CLINICAL APPLICATIONS OF SOMATOSENSORY EVOKED POTENTIALS IN PEDIATRIC NEUROSURGERY

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SUMMARY

This thesis examines the utility of SEP studies in several areas of clinical and operative pediatric neurosurgery. The clinical studies use routine neurophysiological methods to record surface spinal, subcortical and cortical SEPs following electrical stimulation of the tibial and median nerves. Findings in normal children (100) were compared to those in children with surgical myelopathies (26), intracerebral lesions (7) and following major head injury (14).

In normal children characteristic SEP waveforms could be recorded over the lumbar and cervical spine, Erb's point and the scalp. Certain SEP components had latencies that were related to the age and size of the subject, whilst central conduction times showed no significant variation during the first decade. Many children with motor and sphincteric deficits due to congenital and neoplastic spinovertebral disorders had normal SEPs whilst those with impairment of dorsal column modalities had loss of SEPs. Children with intracerebral space occupying lesions often had either loss of short or long latency cortical SEP components and ipsilateral attenuation of waveform amplitude. Following severe head injury many children had normal short latency cortical SEPs and these patients had a favourable outcome. Children with bilateral distortion of the short latency cortical SEP had vegetative outcomes, whilst patients with unilateral distortion of this complex suffered focal neurological deficits.

The studies undertaken during spinal surgery utilized spinal SEPs (26 cases) and cortical SEPs (13 cases) to monitor spinal cord function. Intraoperative monitoring was much quicker and simpler using SEPs recorded from the epidural or subdural plane rather than cortical SEPs. Patterns of spinal SEPs were characteristic of different spinal segments. Distortion and asymmetry of baseline spinal SEPs was seen in several patients with intramedullary tumours. Loss of waveform components during surgery occurred with profound hypotension, overdistraction of the vertebral axis, dorsal midline myelotomy and removal of intramedullary tumors. Persistant loss of waveform components was associated with an acquired neurological deficit. Fluctuations in SEP amplitude were common but were not associated with post operative deficits. An experimental study in sheep revealed that some of this amplitude fluctuation may be attributable to halothane anesthesia.

These studies suggest that SEPs can provide prognostic information following pediatric neurotrauma, and that they are superior to the "wake up" test during spinal surgery for scoliosis. The use of SEPs to evaluate children with intracerebral and spinovertebral disorders, and to monitor spinal function during intramedullary and caudal spinal surgery is however limited by technical, anatomical, clinical and pathological factors.

STATEMENT

I declare that this Thesis is my own composition, and that it is a record of original work that I carried out in the Department of Neurosurgery, Royal Alexandra Hospital for Children, Sydney, Australia in 1983 and 1984. All somatosensory evoked potential recordings and clinical examinations of the patients documented in this Thesis were performed personally.

The Thesis contains no material which has been submitted for the award of any other degree or diploma, and to the best of my knowledge contains no material previously published or written by another person except where due reference is made in the text.

The author consents to the thesis being made available for photocopying if accepted for the award of the degree.

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PUBLISHED WORK

The following articles represent work from this thesis that has been published, is in press or has been presented at international scientific meetings.

1. Whittle, I.R., Johnston, I.H., Besser, M. Monitoring Spinal Cord Function during surgery by direct recording of Spinal Somatosensory Evoked Potentials. Technical Note. J. Neurosurg. 60, 440-443, 1984.
2. Whittle, I.R., Johnston, I.H., Besser, M. et al, Intraoperative Spinal Cord Monitoring during surgery for scoliosis using Somatosensory Evoked Potentials. Aust. N.Z.J. Surg. 54(6), 553-557, 1984.
3. Baines, D.B., Whittle, I.R., Chaseling, R. et al, The Effect of Halothane on Spinal Somatosensory Evoked Potentials in Sheep. Brit. J. Anaesth. 57, 896-899, 1985
4. Whittle, I.R., Johnston, I.H.,Besser, M. Intraoperative recording of spinal somatosensory evoked potentials during surgery for the tethered cord syndrome. Riv. Neurosci. Ped./ J Ped Neurosc. 1 ; 178-186, 1985.
5. Whittle,I.R The uses and limitations of intraoperative spinal cord monitoring with somatosensory evoked potentials (Abstract). J Electrophysiol. Tech. (In press, 1985)
6. Whittle, I.R., Johnston, I.H., Besser, M. Intraoperative recording of cortical somatosensory evoked potentials as a method of spinal cord monitoring during spinal surgery. Aust. N. Z. J Surg (In press, 1985)

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7. Whittle,I.R., Johonston, I.H., Besser, M. Intraoperative recording of spinal somatosensory evoked potentials as a method of spinal cord monitoring during spinal surgery. J Neurosurg. (in press, 1985)
8. Whittle, I.R., Miller, J.D. Somatosensory evoked potential findings following pediatric neurotrauma (Abstract). Brit.J.Surg (In press, 1986).
9. Whittle, I.R., Johnston, I.H., Besser, M. Initial Experience with Intraoperative Spinal Cord Monitoring using Somatosensory Evoked Potentials. Proceedings 40th Annual Scientific Meeting of Neurosurgical Society of Australasia, Adelaide 1-4 Sept, 1983.
10. Whittle, I.R., Johnston, I.H., Besser, M. Variations in Somatosensory Evoked Potentials in Childhood. Proceedings of 1st Asian-Oceania Conference of Child Neurology, Taipei, Taiwan, 17-19 November 1983.
11. Whittle, I.R., Johnston I.H., Besser, M. Intraoperative spinal cord monitoring using somatosensory evoked potentials. Proceedings of the 8th International Congress of Neurological Surgery, Toronto, Canada, July 7-13, 1985

