may diverge laterally so that the dacryon is displaced outwards relative to the optic foramen; in other variants the optic foramina may themselves be abnormally separated. Mild hypertelorism exists in cases of Crouzon and Apert syndromes and a more severe variety in craniosynostosis syndromes associated with midline clefts (Cohen syndrome: David et al 1982).

In hypotelorism the medial orbital walls are closer together (Fig. 10.7a,b) and the interpupillary distance is smaller than normal. This is usually associated with trigonocephaly with premature fusion of metopic sutures.

b. Midface.

(i) Faciostenosis (Fig. 10.18). The chief functional disorder in faciostenosis is of course, constriction of the nasal airway. The defect of the midfacial skeleton is

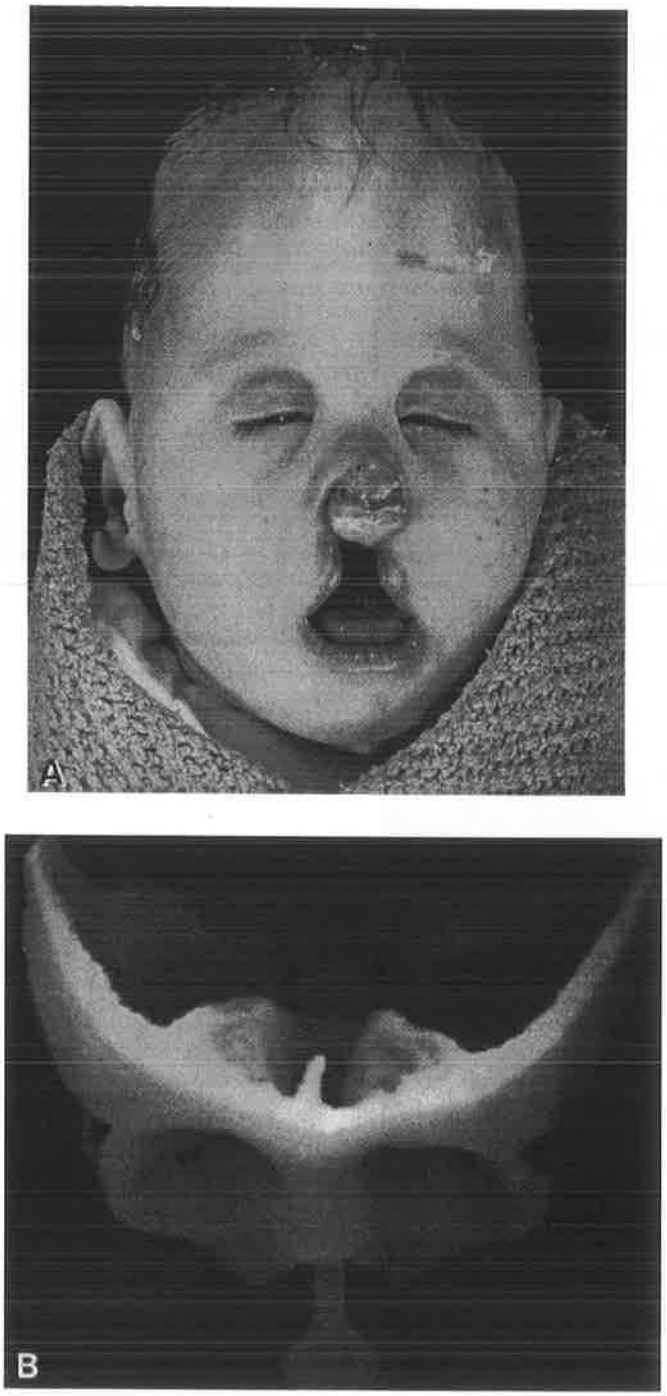


FIG. 10.17. A. *A child with an unclassified craniosynostosis syndrome with oxycephaly, hypotelorism and bilateral cleft lip and palate.* **B.** *3D reconstruction of the same child's skull showing the decreased distance between the orbits and the swept-up sphenoidal ridges producing a severe distortion of the anterior cranial fossa.*

three dimensional and this is very easily appreciated in three–dimensional reconstructions from computerized tomography. There is hypoplasia of antcroposterior maxillary growth; the maxilla therefore lies relatively far back, reducing the nasopharyngeal space. The maxilla is also transversely hypoplastic, resulting in a narrow, high-arched palate, and it is often reduced in vertical height, further reducing the capacity of the nasal cavity and the paranasal air sinuses. The situation may be taken to its extreme with posterior choanal atresia. Severe faciostenosis may threaten life from respiratory obstruction. It is probable that respiratory difficulties cause some otherwise unexplained deaths in infancy, especially in Apert syndrome, which is known to carry a high infant mortality rate.

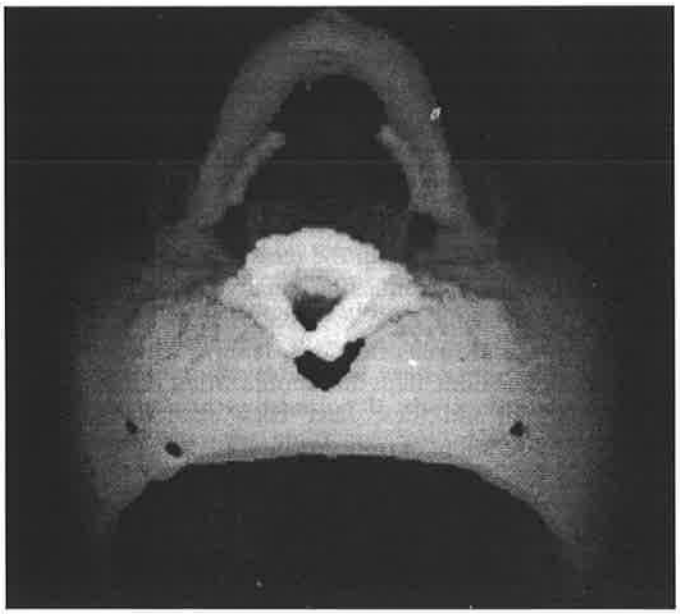


FIG. 10.18. *A basal view of 3D reconstruction of the skull of an infant with Apert syndrome demonstrating faciostenosis. The mandibular outline encloses a tiny maxilla, and there is virtually no identifiable upper airway.*

In our experience, many cases of complex craniosynostosis with faciostenosis show consistent common factors in disturbances of speech. The narrow, high–arched palate precludes accommodation of the otherwise normal tongue, especially if there is hypoplasia of the soft tissues of the palate, or a palatal cleft as in some cases of Apert syndrome. This is likely to cause abnormal tongue placement during speech. Crowding of the nasopharyngeal airway and varying degrees of nasal obstruction have been seen in almost every case of Crouzon syndrome, resulting in hyponasal speech. The hypoplasia or obstruction of the paranasal air sinuses may also reduce resonance. Dental malocclusion further distorts speech.

(ii) Maxillary hypoplasia. Just as cranial vault deformity can be present without craniostenosis, so maxillary hypoplasia can exist without severe disorders of visceral function. Cases of Cohen syndrome and less severe cases of Crouzon syndrome, and some cases of non-syndromal bicoronal synostosis, may exhibit obvious maxillary hypoplasia, but this is either not severe, or occurs only in one dimension, and does not cause upper airway obstruction. Traditionally maxillary hypoplasia is measured by cephalometric analysis and latterly these have been supplemented by biplanar cephalometric analysis and two– and three–dimensional CT scans. The traditional cephalometric analyses are difficult to apply in craniosynostosis problems, particularly in those associated with synostosis syndromes. There are obvious shortcomings in applying two–2 dimensional analyses to a three–dimensional problem; moreover, the reference points in the cranial base which are useful in the assessment of lower facial problems are themselves disturbed in cases of craniosynostosis.

(iii) Scoliosis (Fig. 10.15). Scoliosis resulting from craniosynostosis is most commonly associated with the skull base asymmetry seen in frontal plagiocephaly. Since the base of skull is the template for the face, the basal scoliosis may be reflected in orbital dystopia, asymmetrical positioning of the glenoid fossa, asymmetrical development of the mandible and asymmetry of the maxilla which is possibly secondary to the asymmetry of the mandible. The depression and asymmetry of the central part of the anterior cranial fossa may be reflected in the deviation of the vertical plate of the ethmoid and consequently, the nasal septal cartilage, giving rise to a deviated nose with asymmetry of the piriform fossa. Most cases of craniosynostosis producing facial scoliosis are secondary to skull base deformity. Frontal plagiocephaly often presents with a bulge on the contralateral occipital area and ear advancement on the side of the frontal retrusion reflecting the three–dimensional asymmetry of the face.

(iv) Malocclusion. In the well–known craniosynostosis syndromes of Crouzon and Apert, Saethre–Chotzen and Pfeiffer etc., the midface is usually hypoplastic; the palate is high arched and narrow and the occlusal distortion reflects this, with bilateral cross bite, crowding of teeth and usually class III malocclusion. Anterior open bite is usually found in Apert and Crouzon syndromes. The frontal plagiocephalies with three-dimensional twisting of the face programmed from the cranial base, produce asymmetry of the mandible and subsequent tilting of the occlusal plane.

The lower jaw is relatively prominent, but there is no true mandibular prognathism; it is more correct to say that there is maxillary micrognathia (Converse et al 1977). This may become all too obvious when the maxilla has been advanced by osteotomy. Since the maxillae are hypoplastic in all three dimensions, the palate is narrow and the upper teeth are crowded together. They may occlude on the lingual side of the lower teeth, constituting a bilateral crossbite. Especially in Apert syndrome, the maxillary complex may be tilted upwards, so that the molar teeth occlude, but not the incisors, resulting in an open bite deformity. These dental malocclusions are unsightly and often interfere with efficient mastication and speech.

Principles of Treatment

Treatment is based on a knowledge of the natural history of the disease against a background of an understanding of the pathology and pathogenesis. The decision to operate or not to operate is based on predictions for probability of craniostenosis, orbitostenosis and faciostenosis and on the aesthetic importance given to craniofacial deformity. The surgical relevance of maturity varies in different types of craniosynostosis and this can be important in choosing the time to operate. The growth curves of the brain, the eye and the facial viscera are not parallel and different parts of the cranium reach maturity at different times. Each condition, and indeed each patient, needs individual consideration. It has been helpful to consider the principles of treatment in three, somewhat arbitrary, epochs: the early, the intermediate and the late periods. The early period is the first 12 months of life, or even shorter time in certain types of deformity. The intermediate period extends up to 9 years. The late period concerns patients from 10 years or more (Fig. 10.5).

1. The early period

After the necessary investigations have been done, the surgeon ought to know whether there is any immediate danger to vision in the form of papilloedema or optic atrophy, or any significant X–ray evidence of craniostenosis. Papilloedema is an imperative indication of operation, but is a very rare finding in infancy, except in severe forms of Crouzon syndrome. A marked increase in the convolutional markings under 1 year is also uncommon. We believe this finding likewise justifies operation. Radiological evidence of very extensive sutural fusion is a more usual reason for urging operation. The likelihood of future significant craniostenosis seems to be roughly proportionate to the number of major calvarial sutures involved. If the sagittal, the coronal, the lambdoidal and squamosal sutures are all fused or fusing, then early surgery is imperative. This pattern of fusion may be associated with various types of skull deformity, including oxycephaly and clover-leaf skull deformity (triphyllocephaly). It is often seen as a part of Crouzon syndrome. Fusion of both coronal sutures seems to have much the same significance, especially as these cases sometimes show fusion of the sagittal suture in later months. When the craniosynostosis involves only one major suture, the argument for surgery is the anticipated cosmetic deformity. Isolated premature fusion of the metopic or sagittal sutures, of one coronal or one lambdoid suture is rarely complicated by craniostenosis and, although these forms of craniosynostosis are sometimes associated with mental retardation, there is no firm evidence to suggest that this can be prevented by operation. It has

been reported that single-suture craniosynostosis may be complicated by non-symptomatic elevation in intracranial pressure (Renier et al 1982); the significance of this finding is uncertain, but if there is doubt about the cause of developmental delay in association with craniosynostosis, then intracranial pressure manometry is advisable. The cosmetic appearance may be severely affected and surgery as early as possible will enable the relationship between the growing brain and the covering capsule to re-establish an acceptable cranial shape.

In infants with severe faciostenosis, as in Crouzon syndrome, or particularly Apert syndrome, early major cranial base release for craniosynostosis and fronto–orbital advancement have not in our experience significantly altered the faciostenotic problem and all of these patients have come to midface surgery at a later date. Similar results have been experienced by Marchac & Renier (1985). We believe that in severe craniosynostosis syndromes the facial deformities are primary elements in the disorder of growth, and cannot be expected to respond to frontal and cranial base surgery, even when performed in the first 3 months of life. There has been a recent move (Anderl et al 1983) towards performing surgery on the middle third of the face in infancy to correct faciostenosis, employing the similar concept that early release of the face will facilitate more normal growth. However, there does not appear to be any objective evidence to suggest that the facial sutures play a major role in the restriction of facial growth, or that there is a particular driving force that when unopposed will expand the middle third of the face to normal proportions. There does appear to be a rationale for selected midface advancement in the first epoch in those few cases where it becomes a life-saving necessity for upper airway obstruction. We do not believe that early surgery particularly hinders facial growth, but in our experience early advancement certainly does not maintain the cosmetic appearance. The important evidence to support its use at this early stage would be that further operations were prevented, or that the appearance was made so normal that there was no teasing in later childhood.

Operative procedures in the early phase

Operations in the early periods are based on the original thesis of Ingraham & Matson (1954), that the creation of artificial sutures would allow the growing brain to expand. We accept that craniosynostosis is a malformation of the whole cerebral capsule, dura mater as well as bone, but the empiric success of resections of bones suggests that dural constriction can usually be ignored, except in the most severe deformities.

The linear craniectomies originally recommended by Ingraham & Matson have evolved over the years into a number of standardised resections, some of which entail the elevation of free bone flaps and mobilisation of parts of the orbital margins and these have been expanded into the craniofacial operations of fronto-orbital advancement and the floating–forehead concept of Marchac (1978). While there is debate on how best to implement the concept of capsular release, the principle remains sound.

It is important to achieve complete release of the fronto-orbital complex with fronto–orbital advancement as early as possible during the phase of rapid brain and ocular growth. It seems logical that if the drive of the growing brain is to be used in correcting the balance between the constricting capsule on the growing brain, then struts across the excised coronal sutures, or other forms of fixation retarding the forward advance of the fronto–orbital complex, should be avoided. It is, however, possible to use forms of self–stabilizing osteotomies which fix the advanced fronto-orbital complex to the lateral orbital wall without bridging the newly created suture.

In cases of frontal plagiocephaly due to unicoronal synostosis, it has been found that unilateral orbital advancement is effective in correcting the fronto–orbital deformity and preventing the subsequent facial deformity, if the surgery

is performed radically enough and early enough. Such surgery, left later than 6 months of age, produces poorer results and it is in these later cases that Marchac's approach of bilateral surgery for unilateral disease may be applicable. In our experience, the maxillary hypoplasia often seen in craniofacial syndromes cannot be rectified by early surgical intervention. However, early correction of frontal plagiocephaly does minimize the facial scoliosis.

2. The intermediate period: from 1 to 9 years

In this period intervention may be needed for specific problems, for example craniostenosis, orbitostenosis, rarely faciostenosis and severe psychological trauma. If possible, one should not operate in this period. Cranial deformities are usually too well established to be corrected by simple procedures applicable in infancy, while major craniofacial reconstructions are often too dangerous or distressing, or likely to cause unpredictable disturbances in skeletal growth, especially in the nose and upper dentition. They may also require further major surgery. Nevertheless, there are circumstances in the intermediate period when operation may be justifiable, even imperative. In our philosophy, the younger the patient, the more the tendency should be for operation to be done only for craniostenosis and orbitostenosis; in older patients these operations may be combined with midfacial surgery. Craniostenosis may be present in this period with symptoms and signs of raised intracranial pressure. This may develop as a new symptom, or after an initially successful operation, as other sutures are fused or artificial sutures are closed.

Severe orbitostenosis may also force early action to save ocular function. Psychological stress may swing surgical judgement towards operation in the intermediate period, particularly after the child goes to school. Patients who present for elective cosmetic surgery in this period may have undergone operation in early life elsewhere and have been referred for correction of residual deformities. When these have been in the fronto-orbital region, corrective surgery has been undertaken without delay. When the deformity involves the middle third of the facial skeleton we have withheld operation, wishing to avoid facial osteotomy in this age period. There are practical reasons for waiting until the facial skeleton is nearing maturity: the eruption of the permanent premolar and canine teeth is a convenient, if arbitrary, dateline. After this, the teeth are sufficiently well developed to serve as secure braces for intermaxillary fixation. There are biological dangers in too early facial osteotomy. Operations could interfere with the residual growth potential in the nasomaxillary complex; more importantly, the benefits of early advancement of a hypoplastic maxilla may be lost as continued growth of the normal mandible re–establishes original disproportion between the upper and lower jaws. If operations on the face are done in the intermediate period, it may be necessary to operate again in later life and this fact must be explained carefully to patient and parents.

3. The late period: from the 10th year

From this time onwards, we can deploy the standard craniofacial techniques for the correction of facial deformity. Every technique that is available to the craniofacial team can be used. During this period, the patient's facial skeleton becomes more mature and radical operations can be undertaken to correct orbitostenosis and faciostenosis. Maxillary and mandibular osteotomies can be done and the mobilised bones can be put into better positions without much fear that corrections will be lost as growth progresses. Most experienced craniofacial surgeons have been satisfied with the permanency of facial corrections carried out after the age of 10 years. The operations likely to be necessary in this late period are often formidable. The least demanding are those intended to correct less severe forms of bilateral and unilateral maxillary hypoplasia, or the orbital dystopias associated with plagiocephaly. These deformities can be treated by onlay bone grafts and limited fronto–orbital advancements. More severe degrees of faciostenosis and orbitostenosis require radical correction along the lines

pioneered so brilliantly by Tessier and developed by many of our colleagues in subsequent years. The most major procedures may involve remodelling of the calvarium, frontoorbital complex, midface and indeed the mandible. These are the most difficult and most rewarding procedures now being done for craniosynostosis. They can give enormous benefits, in appearance and psychological well being. Complications are not rare and can be devastating. Nobody in this field can feel complacent; many striking successes have been achieved, but there is need for much greater safety and simplicity. Because these are very large undertakings, they need most careful pre–operative assessment and planning by an experienced team. It has been emphasized that craniofacial malformations are three dimensional in nature and the essential magic of Tessier's approach is the ability to move the craniofacial complex in three dimensions.

Operative principles in the late period

The fundamental principle is to do as much surgery as possible once the extensive exposure of the craniofacial skeleton has been made, so that faciostenosis and orbitostenosis can be corrected in one stage. It is necessary to advance the orbital walls and the entire maxillary complex and to secure this advancement with bone grafts. If there is also hypertelorism or orbital dystopia, the mobilized orbits can be brought together or adjusted vertically. In principle, access is given through a large bicoronal scalp flap with extensive subperiosteal dissection, exposing the frontal region, the nasal bones and the walls of the orbits, malar arches and anterior maxilla down as far as the alveolus. The orbital floor is either exposed through the bicoronal scalp flap, or through a subciliary, or conjunctival incision. The osteotomy is necessary to mobilize the orbital walls and the upper components of the maxillary complex are safeguarded by exposure of the anterior cranial fossa. This is done through a bifrontal bone flap, which is designed to accommodate any frontal remodelling necessary. Extradural dissection as far back as the crista galli and spheroidal ridges allows the neurosurgeon to protect the dura and brain as well as the orbital contents from saw cuts and other manipulations. It is rare these days to need a transoral approach to the maxillary complex, as all the maxillary osteotomies can usually be completed through the bicoronal approach. The necessity of opening into the nasal and paranasal cavities and connecting them with extradural space remains a significant problem even if extramucosal dissections are used as advised by Montaut & Stricker (1977) and others. The difficulties in maintaining asepsis are far greater than in simple cranial reconstructions. Many efforts have been made to close these gaps and indeed to design osteotomies to obliterate this dead space produced by advancement of the anterior cranial base (Anderl 1983).

External forms of craniomaxillary fixation have given way to self–stabilizing osteotomies and the use of mini plates. Rigid fixation of the osteotomies and bone grafts is necessary to minimize infection. Secondary soft tissue and bone surgery is frequently necessary to achieve the best results.

Multidisciplinary Team: Assessment and Management

The requirements of patients with craniofacial abnormalities are very complex and demand a multidisciplinary approach. Many body systems are affected, and every detail of patient management has to be given due attention. Care begins at birth and continues until the patient and his family have been relieved of the burden of the anomaly. A team is needed capable of delivering expert patient care, and representative of all the relevant disciplines. Data, in the form of histories, physical examinations and special investigations, is needed in planning treatment, and such data should be used to the maximum scientific effect to improve present methods of management, still far from satisfactory, and to expand knowledge of the biology of cranial growth and its disorders.

It remains as true today as when first stated by Tessier (1971) that craniofacial surgery should be carried out in regional units with a large referral base. Ideally, a craniofacial unit should be centred in a large teaching hospital with appropriate facilities. If the hospital caters for both adult and paediatric patients, so much the better. However, this is not often the case. All the disciplines involved in the craniofacial unit should be available in the hospital in which the unit is centralized. Such a situation is unobtainable in many centres. Where specialists from other hospitals have to be involved, then facilities should be made available for them to have easy access to the institution. Such a concept involves an organization which transcends the ordinary hospital staff system.

Patient follow–up

Adequate patient follow–up is necessary to monitor results and collect scientific data. As a regional centre, a craniofacial unit serving a large population needs to make appropriate arrangements for patient follow–up by establishing excellent relationships with referring doctors and by arranging for members of the craniofacial unit to visit more distant centres to review previously operated patients and to assess new patients for treatment.

Data collection and research

Data collection on patients during growth, before and after treatment, using standard cephalometry, three–dimensional cephalometry, photogrammetry and three-dimensional CT scanning, is necessary to investigate the many unanswered questions. Such data need to be stored so that they are easily retrievable and periodically subjected to analysis. Much of the information elicited from the initial patient assessment should be followed up. Psychometric testing sheds some light on the effects of physical release of the brain and relief of psychosocial pressures resulting from craniofacial deformity. Follow–up on untreated cases to establish the natural history of these deformities is also important (Barritt et al 1981).

Results and complications

The functional symptoms of craniosynostosis respond well to modern techniques of craniofacial surgery. Raised intracranial pressure has been relieved, orbitostenosis has responded to fronto-orbital advancement in the early epoch and full frontofacial advancement in the older age group. Symptoms of faciostenosis are much improved, the upper airway is less obstructed and dental occlusion is improved by facial advancement. The aesthetic results have shown consistent improvement as craniofacial surgeons and their teams have gathered experience and with the introduction of more sophisticated techniques, such as self–stabilizing osteotomies and some of the newer plating techniques for osteosynthesis. There is mounting evidence of permanent psychological improvement after correction of the deformities resulting from craniosynostosis.

Early surgery for frontal plagiocephaly, resulting from unicoronal synostosis, prevents secondary facial asymmetry. However, early surgery on the skull and anterior cranial base of severe craniofacial syndromes of Apert and Crouzon does not appear to affect the final degree of faciostenosis of these conditions in our experience.

The very innovative techniques which make possible spectacular successes of craniofacial surgery are also the causes of the occasional severe complication. The severest complication of death, brain damage, infection, visual loss and CSF leak have been reported from a number of major units performing this work throughout the world (Tessier 1976, Converse et al 1977, Whitaker et al 1979, David et al 1982). In general terms, it is our experience that very few complications occur in the simple craniosynostoses; most occur in the craniosynostosis syndromes of Crouzon and Apert when the full frontofacial advancements are performed. In this group infection is potentially a most dangerous complication to be feared when the dura is in contact with the open nose and paranasal sinuses.

Those patients most at risk are the older group who have had previous surgery (David & Cooter 1985) and even the most careful precautions and best possible surgical methods do not guarantee freedom from sepsis.

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Craniofacial Infection in 10 Years of Transcranial Surgery

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Infection following transcranial surgery may be devastating. A review of 170 transcranial operations is presented with a focus on postoperative infection and its relationship to patient age, preoperative microbiology, pattern of operation, length of operation, and the use of antibiotic prophylaxis.

The overall postoperative infection rate was 6.5 percent, but the infection rate in adults (23.5 percent) was much higher than in children (2.2 percent). Higher infection rates were found in adults with craniofacial dysostoses undergoing lengthy frontofacial advancements which required tracheostomy airway management. The residual frontal extradural dead space following advancement in adults is a sanctuary to infecting organisms from the respiratory tract — especially *Pseudomonas* transferred from the tracheostomy site into the upper airway and intracranial dead space by ventilation forces.

Operating times for patients who became infected were 2½ hours longer than average operating times for transcranial operations. Preoperative microbiology of the craniofacial region was not a good predictor of subsequent infection.

Recommendations include operative intervention at an early age, short preoperative hospital stay, antibiotic prophylaxis to include gram-negative cover, surgical measures to either fill or isolate the dead space, and strict tracheostomy care — preferably with the patient being barrier-nursed.

Postoperative wound infection is an important major complication of any surgery, but it may be particularly devastating following transcranial surgery. Complications of craniofacial surgery have been reported by Converse et al.,[1] Munro and Sabatier[2], Whitaker et al.,[3] Whitaker, Schut, and Randall,[4] Whitaker et al.,[5,6] Murray et al.,[7] and Tannieres et al.[8] Infection is frequently mentioned—especially following complex transcranial operations which combine neurosurgical intervention with manipulation of nonsterile regions.

The South Australian Cranio–Facial Unit, between 1975 and 1984, performed 170 transcranial procedures, but there were no postoperative infections until August of 1982, after the first 90 transcranial procedures.

The objectives of this retrospective study are: to record accurately the infection rate in transcranial surgery, to isolate factors that influenced infection, and to propose methods to reduce the rate or postoperative infection.

Terminology

Many definitions of infection are found in reports of postoperative wound sepsis. Most require the presence of pus or the isolation of microorganisms in conjunction with signs of inflammation (Barnes et al.,[9] Ljunqvist,[10] Evans and Pollock,[11] Meares,[12] Krukowski et al.[13]). However, infection in the craniofacial region may lack obvious inflammation, and the wide variety of local organisms in craniofacial cavities can make sepsis identification difficult.

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The definitions used in this study are as follows:

Infection: A process in the craniofacial region caused by microbiology-proven pathogens requiring change in postoperative management.

Transcranial operation: An operation requiring a combined neurosurgical/plastic surgeon approach to the craniofacial region via a craniotomy.

Historical Aspects

Prior to the midnineteenth century, most surgery was imperiled by infection. Dramatic progress was made only after the causes of infection and its prevention were discovered by Pasteur, Lister, and Koch—the fathers of bacteriology. Domagk and Florey are credited with the development of effective systemic treatment of infection.[14]

The risk of sepsis prevented significant advance in cranial surgery until the late nineteenth century. In citing the operative experience of the neurosurgeon Harvey Cushing, Wright[15] noted that "Cushing's disciples have all acknowledged his meticulous methods beginning with preparation of the scalp, careful operative technique and postoperative care of the wound, and herein may lie in part the reason for his low rate of operative infections." It was the seemingly impenetrable barrier of meticulous neurosurgical technique, espoused by Cushing, which Tessier had to incorporate into facial surgery to establish the discipline of craniofacial surgery. Tessier pioneered a combined intracranial/extracranial approach for the correction of major craniofacial abnormalities.

Transcranial surgery has provided complete surgical access to the anterior cranial fossa and orbits. However, the extensive subperiosteal stripping employed with this approach allows easy progression of any infective process, making eradication extremely difficult.[16]

Materials and Methods

In the period 1975 to 1984, a total of 170 transcranial procedures were performed by members of the South Australian Cranio–Facial Unit. An analysis of all patients is presented with a focus on postoperative infection and its relationship to preoperative microbiology, surgical factors, antibiotic prophylaxis, and postoperative management.

Information was retrieved from hospital case notes, operation records, microbiology records, and Cranio–Facial Unit files.

The age distribution of patients varied between 5 weeks and 58 years, with the majority of patients in the first 2–year age group (Fig. 1). The sample comprised 131 children and 33 adults, 90 males and 74 females (Table I). Adult operations were performed at the Royal Adelaide Hospital, and operations on children to 16 years of age were done at the Adelaide Children's Hospital. There were 170 operations on 164 patients; 6 patients had two transcranial procedures.

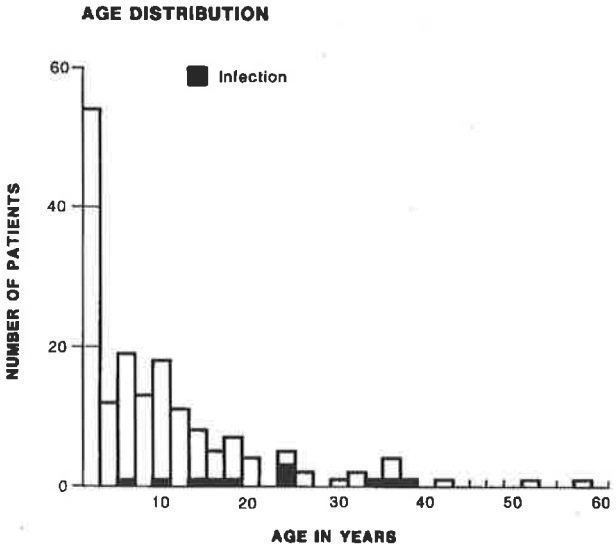

FIG. 1. *Age distribution of transcranial patients.*

TABLE I

Transcranial Operation Patients

Patients	Male	Female	Total
Children:			
Infection	2	1	3
No infection	68	60	128
TOTAL	**70**	**61**	**131**
Adults:			
Infection	3	5	8
No infection	17	8	25
TOTAL	**20**	**13**	**33**
All patients:			
Infection	5	6	11
No infection	85	68	153
TOTAL	**90**	**74**	**164**

The transcranial approach was used to correct deformities of a variety of problems (Table II). The craniosynostosis group was the largest, containing 85 (52 percent) of the 164 patients (Table III). The transcranial operations may be subdivided into five categories (Table IV), with "other" including procedures to repair deformities such as facial clefts, tumor masses, traumatic defects, and the facial anomalies of Treacher Collins and Romberg syndromes. All craniofacial patients had preoperative microbiology swabs of their scalp, conjunctivae, nose, and throat. Results of preoperative swabs were documented and, in patients with postoperative infection, compared with the organisms in subsequent infections.

In the 10 years reviewed, three different types of antibiotic prophylaxis had been employed (Fig. 2). All antibiotic doses were in the therapeutic range appropriate for the patient's age. In the first 5 years (from 1975 to 1979), a regimen of methicillin alternating with ampicillin was used for 72 hours postoperatively. From 1980 to 1982, combined flucloxacillin and ampicillin was given for 48 hours after the procedure. The prophylaxis in the final 2 years was flucloxacillin and metronidazole, the first dose given preoperatively and then continued for 48 hours

postoperatively. Metronidazole was included in 1982 after an anaerobic infection in a 9–year–old boy with Apert's syndrome. Twenty patients had antibiotic prophylaxis at variance with the recommended regimen, e.g., patients with penicillin allergy or those with cardiac lesions requiring extra cover. Six patients did not receive antibiotics.

TABLE II

164 Patients Undergoing Transcranial Surgery

Diagnosis	Children	Adults	Total
Craniosynostosis	70	15	85
Encephalocele	32	1	33
Hypertelorism (not encephalocele)	8	—	8
Orbital dystopia	7	—	7
Facial cleft	5	4	9
Treacher Collins syndrome	2	—	2
Tumor	3	3	6
Romberg	—	1	1
Arthrogryphosis multiplex congenita	—	1	1
Craniofacial fracture	2	7	9
Frontonasal dysplasia	2	1	3
TOTAL	**131**	**33**	**164**

Statistical Analysis

Although chi–squared testing was used for statistical evaluation, the observed number of infected patients was too small to allow reliable analysis.

TABLE III

Transcranial Operations on Patients with Craniosynostoses

	Children	Adults	Total
Crouzon's	18	9	27
Apert's	13	1	14
Saethre-Chotzen	4	—	4
Carpenter's	2	—	2
Pfeiffer's	—	1	1
Waardenberg's	2	—	2
Plagiocephaly	16	2	18
Turricephaly	11	1	12
Trigonocephaly	2	—	2
Unclassified	2	1	3
TOTAL	**70**	**15**	**85**

TABLE IV

Categories of Transcranial Operations

	Children	Adults	Total
Frontofacial advancement	14	11	25
Fronto-orbital advancement:			
Unilateral	21	4	25
Bilateral	31	2	33
Encephalocele removal	32	1	33
Hypertelorism/dystopia correction	25	8	33
Other	13	8	21
TOTAL	**131**	**34**	**170**

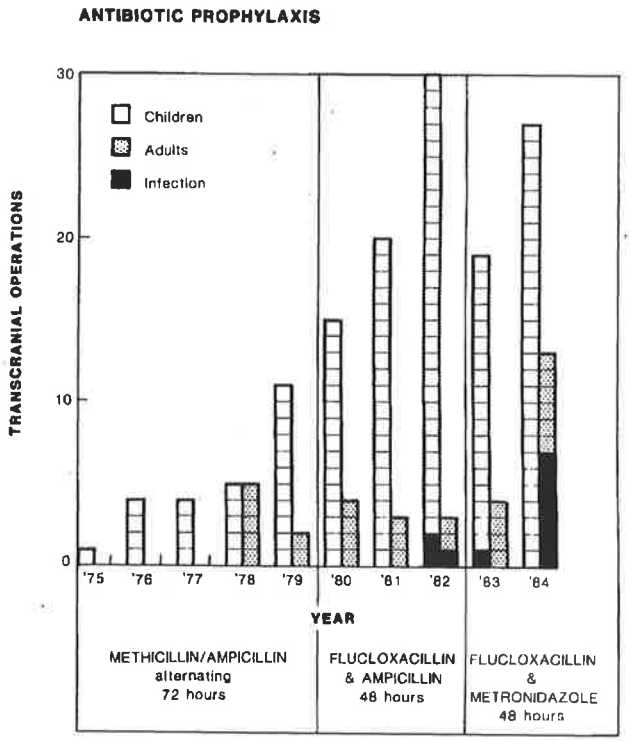

FIG. 2. *Antibiotic prophylaxis regimens.*

Results

All infections occurred after 1981. Of the patients undergoing transcranial surgery, 6.5 percent developed infection—representing an infection rate in children of 2.2 percent but an adult infection rate of 23.5 percent (Fig. 3).

There were 11 infections, and 4 of these were severe. A strong predilection for adults with craniofacial dysostoses undergoing frontofacial advancement was observed, with 8 of the 11 infections occurring in patients with either Crouzon's or Apert's syndrome (Table V). Five patients in the group of 11 sustaining infection had undergone previous transcranial surgery elsewhere; only 3 of the patients with infection had never had surgery to the craniofacial region (Table V). Only 3 children suffered infections, and all cleared with minor surgery and antibiotics. Of 9 adult infections, 4 were severe, with 3 requiring removal of infected frontal bone, but others settling with antibiotics (Table VI). Resolution ranged from 1 week for superficial infections to more than 3 months for severe, smoldering frontal bone infections (Table VI).

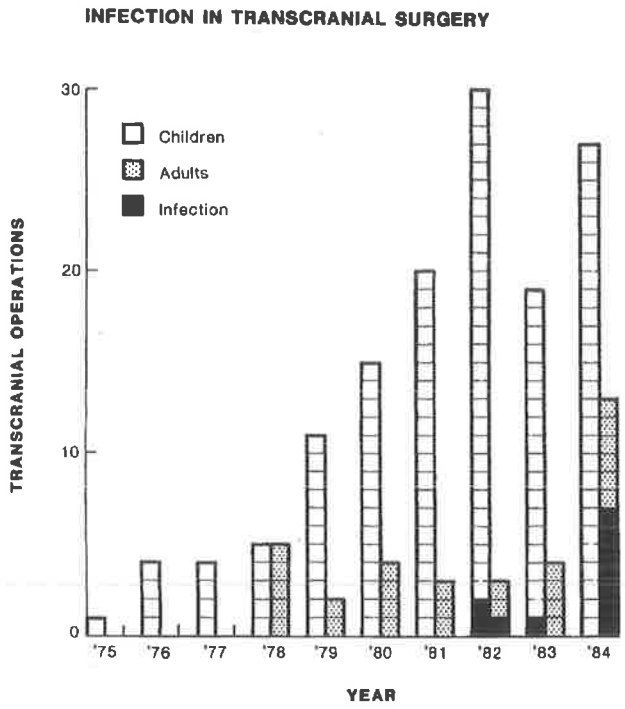

FIG. 3. *Infection in transcranial surgery.*

Five of the infected patients were male and six were female (Table I, with 39 percent of female adults developing infection. Unfortunately, the low number of infected female patients precluded reliable statistical analysis. Table VII, however, illustrates the potential contributors to this high infection trend in females. Of the 11 who developed infection, 4 were referred to the South Australian Cranio-Facial Unit from overseas and 6 presented from interstate. In such cases, lengthy preoperative hospitalisation was often necessary to complete extensive surgical planning.

In the majority of patients, infected wound cultures grew multiple pathogens (Table VI). Despite the polymicrobial nature of infections, only three patients had infecting organisms identifiable on preoperative microbiology. The prophylactic antibiotics flucloxacillin and metronidazole did not provide cover against organisms in four of the infections which developed within the first few postoperative days. These included *Streptococcus faecalis, Pseudomonas aeruginosa,* and *Klebsiella.*

The 170 transcranial procedures in our series involved a total of 1359 operating hours with an overall average of 8 hours per operation. The average operating time for patients who subsequently became infected was 101/2 hours (Fig. 4).

Airway management in 34 patients required tracheostomy—18 children and 16 adults. Of all adults, 47 percent needed a tracheostomy. Eight of the 11 patients with infection had tracheostomies, including the four with severe infection (Tables V and VI).

There were 53 accidental dural tears and 32 planned dural openings. There were 14 postoperative CSF leaks, including 12 in children and 2 in adults. Although both these adults required a further procedure to repair a dural defect, only one was associated with infection, this being a persistent purulent nasal discharge. There were no cases of meningitis.

There was one death in the 11 infected patients, but this resulted from a further operation to repair a tracheostomy site dehiscence. Intraoperative hemorrhage was the cause of death. No cases of blindness or brain damage were recorded in patients sustaining infection.

Discussion

Retrospective studies have their limitations as a means of investigating a clinical problem.[17–20] However, this review was greatly facilitated by rigid protocols formulated at the inception of the South Australian Cranio-Facial Unit in 1975[21] which provided standardised data for each patient.

Our infection rate following transcranial surgery was 6.5 percent. The worst complications reported in craniofacial surgery literature have followed the transcranial approach, and the most common complication is infection.[2,5] In a combined report of the problems and complications in 793 craniofacial operations, Whitaker et al.[5] reported an infection rate of 6.2 percent following 421 intracranial operations. In an analysis of 12 years of craniomaxillofacial surgery by Munro and Sabatier,[2] infection was found to be the greatest problem and occurred in 5.3 percent of operations involving intracranial exposure.

TABLE V

Eleven Patients with Craniofacial Infections Following Transcranial Surgery

No.	Age/Sex Diagnosis	Opration	Date	Length of Operation (hours)	Tracheostomy (days)	Dural Tear	CSF Leak	Surgery to Craniofacial Region Prior to SACFU Operation
1	24/female, Crouzon's	Frontofacial advance	9/8/84	8½	6	–	—	Maxillary advancement, bone graft to orbits, eyelid surgery
2	24/male, Crouzon's	Frontofacial advance	14/10/82	12	9	–	—	Craniectomy
3	23/female, Crouzon's	Frontofacial advance	19/4/84	13	13	+	—	Frontofacial advancement complicated by CSF Leak requiring surgical repair
4	16/male, clefts	Cleft correction	5/7/84	9	—	–	—	Nil
5	33/female, Crouzon's	Frontofacial advance	23/8/84	12	28	+	6 months	Craniectomy, orthognathic surgery, bone grafts to cheeks and nose fat graft to orbit
6	18/female, Crouzon's	Frontofacial advance	16/8/84	10	8	–	—	Craniectomy
7	38/male, Crouzon's	Frontofacial advance	15/11/84	9	8	–	—	Eyelid surgery, Silastic implant, cheek augmentation, removal of Silastic and replacement with iliac bone
8	35/female, Apert's	Frontofacial advance	1/3/84	14	140	+	—	Nil
9	9/male, Apert's	Frontofacial advance	10/8/82	11	8	–	—	Frontal reconstruction cleft palate repair
10	6/male, clefts	Cleft correction	19/10/82	10	—	–	—	Cleft lip repair, facial cleft repair
11	13/female, fibroma	Removal of fibroma	1/3/83	9	—	–	—	Nil

SACFU = South Australian Cranio-Facial unit.

Other authors have presented overall infection rates when reporting complications in craniofcial surgery without delineating infection rates for specific operative procedures.[1,7,8]

The wide surgical exposure of the craniofacial skeleton gained with transcranial surgery provides access to the upper face by means of the anterior cranial fossa, thus allowing considerable manipulation of bony structures. Such exposure requires wide subperiosteal stripping, with large portions of bone denuded of blood supply during the operation. Osteotomies may traverse paranasal sinuses which subsequently communicate with the intracranial cavity. Mobile bone is fixed with wire or metal plates. This postoperative environment promotes any infective process and makes eradication difficult.

In 1971, Davidson, Clark, and Smith[22] prpared a valuable computer analysis of postoperative wound infection with information obtained from 1000 patients and found five factors to be highly significant in determining the development of wound sepsis: (1) a potentially dirty procedure, (2) the presence of wound bacteria at the end of an operation, (3) the ward environment, (4) the patient's age, and (5) the duration of the operation. Davidson, Smith, and Smylie[17] emphasised the multifactorial nature of infection and the fact that different types of procedures are liable to contamination in varying ways. Cruse and Foord,[20] in a 10–year prospective study of over 62,000 wounds, cautioned that "all wounds are of necessity contaminated, but the key to the extent of contamination is the presence of foreign material and the amount of devitalized tissue."

With these factors in mind, a review of 170 transcranial procedures revealed a higher infection trend with longer operations and older patients. The average operating time for patients who became infected was 2 1/2 hours longer than the overall average for transcranial operations (Fig. 4). Adults had an infection rate of 23.5 percent, whereas in children only 2.2 percent developed infection.

Most centers have recorded reductions in infection rate as experience increased. Until 1982 we had no infections, and the majority of infections were in 1984—this directly correlated with an increase in adult frontofacial advancements in that year (Fig. 5). Of the eight patients developing infection following frontofacial advancement, seven had undergone previous surgery to the craniofacial region and five had had transcranial surgery prior to their operation at the South Australian Cranio-Facial Unit (Table V).

TABLE VI

Microbiology and Management of the 11 Infections

No.	Age/Sex, Diagnosis	Infection Site	Organisms	Management	Time to Develop	Infection Duration
1	24/female, Crouzon's	Nasal discharge	*Staphylococcus aureus;* group A nonhemolytic strepto–coccus	Antibiotics	4 weeks	2 weeks
2	24/male, Crouzon's	Right periorbital	*Staphylococcus epidermidis*	Antibiotics and aspiration	13 days	3 weeks
3	23/female, Crouzon's	Frontal bone	*Staphylococcus aureus; Streptococcus faecalis*	Antibiotics and frontal bone removal	2 days	>6 months
4	16/male, clefts	Paranasal abscess	*Klebsiella* spp.	Antibiotics	2 months	1 week
5	33/female, Crouzon's	Scalp wound extradural space	*Klebsiella* spp.; *Enterobacter* spp.; mixed coliforms	Antibiotics and dural repair	4 days	3 weeks
6	18/female, Crouzon's	Scalp wound	*Staphylococcus aureus;* group A nonhemolytic strepto–coccus	Antibiotics	4 weeks	1 week
7	38/male, Crouzon's	Frontal bone	*Pseudomonas aeruginosa*	Antibiotics and frontal bone removal	2 weeks	4 months
8	35/female, Apert's	Frontal bone	*Pseudomonas aeruginosa; Klebsiella* spp.; *Proteus* spp.	Antibiotics and frontal bone removal	3 days	>4 months
9	9/male, Apert's	Right periorbital	*Peptostreptococcus anaerobius; Fusobacterium nucleatum; Peptococcus pre votii*	Antibiotics	5 weeks	5 weeks
10	6/male, clefts	Nasal discharge	*Pseudomonas aeruginosa; Klebsiella* spp.	Antibiotics and bone graft removal	4 days	3 months
II	13/female, fibroma	Nasal discharge	*Staphylococcus aureus*	Antibiotics	14 days	8 days

TABLE VII

13 Adult Females Undergoing Transcranial Surgery

	Yes	No
Infection	5 (39%)	8
Tracheostomy	5*	8
Frontofacial advancement	7*	6
Operating time > 8 hours	8*	5
Operation in 1984	5*	8

*Includes the 5 patients who subsequently became infected.

The strong predilection for adults with craniofacial dysostoses undergoing frontofacial advancements also brought sharp focus onto the resultant intracranial dead space. Transcranial facial and fronto-orbital advancements leave a large frontal extradural space, usually in free communication with the paranasal air sinuses, which is a potential site of infection (Fig. 6). The space following frontal bone advancements in children is accommodated by the expanding brain[16,23] (Fig. 7), but in adults a large aerocele persists (Fig. 8).

Concern about this extradural dead space in adults has been expressed by many surgeons[1, 3, 21 24–26] Tessier[24] suggested that advancements be limited to 10 to 12 mm to avoid large intracranial dead spaces.

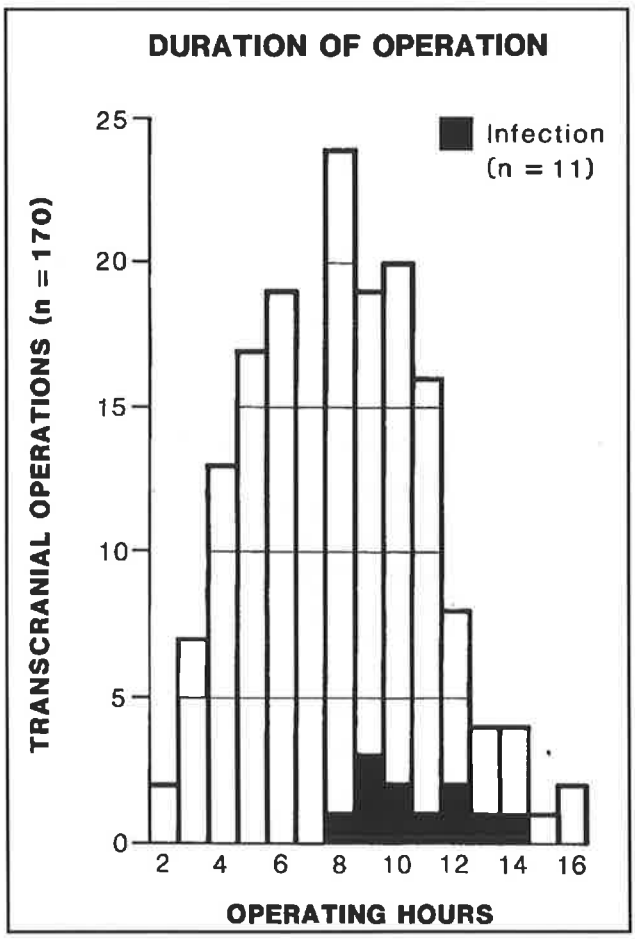

FIG. 4. *Duration of operation.*

Various methods to overcome any communication between the cranial cavity and the nasal cavity have been proposed: Tessier[27] described a two-stage procedure to advance the frontal bone and maxilla separately. Whitaker et al.[6] suggested submucoperiosteal dissection to maintain a barrier of nasal mucoperiosteum between these cavities. Anderl et al.[26] proposed a technique which preserves the complete anterior fossa while simultaneously correcting the

forehead, orbit, and face but maintains separation between cranial and nasal cavities.

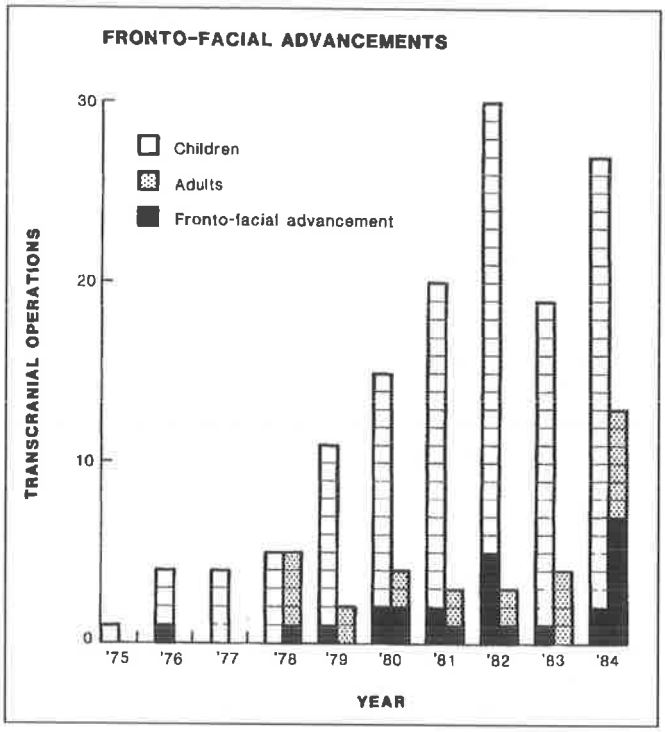

FIG. 5. *Frontofacial advancements.*

At the South Australian Cranio–Facial Unit, a latissimus dorsi free muscle flap has been successfully transferred into a large dead space remaining after transcranial resection of fibrous dysplasia extensively involving the cranial base. Without such a barrier between these cavities, any pathogenic organisms colonizing the respiratory mucosa have easy access to the operative site (Fig. 6). Ventilation may assist the ingress of organisms by forcing air from the respiratory passages into the cranial fossa[16,28] Furthermore, the respiratory tract of the patient with a tracheostomy often becomes infected with *Pseudomonas aeruginosa.*[29,30]

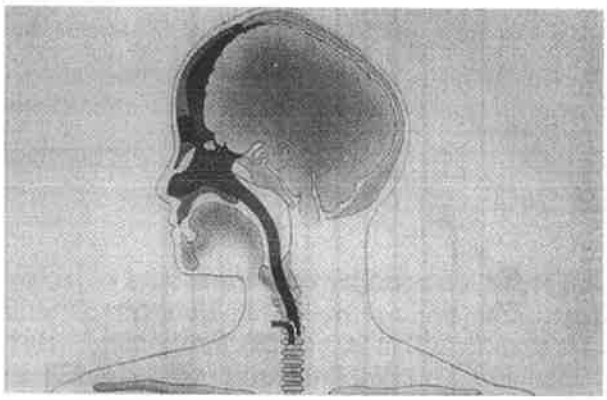

FIG. 6. *Communication between respiratory passages and the intracranial dead space allows easy access of organisms to the operative site.*

Of the four severe infections in our series, all had had tracheostomies and two developed frontal bone osteomyelitis from *Pseudomonas aeruginosa.* One such adult patient with Crouzon's syndrome (case 7 in Tables V and VI) had a frontofacial advancement for which a tracheostomy was required for airway management because nasotracheal incubation was not practical. The patient acquired a *Pseudomonas aeruginosa* crossinfection of his tracheostomy site (from a patient in the same general ward with an indwelling urinary catheter and *Pseudomonas* urinary tract infection). Just prior to removal of his tracheostomy, with the cuff deflated, throat clearing and nose blowing transferred the infection

into his fronto–orbital complex, resulting in osteomyelitis and the need to remove the frontal bone flap.

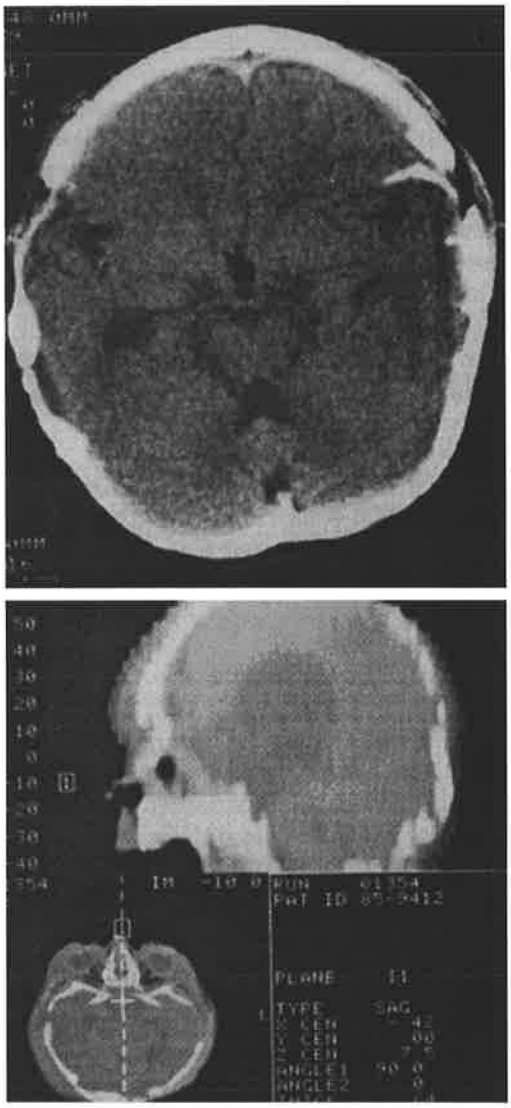

FIG. 7. (Above) *CT head scan of a young boy with Apert's syndrome 10 days after a frontofacial advancement; there is no dead space.* (Below) *CT head scan of a 5-month-old boy with Crouzon's syndrome on the day following a frontoorbital advancement; there is minimal residual space between frontal lobes and frontal bone.*

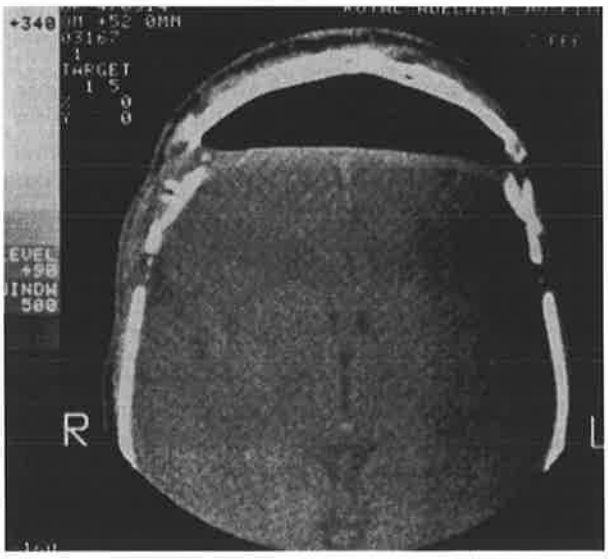

FIG. 8. *CT scan of a 40-year-old man with Crouzon's syndrome 3 months after a frontofacial advancement. There is a large aerocele between the frontal lobes and the frontal bone.*

Policy at the adult hospital has been to transfer patients back to the general ward with tracheostomies in situ. The patients at the children's hospital remain in the intensive care unit until the tracheostomy is removed.

Infection following transcranial surgery may manifest itself with a variety of signs and symptoms. Although most postoperative wound infections in other areas are usually apparent by the fourth or fifth day,[30] infection in the craniofacial region may present without obvious early signs or may have an obscure presentations.[33]

It was anticipated that preoperative microbiology swabs of the patient's scalp, eyes, nose, and throat would provide adequate information for antibiotic choice. However, infections were mainly polymicrobial, and only three patients had infecting organisms identifiable on preoperative microbiology. Becker[34] reviewed 57 patients in a prospective, randomised, double-blind study to establish the value of perioperative cultures and assess antibiotic prophylaxis for surgery of the head and neck. He concluded that a patient at high risk of wound infection could not be defined on the basis of perioperative bacteriology. Although our results support this, preoperative swabs from the craniofacial region may sometimes reveal pathogens, particularly in the nose, which allow eradication prior to surgery.

Prolonged preoperative hospitalisation increases the risk of colonization of hospital bacteria.[20,35] Of the 11 infected patients, 4 were referred to the South Australian Cranio–Facial Unit from overseas and 6 presented from interstate. In such cases, lengthy preoperative stays are necessary to complete extensive preoperative planning.