ABBREVIATIONS USED IN THE TEXT

Ag/AgCl	-	Silver/Silver Chloride
BAER	-	Brainstem auditory evoked response
C	-	Cervical
C3/C4	-	Midparietal point (C3=left, C4=right) of 10/20 International montage system
CNS	-	Central nervous system
CSCT	-	Central somatosensory conduction time
CT	-	Computerized tomogram
Cv2	-	Point over the spine of axis (C2)
Cv7	-	Recording site at point over spine of C7
Cz	-	Sagittal midpoint of 10/20 montage system
dB	-	Decibel
ECG	-	Electrocardiograph
EEG	-	Electroencephalograph
Fpz	-	Anterior frontal (forehead) point of 10/20 montage system
Fz	-	Midfrontal point of 10/20 montage system
GCS	-	Glasgow coma score
Hz	-	Hertz (cycles per second)
ICP	-	Intracranial pressure
L	-	Lumbar
mm Hg	-	Millimetres of mercury
ms	-	Millisecond
m/s	-	Metres per second
N	-	Negative
P	-	Positive

ABBREVIATIONS (Cont)

S	-	Sacral
SEP	-	Somatosensory evoked potential
T	-	Thoracic
uV	-	Microvolt
V	-	Volt
VER	-	Visual evoked response

PREFACE

In the two decades since Clark and colleagues (1961) described the average response computer, further advances in computer and microprocessor based technology have enabled the study of evoked potentials to be transferred from the confines of the research laboratory to clinical medicine. This transformation has been facilitated by the availability of moderately priced, compact, signal averaging units. These machines have the facility for rapid signal acquisition and summation, immediate reproduction or display of the evoked potential waveform, and may often be integrated with computer hardware that enables storage and rapid retrieval of previous studies. Further impetus for electrophysiological studies of the central nervous system has developed with experimental and clinical advances in neuroanatomy, neuropathology and neuroimaging that have enabled a correlation between neurophysiological and structural or ultra-structural findings.

Evoked potential studies are now used as a diagnostic or monitoring tool in a wide variety of disorders in both clinical neurology and neurosurgery (see reviews Courjon et al, 1982; Greenberg & Ducker, 1982; Grundy, 1982; Chiappa & Ropper, 1982a, 1982b; Grundy, 1983; Bunch et al, 1983). Their widespread and increasing clinical importance and application have resulted in a proliferation of publications on VERs, SEPs, BAERs and the establishment of a separate edition of the eminent journal Electroencephalography and Clinical Neurophysiology that is devoted solely to evoked potential studies (Brazier, 1984).

The aim of this treatise was to examine both clinically and experimentally some applications of SEP recording in pediatric neurosurgery. Normative data for short latency spinal, subcortical and cortical SEPs in children was obtained, and changes in these SEPs that occurred with congenital, neoplastic and acquired CNS disorders were studied. New modes of intraoperative monitoring of spinal SEPs were developed, applied and evaluated in both clinical and experimental surgery. The results from these studies were evaluated so that the clinical utility and limitations of SEP recordings in particular aspects of pediatric neurosurgery could be objectively assessed and areas and techniques for future evaluation discussed.

2. INTRODUCTION

2.1 HISTORICAL ASPECTS

2.1.i Short Latency Cortical and Subcortical SEPs

Changes in the electrical activity of the brain in response to sensory stimulation were initially described by Richard Caton, a Lecturer in Physiology at the Royal Infirmary School of Medicine, Liverpool. In a series of experiments between 1875 and 1891 he noticed that electrical currents in certain areas of the rabbit brain were generated by exposure to candle-light and somatosensory stimulation (cited in Brazier, 1984). Further experimentation on the response of the animal brain to both sound and light were performed in the late 19th and early 20th century by Adolf Beck, Vladimir Larionov and Vladimir Pravdich-Neminsky, who is accredited with the first pictures of the EEG and SEP in the literature (Brazier, 1984). This pioneering work must be considered remarkable because of the accuracy of the neurophysiological findings when considering the crude methods of evoking sensory responses (e.g. shouting, magnesium flares and candle-light) and the limitations in contemporary electrodes and galvanometers.

Following the lengthy hiatus caused by two World Wars and the Great Depression, G.D. Dawson from the National Hospital for Nervous Diseases in London developed a novel approach to recording the response of the brain to electrical stimulation of peripheral nerves. His methodology was based upon the hypothesis

that electrical stimulation of the peripheral nerve would produce an evoked potential that was time locked relative to the stimulus, and that recording the evoked potential generated by repetitive stimuli would minimise the effects of random brain-wave activity and extraneous electrical artefact. Initially he used superimposition of brain wave recordings to emphasise the cerebral response to electrical stimulation of the median and ulnar nerves (Dawson, 1947). Later he developed a technique of summating the small amplitude cortical SEP by synchronising the repetitive electrical stimulus to the peripheral nerve with a series of condensers that discharged at a given time interval after stimulus (Dawson, 1954). A similar technique using a rotating drum, and a write-out in which the pen deflection was related to the voltage of the signal, was described by Barlow (1957). This work confirmed that the cerebral response to electrical stimulation of the peripheral nerves was recordable, however technological advances were needed to more readily extract the small amplitude evoked potential (0.1 - 20mV) from background EEG activity.

The advent of the average response computer in 1961, (Clark et al, 1961) heralded the onset of electronic computer averaging units that are currently in use. Advances in computer and microprocessor based technology have facilitated the recording of evoked potentials,so that now non-invasive methods can be utilised. Sensory stimulating units are now synchronised with signal acquisition facilities that enable instantaneous summation and display of the evoked potential, often with simultaneous multiple channel recording over a desired range of frequency

filter settings and epochs. Many of the averaging units are now portable, which allows easier patient access, and are compatible with computer hardware that enables sophisticated and extensive data storage and retrieval. These technological refinements have led to a proliferation of the use of evoked potentials in clinical medicine (Chiappa and Ropper, 1982a).