Chodak and Plaut[19] reviewed 16 years of literature on the subject of prophylaxis with systemic antibiotics in surgery and suggested that antibiotic prophylaxis should be employed only after rigorous trials have supported its efficacy. However, as in craniofacial surgery, there are many surgical procedures where the value of antibiotic prophylaxis has not been adequately studied.[36] Although the use of antibiotic prophylaxis may be controversial, in prolonged transcranial surgery where there is potential contamination and the consequences of infection are dangerous, prophylactic antibiotics seem justified.

Three different antibiotic regimens have been used throughout the 10–year period of this review. The current protocol is flucloxacillin, metronidazole, and tobramycin, with the first dose given prior to operation and then continued for 24 hours postoperatively. The aminoglycoside tobramycin was recently added to provide cover against *Pseudomonas*. All three are given at therapeutic doses intravenously for the first dose, and metronidazole may be given rectally for the subsequent doses with good effect.[37-39]

Dural tears and CSF leaks have long been implicated as strong associates of infection. In conditions requiring transcranial surgery, the dura may be abnormally thin and friable[40] and is easily torn because of scars from earlier surgery or because of abnormal bony contours in the anterior cranial fossa with dural adhesions.[3,21] All dural tears were meticulously repaired, the preferred method being to insert a generous graft of lyophilized dura, temporal fascia, or pericranium into the subdural space and suture the margins of the tear to this graft.[21]

Some clinicians advise against antibiotic prophylaxis to prevent meningitis in patients with a CSF leak for fear of selection of resistant organisms,[41] but following transcranial surgery, CSF rhinorrhea is recognized as a dangerous complication with a high risk of meningitis.[5] The policy of the South Australian Cranio-Facial Unit is to give trimethoprim and sulphamethoxazole (Bactrim or Septrim), since this safe combination achieves adequate CSF levels.[42] We have had no cases of meningitis following transcranial surgery.

Conclusions and Recommendations

1. Early operative treatment is preferred. Following frontal advancement, the child's expanding brain accommodates the extradural dead space with less risk of deep–seated postoperative infection.

2. Preoperative hospital stay should be as short as possible to reduce the risk of colonization of hospital bacteria.

3. Preoperative microbiology swabs will not accurately predict postoperative wound infection organisms, but they are helpful to eradicate potential pathogens preoperatively.

4. Antibiotic prophylaxis for transcranial surgery should provide cover against gram–negative organisms; current policy at the South Australian Cranio–Facial Unit is tobramycin, flucloxacillin, and metronidazole, first dose prior to operation and continued for 24 hours postoperatively. Current dosages are:

> Tobraymcin: Children (over 3 months, normal renal function): 2.5 mg/kg every 8 hours; adults (normal renal function): 80 mg every 8 hours.
>
> Flucloxacillin: Children (3 months to 12 years): 12.5 mg/kg every 6 hours; adults: 500 mg every 6 hours.
>
> Metronidazole: Children (3 months to 12 years): 7.5 mg/kg every 8 hours; adults: 500 mg every 8 hours.

5. In adults undergoing frontofacial advancements, surgical measures should be employed to prevent any communication between the respiratory passages and the intracranial dead space, either by providing a barrier or by filling the space.

6. Strict tracheostomy care is imperative, and postoperative transcranial surgical patients with a tracheostomy tube in situ should be barrier nursed in a single-bed room.

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Hydrocephalus in Crouzon's syndrome

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Abstract

We reviewed 42 cases of Crouzon's syndrome. There were 16 cases with ventricular dilation. We believe that shunt should be inserted after fronto-orbital advancement if there are persistent signs of raised intracranial pressure. However, in cases presenting with severe ventricular dilation and papilloedema. a shunt is inserted prior to fronto-orbital advancement. Medium- or high-pressure systems should be used.

Key words: Crouzon's syndrome — Hydrocephalus — Shunt — Fronto-orbital advance — Intracranial pressure.

In 1912. Crouzon described a syndrome that consisted of a triad: calvarial deformity, facial deformity, and exophthalmos. He emphasised the typical skull deformity as brachycephalic, although other head shapes are seen. Other features are: prominent nose, prognathism, and maxillary hypoplasia. Couzon's syndrome is the most common craniofacial syndrome; it is inherited as dominant and of high penetrance.

We reviewed the cases of Crouzon s syndrome seen in the Cranio-Maxillo-Facial Unit at the Adelaide Children's Hospital, South Australia. We studied all the cases examined by Computerised Cranial Tomography. Our findings are discussed.

Hydrocephalus is common with Apert's syndrome; however it is not described as a usual finding in Crouzon's syndrome. We raise the following questions:

1. Is ventricular dilation common in Crouzon's syndrome?

2. What are the indications for shunting, and when should it be done?

In 1971, Fishman[2] from the Massachusetts General Hospital examined their cases of cranial synostosis and found 14 cases with hydrocephalus: 8 were Apert's syndrome, and only 1 case was described as Crouzon's syndrome.

In 1985, Noetzel[4] from the St. Louis Children's Hospital in Missouri designed a prospective study on the incidence of hydrocephalus and craniosynostosis. Fifty patients were examined by computerised cranial tomography; 77 children with simple craniosynostosis showed no signs of ventricular dilation. In complex craniofacial deformity, computed tomography in 5 children showed ventricular dilation. These were cases of Apert's syndrome and triphyllocephaly *(Kleeblattschädel)*.

In 1987, Mahin Golabi[3] from the University of California (San Francisco), stated that hydrocephalus in craniosynostosis is a rare occurrence. He reviewed 750 cases of craniosynostosis, 10 of which were treated by shunting. Of these 10 cases 8 were Crouzon's syndrome, and 5 showed triphyllocephaly. He described

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the incidence of hydrocephalus in craniosynostosis as 4%. He stated that surgical correction of craniosynostosis would allow for ventricular enlargement, as fused sutures would control the size of the ventricle.

Materials and methods; Results

Forty-six cases of Crouzon's syndrome were treated at the South Australia Cranio-Maxillo-Facial Unit between 1975 and 1987. Of these, 42 cases were examined by computerised axial tomography. Sixteen cases were found with various degrees of ventricular dilation: 11 showed marked dilation, 4 showed moderate dilation, and 1 was described as having mild ventricular dilation. Five cases with marked ventricular dilation were shunted: 2 were shunted before fronto–orbital advancement and 3 after fronto–orbital advance. The two cases shunted prior to fronto–orbital advancement were done in other centres before referral to us. The other 3 cases were shunted after fronto–orbital advancement, as there were signs of persistent, raised intracranial pressure.

Neuropsychological assessments were performed in all the cases. Of the 11 cases with marked ventricular dilation, 9 were of normal intellectual ability and 2 were retarded. Of the 16 cases with varying degrees of ventricular dilation, 13 had negative family histories, while in 3 the family history was positive; 2 out of these 3 were twins.

In summary, 42 cases of Crouzon's syndrome were treated, and 16 showed ventricular dilation; however, only 11 cases showed marked dilation or 26%. The overall estimate of ventricular abnormality was 16 out of 42, which represents 38%.

Discussion

The mechanism of ventricular dilation in Crouzon's syndrome is not clear, and there are various theories. It could be caused by constriction of the subarachnoid spaces by the premature fusion of the sutures[1], or it could be due to obstruction of venous drainage from venous sinuses, as suggested by Renier et al.[5] It was suggested that hydrocephalus is a direct result of impaired CSF flow through the basal cisterns, while Noetzel[4] suggested that hydrocephalus occurs secondary to intrinsic abnormality in the embryology of the brain related to the defective formation of the cranium. This is particularly interesting, as in many cases the hydrocephalus is non-progressive.

Significant ventricular dilation was found in 26% of our cases with Crouzon's syndrome. This is higher than that reported in the literature. We must differentiate between cases with ventricular dilation but no raised intracranial pressure and those presenting with raised intracranial pressure and ventricular dilation. In many of our cases ventricular dilation was asymptomatic and did not necessitate any treatment. Fronto-orbital advancement would increase the cranial volume, and in many cases this was all that was needed.

In cases with signs of increased intracranial pressure such as papilloedema, a shunt should be inserted. The timing of the shunt insertion is important. We know that brain growth contributes to the expansion of the skull: if the shunt is inserted before fronto-orbital advancement, the brain drive which helps to promote skull growth would be absent. Fronto-orbital advancement would increase the cranial volume and assist in reduction of the intracranial pressure. It is not clear whether this would cause further ventricular dilation or, as mentioned by Noetzel[4], the extra space would be taken up by parenchymal expansion.

We believe that in cases with ventricular dilation, unless there is clear evidence of significantly raised intracranial pressure, namely, disc swelling,

cortical thickness of less than 10 mm, or progressive increase in ventricular size, fronto–orbital advancement should be performed first. Following that, the ventricular dilation and intracranial pressure should be monitored for any significant change. The ventricular dilation should be assessed using cranial ultra–sound or CT scan. If the ventricular dilation is progressive and there are persistent signs of raised intracranial pressure, a shunt should be inserted. If the ventricular dilation is not progressive and there are no signs of persistent, raised intracranial pressure, we suggest that no shunt procedure should be carried out. The presence of brain drive helps to achieve better skull contour and moulding.

If there are signs of significantly raised intracranial pressure at the time of presentation, then a shunt procedure should be performed before fronto–orbital advancement. If shunting becomes necessary, high– or medium–pressure systems should preferably be used.

Conclusions

1. Significant ventricular abnormality was seen in 26% of our cases.

2. Ventricular dilation alone does not indicate raised intracranial pressure, and hydrocephalus is often non–progressive. In these cases a shunt procedure is not indicated.

3. Shunt procedures, if indicated, are done after the fronto–orbital advancement, unless the patient presents with significantly raised intracranial pressure; then a shunt is inserted before fronto–orbital advancement.

4. A high– or medium–pressure shunt should be used.

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Surgical Correction of Crouzon Syndrome

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This study analyses the results of surgical treatment in 39 patients with the Crouzon syndrome. Early fronto–orbital advancement and craniectomy were universally successful in relieving raised intracranial pressure and in reducing ocular proptosis. However, definitive cosmetic correction was not achieved, and early cranial surgery was not able to prevent the development of midface hypoplasia.

Thirty-two midfacial advancements have been performed in 30 patients. Sixteen patients had sufficient follow–up data for more than 2 years postoperatively. In all patients, a satisfactory early postoperative result was achieved. In the long–term follow–up group, 11 patients have maintained a satisfactory appearance, while 5 have developed recurrent deformity. Analysis shows this to be associated with a younger age at operation and continued mandibular growth. Frontofacial advancement in adults achieves good long–term results but is associated with a higher incidence of complications.

In 1912, Crouzon described a familial syndrome comprising the triad of calvarial deformity, facial deformity, and exophthalmos.[1] The calvarial deformity results from premature craniosynostosis and is typically brachycephalic, although mesocephalic and scaphocephalic head shapes also may be seen. The facial deformity is characterised by maxillary hypoplasia with relative mandibular prognathism. Exophthalmos is invariably present and may endanger sight.

Craniosynostosis may be accompanied by headaches and failing vision, symptoms attributable to the presence of raised intracranial pressure and presumptive evidence for craniostenosis. Orbital hypoplasia results in a reduction in orbital volume, and this has been termed *orbitostenosis.* The major determinant of exophthalmos, however, is hypoplasia of the orbital floor, a condition attributable to hypoplasia of the maxillae and zygomata. The term *faciostenosis* describes states of midfacial hypoplasia where there are disorders of visceral function, notably airway restriction, speech problems, and dental malocclusion.

In all but the mildest forms of Cronzon's syndrome craniostenosis is treated by early calvarial decompression. Since there is hypoplasia of the anterior cranial fossa, fronto–orbital advancement is performed in addition to coronal and subtemporal craniectomy. This enlarges the orbital roofs and aids control of exophthalmos; aesthetic improvement of the upper face is also achieved. Parasagittal and lambdoid craniectomies performed 2 to 3 weeks later complete the decompression. Rapid expansion of the brain within the first year of life facilitates molding of the mobilized skeleton.[2] However, whether this provides enough remodelling for definitive cosmetic correction remains unproven. Furthermore, McCarthy et al.[3] postulated that early osteotomy of the anterior cranial base will permit normal midface development, but this has not been realised on long-term follow–up.[4]

Osteotomy and advancement of the midface form the basis for treatment of orbitostenosis and faciostenosis in patients with Crouzon syndrome. This was first reported by Gillies and Harrison in 1950.[5] In their patient, the osteotomy lines passed along the inferior orbital margin and anterior lacrimal crest, resulting in only a modest increase in orbital volume. In 1967, Tessier et al.[6] described an operation in which the osteotomies were made posterior to the lacrimal crest, across the orbital floor, and into the lateral orbital wall, i.e., complete craniofacial

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dysjunction. This resulted in an increase in orbital volume and control of exophthalmos. Simultaneous advancement of the frontal bones, orbit, and midface was subsequently reported by Tessier in 1971[7] and later modified by several authors.[8–11]

Although the therapeutic efficacy of midfacial advancement is firmly established, the timing of operation and the effects of further growth remain controversial. Early postulates that early Le Fort III advancement might lead to better midfacial developments have recently been questioned.[12] Indeed, as early as 1974, Hogeman and Willmar[13] expressed the view that early facial advancement will improve the appearance for a time but that the underlying dysplasia will continue to affect facial growth with an inevitable recurrence of the deformity. By contrast, several reports attest to the long–term durability of midfacial advancement in adults.[14–16]

For several years, the Australian Cranio-Facial Unit has adopted a concept of three age-related epochs that we have found useful when considering the timing of craniofacial operations.[17] The early period comprises the first 12 months of life, during which time operations are undertaken to release skeletal restraints on the growing brain and eyeball. During the intermediate period (1 to 9 years), operations are designed to control symptoms of craniostenosis or orbitostenosis and occasionally to relieve psychosocial stress. However, airway obstruction from severe faciostenosis may necessitate urgent midfacial surgery. The late period is from 10 years onward, and it is during this time that established deformities are corrected.

Since 1975, we have managed 47 patients with Crouzon syndrome, 39 of whom have been treated surgically by fronto–orbital and/or midfacial advancement. This represents a unique opportunity to study a comparatively large number of patients with a single disease entity. This paper details the early results, complications, and long-term follow-up of surgery in these patients and analyses the effects of age and further growth on the ultimate surgical result. Excluded from this study are eight patients with mild forms of Crouzon's syndrome, in whom symptoms, at present, have not necessitated surgical treatment. This latter group is under regular review, and it is anticipated that future surgical intervention will be required either to control developing symptoms or to definitively correct established deformities when growth has been completed.

Surgical Management

Multidisciplinary preoperative workup was performed at the Adelaide Children's Hospital by members of the Australian Craniofacial Unit and hospital staff. The surgical treatment was planned using information gained from clinical examination, cephalometric radiographs, and dental models. More recently, three-dimensional CT scanning has been used to precisely define the pathologic anatomy and to permit more specific operative planning. The relationship of the orbital margins to the cornea determines the degree of midfacial and forehead advancement. Preoperative planning predicts the resultant occlusion and forecasts the need for simultaneous Le Fort I or mandibular osteotomies.

The surgical techniques of fronto–orbital advancement, subcranial Le Fort III osteotomy, and frontofacial advancement have been described elsewhere.[17]

Materials and Methods

For purposes of analysis, patients were divided into two operative categories: (1) fronto–orbital advancement with or without decompressive craniectomy and (2) midfacial advancement (fronto-facial advancement or subcranial Le Fort III advancement).

Fronto–Orbital Advancement

Early–Period Surgery. There were six patients in this group (two males and four females), with a mean age at operation of 0.4 years (range 0.3 to 0.8 years). All patients demonstrated radiologic evidence of raised intracranial pressure and/or papilledema (craniostenosis), as well as proptosis of the globes (orbitostenosis). All underwent posterior craniectomy in addition to fronto-orbital advancement.

Intermediate-Period Surgery. There were six patients in this group (two males and four females), with a mean age at operation of 3.9 years (range 1.8 to 5.9 years). Two patients had undergone previous linear craniectomy. Preoperative papilledema was present in four patients (craniostenosis), corneal exposure in one patient (orbitostenosis), and both papilledema and corneal exposure in one other. Five patients underwent fronto-orbital advancement with coronal and bitemporal craniectomy, while in one patient parasagittal and lambdoid craniectomies also were performed.

Midfacial Advancement

Thirty-two midfacial advancements have been performed in 30 patients. There were 13 males and 17 females, with a mean age at operation of 16.6 years (range 8.3 to 38.8 years). Twenty patients had undergone prior surgery, mainly linear craniectomies for craniostenosis. However, fronto–orbital advancement had been performed in three patients, Le Fort I maxillary osteotomy in two patients, and bilateral subcondylar mandibular osteotomy in one patient.

Maxillary hypoplasia with class III malocclusion was a constant preoperative finding. All patients had varying degrees of proptosis, and in two patients, prior dislocation of the globe had occurred. In seven patients the nasal airway was sufficiently compromised to demand obligatory mouth breathing. Obstructive sleep apnea had been documented in one patient, and another had required emergency splitting of the soft palate as a young child.

Twenty–four frontofacial advancements were performed. In two patients bilateral sagittal split osteotomies with mandibular advancement and in one patient simultaneous Le Fort I maxillary osteotomy were required to achieve satisfactory occlusion. Subcranial Le Fort III maxillary osteotomy and advancement was performed in eight patients.

Sixteen patients had sufficient follow-up data available for more than 2 years postoperatively. Assessment of these patients has been by clinical and cephalometric examination.

Cephalometric Method. Lateral cephalometric x–rays were obtained preoperatively, during the early postoperative period, and at the most recent follow–up. These were taken according to a standard technique, and the landmarks were traced onto acetate film.

Midfacial movements were described in terms of x and y coordinates using the anterior nasal spine as the maxillary reference point. The x axis consisted of a line making an angle of 130 degrees to the sella-basion line, with the y axis perpendicular to this line through the origin at the sella. The axes approximated true horizontal and vertical and, by relating these to the posterior cranial base, were little affected by surgery or by subsequent growth. Horizontal maxillary length was measured between the anterior and posterior nasal spines, while mandibular length was measured between articulare and gnathion (Fig 1).

The cephalometric landmarks were digitised using a Hewlett–Packard digitiser and entered onto an Apple computer. The program CEPHS.RECORDER, developed at the University of Adelaide, was used to record and process the data.

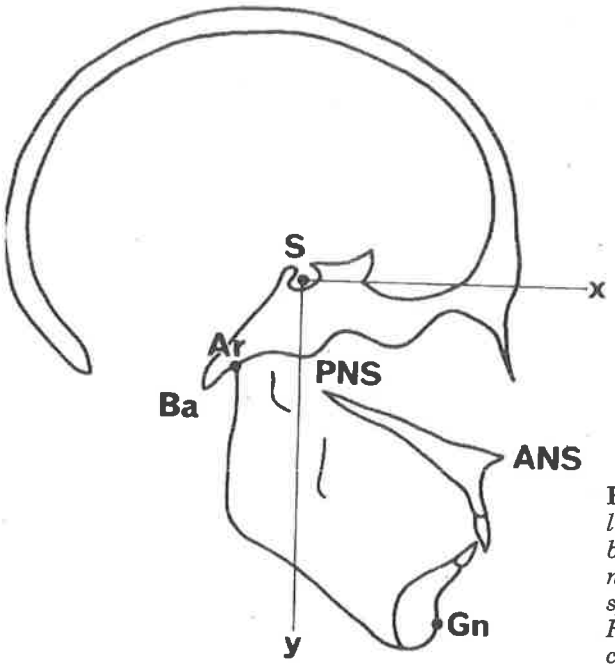

FIG. 1. *The cephalometric landmarks used in this study* (Ba, *basion;* S. *sella;* ANS, *anterior nasal spine;* PNS, *posterior nasal spine;* Ar, *articulare;* On, *gnathion). For a full explanation of the cephalometric method, refer to text.*

Statistical Method. The Mann–Whitney ranksum test was used to compare the differences in patients' ages, whereas the unpaired *t* test was used for all other comparisons.

Results

Fronto–Orbital Advancement

Early–Period Surgery. There were no significant complications related to the surgery. Two patients have died (one from pneumonia at 18 months postoperatively and one from airway obstruction secondary to choanal atresia at 6 months postoperatively), and one Malaysian patient has been lost to follow-up. The remaining three patients have been followed for between 2 and 3 years. No patient has evidence of recurrent raised intracranial pressure or problems with corneal exposure. Considerable calvarial remodelling has been achieved. However, all remain brachycephalic and show some recession of the frontal bone and supraorbital margins relative to the globe. Maxillary hypoplasia is present in all patients (Fig. 2).

Intermediate-Period Surgery. There were no complications. In all patients the symptoms of craniostenosis and orbitostenosis have been controlled. However, three patients required further fronto–orbital advancement (for cosmetic indications) at the time of subsequent midfacial advancement (Fig. 3). The remaining patients have been followed for 1 to 2 years; of these, two possess a satisfactory calvarial and upper facial appearance and one has residual proptosis.

Midfacial Advancement

Early Results. In all patients, the immediate postoperative results were considered satisfactory, with a reduction in proptosis and class I occlusion and an acceptable aesthetic result being achieved.

Complications. There were no deaths. The most devastating complication was blindness. This occurred bilaterally in one patient as a result of an extradural haematoma. The patient had unsuspected clotting abnormalities, and following detachment of the dura from the floor of the anterior cranial fossa, the expanding clot was able to exert traction on the optic nerves without an accompanying change in the conscious state. Unilateral blindness due to undetected intraorbital optic nerve compression occurred in one other patient.

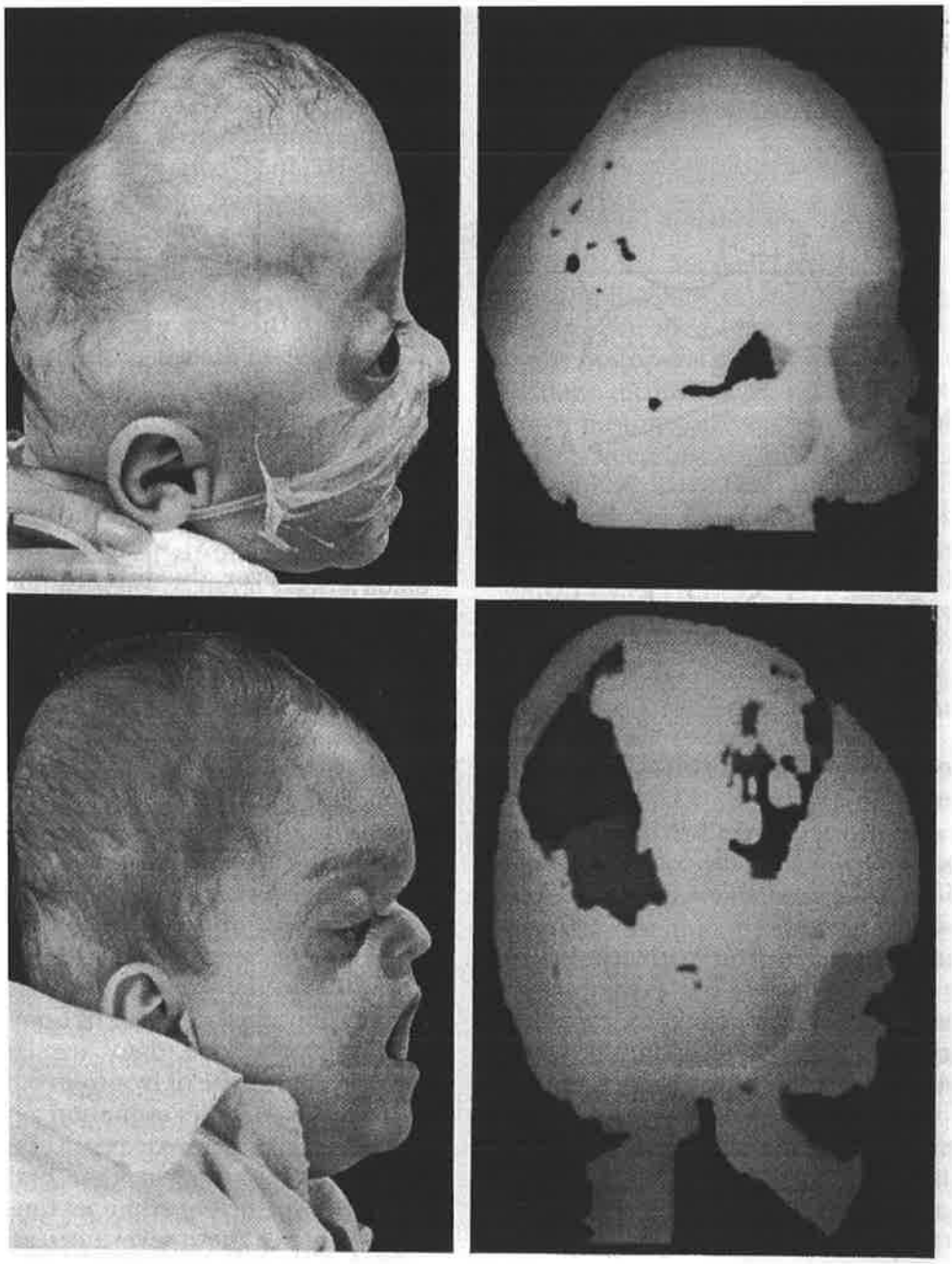

FIG. 2. (Above, left) A *4-month-old boy with Crouzon syndrome showing a triphyllocephalic calvaria and ocular proptosis.* (Above, right) *One year following fronto-orbital advancement and posterior decompressive craniectomy there has been extensive calvarial remodelling. However, the boy remains brachycephalic, and there is still some proptosis. Midfacial hypoplasia is becoming evident.*

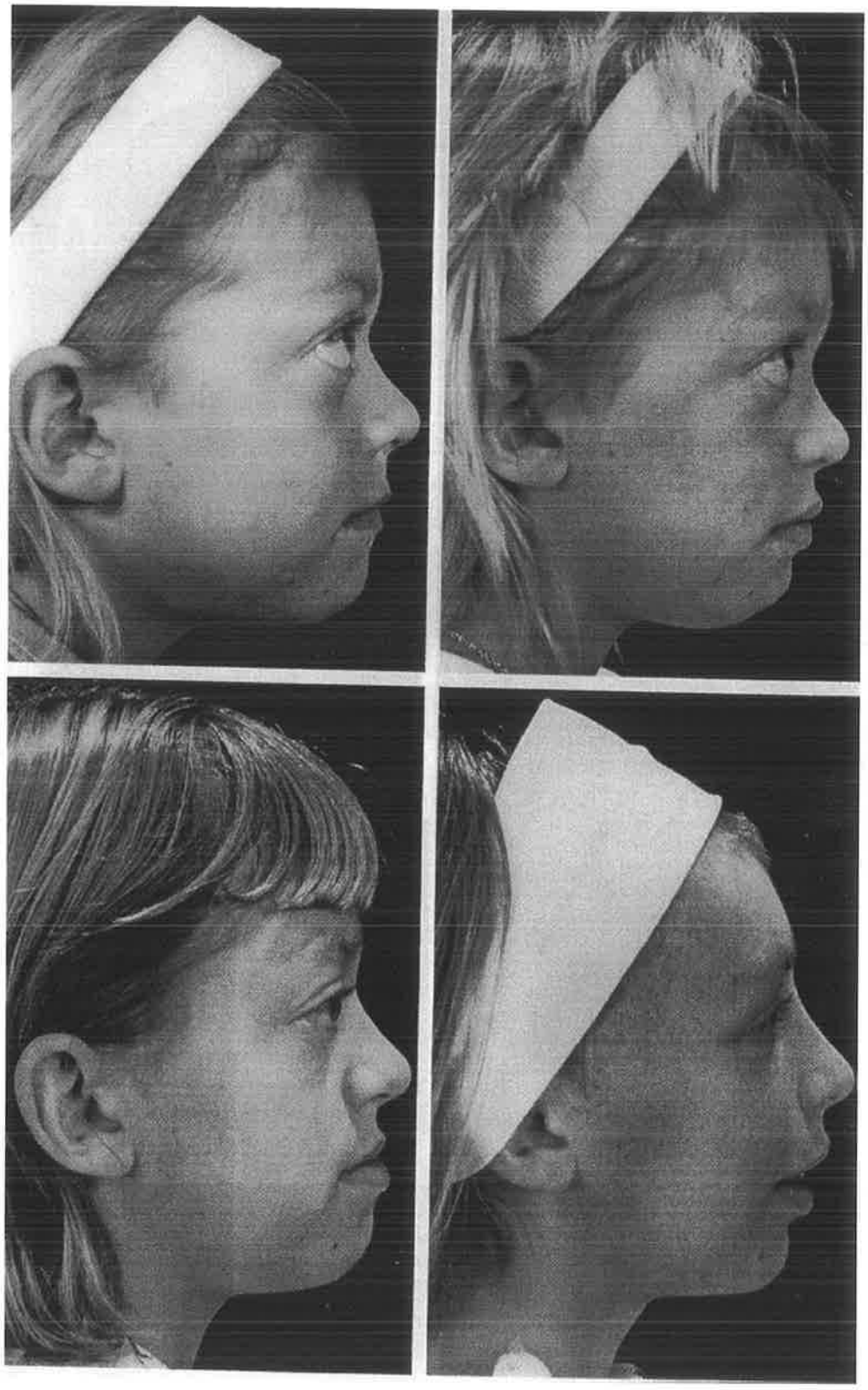

FIG. 3. (Above, left) *A 4-year-old girl with Crouzon syndrome with papilledema and mild ocular proptosis.* (above, right) *The appearance 1 year following fronto-orbital advancement and coronal and bitemporal craniectomy. Raised intracranial pressure has been controlled.* (Below, left) *At the age of 11 years, the patient remains brachycephalic with a recessed forehead and supraorbital margins. Maxillary hypoplasia is also evident.* (Below, right) *The appearance shortly after frontofacial advancement.* (Above, left *from D. J. David, D. E. Poswillo, and D. A. Simpson,* The Craniosynostoses: Causes, Natural History, and Management. *Berlin; Springer-Verlag, 1982. P 255.)*

There were two instances of osteomyelitis necessitating removal of the frontal bone. A single periorbital abscess was controlled by needle aspiration and antibiotics. An extradural abscess developed in association with a CSF leak. There was one wound infection.

CSF leaks occurred in four patients. In one patient this was self-limiting, but three patients required subsequent dural repair (one in conjunction with drainage of an extradural abscess). Postoperative velopharyngeal incompetence necessitating pharyngoplasty was seen in four patients. Transient diplopia and facial nerve palsy (unilateral frontal branch) occurred once each. All complications occurred in adults undergoing frontofacial advancement (Table I).

Long Term Follow-Up. There were 16 patients in this group. Clinically, 11 patients have maintained a satisfactory postoperative appearance of the midface (good long-term result) (Figs. 4 and 5). Four patients who were initially assessed as having a satisfactory appearance with class I occlusion have subsequently experienced a deterioration in their appearance with a recurrence of class III malocclusion (Figs. 6 and 7). A fifth patient still retains an acceptable facial appearance, although recurrence of class III malocclusion has occurred (poor long-term result). The difference in age at operation between these two groups of patients was significant; there was no significant difference in the length of follow-up between the groups, however (Table II). All patients who underwent frontofacial advancement have maintained a satisfactory position of the forehead and supraorbital margins.

The cephalometric data are summarised in Table III. In all patients, forward and downward movement of the midface was achieved at operation. There was no significant difference in the magnitude of the surgical shift between those patients having a good long–term result compared with those having a poor long–term result. Relapses of midfacial position of 1 and 2 mm were noted in three patients in the group having a good long-term result. Continued downward and forward growth of the midface occurred in all patients with a poor long–term result. No significant difference in horizontal maxillary growth *(ANS–PNS)* was seen between the two groups; however, the difference in postoperative mandibular growth *(Ar–Gn)* between the groups was highly significant ($P < 0.001$).

Discussion

When performed during the first year of life, fronto-orbital advancement and decompressive craniectomy provide excellent relief from the symptoms of craniostenosis. However, close follow–up of available patients shows that despite extensive calvarial remodelling, definitive cosmetic correction of the calvaria and upper face has not been achieved. These patients all had severe deformities with soft-tissue constraints limiting the extent of bony advancement. Although objective data are lacking, further growth and relapse of fronto–orbital position may be additional factors militating against a satisfactory long–term cosmetic result. Present evidence does suggest, however, that brain drive alone provides insufficient stimulus for complete skull remodelling in these severe cases. This contrasts with the generally satisfactory improvement in cranial morphology following early fronto–orbital advancement in cases of nonsyndromal craniosoynostosis.

During the intermediate period, fronto-orbital advancement also can control craniostenosis and orbitostenosis. Again, despite some calvarial remodelling, poor long-term cosmetic results were achieved, with three patients requiring repeated fronto-orbital advancement at the time of subsequent midfacial advancement. All these patients were operated on during the first half of the intermediate period (1 to 6 years). By contrast, those patients who underwent frontofacial advancement during the second half of the intermediate period (6 to 10 years)

maintained a satisfactory position of their upper face despite their propensity to recurrent midfacial hypoplasia.

Early fronto–orbital advancement did not prevent the development of subsequent midfacial hypoplasia. However, midfacial osteotomy performed during the intermediate and late periods resulted in an improvement in the position of the midface. Coincident with this was the establishment of normal occlusion and an increase in orbital volume. Although these aims were universally successful in the short term, long-term follow–up of 16 patients has shown a gradual recurrence of deformity in 5 patients.

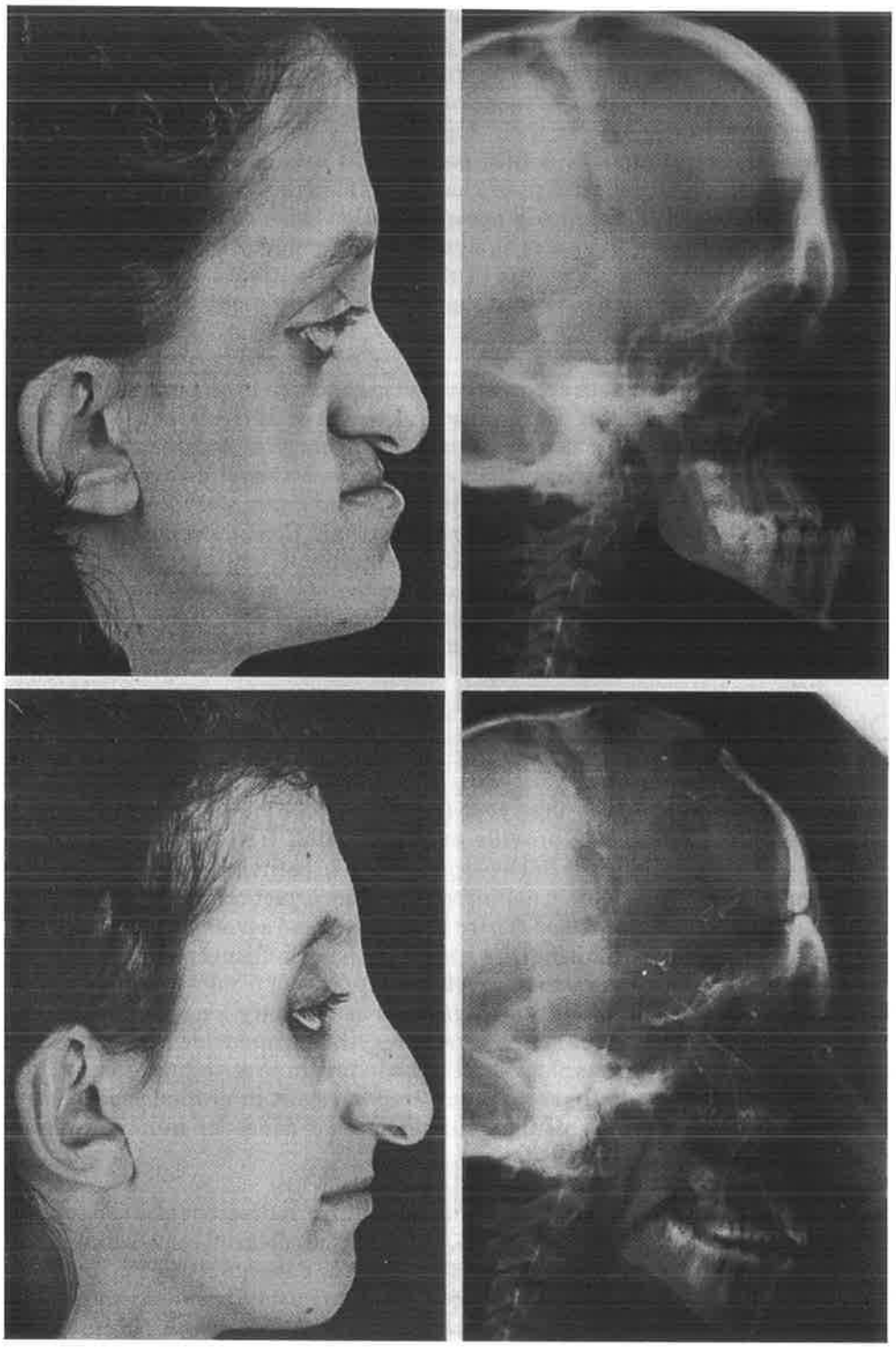

FIG. 4. *An 18–year–old woman with Crouzon syndrome.* (Above) *Preoperative appearance.* (Below) *Early postoperative appearance following transcranial frontofacial advancement.*

Recurrent facial deformity developed in those patients operated on during childhood and early adolescence, i.e., during the intermediate and early late periods. Cephalometric analysis indicates that this is due to continued mandibular growth and not to relapse of the position of the midface. The results also confirm previous reports[14,15] of continued forward and downward growth of the anterior nasal spine away from the cranial base in patients who undergo midfacial advancement during the growth period. However, this is insufficient to keep pace with growth of the mandible. This supports the hypothesis that the midface continues to be affected by the original dysplasia and that early surgical advancement will not "unlock" any constrained potential for normal growth. Although we have not performed frontofacial advancement during infancy, [18] we remain skeptical as to its efficacy in promoting midfacial growth.

Adults undergoing advancement of the midface show a great deal of long-term stability, and this is directly attributable to a lack of further mandibular growth. Although cephalometric relapses in midfacial position of 1 and 2 mm were seen in three patients, these had no effect clinically. Such measurements probably fall within the limits of error of the cephalometric method and, in the present context, are of little significance.

TABLE I

Postoperative Complications Following 32 Midfacial Advancements in 30 Patients

Complication	No.
Blindness	2
Frontal osteomyelitis	2
Extradural abscess	1*
Periorbital abscess	1
Wound infection	1
CSF leak	4*
Velopharyngeal incompetence	4
Diplopia	1
Facial palsy	1

* CSF leak and extradural abscess occurring in the same patient.

Although the likelihood of recurrent deformity is high when midfacial advancement is performed during childhood and early adolescence, there are certain situations where early surgery is mandatory. Severe faciostenosis with obstructive sleep apnea is an absolute indication, and when craniostenosis and ocular proptosis coexist, full frontofacial advancement may be required. These operations provide excellent palliation of symptoms, but they should be undertaken in the knowledge that further midfacial surgery may be needed to achieve the definitive cosmetic result.

Older patients who underwent frontofacial advancement demonstrated a great deal of long-term stability at the expense of a higher incidence of complications, particularly infections. Whereas in children the extradural space left following frontal bone advancement is rapidly filled by the expanding brain, in adults a large aerocele persists, usually in direct communication with the nasal passages. Tessier postulated that limiting the frontal advancement in adults to 10 to 12 mm will lessen the incidence of infection,[19] but this has yet to be proven. Submucoperiosteal dissection of the nasal mucosa,[10] bony frontofacial separation,[11] and the use of galeal–frontalis flaps[20] are conceptually attractive as a means of preventing contamination of the extradural space, but these too are unproven modalities. Although we have had no experience with these latter techniques, the galeal–frontalis flap, however, does seem to provide the most logical solution. Because of the devastating consequences of intracranial infection, we intend employing its use in future frontofacial advancement. Scarring of tissue

planes, the legacy of previous surgery, contributes to the increased complication rate in adults.[21]

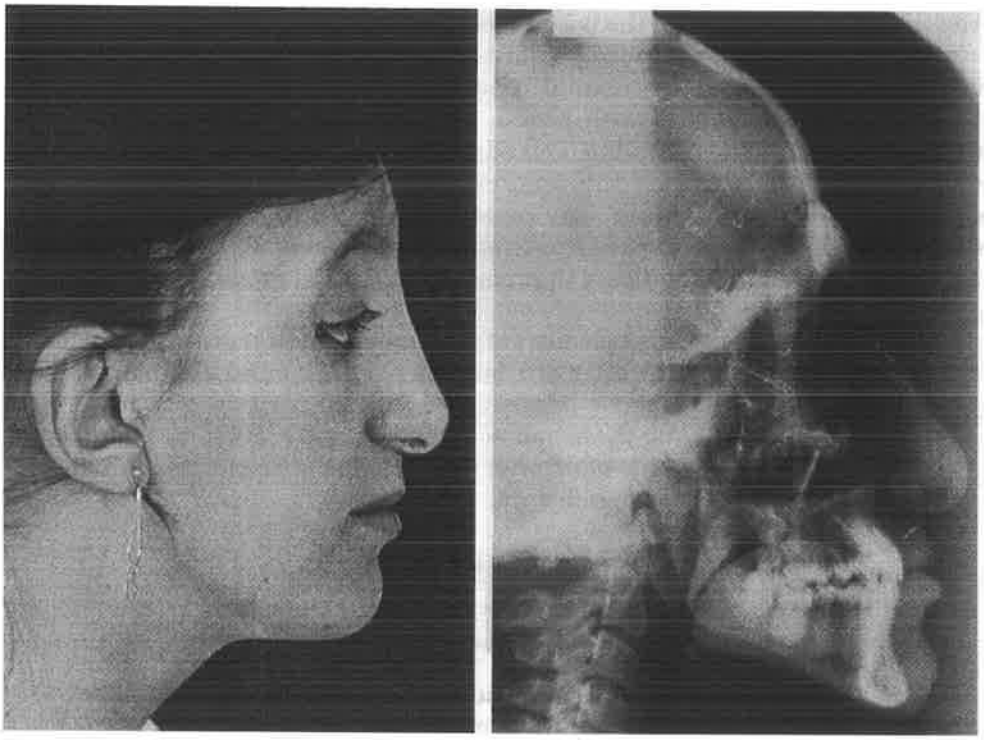

FIG. 5. *At 3 years postoperatively, the position of the midface has been maintained and there has been no mandibular growth. A rhinoplasty has been performed in the interim (see Fig. 4).*

TABLE II

Age at Operation and Length Of Follow–Up of 16 Patients Followed for More than 2 Years after Midfacial Advancement (in years)

	Good Long–Term Result (*N* = 11)		Poor Long–Term Result (*N* = 5)	
	Range	**Mean**	**Range**	**Mean**
Age at operation	10.3–38.8	20.7	8.3–11.2	10.0
Length of follow-up	2.3–8.7	3.9	3.4–7.0	5.7

Under ideal circumstances, the brain and vision should be protected by early fronto–orbital advancement and decompressive craniectomy. If advancement of the frontal bone and supraorbital margins can be maintained, subcranial midfacial advancement can be implemented at a time when mandibular growth has ceased. This avoids a third operation and minimises the likelihood of intracranial infection. Unfortunately, in the more severe cases, this ideal cannot be achieved.

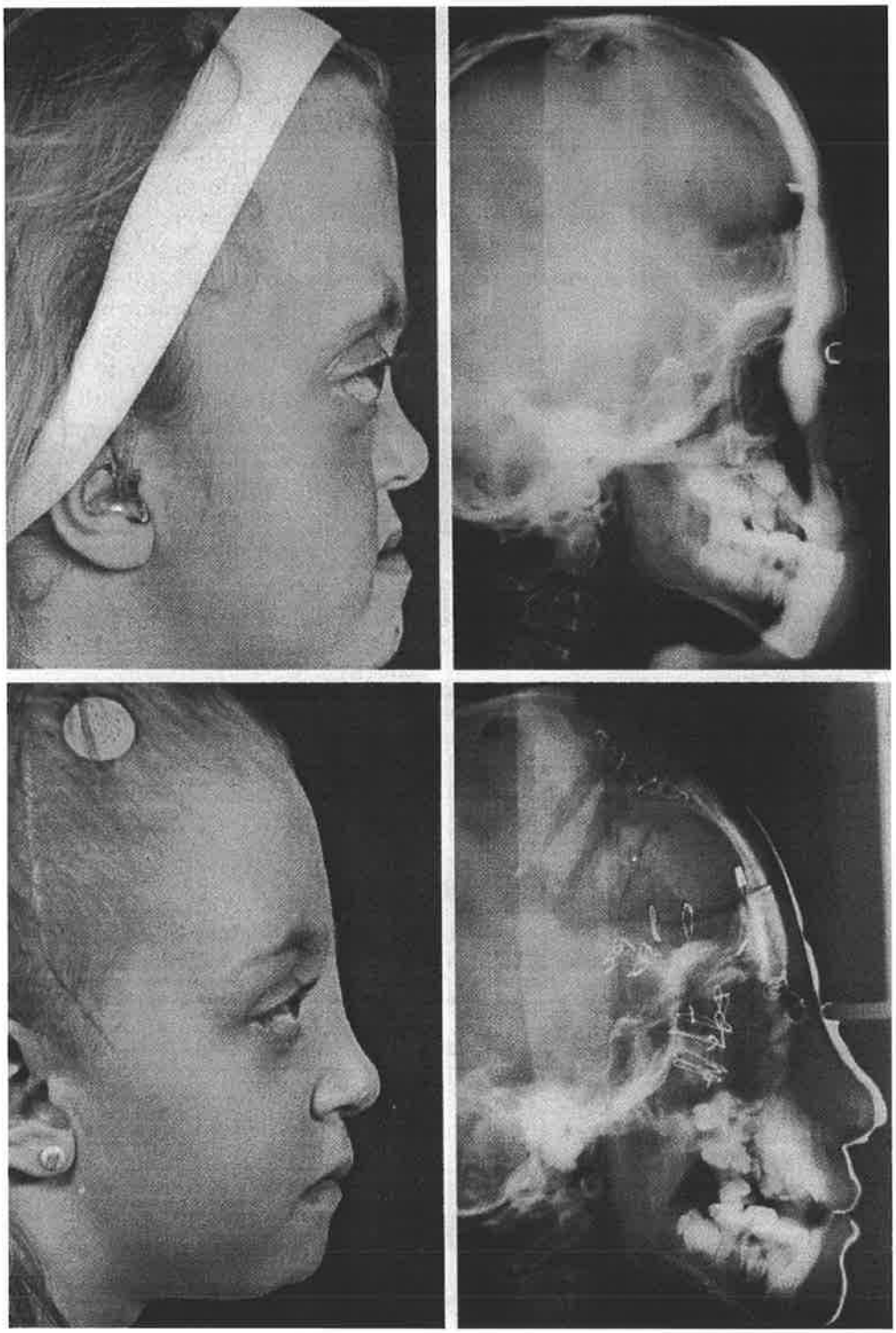

Fig. 6. *An 8-year-old girl with Crouzon syndrome. (Above) Preoperative appearance. (Below) Early postoperative appearance following transcranial frontofacial advancement.*

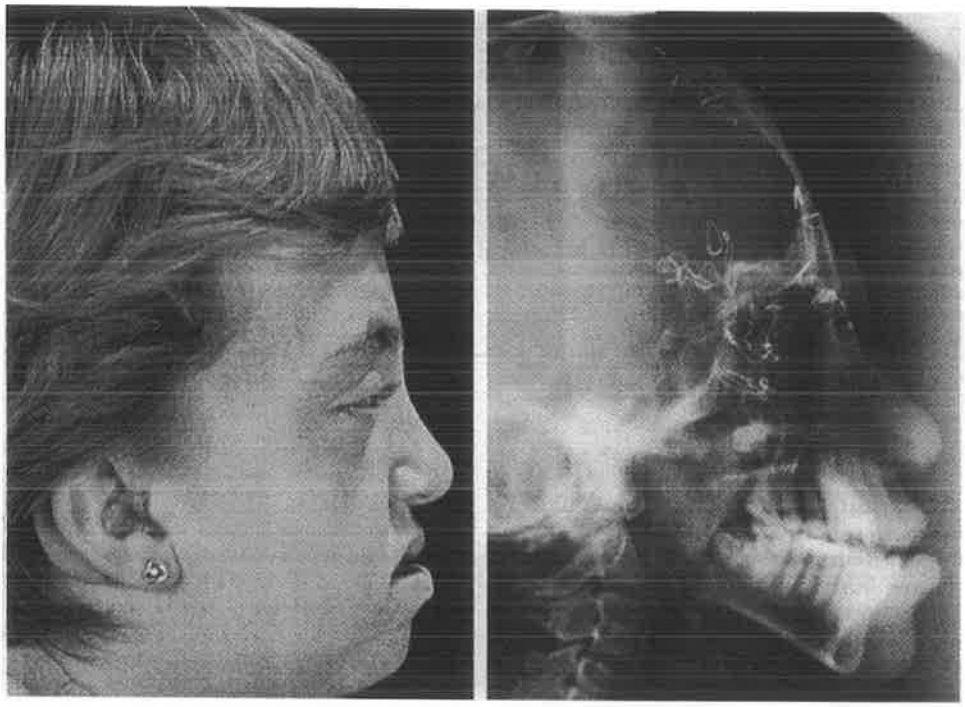

FIG. 7. *At 6 years postoperatively, there is recurrent midfacial deformity. Cephalometric analysis indicates that this is due to continued mandibular growth. The position of the forehead and supraorbital margins has remained satisfactory (see Fig. 6).*

TABLE 111

Cephalometric Data on the 16 Patients Followed for More than 2 Years after Midfacial Advancement (in millimetres)

	Good Long-Term Result (N = 11)		Poor Long-Term Result (N = 5)	
	Range	**Mean**	**Range**	**Mean**
Operative horizontal midface advancement *(x)*	2–15	7.6	3–11	8.0
Operative vertical midface advancement *(y)*	4–15	8.0	1–16	6.6
Postoperative horizontal midface movement *(x)*	–2–4	0.8	1–12	5.4
Postoperative vertical midface movement *(y)*	–2–4	0.7	1–8	4.6
Postoperative horizontal maxillary growth *(ANS–PNS)*	0–2	0.4	0–2	1.2
Postoperative mandibular growth *(Ar–Gn)*	0–5	1.3	9–19	15.0

Note. A negative movement constitutes a relapse.

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Crouzon Twins with Cloverleaf Skull Malformations

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We describe unique monozygotic twins with Crouzon's disease and cloverleaf-shaped skull deformities who have been closely followed since birth. Their abnormal skull shapes were identified during antenatal ultrasound examination. The twins had gross exophthalmos and hydrocephalus with papilledema, so early calvarial decompression surgery was required. Although born to healthy parents of normal appearance, a third cousin of the twins had a milder form of Crouzon's disease, and there was a family history of high–arched palate on the twins' paternal side. Because Crouzon's disease is an uncommon condition and the cloverleaf skull shape is unusual in patients with Crouzon's disease, identical twins with this constellation of deformities must be exceptionally rare.

Key Words: Crouzon's disease, birth defects, craniofacial

Crouzon's disease is characterised by craniosynostosis, shallow orbits with ocular proptosis, and maxillary hypoplasia. Skull shape depends on the sequence of premature sutural fusion, and multiple sutures are usually involved. The most common skull shapes seen are brachycephalic and scaphocephalic, but trigonocephalic and triphyllocephalic (cloverleaf skull) may occur, albeit rarely.[7,10]

Clinical Record

Male twins with Crouzon's disease were born at 36 weeks' gestation following a normal pregnancy. The clinical suspicion of twins early in the pregnancy had been confirmed by maternal abdominal ultrasound at 22 weeks' gestation. Further ultrasound examination at 35 weeks' gestation clearly identified a cloverleaf head shape in coronal section (Fig 1A). In addition, severe exophthalmos was evident (Fig 1B).

Family history revealed that a female third cousin of the twins had required surgical treatment for a less severe form of Crouzon's disease; her father had brachycephaly, a high arched palate, and slight ocular proptosis. On the twins' paternal side there was a family history of high palatal arches; both parents, however, had normal facies (Fig 2).

At birth the twins exhibited the features of Crouzon's disease (Fig 3). Initial radiographs demonstrated their shallow orbits and widespread calvarial synostosis with temporal bulging. Three-dimensional reconstructions revealed their remarkable shape similarities (Fig 4). For each twin, a two–stage operative plan was devised to decompress their calvaria to remedy craniostenosis with progressive papilledema.

Because Twin 1 had worse ocular proptosis, a frontoorbital advancement was performed first. This procedure proved difficult to maintain due to thinning of the calvaria and the marked degree of orbitostenosis, complicated by bilateral ocular prolapse, which required temporary tarsorrhaphies. Conversely, Twin 2 had initially worse posterior lacunae, so posterior craniectomies were performed.

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On review at 6 months of age (6 weeks after their first stage of surgery), Twin 1 still had marked ocular prolapse, but his papilledema had begun to subside. The frontal advancement was not well maintained on the left side. Twin 2 had a flattened occiput. The second stage of surgical decompression for each twin was performed at 8 months of age; Twin 1 underwent posterior craniectomies and Twin 2 underwent frontoorbital advancement. On review at 10 months of age, it was evident that the second operation had been the most successful in each twin. Although hydrocephalus had been noted on earlier ultrasound scans, it increased despite two-staged decompression; therefore, ventriculoperitoneal shunts were inserted.

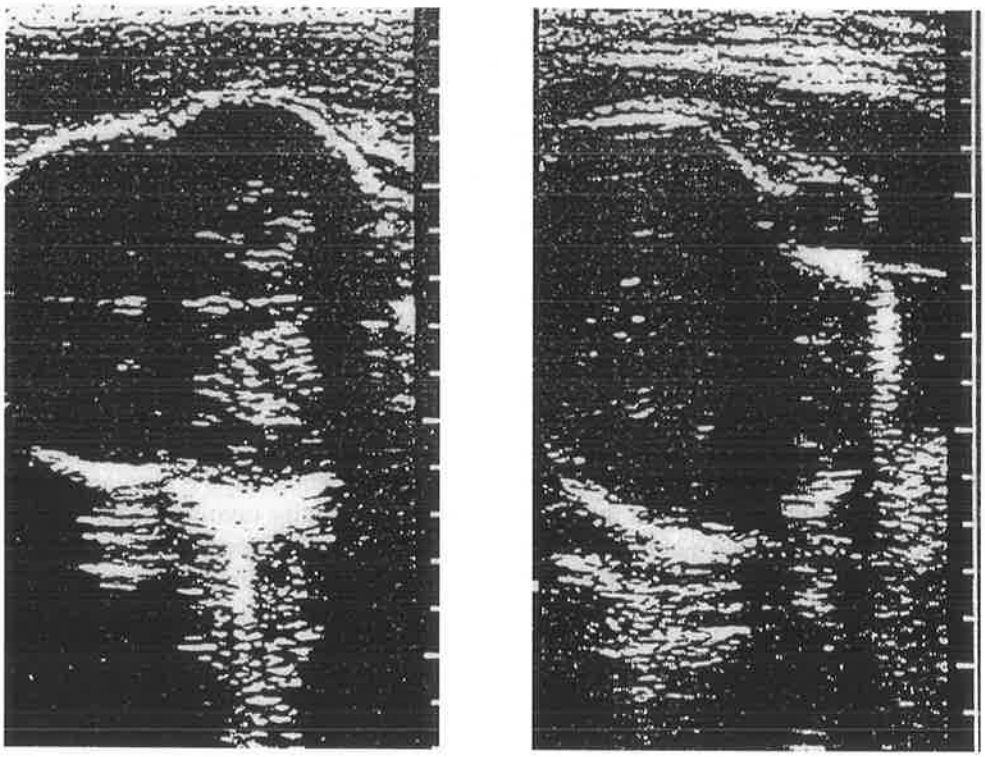

FIG. 1. *Ultrasound examination at 35 weeks' gestation showed the cloverleaf skull* **A.** *and severe exophthalmos* **B.**

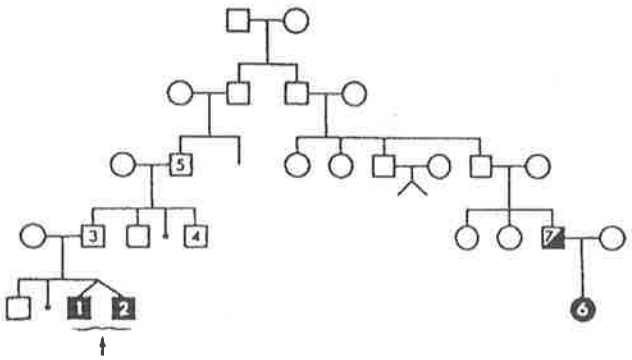

FIG. 2. *The family tree. 1, 2 = Twins 1 and 2; 3 = high arched palate; 4, 5 = partial syndactyly; 6 = Crouzon's disease, 7 = brachycephaly. high arched palate, slight ocular proptosis.*

At 15 months of age formal sleep studies were performed on both twins, which revealed evidence of moderate airway obstruction in Twin 2 and severe airway obstruction in Twin 1. Twin 1 had an oxygen saturation of less than 90% for 78% of sleep time, less than 80% for 61% of sleep time, and less than 70% for 1% of sleep time. Both twins underwent adenotonsillectomy at 3 years of age. This procedure resulted in remarkable improvement in their nocturnal obstructive

sleep pattem. Twin 2 was virtually normal and Twin 1 had improved oxygen saturation (i.e., >90% only 51% of the time, although he still occasionally dropped as low as 65%). At 5 years of age Twin 2 still has no evidence of airway obstruction; however, Twin 1 has deteriorated, and an early midface advancement is currently being planned.