Brazier (1960) was the first to apply computer averaging techniques in experimental neurology. Subsequently many other workers have used similar techniques in the clinical and experimental evaluation of somatosensory pathways. This work has involved analysis of long and short latency components of both subcortical and cortical SEPs recorded in response to peripheral nerve stimulation (Williamson et al, 1970; Jones, 1977; Krichevsky and Wiederholt, 1978; Desmedt and Cheron, 1980; Allison et al, 1980; Anziska and Cracco, 1981; Lesser et al, 1981; Ganes et al, 1982; Lueders et al, 1983; Yamada et al, 1984; Emerson et al, 1984). These studies have delineated the field distribution and morphology of the subcortical SEPs recorded over the midclavicular region (Erb's point), the cervico-medullary junction (Cv2) and scalp recorded cortical SEP components. Most studies have concerned the electrical response of the somatosensory pathways to median nerve stimulation however, the ulnar, tibial, peroneal, and other smaller peripheral nerves have also been studied (Tsumoto et al, 1972; Dimitrijevic et al, 1978; Jones and Small, 1978;Cracco et al, 1979; Eisen et al, 1980; Chehrazi et al, 1981; Vas et al, 1981; Rossini et al, 1981; Lueders et al, 1983; Iragui, 1984; Tsuji et al, 1984a and 1984b).

Much of the more recent work in SEP neurophysiology concerns definition of the neuroanatomical generator sites and the spatial distribution of various SEPs. Multiple recordings from different montages and intraoperative intracranial recordings are being used to clarify the origin and topography of near and far field SEPs (Desmedt and Cheron, 1980 and 1981; Lesser et al, 1981; Suzuki and Mayanagi, 1984; Yamada et al, 1984; Emerson et al, 1984; Small and Mathews, 1984). Such fundamental considerations are essential for the successful application of SEP waveforms in clinical medicine.

An evaluation of the rapidly proliferating literature on SEPs is compounded however by the problem of the diversity of stimulating and recording techniques used, prescribed and pioneered by various laboratories. These methodological considerations have led to some confusion and difficulty in interpreting and comparing work describing the response of a particular somatosensory pathway. Although evoked potential data has to some extent been standardised by the used of specific recording montages, adopted from the International 10/20 recording system (Jasper, 1958) the relative advantages of near field and far field SEP recording techniques using these montages in clinical medicine is still being evaluated (Anziska and Cracco, 1981; Lueders et al, 1983; Small and Mathews, 1984).

2.1.ii SPINAL SEPs

Experimental neurophysiologists have for a long time been able to record evoked potential changes in the spinal cord of animals

following stimulation of the dorsal roots (Gasser and Graham, 1933). Subsequently the study of evoked field potentials recorded both from surface spinal cord electrodes and intramedullary micro-electrodes in response to stimulation of peripheral nerves, visceral nerves and the sympathetic chain have been well documented in a variety of animal species (see Yates et al, 1982). The first recordings of evoked potentials in the human spinal cord utilised intra-thecal electrodes (Magladery et al, 1951). This invasive mode of recording also used by Caccia et al (1976) Ertekin (1976, 1978) and Shimoji and colleagues (1977) has not been widely adopted because of the potentially serious problems that could emanate from CSF leakage, infection and inadvertent spinal cord injury or contusion (Delbeke et al, 1978).

With the development of computer averaging techniques it became possible to record the small amplitude spinal evoked potentials by non-invasive methods. This led to the early studies describing surface recordings of spinal SEPs in humans (Liberson and Kim, 1963; Cracco, 1973) and later comparative studies of subdural, epidural and cutaneous recordings of lumbosacral spinal SEPs (Ertekin, 1978).

Studies of the cervical and lumbar spinal SEPs in humans and the evoked electrospinogram in other mammals have allowed comparison of the waveform morphology and putative generator sites (Happel et al, 1975, Shimoji et al, 1977; Delbeke et al, 1978; El-Negamy and Sedgwick, 1978). Animal experimentation has also been undertaken to clarify the spinal funiculi mediating rostral

transmission of the SEP. Localisation of afferent conduction has been largely confined to the dorsal columns however the spinocerebellar and spinothalamic tracts may also contribute (Singer et al, 1970; Sarnowski et al 1975; Cusick et al, 1979; Cohen et al, 1981, Simpson et al, 1983; Snyder and Halliday, 1984). These neuroanatomical correlates of the spinal SEP have important implications in clinical practice. As well as changes in waveform latencies which may be seen in demyelinating or neuropraxic disorders, it is possible that differential spinal dysfunction could lead to discrete changes in the morphology of the spinal SEP (Cohen et al, 1981; Bennett, 1983; Schramm et al, 1983)

Normative data for spinal SEPs recorded with both bipolar and monopolar surface montages has now been documented (Cracco, 1973; Cracco et al, 1975; Delbeke et al, 1978; El-Negamy and Sedgwick, 1978; Hashimoto et al, 1984). These studies have enabled changes induced by various neuropathological disorders to be studied. However the major clinical application of spinal SEPs to date has been their use as an intraoperative assessment of spinal cord function.

2.1.iii INTRAOPERATIVE SPINAL MONITORING USING SEPs

The possibility of having some method of assessment of the anatomical and functional integrity of the nervous system under anaesthesia is particularly enticing to spinal surgeons. Despite technological advances in micro-instrumentation, neuro-anaesthesia, and innovative mechanical systems for correcting

spinal deformities, the risks of iatrogenic spinal cord damage during spinal surgery remain considerable (Levy, 1983).

Patients undergoing spinal medullary surgery and surgery to correct scoliosis with the powerful instrumentation that is now available, are particularly at risk. It has been estimated that there is at least a 0.75% incidence of major neurological damage following correction of scoliosis during Harrington instrumentation (MacEwan et al, 1975) whilst the morbidity associated with removal of an intra-medullary neoplasm or vascular malformation is estimated at 20% (Levy, 1983).

Although Perot (1972) postulated the use of SEPs for monitoring neural integrity intraoperatively, and Croft and associates (1972) developed an experimental system of intra-operative SEP monitoring in animals, Vauzelle and colleagues (1973) described the first system of assessment of spinal cord function during operations in humans. The system they developed for intraoperative assessment of spinal cord function in patients undergoing surgery for the correction of scoliosis, was termed the "wake-up" test. This test involved lightening the level of anaesthesia during surgery to a level such that the patient moved the limbs to command. If satisfactory movement was observed and ipso facto spinal cord function deemed to be satisfactory the anaesthetic was recommenced and the surgical procedure completed. Prior to this mode of intra-operative assessment, early post-operative clinical examination followed by removal of the distraction or compresssion rods, if a neurological deficit had occurred, was the accepted technique of minimising spinal cord

damage following instrumentation for scoliosis.