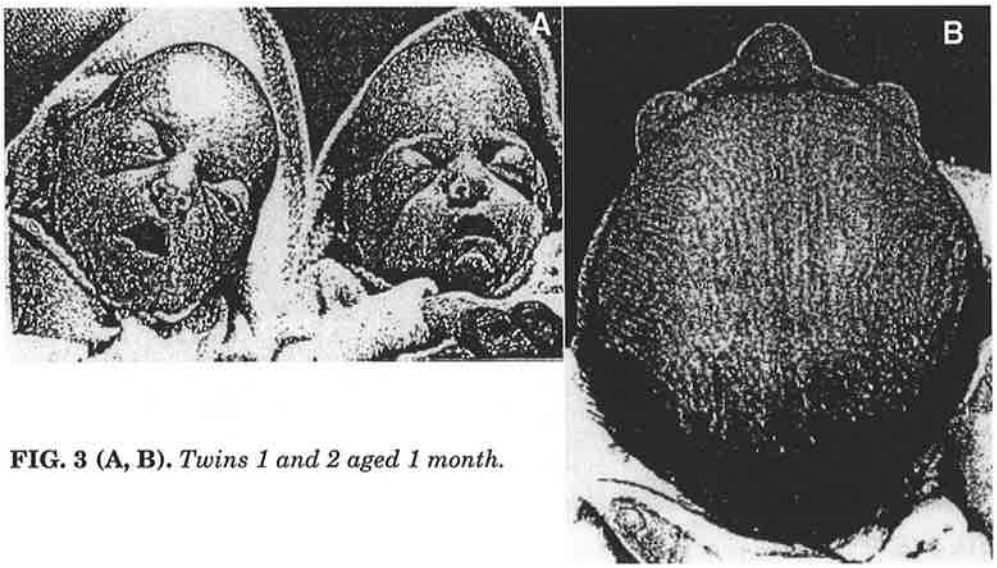

FIG. 3 (A, B). *Twins 1 and 2 aged 1 month.*

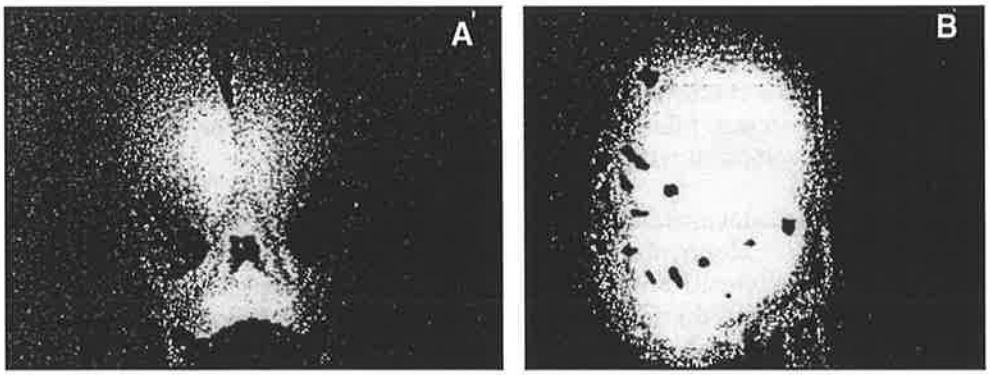

FIG. 4 (A, B). *Three-dimensional computed tomographic reconstructions preoperatively.*

Neuropsychological evaluation was performed regularly at 12 months; 2 years, 10 months; 3 years, 10 months; and 5 years of age. They have continued to achieve similar scores. Currently they are rated approximately 12 months behind their age group. They are farthest behind in language use and understanding, but self-help and gross motor abilities are normal. Twins 1 and 2 are shown at 4 years of age in Figure 5.

Discussion

Patients with Crouzon's disease may exhibit any one of a variety of calvarial deformities; however, triphyllocephaly (cloverleaf skull shape) is uncommon.[7,10] The cloverleaf skull shape was pictorially recorded initially by Vrolik in 1949[20]; however, it was not until 1960 that the "Kleeblattschadel-Syndrom" was described by Holtermuller and Wiedemann.[16]

Although the similarity of the craniofacial features of patients with Kleebattschadel syndrome to those of patients with Crouzon's disease had been previously noted,[1.,8,13,21] it was Hall and colleagues[15] in 1972 who suggested that some Kleeblattschadel patients may in fact have a severe form of Crouzon's disease. This viewpoint was endorsed by Cohen,[5] who further suggested that the Kleeblattschadel anomaly should be regarded as a symptom that may occur in a

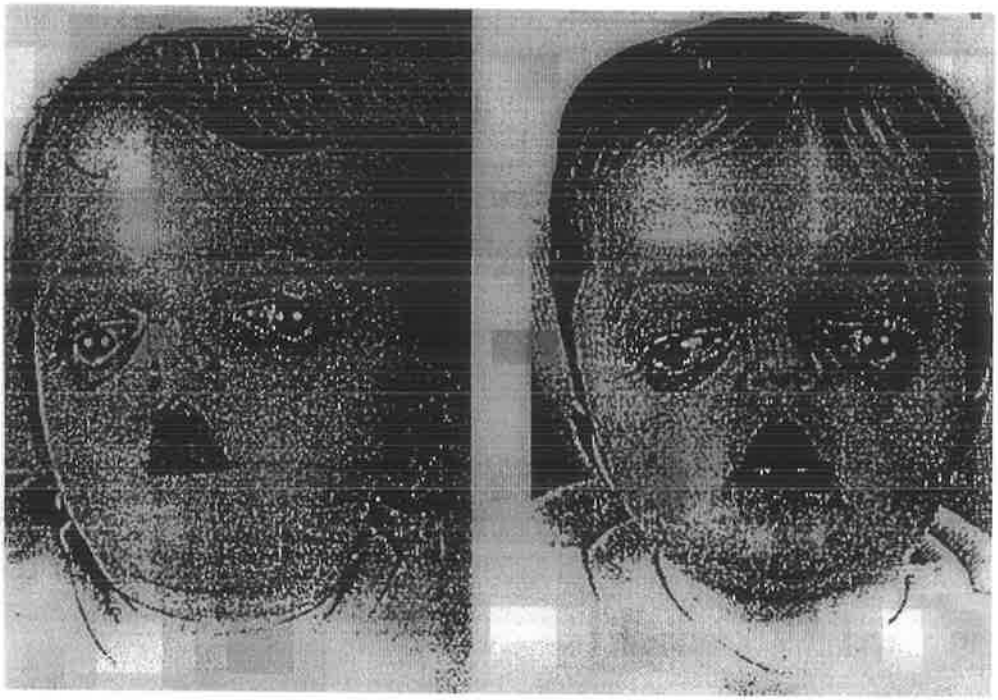

FIG. 5 (A, B). *The twins at age 4.*

variety of disorders. He later delineated these disorders to include Crouzon's disease, Carpenter's syndrome, Apert syndrome, and Pfeiffer's syndrome, as well as patients subjected to bilateral subtemporal decompression or as an isolated anomaly or in association with bony ankylosis of the limbs.[6,7]

Crouzon's disease affecting one dizygotic twin was reported by Fogh–Anderson in 1943.[14] Monovular twins with Crouzon's disease were described by Loffredo and associates,[18] and those twins both had oxycephalic skull shapes. Monozygotic twins with thanatophoric dwarfism who did not have cloverleaf skull deformities were described by Horton and co-workers.[17] The unique twins presented here were both monozygotic and had severe cloverleaf skull deformities.

The twins' abnormal head shapes were identified during antenatal ultrasound scanning (see Fig. 2). In addition to the plain antenatal radiographic recognition of cloverleaf skull shapes,[3,12] their recognition in utero by ultrasonography has been described previously by Brahman and associates[4] and by Banna and colleagues.[2]

It is interesting to explore the inheritance of Crouzon's disease in this family. Crouzon's disease clearly follows an autosomal dominant mode of transmission, and variability of expression is common.[19,20] The twins were born to parents of essentially normal appearance who also had a normal healthy son. A third cousin of the twins had mild Couzon's disease, however, and was treated at our Unit. The twins' paternal side had high arched palates. Thus it would appear that this family exhibits autosomal dominant inheritance with variable penetrance. This possibility was described by Crouzon.

The prognosis of patients with cloverleaf skull deformity is not known. With no treatment, prognosis is poor and few survive.[12] The Australian Cranio–Facial Unit has adopted a policy of early frontoorbital advancement and posterior craniectomy in the treatment of Crouzon's disease to relieve raised intracranial pressure and to reduce ocular proptosis.[11] The unique twins presented here required both procedures to relieve these manifestations. Further follow-up and reporting may clarify the long-term benefit of this approach.

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Obstructive Sleep Apnea in Apert's and Pfeiffer's Syndromes: More than a Craniofacial Abnormality

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Nine acrocephalosyndactyly type I patients (Apert's syndrome) and three acrocephalosyndactyly type V patients (Pfeiffer's syndrome) were evaluated for the relative importance of upper and lower airway abnormalities in the generation of obstructive sleep apnea. All patients were found to have a combination of upper and lower abnormalities. The influence of lower pathology was greater in the infants, and the influence of upper airway, specifically pharyngeal, was greater in the adults. A comparison between preoperative and postoperative polysomnography revealed little improvement with standard craniofacial advancements. Furthermore, three patients are described who succumbed to pulmonary death despite tracheostomy. Conservative treatment with prone or lateral positioning and medical pulmonary regimens is advocated. Finally, the pathogenesis of this diffuse airway pathology is discussed.

Recent articles have addressed the causes of sleep apnea in various craniofacial syndromes.[1–3] Most of the attention has been focused on the upper airway and its manipulation. Occasional case reports have documented the marked lower airway pathology in both Crouzon's and Apert's syndromes.[4,5] These reports, however, are based on autopsy findings. With improved podiatric fibreroptic endoscopy, even the smallest infants may be examined during dynamic respiratory function. In addition, multichanneled polysomnography has added diagnostic values to the clinical diagnosis of sleep apnea. In this paper we report our findings on the causes of sleep apnea in acrocephalosyndactyly type I (Apert's syndrome) and acrocephalosyndactyly type V (Pfeiffer's syndrome).

Patients and Methods

The study group includes 12 consecutive patients evaluated by the first author (Mixter): nine Apert's and three Pfeiffer's patients ranging in age from 1 month to 48 years and from 1 month to 30 years, respectively. Two Apert's patients were from the Australian Craniofacial Unit, and the remainder were from the University of Wisconsin Craniofacial Anomalies Clinic. Method of study included bronchoscopic data from nine patients, bronchoscopic and autopsy reports from two patients, and bronchographic and autopsy data from one patient. Eight patients were studied preoperatively and postoperatively by multichannel polysomnography. Surgical interventions included three intranasal dilatations and placement of stents, two palate repairs, three extracranial Le Fort III procedures, one monoblock and one Le Fort III, Le Fort I, and mandibular setback.

Results

Airway Abnormalities (Table I)

All patients suffered from a combination of upper and lower airway disease. The severity of the lower airway disease appears to correlate with the severity of the

craniofacial anomalies and the severity of the upper airway disease; i.e., Pfeiffer's syndrome patient K.H. had a marked pancraniosynostosis cranial deformity, severe midface hypoplasia, choanal atresia, and severe laryngo–, trachea–, and bronchomalacia. The older patients experienced fewer symptoms from lower airway pathology than did the younger patients. Because of the extremely small size of the infant airways, these patients suffered from frequent bouts of tracheitis and bronchitis. As airway swelling increased, obstructive symptoms also increased. In addition to treatment with antibiotics, bronchodilators, and airway humidification, aerosolized racemic epinephrine and inspired helium/oxygen combinations also improved airway obstruction. Those patients who did require intubation were extremely difficult to wean and extubate because of airway edema. In comparison, the adult patients experienced fewer symptoms referable to lower abnormalities. While lower airway malacia was still present, the greater size and increased rigidity of the airway lessened the impact of collapse. For unknown reasons, however, the degree of pharyngeal collapse varied from mild (patient D.P.) to severe (patient C.Y.).

TABLE I

Airway Abnormalities in Apert's and Pfeiffer's Syndromes

Patient	Age	Hospital*	Diagnostic Studies	Midface Hypoplasia†	Relative Glossomegaly†	Choanal Stenosis†	Pharyngeal Collapse	Laryngo†malacia†	Tracheomalacia†	Bronchomalacia†	Additional Findings
Acrocephalosyndactyly type (Apert's syndrome):											
BD	1 mo	ACFU	Bronchogram Autopsy	++	++	++	++	++	++	++	Pancraniosynostosis, progressive respiratory insufficiency despite tracheostomy, death
SB	1 mo	UW	Bronchoscopy	+	+	+	+	+	+	+	Cleft palate
BW	1 mo	UW	Bronchoscopy	+	+	+	+	+	+	+	
DV	1 mo	UW	Bronchoscopy	+	+	+	+	+	+	+	Cleft palate
ML	5 yrs	UW	Bronchoscopy	+	+	+	+	+	+	+	
DW	5 yrs	UW	Bronchoscopy	+	+	++	++	++	++	+	Respiratory arrest at birth, anoxic brain injury, tracheostomy
BH	7 yrs	UW	Bronchoscopy	+	+	+	+	+	+	–	
DL	30 yrs	ACFU	Bronchoscopy	++	++	++	+	+	+	+	Tracheal stenosis, postoperative tracheostomy, death
CY	48 yrs	UW	Bronchoscopy	++	++	++	++	+–	––	–	
Acrocephalosyndactyly type V (Pfeiffer's syndrome)											
KH	1 mo	UW	Bronchoscopy Autopsy	++	++	++	++	++	++	++	Pancraniosynostosis, progressive tracheal injury, postoperative tracheostomy, death
SF	4 yrs	UW	Bronchoscopy	++	++	+	+	+	+	–	Kleeblattschadel
DP	30 yrs	UW	Bronchoscopy	++	+	+	+–	–	–	–	Prognathic, rudimentary tracheal rings, protective tracheostomy

* UW = University of Wisconsin; ACFU = Australian Craniofacial Unit.

†++ = severe; + = mild; +– = variable study; - = not present.

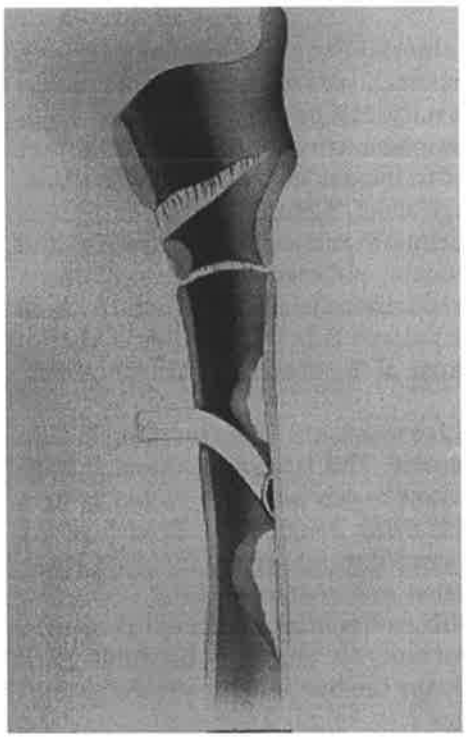

FIG. 1. *Inappropriate tracheostomy tube angle resulted in posterior wall damage.*

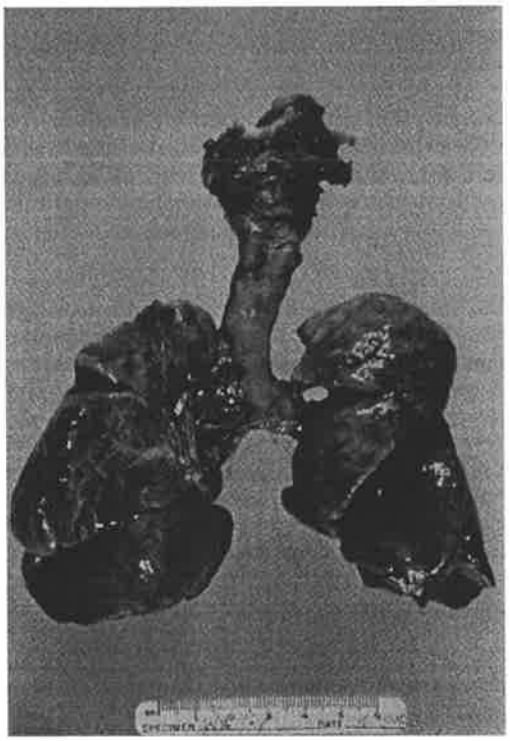

FIG. 2. *Tube change on postoperative day 2 caused inversion of the Bjork valve.*

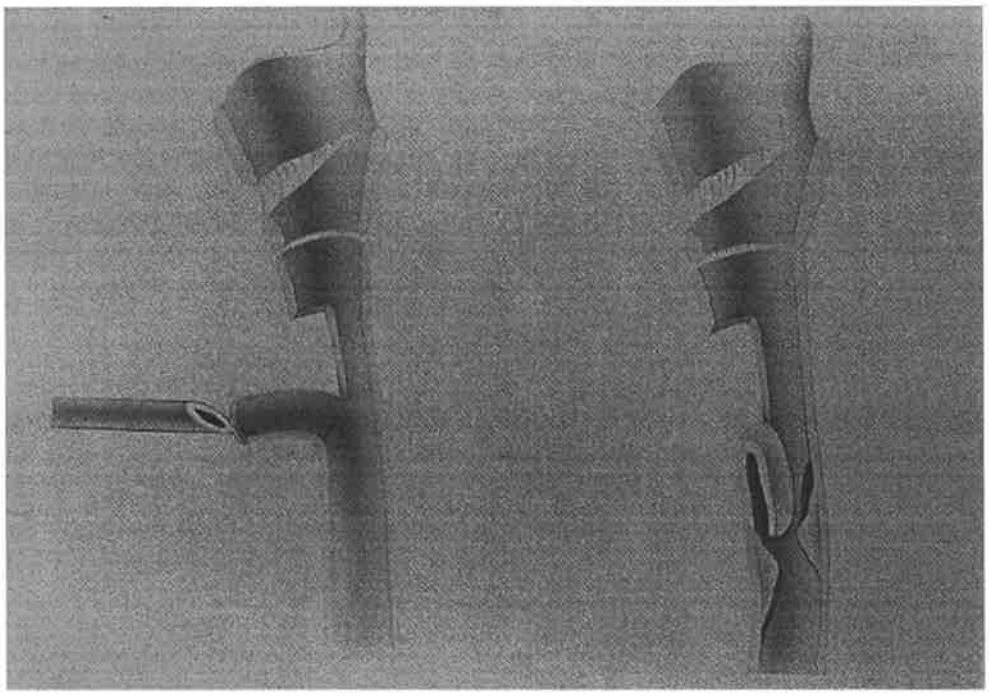

FIG. 3. *Autopsy specimen of Pfeiffer's syndrome patient K.H.: cartilaginous trachea without rings, subglottic stenosis, bronchomalacia.*

Predisposition to Tracheal Injury

Of particular concern in our series was the vulnerability of the trachea to injury. Pfeiffer's syndrome patient K. H. underwent tracheostomy after Bronchoscopy revealed severe upper and lower airway deformity. His postoperative course was marked by progressive respiratory obstruction because of posterior wall injury by the tracheal cannula (Fig. 1). In an effort to bypass the area of obstruction, progressively longer tracheal cannulas were used. Ultimately, granulations extended beyond the carina down into the primary and secondary bronchi and K.H. succumbed to respiratory obstruction, anoxia, and cardiopulmonary failure. Autopsy evaluation revealed a completely cartilaginous trachea without tracheal rings (Fig. 2). The death of Apert's syndrome patient B.D. was similar to that of K.H. but less well documented clinically because of the absence of bronchoscopy.

Apert's syndrome patient D.L. underwent an anterior Bjork flap tracheostomy before a frontofacial advancement. The tube was changed with some difficulty on day 10. On day 14 the patient began coughing violently first with blood-tinged sputum and ultimately with frank hemoptysis. Bronchoscopy revealed marked tracheal obstruction due to granulations and an inverted Bjork flap (Fig. 3). D.L. underwent tracheal resection and reanastomosis. Operative findings included a cartilaginous trachea with rudimentary tracheal rings. On postoperative day 7 after the tracheal resection, an anastomotic dehiscence developed, and despite further attempts to salvage the tracheal repair, the patient died from pulmonary sepsis.

Polysomnographic Evidence of Sleep Apnea

It was a surprise that not all patients who gave a clinical history of sleep apnea had confirmation when multichannel polysomnography was performed. Pfeiffer's syndrome patient D.P. complained of a lifelong history of fatigue and falling asleep on the job. Witnesses confirmed loud snoring but did not document apneic episodes. D.P's sleep study revealed that he had some episodes of hypopnea but no prolonged apneas. In general, the severity of the sleep apnea correlated with the severity of upper and lower airway pathology. Of interest, though, is that Apert's syndrome patient C.Y. had the most abnormal sleep study and yet had mild lower airway disease. His anatomic site of obstruction was posterior pharyngeal collapse, as documented by bronchoscopy and videofluoroscopy. It was astonishing that C.Y. had no systemic symptoms (i.e., hypertension, right heart strain, etc.) despite sleep *Pa*O2 values below 60 percent for 48 years!

TABLE II

The Effect of Facial Surgery on Sleep Apnea

Patient	Age	Hospital	Surgery	Polysomnography* Preoperative	Polysomnography* Postoperative	Comments
ACS 1 (Apert)						
BW	1 mo	UW	Choanal dilation and stents	+	+	
SB	1 mo	UW	Choanal dilation and stents	+	+	
	2 yrs		Palate repair	+	+	
DV	1 mo	UW	Choanal dilatation and stents	+	+	
	18 mos	UW	Palate repair	+	+	
BH	7 yrs	UW	Le Fort III	±	±	
ML	9 yrs	UW	Le Fort III	++	+±†	Requires night CPAP
CY	48 yrs	UW	Le Fort III	++	++	Requires night CPAP
ACS V (Pfeiffer)						
SF	5 yrs	UW	Monoblock	+	+	
DP	30 yrs	UW	Le Fort III Le Fort I Mandibular set-back Reduction genio-plasty	-	-	

* ++ = severe sleep apnea; + = mild sleep apnea; ± = hypopnea; - = no sleep apnea. † Slight improvement.

The Effect of Surgical Interventions on Documented Sleep Apnea

In general, procedures that altered upper airway anatomy had very little influence on sleep apnea (Table II). The infants who had intranasal dilatation of choanal atresia and placement of stents had some improvement in feeding but very little improvement in desaturations when sleeping. The most significant improvement resulted from postural positioning; i.e., prone improved obstructive symptoms more than lateral, which was better than supine. The influence of Le Fort III osteotomy was similar. The case of Apert's syndrome patient M.L. was representative (Fig. 4). The patient had profound sleep apnea preoperatively with some improvement in the lateral and prone positions. A sleep study 3 months after a 15–mm Le Fort III advancement (Fig. 5) revealed a slight improvement in obstructive symptoms. The patient underwent behavioral training to encourage her to sleep either prone or on her side (a backpack filled with two pillows also was placed before bed to discourage supine sleep). In addition, M.L. tolerated the use of continuous positive–pressure nose/mouth mask ventilation (CPAP) at night (Fig. 6). Almost immediately she experienced an improvement in her symptoms, including a 10–lb weight gain and restful sleep up to 9 hours.

Apert's syndrome patient C.Y. underwent a 17–mm Le Fort III advancement. After extubation, he experienced such severe sleep apnea that he consistently desaturated below 60 percent while asleep. CPAP was initiated immediately, with improvement of symptoms. C.Y. has, however, been unable to sustain use of apparatus at home because of claustrophobia and mask discomfort.

Palatoplasty and mandibular setback are procedures undertaken with some trepidation in these patients. The effect of palatoplasty was less than expected in Apert's syndrome patients S.B. and D.V. Surgery was, however, delayed until age 2 years, when the incidence of respiratory infections had decreased and their sleep studies in the prone and lateral positions had improved. Similarly, the net effect of midface advancement and mandibular setback on Pfeiffer's syndrome patient D.P. was negligible. If he *had* demonstrated preoperative sleep apnea, however, we might have been less inclined to perform a setback—or combined it with a tongue reduction.

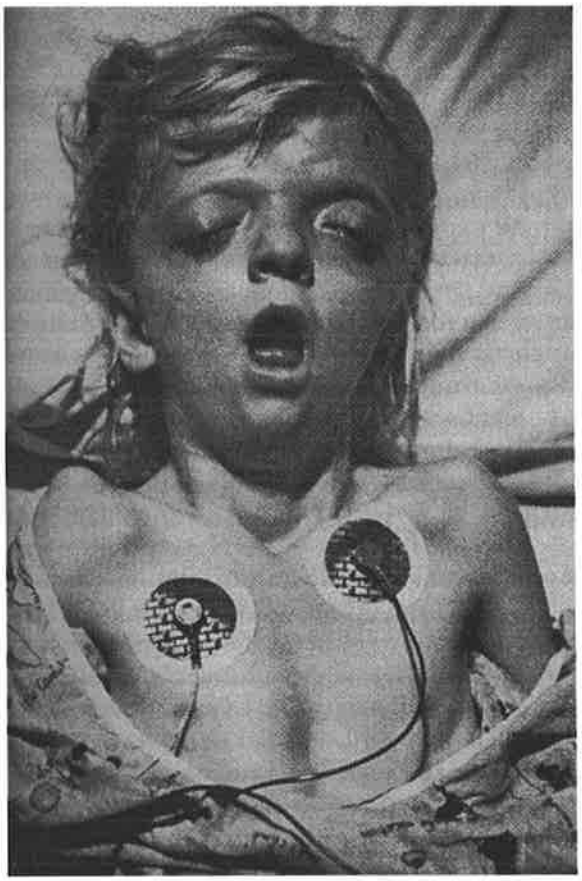

FIG. 4. *M.L. exhibiting severe obstructive symptoms after mild sedation.*

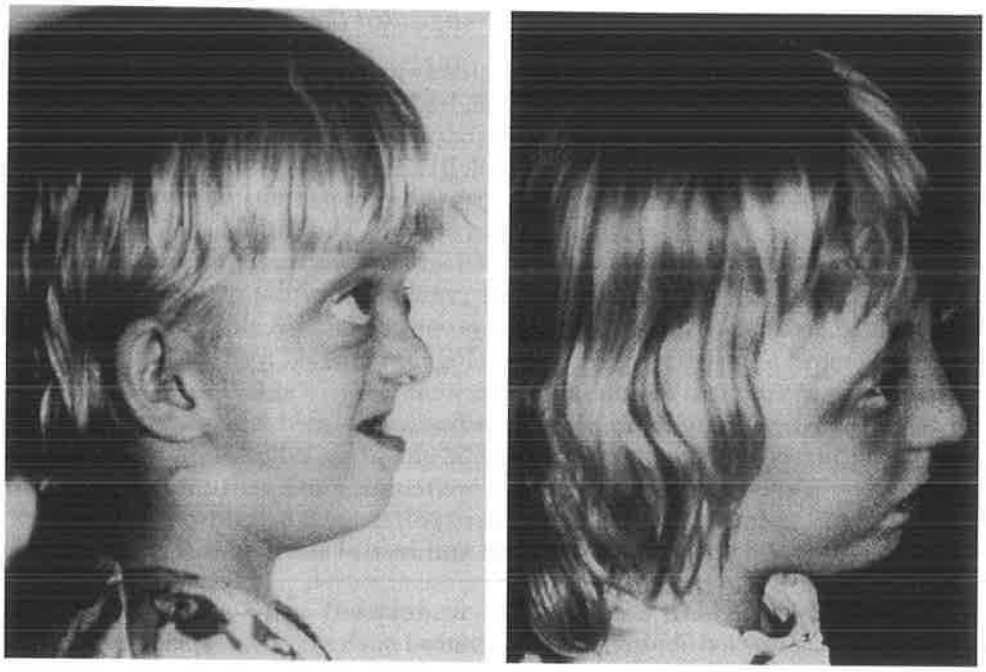

FIG. 5. *M.L. preoperatively* (left) *and after 15–mm Le Fort III advancement* (right).