The application of the "wake-up" test was therefore a major advance in obviating iatrogenic spinal cord damage. Despite the wide application and success of the "wake-up" test, several problems are inherent in its use (Engler et al, 1978; Grundy, 1983). The level of anaesthesia required for adequate co-operation in the test is such that patient movements may dislodge the endotracheal tube, intravenous, intra-arterial or other life support or monitoring lines, compromise the sterility of the surgical field, or be associated with pulmonary air embolism. The required level of co-operation may be difficult to obtain in children, the mentally retarded and deaf patients. There is also the disquieting possibility of patient awareness with psychological sequelae during lightening and re-induction of the anaesthetic (Hall et al, 1978). Furthermore the patient may have suffered an irreversible neurological insult prior to the performance of the wake-up test, or indeed may suffer an insult after a normal test.

The shortcomings in the "wake-up" test plus limitations of its use to orthopaedic surgery led to the development of various methods of intraoperative monitoring using SEPs. The earliest descriptions of the use of SEP recording as a mode of spinal cord function monitoring came from Case Western Reserve University where Nash and colleagues recorded SEPs during orthopaedic correction of scoliosis using Harrington instrumentation and other complex spinal procedures (Nash et al, 1972; Nash et al, 1974; Nash et al, 1977; Brown and Nash, 1979). Further

refinements in the systems developed by these and subsequent workers now allow almost continuous intra-operative spinal cord monitoring (see Grundy, 1983).

The earliest systems of intra-operative spinal cord monitoring using SEPs both in orthopaedic and spinal neurosurgery, were based upon the principle that stability of the scalp recorded cortical SEP to peroneal or tibial nerve stimulation correlated with the preservation of neural integrity (McCallum et al, 1975; Engler et al, 1978; Owen et al, 1979). Unfortunately in a high percentage of cases, technical problems precluded satisfactory monitoring using the cortical SEP as a parameter of spinal cord function (Allen et al, 1981; Uemato and Tolo, 1981; Raudzens, 1982; Jones et al, 1983). Furthermore, variables such as changes in cerebral blood flow, blood pressure and anaesthetic agents were shown to significantly attenuate, modify or abolish the cortical SEP (Clark and Rosner, 1973; Worth et al, 1982; Raudzens, 1982).

In view of these practical problems experimental work was commenced that examined modes of assessing spinal cord function intraoperatively by recording spinal SEPs. Nordwall and colleagues (1979) effectively demonstrated that good amplitude spinal SEPs to lower limb peripheral nerve stimulation could be recorded from electrodes inserted in the vertebral bone. The spinal SEP had the advantage of being considerably larger in amplitude than the cortical SEP and consisted of a short latency response with a waveform of only several milliseconds duration. It was consequently easier to acquire and interpret than the

polyphasic cortical SEP. It was not significantly attentuated by therapeutic hypotension, nitrous oxide or halothane in concentrations of up to 4%, and yet attentuated significantly with spinal cord compression or ischaemia (Nordwall et al, 1979; Schramm et al, 1979; Bennett, 1983). Subsequent experimental and clinical studies have confirmed that the spinal SEP is an accurate parameter of spinal cord function and can be recorded using a variety of simple techniques (Grundy, 1983; Bunch et al, 1983; Schramm et al, 1983). Much of the current research on intraoperative spinal cord monitoring using SEPs is concerned with clinical evaluation of the multiple methods of monitoring that have been described.

2.2 METHODOLOGICAL ASPECTS OF RECORDING SPINAL SUBCORTICAL & CORTICAL SEPs

With the technological advances that have occurred in the last two decades, recording SEPs has become a routine procedure in most neurophysiological units. Methodological aspects are particularly important however in the categorisation and standardisation of the SEP. The temperature of the patients' limbs, mode of SEP generation, stimulus intensity, rate of stimulation, electrode type, recording montage, frequency bandpass and epoch over which averaging occurs may cause factors that contribute to variations in the acquired SEP waveforms. These factors become important when comparing SEP data from different units, and in the interpretation of the waveform associated with various neuropathological disorders. Although SEP data has to some extent been standardised by the use of

recording montages adopted from the International 10/20 recording system (Jasper, 1958), clinical application requires collation of normative data using defined recording conditions and techniques before electrophysiological changes attributable to neural lesions may be evaluated.

2.2.i Stimulus Modality

Various methods of eliciting a SEP have been described. These utilise electrical stimulation of peripheral nerves or electrical or mechanical cutaneous stimulation. Most units employ a technique of transcutaneous or percutaneous electrical stimulation with the electrodes placed directly over the path of a major peripheral nerve. The electric shock may be delivered through saline soaked felt pads mounted in a saddle-shaped plastic stimulating unit (Jones and Small, 1978; Lastimosa et al, 1982; Ganes, 1982), metal surface disc or cup electrodes attached to the skin with tape, gels or colloidin (El-Negamy and Sedgwick, 1978; Cracco et al, 1979; Hashimoto et al, 1984; Iragui, 1984), needle electrodes (Shimoji et al, 1977) or ring electrodes placed around a digit (Pratt et al, 1979; Eisen et al, 1980). If cortical SEPs from segmental dermatomal stimulation are to be studied, bilateral cutaneous electrical stimulation is applied using any of the aforementioned techniques (Jorg et al, 1982). Convention has led to the positioning of the cathode proximal to the anode.

Mechanical stimulation of the fingernail has been described as an alternative method of eliciting SEPs (Pratt et al, 1979; Woolsey

et al, 1979)). This method of stimulation is alleged to be a more natural cutaneous stimulus without the discomfort associated with electric shock stimulation. The SEP generated by mechanical stimulation has the same general waveform as that generated by peripheral nerve stimulation, however, standardisation of stimulus is difficult and the SEPs are of lower amplitude, have different interpeak latencies and occasionaly have fewer components (Pratt et al, 1979; Cohen and Pratt, 1985).

2.2.ii Stimulus Site

The peripheral nerves most commonly stimulated by percutaneous electrical stimulation are the median and ulnar nerves, and the digital nerve to the index finger in the upper limb, and the posterior tibial and peroneal nerves in the lower limb. It has been demonstrated that SEPs can also be generated by stimulation of intercostal nerves (El-Negamy & Sedgwick, 1978; Pratt et al, 1979; Jorg et al, 1982), the saphenous, musculocutaneous, superficial radial and sural nerves (Phillips and Daube, 1980; Eisen et al, 1980), the trigeminal nerve (Stohr et al, 1979; Bennett and Janetta, 1980; Bennett and Lunsford, 1983) and the pudendal nerve (Chiappa and Ropper, 1982a). Although some of these procedures have the advantage of recording SEPs generated by stimulating a pure sensory nerve, as opposed to a mixed nerve, they are technically demanding and except in specific clinical situations offer little advantage over standard major peripheral nerve stimulation.

Access to the median and ulnar nerves is readily obtained at the

wrist, cubital fossa and medial epicondyle. Satisfactory location of stimulating electrodes can be confirmed by a readily visible forearm, hypothenar or thenar muscle twitch at appropriate stimulus intensity. Both these nerves have extensive contributions from the roots of the brachial plexus, cervical spinal cord segments and extensive parietal cortical representation. These anatomical considerations ensure that a readily evoked SEP waveform may be recorded at Erb's point, over the cervical spine, and from the scalp overlying the hand somatosensory cortex (C3/C4).

Generating spinal and cortical SEPs from lower limb stimulation is routinely obtained by using either the tibial, peroneal or posterior tibial nerve stimulation. These nerves may be stimulated in the posterior fossa, posterior to the head of the fibula, or behind the medial malleolus of the ankle respectively. Accurate placement of the stimulating electrode can be confirmed by ready observation of rhythmic eversion and dorsiflexion of the foot (peroneal nerve) or plantar flexion of the foot and toes (tibial nerve) with moderate stimulus intensity. Unlike the situation in the upper limb, it is much more difficult to elicit spinal SEPs to digital (toe) stimulation even when recording from the lumbosacral epidural space (Shimoji et al, 1977).

2.2.iii Stimulus Parameters

The frequency and intensity of peripheral nerve stimulation that is delivered to elicit an SEP have both practical and theoretical considerations. From the practical viewpoint, the more rapidly

the peripheral nerve is stimulated, the more rapidly the waveform is aquired, and the greater the stimulus intensity, the greater the amplitude of the subcortical evoked potential (Lesser et al, 1979; Tsuji et al, 1984a). Apart from the patient discomfort that will be associated with rapid, high intensity stimulation, there are also theoretical considerations in the control of the stimulus parameters.

It has been demonstrated that the amplitude of the Erb's point SEP to median nerve stimulation, and the thoracolumbar spinal SEP to tibial nerve stimulation, have a close relationship with stimulus intensity until a maximal response is elicited at approximately twice the motor thresholh intensity (Lesser et al, 1979; Tsuji et al, 1984b). It is considered that at such stimulus intensities, all the fibres in the peripheral nerve contributing to the SEP are stimulated, since there are no additional components to the SEP waveform with higher stimulus intensities. This data would suggest that the early increase in SEP amplitude with increasing stimulus intensity is due to activation of increasing numbers of afferent fibres and that motor fibres do not contribute significantly to the subcortical SEPs (El-Negamy and Sedgwick, 1978; Delbeke et al, 1978; Dimitrijevic et al, 1978; Phillips and Daube, 1980).

Despite the augmentation of the Erb's point, cauda equina, and conus SEPs with increasing stimulus intensity, it has been shown that the amplitude of the more rostral SEPs have a less direct relationship to stimulus intensity (Tsumoto et al, 1972; Lesser et al, 1979; Ganes, 1982; Tsuji et al, 1984). The conclusion

from these authors is that a stimulus intensity of approximately three times the sensory threshold is sufficient to elicit an optimum SEP waveforms.

Bilateral lower limb stimulation has been shown to augment the lower spinal SEPs to an amplitude of approximately twice that obtained with unilateral lower limb stimulation, and also to decrease the incidence of failed recordings (Delbeke et al, 1978; Dimitrijevic et al, 1978; Schiff et al, 1984; Tsuji et al, 1984a). Bilateral lower limb stimulation has also been reported to enable surface recording of spinal SEPs up to the cervicothoracic junction (Lueders et al, 1981).

The frequency of peripheral nerve stimulation determines to some extent the morphology of the evoked potential. At short interstimulus intervals, saturation of synaptic transmission occurs. Presynaptic components of the SEP waveform may therefore be differentiated from the components that are generated by postsynaptic potentials, since the latter will attenuate with higher frequency stimulation. These considerations have been used by various workers to elucidate the neuroanatomical generators of various components of the SEP. Using subtraction techniques of waveform analysis and either paired stimuli with short interstimulus intervals, or high frequency stimulation rates, it has been demonstrated that the Erb's point SEP to median nerve stimulation and the cauda equina SEP, show no attenuation with high rates of stimulation, thus indicating a presynaptic origin. Conversely the spinal SEP to median nerve stimulation recorded over Cv7 and the thoracolumbar SEP to lower

limb stimulation both attenuate at high frequency stimulation, thus indicating a postsynaptic origin (Dimitrijevic et al, 1978; El-Negamy and Sedgwick, 1978; Ganes, 1982). In view of these findings, most units employ a stimulus frequency of 3-5 Hz.

2.2.iv Recording methodology

After satisfactory generation of the SEP, recording electrode characteristics and montage must be considered from both practical and theoretical viewpoints. Practical problems include the cost of electrodes, their recording characteristics and method of application to the patient which is important in terms of patient comfort and surface-electrode interface artefact.

Subdermal needle electrodes of various metallic alloys have been used extensively (Hume et al, 1982; Jorg et al, 1982; Lueders et al, 1983). More recently disposable Ag/AgCl ECG stick-on disc electrodes (El-Negamy and Sedgwick, 1978; Eisen et al, 1980; Ganes, 1982), or strips of Ag/AgCl foil (Delbeke et al, 1978) have become widely available. Standard EEG and cup-shaped silver or tin electrodes, filled with conduction paste and maintained in postion with colloidin, have also been used in the recording of scalp cortical SEPs (Cracco et al, 1979; Pratt et al, 1979; Hume et al, 1982). Although these electrodes have different electrical properties, with stainless steel needles being theoretically the worst (Cooper et al, 1980), they all have been used successfully in clinical practice.