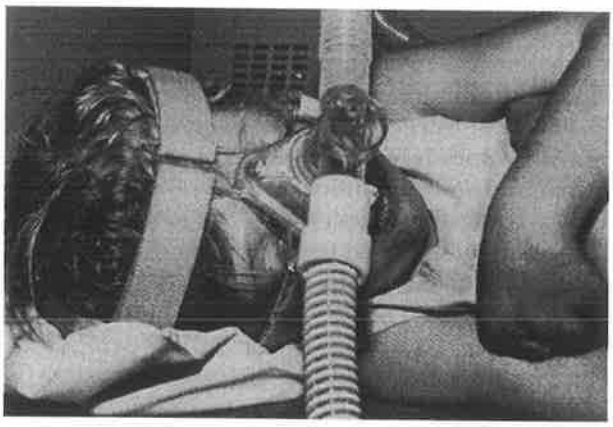

FIG. 6. *M.L. with CPAP apparatus in place.*

Discussion

In 1973, Solomon et al.[6] discussed the significance of Apert's syndrome and palatal mucopolysaccharides. They speculated that these abnormal mucopolysaccharides may represent a critical abnormality in the formation of ground substance and thus cartilage and bone matrix throughout the entire body. Clearly, Apert's and Pfeiffer's syndrome patients do have abnormalities of almost all cartilaginous structures, including the cranial base, midface, respiratory tree, elbows, hands, knees, and feet. While the exact biochemical abnormality has not been elucidated, the genetic defects responsible for these disorders must affect compounds that influence diverse cartilaginous structures.

Airway abnormalities also may be present in other craniofacial syndromes (Table III). While serious lower airway pathology occasionally occurs in patients with various craniomaxillofacial conditions, the vast majority of those studied (Table IV) have no abnormalities. The notable exception was Larsen's syndrome, which is characterized by nasomaxillary hypoplasia, joint hypermobility and dislocation, short stature, and scoliosis. All four of our Larsen's syndrome patients (including one pair of identical twins) suffered from trachea– and bronchomalacia as severe as that seen in Apert's and Pfeiffer's syndromes.

The airway management of craniofacial patients with combined upper and lower airway pathology is challenging — not only because of edema caused by recurrent respiratory infections and surgery, but also because of the vulnerability of these abnormal tracheas to trauma, as exemplified by the three deaths in our series. With improvement in intubation techniques and postoperative management of the airway, however, tracheostomy may be reserved for the unusually difficult cases. Conservative treatment of obstructive apnea in the infant may be improved by lateral and prone positioning, intranasal dilatation and stents, bronchodilators, aerosolized racemic epinephrine, and helium/oxygen mixtures. In the event that tracheostomy is necessary, custom fabrication of tracheostomy tubes may lessen the traumatic effect of chronic intubation.

In the older patient in whom lower airway abnormalities are less important, CPAP may be the only alternative to tracheostomy. After the initial period of claustrophobia and restlessness, most patients adjust to the somewhat cumbersome apparatus. CPAP therapy should be combined with humidification to lessen the drying effects, which result in nose bleeds, inspissated secretions, etc. Finally, endoscopic evaluation of the airway and polysomnography are useful as a preoperative measure in the prediction of anesthetic difficulties and for proper surgical planning specifically where mandibular setback surgery is contemplated.

TABLE III

Other Craniomaxillofacial Conditions with Lower Airway Pathology

Syndrome	Number of Patients	Airway Findings		Surgical Procedures
		Upper	Lower	
Larsen's	4	Nasomaxillary hypoplasia Micrognathia/micro–genia Choanal stenosis	Pharyngeal collapse Laryngo–, Tracheo–bronchomalacia	Tracheostomy × 2 (CPAP × 1)
Complex craniosynostosis, complex facial clefting, club feet	1	Midface hypoplasia Choanal stenosis	Pharyngeal collapse Laryngo–, tracheo–, bronchomalacia	Tracheostomy, facial bipartition advancement
Complex craniosynostosis, thyroid insufficiency, juvenile diabetes	1	None	Tracheomalacia	Tracheostomy, fronto–orbital advancement
Pierre-Robin malformation sequence (two of eight broncho–scoped)	2	Cleft palate Glossoptosis Micrognathia/micro–genia	Laryngo-, tracheo-malacia	Tracheostomy after glossopexy and lip–tongue lesion adhesion failed

TABLE IV

Craniomaxillofacial Conditions without Lower Airway Pathology

Isolated craniosynostosis	15
Cleft lip/palate	10
Craniofacial microsomia	6
Goldenhar's syndrome	2
Crouzon's syndrome	2

Summary

Patients with Apert's and Pfeiffer's syndromes have an exceedingly high frequency of documentable upper and lower airway abnormalities when such difficulties are actively evaluated. Virtually all those in a nonselected sequential series of patients evaluated at the University of Wisconsin and at the Australian Craniofacial Unit showed some combination of clinically significant upper and lower airway dysfunction. Surgical manipulation of the upper airway had very little influence on symptoms. Tracheostomy resulted in the death of three patients. Conservative pulmonary treatment and CPAP may be preferable to tracheostomy in these patients.

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Aggressive Surgical Management of Sleep Apnea Syndrome in the Sydromal Craniosynostoses

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Obstructive sleep apnea syndrome (OSAS) frequently develops in patients with craniosynostosis and associated midfacial stenosis. In the past, conservative measures or tracheostomy have been used to manage this condition. Although the course of OSAS in these patients is multifactorial, a major factor is the narrow nasopharyngeal space. Aggressive surgical intervention to enlarge the nasopharyngeal space can reduce the severity of OSAS and therefore avoid the need for tracheostomy. Surgical approaches include adenotonsillectomy, uvulopalatopharyngoplasty, and midface advancement.

Obstructive sleep apnea syndrome (OSAS) occurring in patients with craniosynostosis syndromes has received attention from few authors in the literature. The etiology of this obstruction is multifactorial and may result from narrowing of the nasopharyngeal space associated with midface hypoplasia [1] or from lower airway pathology in both Crouzon's disease and Apert syndrome [1,2].

Authors of recent reports have highlighted the severity of the problem [1–3] and have concluded that the surgical option of Le Fort III osteotomy and midfacial advancement has shown disappointing results in improving OSAS [2,3]. We review 5 patients with craniosynostosis syndromes in whom OSAS developed in 1990. The group is comprised of 2 patients with Crouzon's disease, 1 patient with Apert syndrome, and 2 patients with Pfeiffer syndrome; their treatment illustrates the range of options available in treating this challenging condition.

Patient 1

This baby girl was born with Apert syndrome and was transferred from the maternity hospital to the Adelaide Children's Hospital due to repeated apneic episodes. Sleep studies carried out at 3.5 weeks of age monitored 3 hours of sleep; during this time there were 8 episodes of periodic breathing during which oxygen saturation decreased significantly. Twenty apneas longer than 10 seconds occurred.

Home oxygen therapy was instituted; there were no subsequent prolonged apneas or desaturations, although periodic breathing was still quite marked. After 3 months of home oxygen therapy, further sleep studies revealed that the respiratory status had improved such that home oxygen therapy was no longer required.

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Patient 2

This baby boy was born with Crouzon's disease following a normal pregnancy. At birth the child was noted to have the cloverleaf skull deformity. A two–stage operative plan was devised. Posterior craniectomies were initially performed at 4.5 months of age, followed by a frontoorbital advance 6 weeks later.

At 2 years of age a sleep study was performed, which showed he had frequent obstructive apneas and 8 frank obstructive apneas. Paradoxical breathing was noted, and desaturation below 90% occurred 3% of the time and below 94% for 18% of the time. Adenotonsillectomy was performed at 3 years of age. A subsequent sleep study showed this procedure to have been remarkably successful. Oxygen saturation was less than 94% for 0.8% of the time and never decreased below 90%. On latest review at 5 years of age, his sleep studies remain virtually normal.

Patient 3

This baby boy was born with Crouzon's disease (twin brother of Patient 1) following a normal pregnancy. He also had the cloverleaf skull deformity. He underwent frontoorbital advancement at 4.5 months of age, followed by posterior craniectomies 6 weeks later. Sleep studies as early as 1 year of age showed OSAS; by 2.5 years of age, the OSAS had deteriorated to show an oxygen saturation of less than 94% for 99% of sleep time, less than 90% for 78% of sleep time, less than 80% for 6% of sleep time, and less than 70% for 1% of sleep time. Following adenotonsillectomy, there was marked and significant improvement in these values (to 11%, 5%, and 0.5%).

He was followed up at 6–month intervals and remained stable until 5 years of age, when overnight oximetry values again deteriorated and revealed marked and significant periods of desaturation. Interestingly, his twin brother (Patient 1) continues to exhibit normal oxygen saturation. It was decided to again surgically increase the nasopharyngeal space. Consequently, uvulopalatopharyngoplasty was performed. Formal sleep studies 2 months after surgery showed significant improvement. His apnea/hypopnea index was 2.4/hour, which is within the normal range. Saturation was less than 94% for 1% of the time and less than 90% for 0.3% of the time; the minimum saturation was 82%.

Patient 4

This baby boy was born with Pfeiffer's syndrome, the result of a sporadic mutation. He was initially treated with strip craniectomies of sagittal and coronal sutures at age 5 months. At 10 months of age he required a ventriculoperitoneal shunt for hydrocephalus.

He was referred to the Australian Cranio–Facial Unit at 8 years of age, and frontoorbital advancement was performed at 8 years, 10 months. Shortly thereafter, he was noted to have OSAS. He exhibited classical complete airways obstruction, with rapid desaturation, and also the nonperiodic type with progressive hypoventilation. His oxygen saturation was below 90% for 17% of the time; his minimum saturation was 57%. In view of these results, a two–stage operative plan was devised: uvulopalatopharyngoplasty followed by Le Fort III facial advancement if the initial procedure did not produce the desired result.

Uvulopalatopharyngoplasty was performed and resulted in a moderate improvement in his OSAS; however, the results were not satisfactory and therefore it was decided to proceed with subcranial Le Fort III facial advancement,

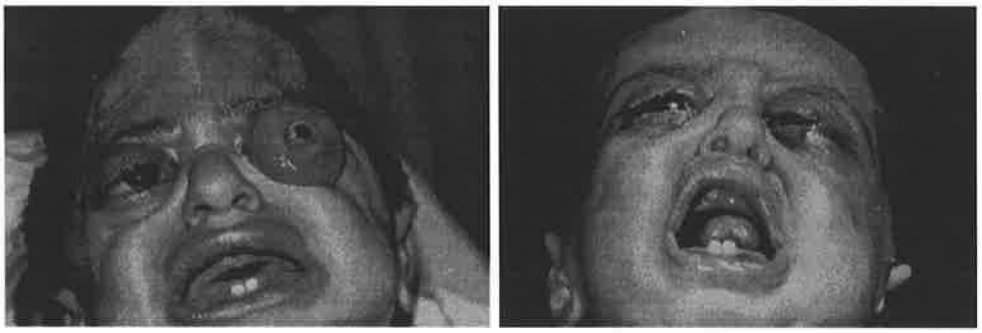
resulting in midface advancement of approximately 20 mm. Sleep studies 2 months after surgery showed significant improvement. The control of breathing had improved; only one 15 second spontaneous apnea was noted. There was still evidence of mild snoring with snorts and arousals, predominantly in the later half of the night.

Patient 5

This 11–month–old boy was born with Pfeiffer's syndrome and was referred from Oman. He had severe craniosynostosis, exorbitism, and upper airways obstruction. On arrival he was noted clinically to have gross upper airways obstruction and was immediately admitted to the intensive care unit. A formal sleep study was not performed; however, monitoring showed frequent prolonged apneic episodes, and oxygen saturation frequently decreased below 50%.

Due to the severity of his apneic episodes, the decision was made to proceed with frontofacial advancement (Figure). Despite the risks associated with this procedure, it was believed preferable to sending the child back to Oman with a tracheostomy in situ.

Sleep studies in the form of two nap studies were performed one month after frontofacial advancement. These studies were essentially normal and showed a marked and significant improvement.



Patient 5 shown before and after frontofacial advancement.

Discussion

The relationship between upper airways obstruction and pulmonary hypertension, cor pulmonale, and congestive cardiac failure was first described in 1956 by Burwell and colleagues [4] and later by Noonan [5] and Menashe and associates [6] in 1965. In essence, a person with OSAS suffers from hypoxic episodes during sleep. Sequelae of OSAS include daytime fatigue, failure to thrive, cor pulmonale, reduced mental capacity, and permanent brain damage [7].

Schafer described three levels at which upper airways obstruction could occur [2]. (1) Choanal atresia: found in facial clefting, hypertelorism, and Treacher Collins syndrome. (2) Malformations of the cranial base and midface: obstruction due to hypoplasia of the maxilla in all three dimensions. This malformation is found in the craniosynostosis syndromes. (3) Hypoplasia of the lower jaw [2].

Natural growth of the craniofacial skeleton implies that nasopharyngeal obstruction cannot be considered static; it will change with growth, either increasing or decreasing in severity. In addition, overgrowth of the soft palate in Crouzon's disease or Apert syndrome may result in increasing respiratory obstruction with age, in conjunction with growth of the tonsils and adenoids [2].

Relatively few authors have published their views on treatment options. Mixter and colleagues [3] reviewed 12 patients with Apert and Pfeiffer's syndrome. They found that procedures that altered upper airways anatomy had little effect on sleep apnea. These procedures included intranasal dilation of choanal atresia and Le Fort III facial osteotomy. The most significant improvements resulted from postural positioning. They advocate conservative treatment in infants and tracheostomy where necessary. In older patients, continuous positive airway pressure may be the only alternative to tracheostomy.

Lauritzen and associates [1] found that tonsillectomy, midface advancement, and Le Fort III facial osteotomy were all ineffective. They advocate tracheostomy for all procedures requiring general anesthesia, such as surgery, computed tomographic scans, and cephalometric radiographs.

The initiative to explore alternatives to tracheostomy was inspired by the death of a child with Pfeiffer's syndrome from complications related to the tracheostomy. In addition, we believe tracheostomy should be avoided when possible, not only in view of the well–documented complications, but also because of the secondary difficulties with socialisation, teasing, and speech development. By recognising the changing dynamics of the nasopharyngeal space, surgical intervention can be tailored in the most appropriate way.

Soon after birth, patients with a craniosynostosis syndrome should be examined by a respiratory physician and undergo a normal sleep study as part of their initial assessment. Sleep apnea can be managed by home oxygen therapy or, if severe, frontofacial advancement. The most dangerous period is at 3 to 5 years of age, when growth of the adenoids and tonsils can compromise the already narrowed nasopharyngeal space. Combined adenotonsillectomy should be employed at this point. If further deterioration occurs, uvulopalatoplasty should be employed to try and delay the necessity for early Le Fort III facial advancement. Although ideally the facial advancement is usually postponed until the early teenage years, it may be necessary at an earlier age to improve the respiratory obstruction, with the understanding that early Le Fort III advancement may have to be repeated at a later date.

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The Progress Of Craniofacial Surgery Reflected In The Management Of Craniosynostosis

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Introduction

The spectacular beginnings of Craniofacial Surgery exemplified by Gillies and Harrison's [1] report in 1950–1951 of the operative correction case of Crouzon syndrome developed their full technical potential with Tessier's [2] establishing the discipline in the late 60's and early 70's. Original emphases were clearly placed on the technical expertise and team work necessary to achieve the surgical tour de force. Since then the increasing emphasis on widening the scope of the team has led to a shift from the technical to a study of the pathology of the condition's involved [3,4]. The more patients sent to and managed by centralised units the more opportunity there has been to study the natural history of disease processes. An increasing number of articles appear which place emphasis on the pathology, natural history, and long term results of treatment.

This change in emphasis can be illustrated by tracing the progress made in the management of the Craniosynostoses.

Material and Methods

The Australian Craniofacial Unit experiences based on the management of 500 cases of craniosynostoses from 1975 to 1990.

Etiology and pathogenesis
Premature fusion of the cranial sutures (craniosynostosis) may be associated with gross cranial or facial deformity, and may also cause serious constriction of the developing brain (craniostenosis), eyes (orbitostenosis) and facial visura (faciostenosis). Craniostenosis can result in raised intracranial pressure, with impairment of vision and cerebral function.

Deformities of the facial skeleton may be still grotesque and may result in visual impairment, constriction of the upper airway and dental malocclusion. Premature sutural fusion is not a disease entity; it is a non–specific pathological process with many causes. Craniofacial Surgery is chiefly concerned with primary or idiopathic craniosynostosis. Simple craniosynostosis is the commonest form of primary craniosynostosis; the premature fusion effects one or more calvarial sutures, with an associated deformity of the skull vault. Such cases are usually sporadic but familial forms of simple craniosynostosis do occur. Complex craniosynostosis includes the import monogenic craniofacial syndromes such as Crouzon syndrome, Apert syndrome, Pfeiffer syndrome, Saethre–Chotzen syndrome, Carpenter syndrome and Cohen syndrome (cranio–fronto–nasal dysplasia). We believe [5] that primary premature sutural fusion is best regarded as an important and treatable local manifestation of an underlying growth defect in the mesenchymal capsule of the developing brain. This defect may be regional, as in the simple craniosynostoses, or generalised, as in the syndromal

craniosynostoses. In varying degrees the membranous bones of the skull vault, the cartilaginous bones of the base, and the dura mata may all be involved. Regional growth defect or growth imbalance may deform the skull vault and face, together or independently; generalised growth defect may reduce the volumetric capacity of the cranial cavities causing constriction of brain, ptosis and narrowing of the nasal air passages. Future research may well amplify or modify this concept; at present it provides a rationale for surgical intervention.

Treatment strategies

In planning treatment one must consider first the age of the patient. There are 3 surgical epochs [5,6].

(1) Early period; up to twelve months
In this period, the velocity of the cerebral growth is greatest in the craniosynostosis and may be most detrimental. The classical principle of operation was to excise the premature suture (s) by lineal craniectomy or cranicctomies and to allow the growing brain to expand, stretching the dura at the site of the new suture and reshaping the head. Modern operative management is based on this concept of intervention in the period of rapid growth but linea craniectomies are supplanted by early remodelling procedures in the fronto–orbital region and elsewhere, appropriate to the deformity. Each case requires separate consideration; as a generalisation, one can say that the calvarial deformities due to craniosynostosis can and usually should be treated surgically in the early period, where as the facial deformities are best treated expectantly. Craniostenosis, if evident in infancy requires decompression, and this can be achieved by generous fronto-orbital advancement and multiple suture excisions, giving lateral and posterior expansion, under the driving force of the growing brain.

(2) Intermediate period; one to ten years

After the second year, the loss of the cerebral growth slows. Nevertheless, severe craniosynostosis may still cause craniostenosis, leading to papilloedema and possibly visual failure. Simple sutural excision has no place; it is necessary to expand the cranial capacity by large bilateral decompressive craniotomies, or by generous frontal advancement or by a combination of these procedures. Orbitostenosis may require surgical treatment in this epoch, but if possible, faciostenosis should be treated expectantly; occasionally a severe degree of maxillary hyperplasia may require a midfacial advancement but this deformity is very likely to recur after too early operative treatment.

This is an unfavourable epoch from the psychological view point; young children may suffer considerable emotional disturbances when subjected to major operations, the need for which may be hard to explain to them. However, in the latter years of this epoch, severe psychosocial problems may sometimes necessitate operation ahead of the ideal schedule.

(3) Late period; from the tenth year
Cerebral complications are unlikely in this epoch, but facial deformities often need correction. In Crouzon syndrome and other craniofacial syndromes, proptosis and maxillary hypoplasia are especially likely to need operative treatment [7]. The operative procedures (orbital relocation, maxillary osteotomy, etc.) are very demanding but older children can appreciate the need to correct deformity, and from the psychological view point, this epoch is a favourable time. These craniofacial reconstructions entail considerable operative risks and are best treated in specialised multi disciplinary Craniofacial Units.

Hydrocephalus
The hydrocephalus often associated with severe craniosynostosis may arrest spontaneously. It is therefore usually appropriate to treat the craniosynostosis

first and to perform a ventriculo peritoneal or other shunt, if this is unavoidable, after the head has begun to remold itself

Results

1. Isolated premature fusion of the sagittal suture, lambdoid sutures and the metopic sutures are best treated early in the first year. The latter responds best to the more radical procedures involving the orbital margins and separation of the bony elements at the midline.
2. 'All that is plagiocephaly is not craniosynostosis'. There should be increased emphasis on making the correct diagnosis before surgical intervention and the pathology of craniosynostosis should be confirmed by sutural biopsy. The so called unicoronal synostosis causing frontal plagiocephaly responds well to a unilateral operation for unilateral disease in the first 2 to 3 months of life; much later than this a bilateral operation gives the best results.
3. Facial deformities, secondary to frontal plagiocephaly due to craniosynostosis, respond to cranial surgery.
4. Craniosynostosis syndromes (Crouzon, Apert, Pfeiffer, Saethre–Chotzen, Cohen) require staged surgical procedures throughout their growth period and early surgery often has to be repeated in severe cases to produce the appropriate aesthetic results in the fronto-facial region.
5. There is an increasing knowledge of the morphology and natural history of the Craniosynostosis syndromes since the advent of centralised units studying these conditions, eg. the emergence of some cases of Apert syndrome with normal or near normal intelligence who have produced families and greater detail of the morphological distinction in the Craniofacial skeleton between these groups.
6. A more acute awareness of the short term and long term problems associated with faciostenosis resulting in cerebral hypoxia and the acute awareness of the dangers of infant fronto-facial advancement as a method of solving these problems.
7. The problems of lower airway pathology coexisting with faciostenosis [8], the management of Triphyllocephaly (clover leaf skull), is fraught with danger and the prognosis is usually poor.
8. The surgery of fronto–facial advancement needs to include all the strategies necessary to separate nasal cavity from the extradural space [9].

Conclusions

The progress in Craniofacial surgery over the last 20 years is well illustrated with reference to the pathology and management of craniosynostoses. Need for multidisciplinary centralised teams involved in teaching, research and service are confirmed, as the emphasis is transferred from the difficult technical surgery, are problems of producing good long term results against a background of understanding the pathology and natural history of the disease.

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Phenotypic Variation in Acrocephalosyndactyly Syndromes: Unusual Findings in Patient with Features of Apert and Saethre-Chotzen Syndromes

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The acrocephalosyndactyly syndromes have presented diagnostic challenges because of overlap in their clinical manifestations. We present a patient with features most suggestive of Apert syndrome, but with a pattern of syndactyly not previously described. In contrast to the complex syndactyly reported as a universal feature of this syndrome, this patient shows close to total simple syndactyly of the index through ring fingers of each hand. Differential diagnoses are discussed. Because the features are reminiscent of Apert syndrome, we suggest that a new classification of hand morphology should be added to include the pattern described here.

KEY WORDS: *acrocephalosyndactyly, Apert syndrome, Craniofacial syndromes, craniosynostosis, Pfeiffer syndrome, Saethre–Chotzen syndrome, syndactyly*

Classification of the acrocephalosyndactyly syndromes is highly controversial. Several authors suspect that some of the distinctions are due more to the perception of clinicians than to genetic differences (Jackson et al., 1976; Schinzel et al., 1983; Tsukahara et al., 1985; NiemannSeyde et al., 1991). Despite the variability in expression of these syndromes, their broad overlap, and the possibility that some of them may be but differing clinical presentations of the same genetic mutations, it is helpful to attempt some logical classification for the purposes of education, prognosis, and advice to parents and patients.

We describe a patient with acrocephalosyndactyly, not entirely consistent with any previously defined type, who shows an unusual pattern of syndactyly in the hands.

Case Report

S.G. is a female child of Maltese and Australian Caucasian descent who was noted at birth to have significant facial asymmetry and syndactyly of hands and feet. She was 4 months old at the time of examination. The parents also reported that at birth the frontal hairline extended inferiorly onto the forehead to a level almost confluent with the eyebrows. She was noted to have frequent breathing difficulties, gastroesophageal reflex, and copious discharge from the eyes. She was referred to the Australian Craniofacial Unit by her paediatrician with a provisional diagnosis of Apert syndrome. The pedigree and gestational history are noncontributory.

Dr. Cantrell is a Fellow of Craniofacial Surgery, Dr. Moore is a Craniofacial Surgeon, Dr. Trott is Deputy Head of Unit, Dr. Morris

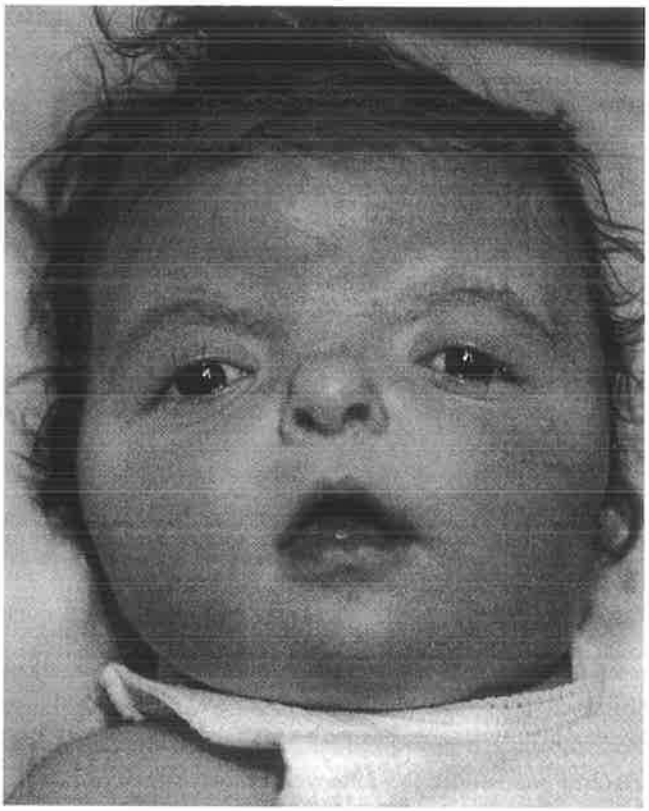

FIG. 1. *The patient at 4 months of age showing facial appearance characteristic of Apert syndrome. Note frontal bossing and turribrachycephaly, inferiorly situated frontal hairline, and mild hypertelorism.*

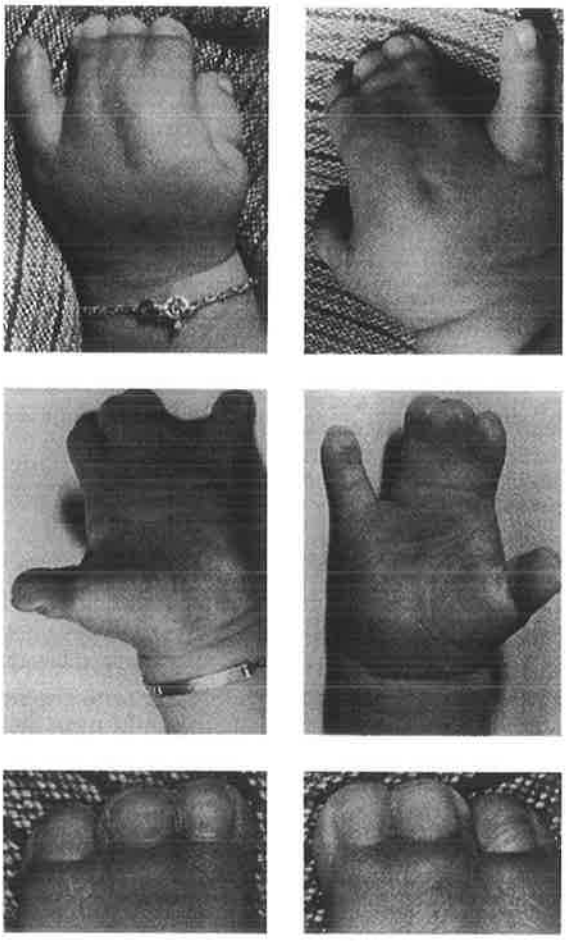

FIG. 2. *Views of the hands showing near-total soft tissue syndactyly of the second, third, and fourth fingers, with preservation of separate nails.*

Examination revealed a generally healthy and apparently bright child with striking deformities. The cranial shape was markedly turribrachycephalic, with frontal bossing and a recessed supraorbital ridge. The frontal hairline, while less pronounced than at birth, remains inferiorly displaced (Fig. 1). The posterior hairline showed the same displacement, albeit to a lesser extent. There was orbital dystopia and possibly a degree of hypertelorism, bilateral ptosis with the left eyelid more affected than the right, and antimongoloid slant of the palpebral fissures. Strabismus was also noted, and the lacrimal puncta were stenotic. The ears appeared inferiorly situated, but the helical formation was normal and not significantly different from that of either parent. The palatal vault was highly arched. A small cleft of the posterior soft palate was evident.