With the use of surface electrodes, preparation of the skin with

alcohol and a dermabrasive preparation is particularly useful to reduce inter-electrode impedance (Cooper et al, 1982). Furthermore, for optimal recording characteristics, electrodes should be non-polarisable and have short leads connecting them with the pre-amplifier.

The recording montage is of vital significance when acquiring SEPs, since the morphology of the SEP waveform is influenced by the positions of the electrodes relative to the generator sites of the evoked potentials (Desmedt and Cheron, 1981). Many subcortical neurones generate far field potentials which can be augmented or attenuated by using particular recording montages in which the electrodes are either widely separated or closely apposed. Such variations in recording montages together with variations in stimulus frequency have been used to localise the generator sites and mode of generation of various components of the SEP waveform (Anziska and Cracco, 1981; Ganes et al, 1982; Emerson et al,1984). From the clinical viewpoint however, a few standard recording sites may be used to obtain basic spinal, subcortical and cortical SEP data.

The common recording montages for SEPs from upper limb peripheral nerve (median nerve, ulnar nerve) stimulation are:

1. C3/C4 referenced to Fz or Fpz for cortical SEP (Eisen et al, 1980; Hume et al, 1982; Cullity et al, 1976; Pratt et al, 1979, Willis et al, 1984). Other workers have used a point 3 cm behind C3/C4 referenced to Fz or Fpz (Stohr et al, 1983). C3/C4 referenced to noncephalic electrodes such as

linked ears, or a shoulder have also recently been described so that near field cortical and far field subcortical SEPs may be recorded simultaneously (Leuders et al, 1983).

2. Midline cervical spine surface electrodes position from Cv7 to Cv2 referenced to either Fz, Fpz or a noncephalic electrode for spinal and subcortical SEPs (Eisen et al, 1980; El-Negamy and Sedgwick, 1978; Emerson et al, 1984; Lueders et al, 1983).

3. Erb's point referenced to either Fz, Fpz or a noncephalic electrode.

The common recording montages for SEPs to lower limb peripheral nerve stimulation are:-

1. Spinal recording electrode over L4/5 and T12/L1 referenced to either the contralateral iliac crest (Phillips and Daube, 1980; Leuders et al, 1983; Tsuji et al, 1984b), or T6 (Delbeke et al, 1978; Dimitrijevic et al, 1978; Lastimosa et al, 1982). Sequential spinal electrodes linked in a bipolar fashion or referenced to Cz or Fz have also been described (Jones and Small, 1978; Cracco, 1973; Cracco et al, 1979; Hasimoto et al, 1984). In general it has been shown that the spinal SEP amplitude increases with the distance of the reference electrode from the recording electrode (Phillips and Daube, 1980). However, with increasing inter electrode distance recording artefact also increases from ECG and EMG interference.

2. Cortical SEPs have been recorded with montages that range from Cz referenced to Fpz (Eisen et al, 1980), Cz' (a point 2cm posterior to Cz) referenced to Fpz (Lastimosa et al, 1982), Fz referenced to the mastoid (Stohr et al, 1983), Cz referenced to Fz (El-Negamy and Sedgwick, 1978). Fz referenced to linked ears (Tsumoto et al, 1972), and Cz' referenced to a non-cephalic electrode (Vas et al, 1981). Feinsod and colleagues (1982) have recorded cortical SEPs to peroneal nerve stimulation at both C3/C4 and Cz, referenced to linked ears, and noted little difference in waveform configuration.

2.2.v Frequency Bandpass

Processing of the acquired SEP signal in terms of frequency bandpass has a major influence on both the morphology of the SEP waveform and the subcomponent peak latencies. The changes induced by altering frequency filters have been well documented (Desmedt et al, 1974; Tsuji et al, 1984b). These workers have demonstrated that whilst subcortical SEPs may be satisfactorily recorded with a band pass restricted to 30-150Hz, cortical SEPs require an upper band pass of 1,000Hz to avoid phase shifts, distortion and voltage attenuation of the major short latency peaks. Too much high frequency attenuation may lengthen the latency of some components by up to 2 ms, whilst too much low frequency attentuation may increase the amplitude of the later duration components with a decrease in amplitude of the shorter latency components (Tsuji et al, 1984b; Nuwer and Dawson, 1984).

2.2.vi Epoch Duration

The period or epoch over which the evoked potential is averaged will also influence definition of the SEP. The quality and definition of an evoked potential averaged over a long period will depend upon the dwell time and other technical characteristics of the micro-computer system used for averaging. Long epochs will result in suboptimal definition of short latency components, although the long latency components will be recorded. In clinical medicine the shorter latency components (less than 40 ms) are considered more significanrt because of putative electrophysiological-neuroanatomical correlations. The long latency components do not have well defined generators and are less stable than short latency components and are influenced by awareness and other higher cerebral functions (Desmedt and Manil, 1970; Cracco, 1972; Tsumoto et al, 1972; Willis et al, 1984).

From this review of methodological influences on SEP waveform, it can be realised that it is difficult to interpret some papers recorded in the literature in which methodological details are inadequately described (Blair, 1971; Cullity et al, 1976; Riegel et al, 1976; Duckworth et al, 1976; Allen et al, 1981; Grundy et al, 1982). Such omissions led to a declaration concerning standard requirements for evoked potential papers that are to be published (Donchin et al, 1977).

2.2.vii Methodological and Technical Considerations for Intraoperative SEP Monitoring

Recording SEPs in the theatre environment has its own particular advantages and disadvantages. The major advantages of intra-operative recording from the anaesthetised patient are that muscle artefact is abolished by the use of muscle relaxants and methods of stimulation and recording may be more intense and invasive than in the conscious patient. These consideration are counterbalanced by the problems of electrical noise in the theatre environment, limited patient access once the operation has commenced, and the variable and unpredictable effects of anaesthetic agents and random changes in blood flow on cortical and subcortical SEPs (Clark and Rosner,1973; Thornton et al, 1984). Furthermore, the mode of monitoring must complement the surgical procedure. General desiderata require that the mode of intraoperative recording is safe, simple in execution, not disruptive to the surgical team and produces a reliable, reproducible record that can be readily acquired and interpreted (Grundy, 1982).