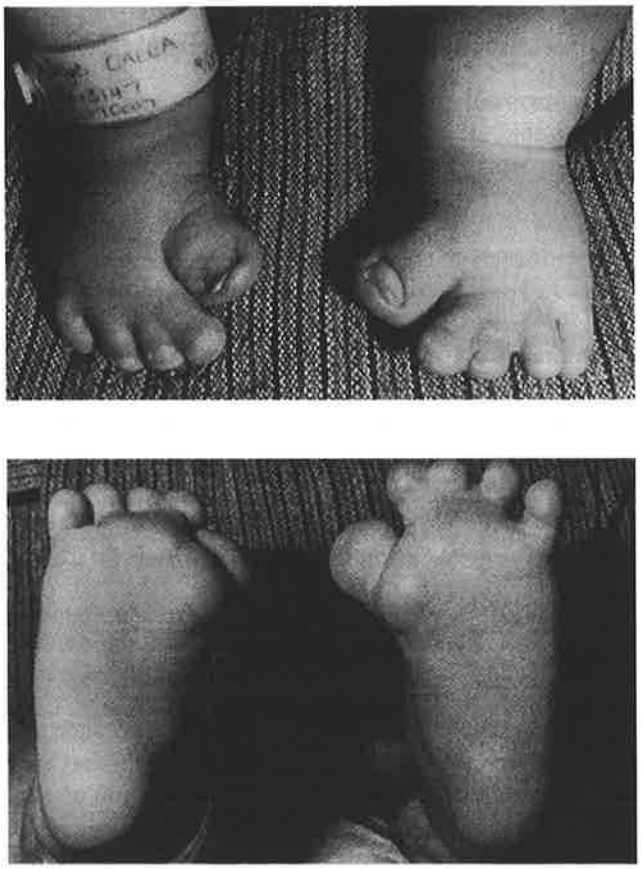

FIG. 3. *Views of feet showing how great toes are medially deviated, and partial syndactyly of the second and third toes.*

The thumbs were broad and radially deviated, and the little fingers showed mild clinodaclyly. There was near–total cutaneous syndactyly of the index, middle, and ring fingers on each hand, with preservation of separate well–formed nails and paronychial folds. On the left hand, there was also proximal webbing of the ring and little fingers. A degree of furrowing in the skin delineates the digits (Fig. 2). The great toes were broad and showed tibial (medial) deviation. The left great toe had a width of 20 mm compared with 10 mm for the adjacent toe (ratio of 2.0); the respective values for the right foot were 18 and 11 mm with a ratio of 1.64. The second and third toes on each foot showed partial syndactyly (Fig. 3).

Radiographs of the head demonstrated bicoronal synostosis and turricephaly with accompanying features: diminished cranial length, frontal bossing, and upward slanting lesser sphenoid wings (Fig. 4). The same films showed maxillary hypoplasia and relative compromise of the upper airway. Coronal sutures could not be identified.

Three-dimensional reconstructions of the computed tomography (CT) data illustrate the deformity (Fig. 5) as well as widely patent metopic and sagittal

sutures and associated fontanelles. Parietal foramina may be present. The scans from which these are derived also suggested hypoplasia of the corpus callosum.

Hand radiographs (Fig. 6) are notable for the absence of complex syndactyly. The proximal interphalangeal joints of each middle finger are absent, but whether this is due to early fusion or congenital absence is unknown. Bone formation was otherwise unremarkable with intact cortical delineation of all distal phalanges. The deviation of the thumbs is demonstrated, but the proximal phalanges are within the limits of normalcy. The appearance of the feet (Fig. 7) confirms the clinical description and, again, shows the syndactyly to be limited to soft tissues. No CT or magnetic resonance imaging (MRI) data for the extremities are available.

Nocturnal pulse oximetry studies confirmed multiple severe episodes of desaturation. Shortly after admission, the tear ducts were dilated and adenoidectomy was performed in conjunction with further splitting of the posterior palate. The airway compromise was resolved as confirmed by observation and repeat oximetry. Lambdoid craniectomies and fronto–orbital advancement with cranial vault reshaping were performed and resulted in an early improvement in cranial morphology.

The minimal syndactyly of the left fourth web space and the almost complete syndactyly of the second web space bilaterally were released and resulted in freely mobile digits. Because the nails and paronychial folds were separate and individually well formed, no reconstruction of this area is indicated. Surgical exposure and histological assessment of tissue from all of the joined areas failed to show any bony or cartilaginous tissue between the digits, confirming the proper diagnosis of a simple syndactyly (Upton, 1990). Release of the middle and ring fingers is planned in the near future.

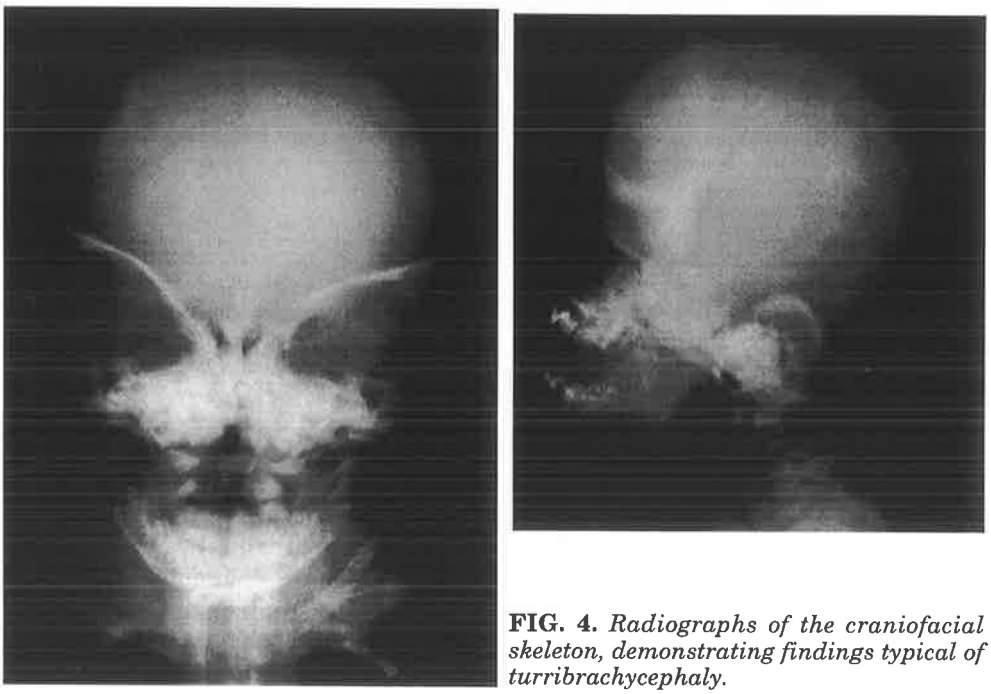

FIG. 4. *Radiographs of the craniofacial skeleton, demonstrating findings typical of turribrachycephaly.*

Discussion

The clinical and radiographic craniofacial findings in this child prompted an initial diagnosis of Apert syndrome. Indeed, the cranial shape and conformation of the hands resemble Apert syndrome with a type 1 (*obstetrician's)* hand (Upton, 1991). However, osseous syndactyly of the distal phalanges in the hands, absent in this case, is considered an obligate universal feature of Apert syndrome (Upton, 1991). This is certainly consistent with the collected experience at our institution (David

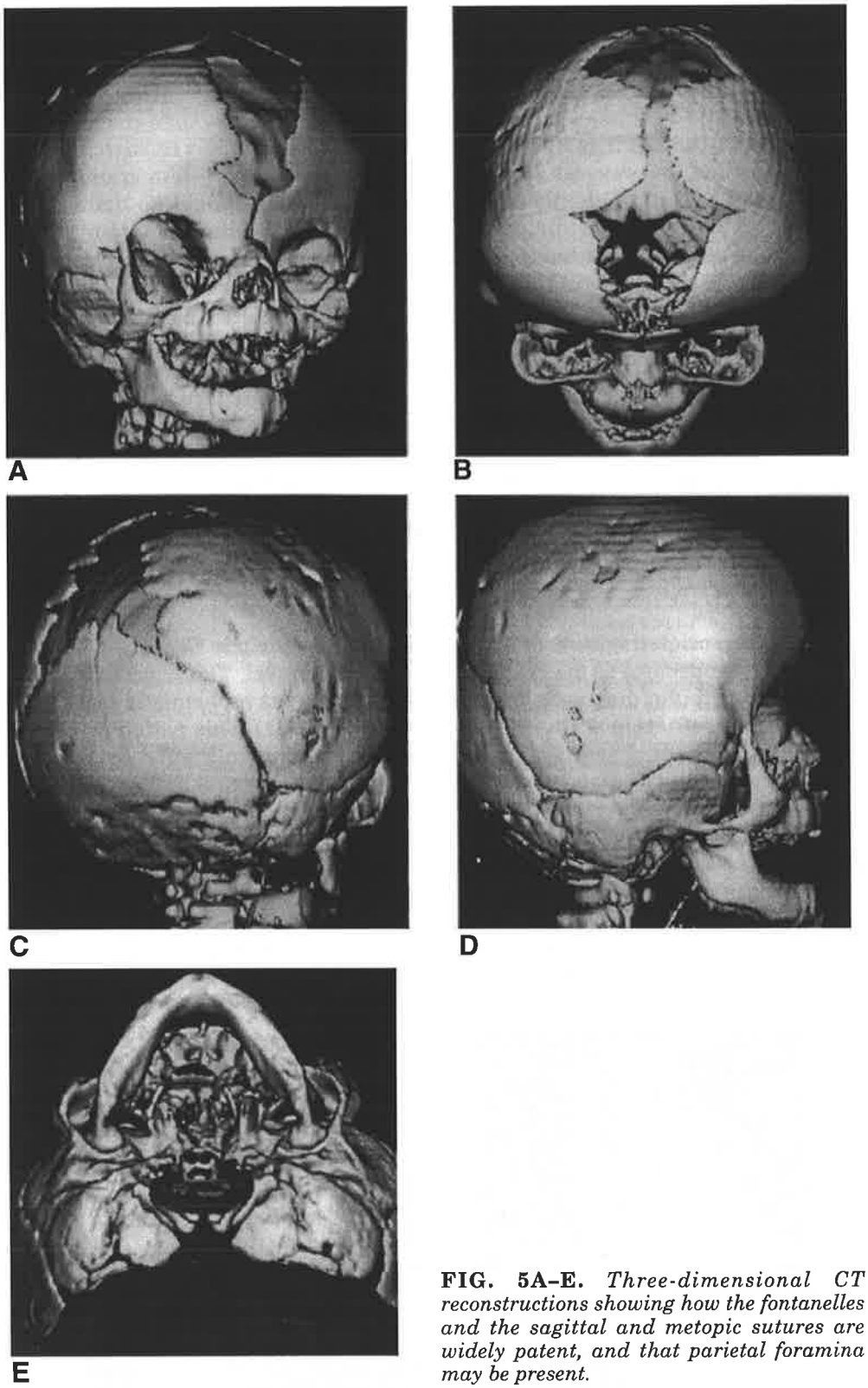

FIG. 5A–E. *Three-dimensional CT reconstructions showing how the fontanelles and the sagittal and metopic sutures are widely patent, and that parietal foramina may be present.*

et al., 1982; Holten, 1994) as well as in all other reports. It has been suggested that this fusion takes place during the first years of life (Gorlin et al., 1990), but the literature indicates that some degree of complex syndactyly, at least including cartilaginous bridging, will be present from birth (Hoover et al., 1970; Upton, 1991). This should not be confused with interphalangeal fusion within the individual digits (symphalangism), which does occur progressively in the first 4 to 6 years, or with fusion of adjacent metacarpals, which has been observed (Schauerte and St. Aubin, 1966) to continue over many years. In the acrocephalosyndactyly literature, we have been able to find only two individuals who were published as Apert patients but did not have true complex syndactyly

in the hands (Hoover et al., 1970). Unfortunately, their exact diagnosis in the Apert category is not clear as no details and no differential diagnoses are provided. It is noteworthy that at the time these were described relatively little literature existed regarding other craniosynostosis syndromes.

Because the findings in this patient are, in general, most consistent with Apert syndrome, we suggest that the constellation of Apert–like craniofacial features and extensive simple syndactyly of the hands should be defined as consistent with the Apert diagnosis. Description of the hand anatomy then becomes the obstacle. We have found Upton's classification system for Apert syndrome hand morphology (Upton, 1991) to be a straightforward and clinically useful guide and suggest that it be considered the standard. Its application in this case is diminished, however, because the absence of complex syndactyly contradicts all the defined categories. It might be argued that this difference is not important enough to warrant the creation of a new category, but we believe that overlooking contradictory features would erode the validity of the entire classification scheme. We would suggest, therefore, that the usefulness and accuracy of the present system is best preserved by defining extensive simple syndactyly as "type O" hand morphology. This places it in order as the least severe of the known syndactyly patterns in Apert syndrome. Thus, complex syndactyly of the hands should not be considered an obligate diagnostic feature in such a case.

A differential diagnosis of our case should also include Saethre–Chotzen and Pfeiffer syndromes as many of her features are typical. The argument could be made for a Pfeiffer diagnosis, but we find several areas problematic according to the present definition of this syndrome (Cohen, 1993a). This patient's widely patent fontanelles and sagittal suture would be unusual in Pfeiffer syndrome. (No diagnosis would be dismissed simply because of an unusual finding; the only thing of which we feel certain is that none of these syndromes are as clinically distinct as might once have been believed.) Syndactyly has not been described to this extent. Also, while she does have broad thumbs, the usually marked abnormal shape of the proximal phalanx of the thumb is absent in this case. It is unfortunate that, even in the most recent literature, there is no clear definition of "Pfeiffer" thumbs. Finally, if Cohen's criteria for hallucal ratios (Cohen, 1993a) are applied, both toes do not fit this diagnosis.

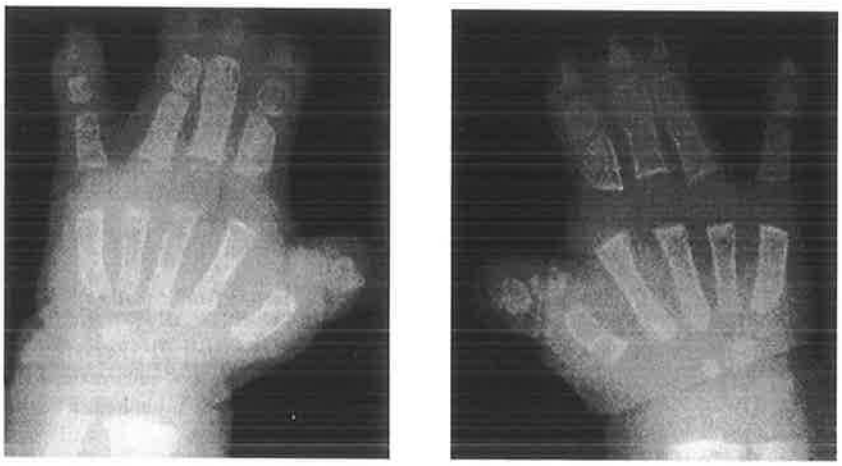

FIG. 6. *Hand radiographs show that there is no confluence of phalanges between adjacent digits. The proximal interphalangeal joint of each middle finger is absent.*

Neither is the overall picture typical of Saethre–Chotzen syndrome, but if one were constrained not to contradict previous literature this would be the only choice remaining (Pantke et al., 1975). This severity of turricephaly would be unusual for Saethre–Chotzen, however, and syndactyly this extensive is unknown. Hypoplasia of the corpus callosum is not a helpful distinction: hypoplasia or agenesis has been described in Apert, Crouzon, and Greig syndromes as well as

craniofrontonasal dysplasia and frontonasal dysplasia (Kapusta et al., 1992) and can be seen in any syndrome or as an isolated finding (Cohen, 1993). CNS abnormalities documented in the usually "mild" Saethre–Chotzen syndrome have included retardation, epilepsy, cerebral cortical atrophy, enlarged ventricles, and wide interhemispheric fissures (Cantrell et al., 1994). This present unusual case also reinforces the observation that upper airway obstruction, which have been problematic in several craniosynostosis syndromes (Mixter et al., 1990; Moore, 1993), should probably be considered applicable to all of them.

Until genetic markers can delineate the true distinctions and commonalities between the presently recognised acrocephalosyndactyly syndromes, we must continue to make the most careful observations we can and revise our definitions when necessary. This case does not fit accurately into any current categories. Although many of our patient's features are typical of Saethre–Chotzen syndrome and none are contradictory, we feel that her overall condition most strongly suggests Apert syndrome and consider this the most accurate diagnosis. In discussion generated by this case, it seems evident that other clinicians may also have observed similar features in children with Apert syndrome as the "best diagnosis." If this is correct, then it is necessary to redefine Apert syndrome to include this pattern of near-complete simple syndactyly, perhaps designated as a "type O" hand formation. The same logic would of course apply to its inclusion with either of the other syndromes mentioned. The unanswered question, in either case, is the prognosis for mental function and possible progressive dysfunction of the interphalangeal joints, both thought to be poorer with an Apert syndrome diagnosis.

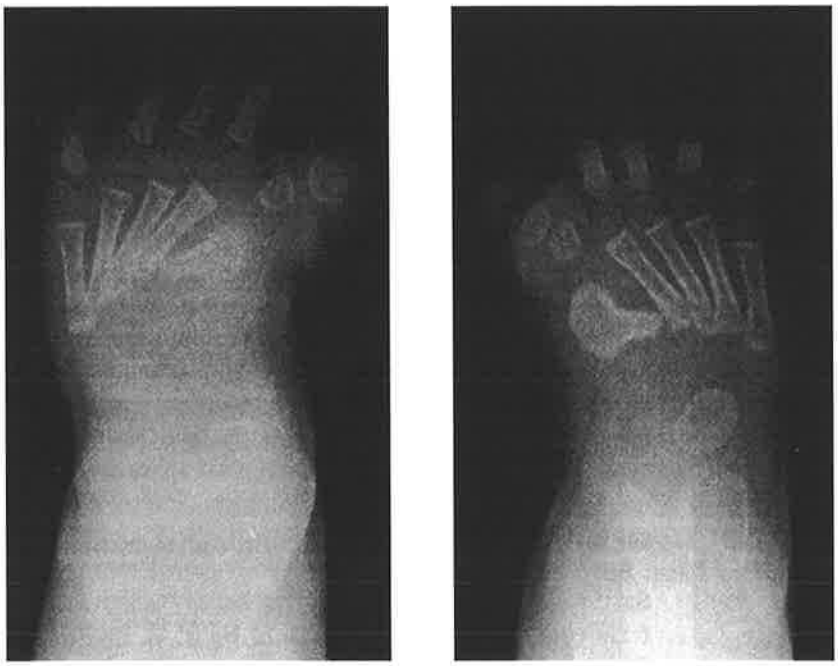

FIG. 7. *Radiographs of the feet show significant medial deviation of the metatarsals and phalanges, with separate bones.*

Acknowledgments

We thank Mr. Kent Wallis for his guidance in the surgical treatment of syndactyly in the acrocephalosyndactyly syndromes. Dr. Cantrell is the recipient of a clinical surgery fellowship from the Oral and Maxillofacial Surgery Foundation.

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Pfeiffer Syndrome: A Clinical Review

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The combination of bicoronal craniosynostosis, broad thumbs and great toes, and partial variable soft tissue syndactyly of the hands and feet (i.e., Pfeiffer syndrome) classically followed a benign clinical course. A review of the clinical features of those Pfeiffer syndrome patients presenting to our unit confirm another subgroup in whom the craniofacial manifestations are more extreme, with a significant risk of early demise. The early aggressive surgical management of craniostenosis, hydrocephalus, exorbitism, faciostenosis, and upper airway obstruction has provided the potential for prolonged useful survival in these cases.

KEY WORDS: *acrocephalosyndactyly, craniosynostosis, Pfeiffer syndrome*

Recent reports on Pfeiffer syndrome have identified a subpopulation in which the clinical features are extreme, the prognosis poor, and early demise in infancy common despite extensive craniofacial surgery (Cohen, 1993). These observations are in stark contrast to the relatively benign course outlined by Pfeiffer of the eight individuals in three generations of one family who manifested craniosynostosis, broad thumbs and great toes, and a variable partial soft tissue syndactyly of the hands and feet when he first described the syndrome that now bears his name (Pfeiffer, 1964). Subsequently classified as acrocephalosyndactyly type V (Tentamy and McKusick, 1969), Pfeiffer syndrome has been likened to Apert's syndrome with less severe expression, or alternately to Crouzon syndrome with superimposition of limb anomalies. Failure to demonstrate transition from one to the other in pedigree studies confirms the distinct nature of Pfeiffer syndrome.

Detailed study of the clinical features, radiographic findings, natural history, and management approaches to Pfeiffer syndrome is limited. A critical review of the accumulated case reports from the literature has seen Cohen (1993) subgroup Pfeiffer syndrome according to clinical features, associated low frequency anomalies and outcome. Thus type 1, which corresponds to the classic Pfeiffer syndrome, is associated with a satisfactory prognosis, whilst types 2 and 3, subgrouped according to the presence or absence of the cloverleaf skull anomaly, have a uniformly poor outcome with death in infancy common. In contrast, it is the management of the faciostenosis and upper airway compromise in these severe cases which provides the possibility for survival beyond this time with the potential for unimpaired intellectual development (Lodge et al., 1993; Moore, 1993).

This paper details the clinical and radiographic findings, treatment, and outcome in a consecutive series of 14 patients with Pfeiffer syndrome examined by the members of The Australian Craniofacial Unit.

Dr. Moore is Plastic Surgeon, Dr. Cantrell is Craniofacial Fellow, Dr. Trott is Craniofacial Surgeon, and Dr. David is Head of Unit. All Drs. are at The Australian Craniofacial Unit, North Adelaide, South Australia. Submitted April 1994; Accepted August 1994.

Patients and Methods

Since 1980, 14 patients with Pfeiffer syndrome have been evaluated by The Australian Craniofacial Unit. Two patients were assessed at an overseas clinic, without undergoing a complete multidisciplinary examination and died prior to transfer to this unit. The remaining patients have all had a detailed clinical examination with specific attention to craniofacial, neurological, ophthalmologic, upper airway and trunk, and limb skeletal anomalies.

Plain radiographic examination, and since 1983, complete two–dimensional computed tomographic (CT) scan assessment of the Craniofacial region with production of three-dimensional CT reconstructions, has been standard.

Clinical assessment of the airway by pediatric respiratory physicians has been augmented by endoscopy of lower respiratory tract, oximetry, and formal sleep studies using multichannel polysomnography. Where hypopnea and apnea are frequent and oxygen desaturations marked, early referral for airway management has been instituted.

Review by a medical geneticist to confirm the diagnosis has occurred in all cases. Classification according to the three subgroups of Cohen (1993) has been possible.

The approach to treatment has evolved over the period during which the patient population has been collected. The general philosophy remains for early release in infancy of craniosynostosis, correction of hydrocephalus, orbitostenosis and preservation of vision, and aggressive management of faciostenosis where it manifests as upper airway compromise. Subsequent surgical intervention during the intermediate years (1–10 years) is directed at the cranial, orbital, and facial levels as determined by clinical requirement. Final definitive midfacial and mandibular surgery is reserved until completion of growth.

The long-term outcome in this group of Pfeiffer syndrome patients using this approach is compared with previous reports and proposes recommendations for future management.

Results

The general features of the patients in this series are detailed inTable 1.

Most presented to this unit as new, sporadic cases less than 12–18 months old. Presentation during the intermediate years (1–10 years) occurred in three patients, while two cases were reviewed late (greater than 10 years) (Fig. 1).

Classical Pfeiffer syndrome (type 1) was observed in three patients including a mother and daughter who were the only familial cases in the series (Figs. 1 and 2). Four patients presented with the cloverleaf skull anomaly (type 2). Some features of these cases have been reported previously (Lodge et al, 1993) (Fig. 3). The remaining seven cases presented with clinical evidence of cranio-orbito-faciostenosis, without the cloverleaf skull anomaly and hence equated to the type 3 group (Fig. 4).

Two infants with type 3 deformity (severe craniostenosis extreme proptosis, and upper airway obstruction) were examined at outreach clinics, but they died before transfer to the unit could be facilitated.

TABLE 1

Patient Data

	Case	Sex	Age at Referral	Prior Surgical Intervention	(Number)
Cohen Type 1					
1.	ST	M	23 years	Yes	(1)
2.	SK	F	27 years	Yes	(1)
3.	NK	F	Birth	No	
Cohen Type 2					
4.	NP	F	11 weeks	No	
5.	GB	F	30 weeks	No	
6.	JB	M	8 years	Yes	(5)
7.	JV	F	Birth	No	
Cohen Type 3					
8.	Bl	F	20 months	Yes	(6)
9.	AF	M	3 months	No	
10.	LR	M	3 months	No	
11.	AR	M	13 months	No	
12.	GP	M	2 months	No	
13.	YH	F	8 months	Yes	(3)
14.	RA	F	1 month	No	

Five patients had undergone linear craniectomies and/or ventriculoperitoneal shunting prior to referral to this unit (see Table 1).