Methods of eliciting spinal, subcortical and cortical SEPs intra-operatively include the conventional percutaneous modes of peripheral nerve stimulation, or more invasive techniques utilising spinal cord or cauda equinal stimulation. If surface stimulating electrodes are used then accurate placement over the path of the peripheral nerve must be confirmed prior to commencement of operation. This may be achieved either by pre-operative localisation and marking of stimulation sites with an

indelible crayon or pre-anesthetic positioning of the stimulating electrodes (Engler et al, 1978; Spielholz et al, 1979; Worth et al, 1982).

Percutaneous or transcutaneous modes of stimulation may in some cases be inadequate to elicit a readily recordable SEP if there is major spinal pathology associated with a neurological deficit (Allen et al, 1981; Uemato and Tolo, 1881; Raudzens, 1982; Riegel et al, 1976; Macon et al, 1982). Intra-operative monitoring also requires a stable and reliable mode of stimulation since for many operations there is no access to the stimulation sites following draping. Subsequently modes of epidural spinal cord stimulation (Tamaki et al, 1981; Shimizu et al, 1982; Macon and Poletti, 1982; Kaschner et al, 1984) and direct stimulation of the cauda equina (Lueders et al, 1982) have been described. Stimulation may be delivered so that the spinal cord potentials travel in a caudo-rostral manner or in a rostro-caudal fashion (Tamaki et al, 1981; Shimizu et al, 1982).

These modes of electrical stimulation of the peripheral and central nervous system give rise to short duration, short latency, large amplitude spinal SEPs. However, the cortical response to similar stimulation under anaesthesia is much more variable both in latency and amplitude (Hahn et al, 1981; Maccabee et al, 1982; Worth et al, 1982; Jones et al, 1983).

Since intra-operative monitoring is mainly concerned with the stability of a baseline SEP waveform, stimulus intensity, frequency of stimulation, and amplifier bandpass must be

maintained at constant settings during the surgical procedure. As previously discussed, changes in these parameters will alter the spinal SEP waveform. For example, increasing stimulus intensity may cause a corresponding increase in the amplitude of the evoked potential, and a more complex ascending spinal SEP waveform often with more later components (Nordwall et al, 1979; Macon and Poletti, 1982; Shimizu et al, 1982; Jones et al, 1982).

The morphology of the ascending spinal SEP is less dependent upon stimulus frequency than segmental spinal SEPs to median nerve and peroneal or tibial nerve stimulation. It has been shown that with stimulus frequencies of up to 30 Hz the amplitude and morphology of the ascending spinal cord potential are stable (Macon and Poletti , 1982; Jones et al, 1982, Shimizu et al, 1982). However, with stimulus frequency greater than 30-50Hz, there is a gradual decrease of ascending spinal cord potential amplitude (Nordwall et al, 1979; Jones et al, 1982).

Because of the fairly high electrical "noise" levels in the operating theatre, the signal is usually amplified over a narrow band width of approximately 200 - 2000 Hz. It has been shown however that with wider band widths (eg 20Hz-10KHz) there is a slight increase in amplitude, and better resolution of some components of the ascending spinal cord potential (Jones et al, 1982).

The method of recording the generated SEP signal must also satisfy many of the desiderata previously enumerated. Recording electrodes must be stable, and not subject to major impedance

changes or polarisation. Compromises may therefore have to be made in terms of electrode stability and its recording characteristics. It is well known that stainless steel subdermal electrodes have inferior recording qualities to Ag/AgCl disc stick-on electrodes (Cooper et al, 1980). However, the practical problem of dislocation of the electrode due to sweat or other fluids decreasing electrode adhesion during surgery means that needle electrodes may be preferable in certain types of surgery. Certainly both types of electrodes have been used to satisfactorily record scalp cortical SEPs during operations (Engler et al, 1978; Spielholz et al, 1979; Raudzens, 1982; Worth et al, 1982; Maccabee et al, 1982; Jones et al, 1983).

Because of the availability of more invasive methods of recording afforded by the surgical procedure, several innovative methods of recording have been described. It has been clearly shown that the closer the recording electrode is to the spinal cord, the larger and more discrete is the SEP configuration and amplitude (Happel et al, 1975; Caccia et al ,1976; Ertekin, 1978). This proximity of recording electrode to the spinal cord generator site is particularly important in monitoring the cervico-thoracic ascending spinal cord potential to lower limb stimulation since at this level the signal is both temporally dispersed and of low amplitude. (Jones et al, 1982, Bunch et al, 1982).

Since intra-operative monitoring is performed mainly to detect changes in the baseline SEP waveform, ad hoc electrode positioning and types of recording may be used. In addition to the routine methods of recording scalp cortical SEPs several

different methods of recording spinal SEPs have been described. These include electrodes inserted into the spinous processes of vertebral bodies (Nordwall et al, 1979; Maccabee et al, 1982; Lamont et al, 1983; Jones et al, 1983), recording electrodes positioned in the epidural space (Jones et al, 1983; Bunch et al, 1983; Tamaki et al, 1981; Macon and Poletti, 1982), recording electrodes inserted in the interspinous ligaments (Hahn et al, 1981) and SEP recording utilising montages that record both spinal and cortical SEPs concurrently (Leuders et al, 1981; Hahn et al, 1982).