The clinical findings, natural history, and management approach to Pfeiffer syndrome as displayed in this consecutive series is detailed as follows:

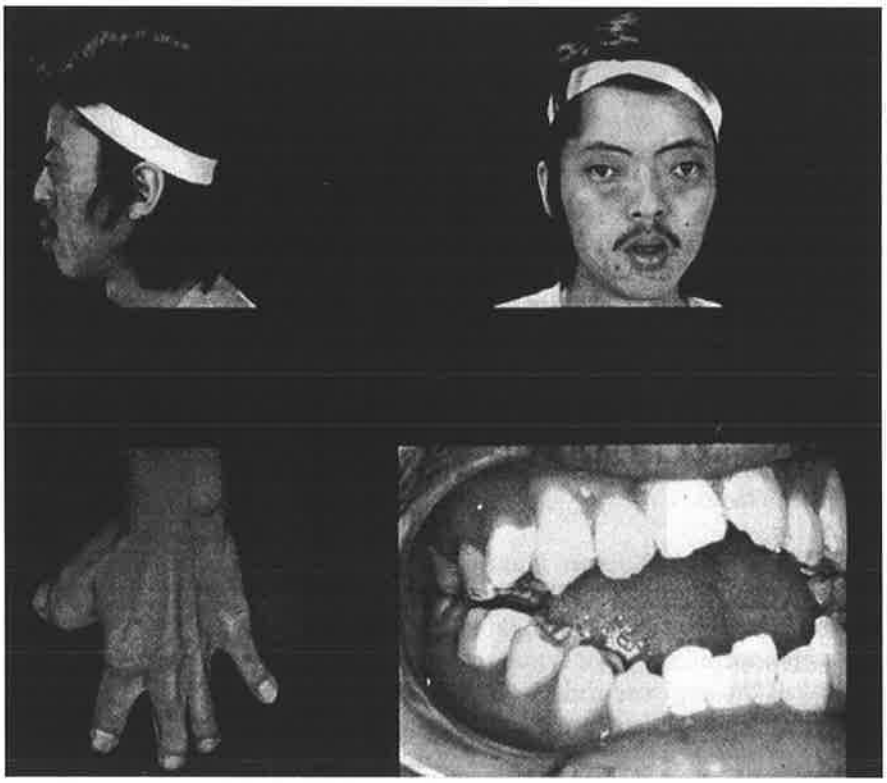

FIG. 1. *Patient 1: Classical Pfeiffer syndrome type 1, presenting having only had craniectomies as an infant (upper left & right). Typical hand features of broad, radially deviated thumbs, and partial soft tissue syndactyly (lower left). Midfacial hypoplasia with anterior open bite deformity (lower right).*

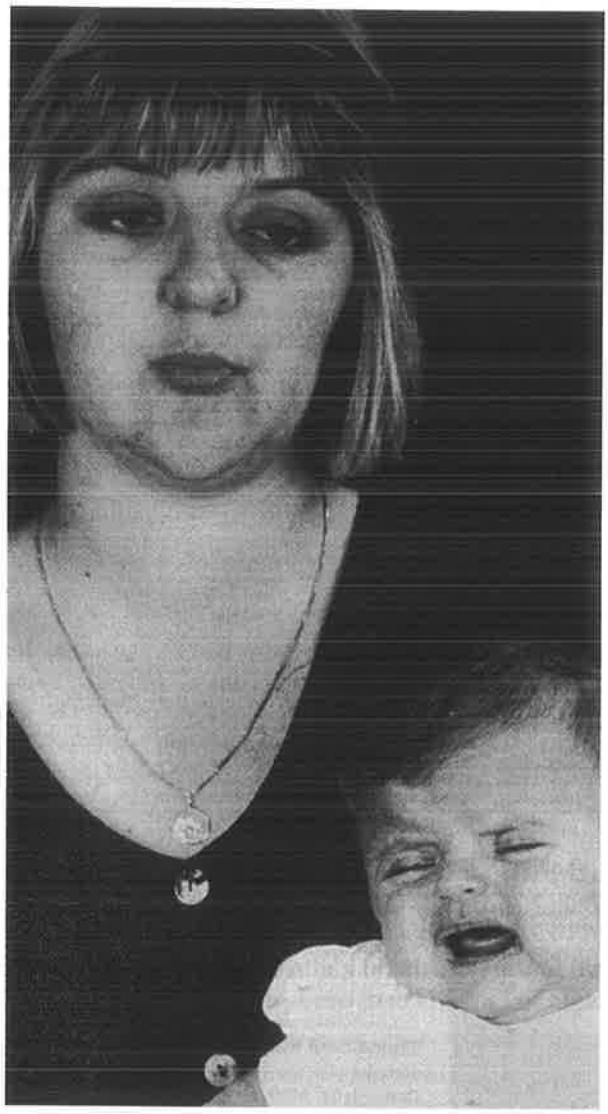

FIG. 2. *Patients 2 and 3: Pfeiffer syndrome—familial cases; mother and daughter both type 1 cases.*

1) Calvarium and Cranial Base (Table 2)

Turricephaly was the calvarial deformity most frequently noted. Bilateral coronal synostosis was the radiologic and histologic norm in this series with variable involvement of other sutures concordant with the severity of calvarial distortion.

The sole infant type 1 case viewed had been diagnosed on antenatal ultrasound (the only familial case in this series). The two remaining type 1 cases were referred late, having previously undergone bicoronal linear craniectomies as infants. Moderate hypertelorism and turricephaly were the significant clinical features at this time.

All of the group with severe manifestations but no cloverleaf anomaly (type 3), presented early with consistently documented bicoronal synostosis. Coincident metopic and anterior sagittal synostosis was noted in one case producing an unusual trigonocephalic appearance, or frontal gibbosity, while another had involvement of the sagittal and lambdoid sutures (see Fig. 4).

The four patients with cloverleaf skull anomaly (type 2) all had extensive premature sutural fusion, with characteristic bitemporal bulging (see Fig. 3).

TABLE 2

Patterns of Craniosynostosis

	Radiographically Fused Suture			
	Bicoronal	**Metopic**	**Sagittal**	**Lambdoid**
Cohen Type 1 (n= 1)	1	—	—	—
Cohen Type 2 (n=4)	4	—	4	4
Cohen Type 3 (n=4)	4	1	1	3

(2) Ventricular and Cerebral Anomalies

Clinical neurologic and CT data were available for 11 cases assessed. The type and pattern of ventricular abnormality is detailed in Table 3.

A normal size and pattern to the ventricular system was noted only in a single case. Most cases manifested marked ventricular dilatation early, which was persistent after fronto-orbital advancement of such severity as to require surgical intervention.

One case in the cloverleaf skull group remains with chronic uncontrolled hydrocephalus following repeated shunt revision. This child is severely retarded aged 4.5 years (see Fig. 3).

Blindness occurred in two cases, where management of raised intracranial pressure was inadequate.

Macroscopic cerebral anomalies in this series were uncommon and minor in degree. Magnetic resonance imaging (MRI) scan data is available only on the most recent case (case 14) who was both blind and deaf on late presentation here. No significant primary cerebral anomaly was evident in this case.

Adequate psychometric evaluation was available only in two cases (cases 1 and 6) both of whom function in the average intelligence range (IQ 90–109).

(3) Orbital and Ocular Anomalies

Orbitostenosis, marked by extreme proptosis, was evident in all the type 2 and type 3 patients (see Figs. 3 and 4) (Table 4). Midfacial retrusion is coincident in these cases and this deficiency of inferior support contributes significantly to the exorbitism. A variety of secondary eyelid and globe anomalies develop as a consequence of the foreshortened orbit (antimongoloid obliquity of the palpebral fissure lid retraction, globe prolapse, and corneal scarring) with the attendant risk to maintenance of vision.

The type 1 cases have mild degrees of exorbitism which is of little consequence to maintaining the visual pathways.

Two patients manifested blindness consequent upon unrecognised or untreated raised intracranial pressure as detailed above.

TABLE 3

Ventricular Abnormalities

Ventriculomegaly	Ventricular Pattern			
	Normal Ventricles	Mild	Moderate	Marked
Cohen Type 1 (n=2)	1	1	0	0
Cohen Type 2 (n=4)	0	0	0	4
Cohen Type 3 (n=5)	0	1	1	3

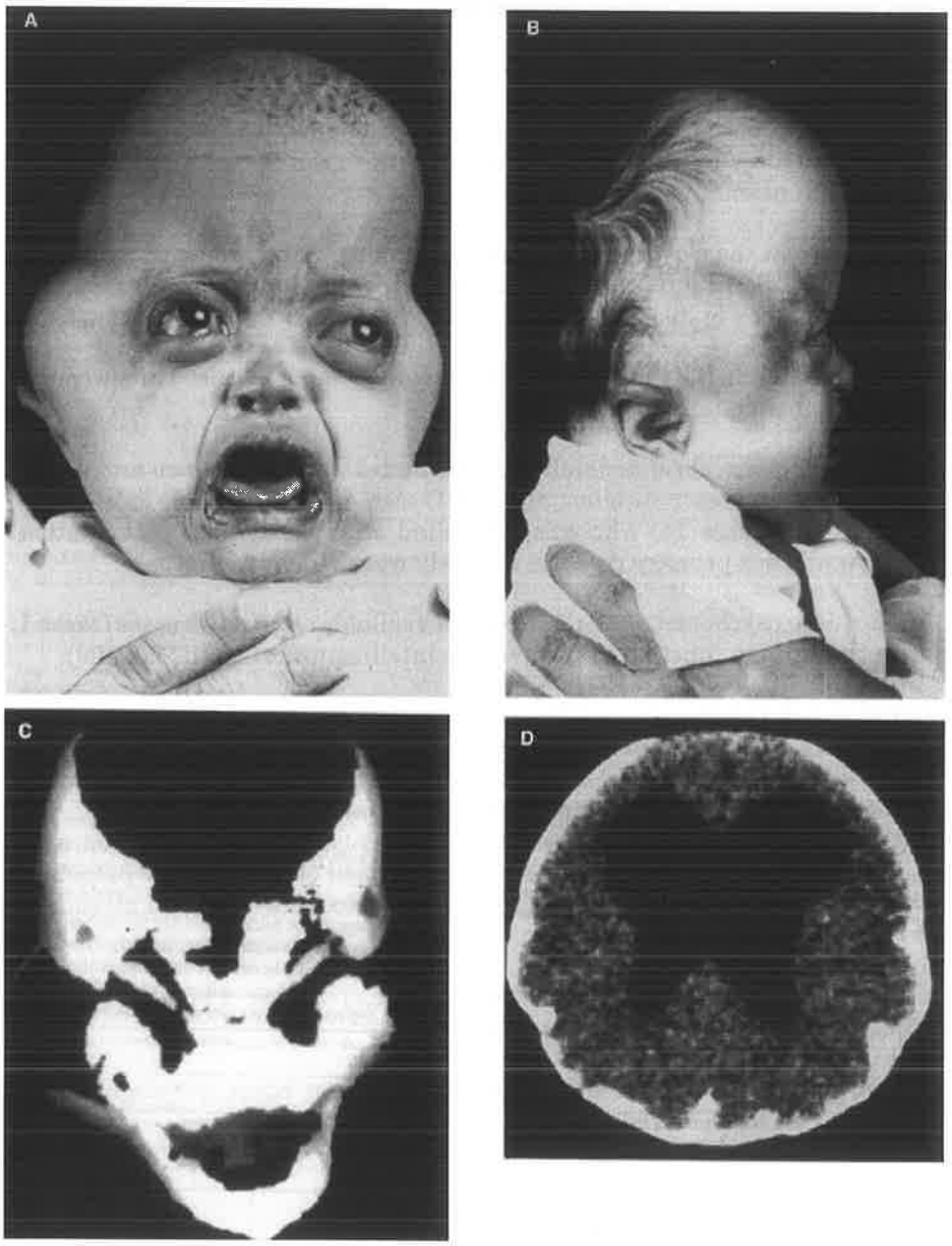

FIG. 3. *Patient 4: Pfeiffer syndrome type 2.* **A.** *Cloverleaf skull appearance.* **B.** *Gross anteroposterior shortening of the cranial base and marked proptosis.* **C.** *3D CT scam confirms the bitemporal and sagittal bulging.* **D.** *2D CT brain scan reveals the significant ventricular dilation.*

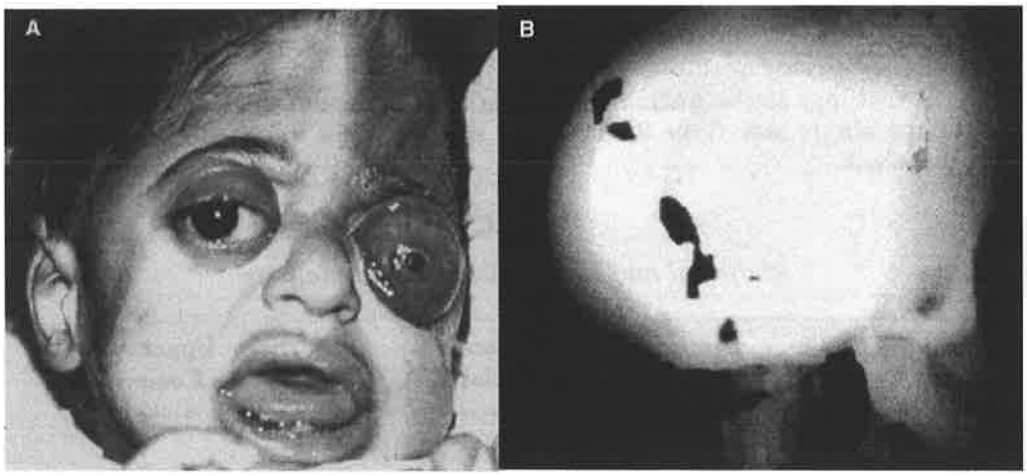

FIG. 4. *Patient 11: Pfeiffer syndrome type 3.* **A.** *Unusual trigonocephalic appearance, with metopic synostosis accompanying the bicoronal synostosis. Extreme proptosis with prolapse of the left eye.* **B.** *Lateral 3D CT scan confirms the atypical frontal appearance.*

TABLE 4

Ophthalmologic: Major Abnormalities

	Orbitostenosis (Proptosis, Exorbitism)	Blindness
Cohen Type 1 (n = 3)	0	0
Cohen Type 2 (n=4)	4	0
Cohen Type 3 (n = 7)	7	2

(4) Midfacial and Upper Airway Anomalies (Table 5)
Midfacial hypoplasia was evident in the untreated cases which presented early. In the older patients midfacial retrusion, class III malocclusion, and open bite deformity become more marked.

The principal early functional consequence of the facial anomalies is that of significant upper airway compromise and obstructive sleep apnea. Upper airway obstruction as assessed by overnight monitored pulse oximetry, or formal overnight sleep studies, was present in five of the nine of the early presenting cases. These patients all required surgical procedures to secure their airway (Moore, 1993).

Two of the early presenting group had mild airway obstructive symptoms, which did not demand aggressive surgical intervention. The remaining two early presenting cases did not demonstrate obstructive sleep apnea.

The two infant Pfeiffer syndrome type 3 cases seen in overseas clinics had severe upper airway obstruction, which was a major factor in their demise prior to transfer to this unit.

The only case referred during the intermediate period had previously undergone adenotonsillectomy for upper airway obstruction. Sequential soft tissue and skeletal surgery has been necessary to ensure continued upper airway patency.

Neither of the two late presenting cases (both type 1) had airway obstructive symptoms.

(5) Other Features

i *Lower airway anomalies*
A cartilage sleeve anomaly affecting the tracheobronchial tree was noted in a single case (type 2). This was significant in the early death of this patient.

TABLE 5

Midfacial and Upper Airway Abnormalities

	Midface Hypoplasia	Upper Airway Compromise
Cohen Type 1 (n=3)	3	0
Cohen Type 2 (n = 4)	4	3
Cohen Type 3 (n=7)	7	5

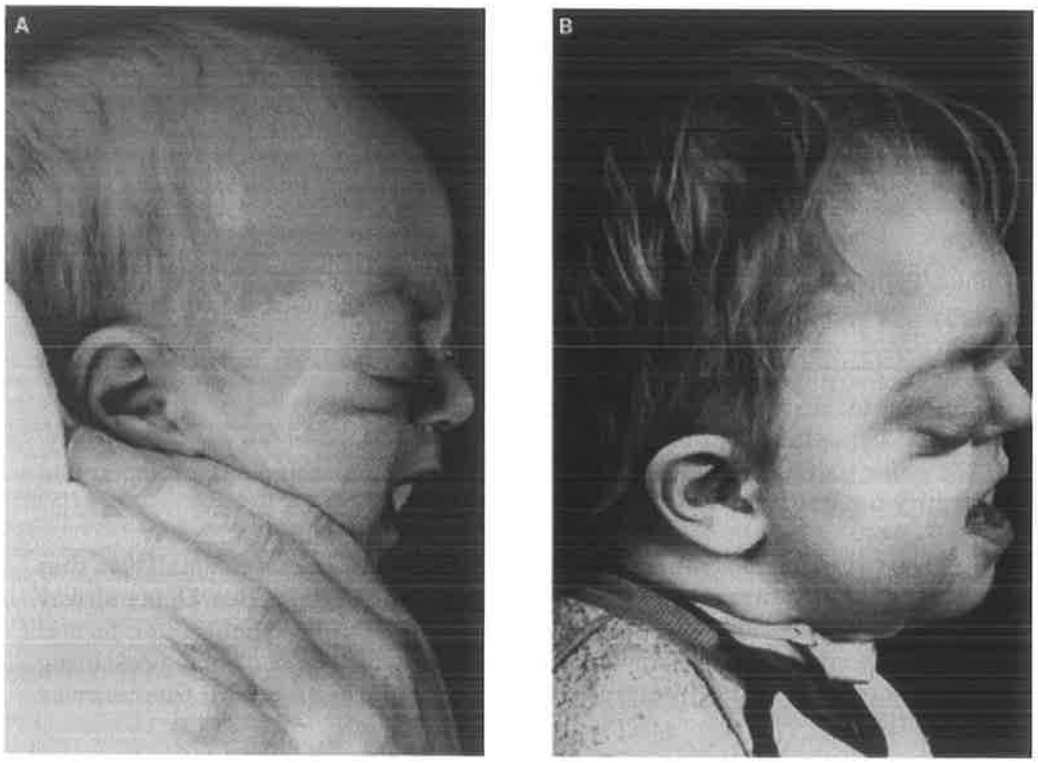

FIG. 5. *Patient 9: Pfeiffer syndrome type 3.* **A.** *Right lateral view at 3 months. Significant proptosis, but a relatively normal, if small head.* **B.** *Age 3 years, following infant fronto-orbital advancement, now has gross proptosis, midfacial retrusion, and tracheostomy for airway obstruction.*

ii *Otologic anomalies*
Idle ear effusion in conjunction with the stenotic pharyngeal space was a universal finding. An unrelated significant hearing deficit was noted in one type 3 case.

iii *Spinal anomalies (Table 6)*
Vertebral architectural anomalies are common without a localizing pattern. Cervical spine fusions are frequent the pattern of fusion complex. Isolated anomalies were evident at lower levels, including two cases of sacrococcygeal eversion.

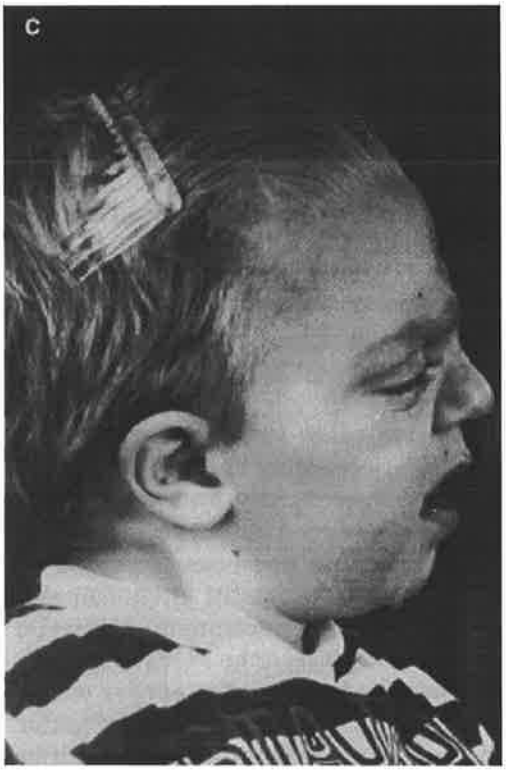

FIG. 5c. *Patient 9: Pfeiffer syndrome type 3.* **C.** *Age 5.5 years following Le Fort III advancement with improved facial profile, and tracheostomy removed after resolution of airway symptoms.*

iv *Limb anomalies*

All cases have demonstrated the diagnostic symmetric, broad, radially deviated thumbs, and broad medially deviated great toes (see Fig. 1). Elbow ankylosis was the next most frequently recorded limb anomaly, in four patients (all type 2 and 3 cases).

TABLE 6

Spinal Anomalies

	Cervical	*Thoracic*	*Lumbar*	*Sacrococcygeal*
Cohen Type 1 (n=2)	0	0	0	0
Cohen Type 2 (N=4)	4	1	1	1
Cohen Type 3 (N=5)	3	0	0	1

Surgical Intervention

The surgical procedures performed on this series is outlined in Table 7. The variation in age of presentation, as well as severity of phenotypic expression contributes to the types of intervention required. Two cases had no surgical procedures, these patients died of airway-related complications prior to transfer.

Outcome

Follow-up into adulthood is available in only two Cohen type 1 cases, both of whom function normally. Morbidity, particularly in regard to the surgical treatment of the ventricles and the upper airway, is detailed in Table 8.

Discussion

The definition and understanding of Pfeiffer syndrome has undergone a transition in recent years. That it is not Crouzon syndrome, with superimposition of limb anomalies, or Apert's syndrome, with less severe expression, but rather an entity in its own right, is evident both on clinical presentation and family studies. The constellation of clinical features which comprise Pfeiffer syndrome seemed to be associated with a comparatively benign course as reported in the initial familial cases (Pfeiffer, 1964). In contrast, a recent review (Cohen, 1993) has identified groups of Pfeiffer syndrome patients who are characterised by severe neurologic compromise, poor prognosis, and early death (type 2 and 3).

This series, representing the consecutive cases reviewed in a single institution, can be subgrouped in a similar manner according to clinical features. The type 1 or classic Pfeiffer syndrome has been infrequently seen in this unit. These are the only familial cases reviewed and have a good long-term prognosis. Cranial vault deformity is characteristically mild and has been managed by craniectomy of the involved sutures and early fronto–orbital advancement. Cerebral, ventricular, and oculo–orbital anomalies are uncommon. Midfacial hypoplasia is less marked and corrected by either Le Fort III or Le Fort I osteotomy or both at the completion of growth. Sometimes the treatment is earlier where psychosocial, or the less frequent functional problems seen with type 1 Pfeiffer syndrome precipitate it. The mother of the only child seen with type 1 Pfeiffer syndrome has not had any midfacial surgery.

TABLE 7

Surgical Intervention

Case	Orbito-Cranial	Orbito-Facial	Other
Cohen Type 1			
1	—	Le Fort III	—
2	Coronal craniectomy	—	—
3	Fronto-orbital advancement	—	—
Cohen Type 2			
4	Fronto-orbital advancement Ventriculo-peritoneal shunt	—	—
5	Fronto-orbital advancement Ventriculo-peritoneal shunt	 Tarsorrhaphies	Tracheostomy
6	Fronto-orbital advancement	Le Fort III	Uvulopalatoplasty
7	Fronto-orbital advancement Lambdoid craniectomy Ventriculo-peritoneal shunt Decompressive craniectomy	—	Adenoidectomy Palatal split
Cohen Type 3			
8	Fronto-orbital advancement Lambdoid craniectomy	Le Fort III Optic nerve decompression	Tracheostomy Adenotonsillectomy
9	Front-orbital advancement Ventriculo-peritoneal shunt	Tarsorrhaphies Fronto-facial advancement	Tracheostomy
10	Fronto-orbital advancement Ventriculo-peritoneal shunt	—	—
11	Fronto-orbital advancement	Le Fort III	Uvulopalatoplasty Palatal split
12	—	—	—
13	Front-orbital advancement Ventriculo-peritoneal shunt	—	Uvulpalatoplasy
14	—	—	—

TABLE 8

Morbidity and Outcome

Case	Morbidity	Outcome
Cohen Type 1		
1	—	Alive age 36 years
2	—	Alive age 29 years
3	—	Alive age 2 years
Cohen Type 2		
4	Gross hydrocephalus and intellectual retardation	Alive age 4½years
5	Hydrocephalus	Died age 15 months
6	Persisting upper airway obstruction	Alive age 12 years
7	—	Alive age 2 years
Cohen Type 3		
8	Blind	Alive age 9 years
9	—	Alive age 6 years
10	—	Alive age 3 years
11	—	Alive age 4 years
12	Upper airway obstruction	Died age 2 months
13	Hydrocephalus, blind and severe intellectual retardation	Alive age 18mths
14	Upper airway obstruction	Died age 1 month

The prognosis for types 2 and 3 Pfeiffer syndrome was described by Cohen (1993) as being uniformly poor with early demise. While this is the outcome where each of the elements of the craniofacio–orbitostenosis are not able to be dealt with adequately, we have seen prolonged survival with an acceptable outcome. Head shape and hydrocephalus aside, both groups (types 2 and 3) appear to behave similarly with respect to the frequency of intracranial anomalies, ocular proptosis, upper airway compromise, and vertebral and limb anomalies. In both types, the premature fusion of multiple sutures demands early extensive release and cranial vault remodeling. Ventricular shunting and repeated cranial vault decompression has been necessary in a number of cases. Despite these maneuvers, uncontrolled hydrocephalus, persistently raised intracranial pressure and blindness. A diminished level of general neurologic function also occurred.

The short anterior cranial base, with consequent extreme anteroposterior shortening of the orbit and midfacial retrusion, means that support and protection of the globe can only be anticipated after advancement of the midface. Tarsorrhaphy, canthal ligament release, and levator lengthening provide only short-term solutions to the coverage of the proptotic globe.

Airway obstruction in these cases is the result of inputs at multiple levels of both upper and lower airway (Stone et al., 1990; Cohen and Kreiborg, 1992; Moore, 1993). In the early months of life, uncorrected airway compromise can result in death: the major factor in the death of the two type 2 cases seen in overseas clinics. In the longer term, the chronic upper airway compromise potentially contributes significantly to cardiac and neurologic morbidity. Chronic hypoxemia, in the clinical environment where intracranial pressure is elevated and difficult to manage, may become a major factor in impaired neurologic development and long-term intellectual outcome. A staged combination of soft tissue and skeletal surgical interventions have proven useful in "buying time" for these cases (Moore, 1993). Where previously a clinical presentation with

extreme upper airway obstruction meant a direct step to tracheostomy, an alternative approach involving surgical enlargement of the nasopharyngeal space has been successful. Resolution of airway obstruction was produced by resection of the soft palate, posterior to the levator palati muscle, proceeding where necessary to the creation of a soft palate "pseudocleft" and/or adenotonsillectomy. Where airway obstructive symptomatology is recurrent after these soft tissue interventions, upper level midfacial advancement has been used with good effect. The initial soft tissue surgery delayed this procedure and allowed further maturation of the facial skeleton with improved spatial stability (Moore, 1993). The temporal nature of the changes in the upper airway patency, with the superadded alterations to the adenotonsillar lymphoid tissue, means airway function requires constant monitoring to minimize this significant potential threat to long–term survival and optimal intellectual function. Hearing loss has been recorded in Pfeiffer syndrome (Martsolf et al., 1971; Cremers, 1981). Conductive type hearing loss is possible consequent upon ankylosis of the auditory ossicular chain (Cremers, 1981). A significant bilateral hearing impairment was noted in one infant in this series, however, the anatomy of the middle ear cleft was not adequately imaged. Hearing loss needs to be considered in these cases as it represents another potential impairment of sensory input and neurologic development.

Few patients in this series have been followed to adulthood. The two type 1 cases both appear to function in the normal intellectual range, as does the type 2 teenage patient. The type 2 and 3 patients treated during the early period in the evolution of our management plan have had a worse survival and intellectual functioning outcome. The later patients, managed by aggressive early multilevel and multisystem intervention, are now surviving beyond infancy with the prospect of long-term functioning in the normal range.

To concentrate solely on the craniofacial anomalies of Pfeiffer syndrome risks ignoring the other clinical features which may impact negatively on function. Major cartilaginous anomalies of the trachea have been reviewed previously in Crouzon, Apert's, and Pfeiffer syndrome (Stone et al., 1990; Cohen and Kreiborg, 1992). One case in this series manifested an extensive cartilaginous sleeve abnormality involving the trachea, which was a significant factor in an ultimate early demise.

The classically described thumb and great toe findings (all cases) are among the other cartilaginous anomalies. Despite their requirement for a valid diagnosis, considerable variation exists in both the soft tissue syndactyly and bony anomalies. To help clarify the requirements for a diagnosis of Pfeiffer syndrome, Cohen (1993) proposed a width ratio of 1.91 (range 1.72–2.23) between the great toe and second toe in affected individuals. Similar criteria for the thumb have not yet evolved, as imaging of the hand has often been inadequate.

More proximally, elbow ankylosis was a feature, particularly of the more severe cases (Cohen, 1993). While no surgical corrections have been performed, elbow immobility may impact markedly on the ability to function independently.

Vertebral anomalies were common in the cervical region, without identifying the consistent patterns of Crouzon or Apert's syndrome (Hemmer et al., 1987; Kreiborg et al., 1992). Appropriate assessment of instability is indicated, particularly where major craniofacial surgical movements are planned.

The natural history of Pfeiffer syndrome can be better defined and medical intervention and support prioritized with careful documentation of the patterns of cranial vault and base growth disturbance, cerebroventricular anomalies, orbitofacial deformities, and extracranial features. A relatively benign form of Pfeiffer syndrome (type 1 or classical) exists. In these cases, staged sequential management of the craniosynostosis in infancy and the orbito–faciostenosis in later years produces a good functional and esthetic outcome. The alternative

sinister form of Pfeiffer syndrome is characterised by multiple suture craniostenosis and hydrocephalus. The pattern and temporal evolution of suture involvement determines the cloverleaf (type 2) or craniostenotic, but non-cloverleaf (type 3), head shape. In these extreme cases, the cranio–orbito–faciostenosis demands aggressive early suture release, hydrocephalus management, orbital expansion, and airway decompression to permit survival to childhood and beyond. Instead of uniform early demise, it has been possible with this approach, to achieve prolongation of life into childhood and beyond with a good level of function in more than half of our cases. Ongoing detailed assessment and intervention in hydrocephalus and upper airway compromise remains essential if progress is to continue.

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