2.3 MORPHOLOGY OF SPINAL, SUBCORTICAL AND CORTICAL SEPs

2.3.i General Considerations

The morphology of SEP waveforms recorded following electrical stimulation of a major peripheral nerve are generally constant if parameters of stimulation and recording are remain stable. There may be some intertrial variation of peak amplitudes and latencies due to differences in ambient temperature and levels of cortical arousal (Desmedt and Manil, 1970; Cracco, 1972; Tsumoto et al, 1972) These differences are particularly marked in the longer latency components, (greater than 40 ms post stimulus). The short latency SEP components (less than 40 ms post stimulus) can however be readily identified according to their post-stimulus latencies (Hume and Cant, 1978). These components are labelled as positive (P) or negative (N) deflections relative to the active electrode (Donchin et al, 1977). Several of these components can be correlated with precise neuroanatomical generators whilst the

origin of other components remains contentious (Allison et al, 1980; Chiappa and Ropper, 1982a; Yamada et al, 1983). The post stimulus onsets of these components do however vary with parameters such as limb length, patient age and stimulus site (Eisen et al, 1980; Lastimosa et al, 1982; Tsuji et al, 1984b).

Since the field potentials generated by ascending afferent volleys and their synaptic connections may be recorded as near and far field potentials, the morphology of the SEP and the relative contributions of far and near field potentials is dependent on the geography of the recording electrode montage (Desmedt and Cheron, 1982). For example, scalp recorded cortical SEPs to median nerve stimulation may be recorded from most linked International 10/20 recording positions on the scalp both contralateral and ipsilateral to the site of stimulation (Yamada et al, 1984). If however an International 10/20 scalp electrode is referenced to a non-cephalic electrode, the scalp recorded SEP may also reflect subcortical far field potentials generated by the peripheral nerve stimulation (Desmedt and Cheron, 1981 and 1982; Lueders et al, 1983; Small and Mathews, 1984). These far field, volume conducted potentials cannot be recorded with a scalp-scalp recording montage because of cancelation effects. The delineation and topography of far field potentials and the contributions of motor afferents to cortical SEPs (Gandevian et al, 1984) are not considered in this treatise. Consideration here is given to recording sites that allow optimal and simple acquisition of short latency SEP waveforms, since in clinical practice it is pathological changes in the primary generator sites and the associated near field SEP waveform distortion that

will allow major anatomical-electrophysiological correlations.

Recording sites close to the presumed generators of the SEP in response to upper limb peripheral nerve stimulation are Erb's point (above the mid-clavicular point), which overlies the brachial plexus; at the posterior midline of the neck at the 7th cervical vertebra which overlies the cervical spinal cord enlargement; the posterior midline of the neck over the spine of the axis (Cv2) which is close to the gracile and cuneate nuclei; and the scalp overlying the primary hand and brachial somatosensory cortex (approximately C3/C4).

The recording sites and anatomical generators of the SEP following lower limb stimulation are the posterior midline around L4/5 which overlies the cauda equina; the posterior midline over the thoraco-lumbar junction which overlies the conus medullaris spinalis; and the scalp overlying the primary leg somatosensory cortex (approximately Cz).

These recording electrodes may be referenced to a cephalic (Fz or Fpz) or non-cephalic (iliac crest, T6, angle of scapula, linked ears or knee) electrode. As previously stated a scalp recording electrode referenced to a non-cephalic electrode may enhance the far field potentials generated by subcortical pathways mediating transmission of the afferent volley.

2.3.ii The Erb's Point SEP

In adults approximately 9 ms following stimulation of the median nerve or ulnar nerve at the wrist a major negative wave (N9) is recorded over Erb's point (Fig 2.1). This potential represents an ascending volley in low threshold large diameter sensory fibres as they traverse the brachial plexus (Jones 1977; El-Negamy and Sedgwick, 1978; Lesser et al, 1981; Ganes 1982; Chiappa and Ropper, 1982a; Iragui, 1984). The N9 peak is a presynaptic potential that has an amplitude which is closely related to stimulus intensity (Lesser et al, 1981). This wave therfore represents transmission, both orthodromic and antidromic in the fibres comprising the middle (median nerve) or lower (ulnar nerve) trunk of the brachial plexus.

2.3.iii Cervical cord and Cv2 SEPs to upper limb stimulation

After median nerve stimulation at the wrist a negative wave can be recorded from the posterior midline over Cv2 (Fig 2.1). In adults the peak latency is approximately 13 ms (N13) with inflections at 11 ms (N11) on the ascending potential gradient and 14 ms (N14) on the descending potential gradient. From studies of polarity reversals and field potential distribution the following generators are postulated: N11 represents the ascending volley as it enters the dorsal root entry zone of the spinal cord, with possible contributions from the grey matter in the dorsal horns (Lesser et al, 1981; Hume et al, 1982; Anziska and Cracco, 1981; Chiappa and Ropper, 1982a; Suzuki and Mayanagi,

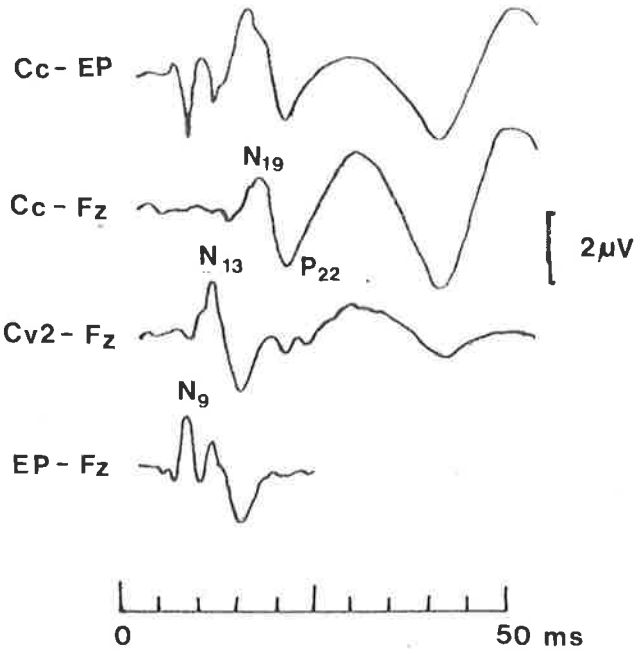


Fig 2.1 Representative short latency SEPs, following median nerve stimulation, recorded over Erb's Point (EP), Cv2 and the scalp overlying the primary somatosensory cortex (CC). Recording the cortical SEP referenced to an electrode at Erb's Point (CC-EP) causes phase reversal of the N9 and N13 peaks. Reproduced from Chiappa and Ropper, 1982.

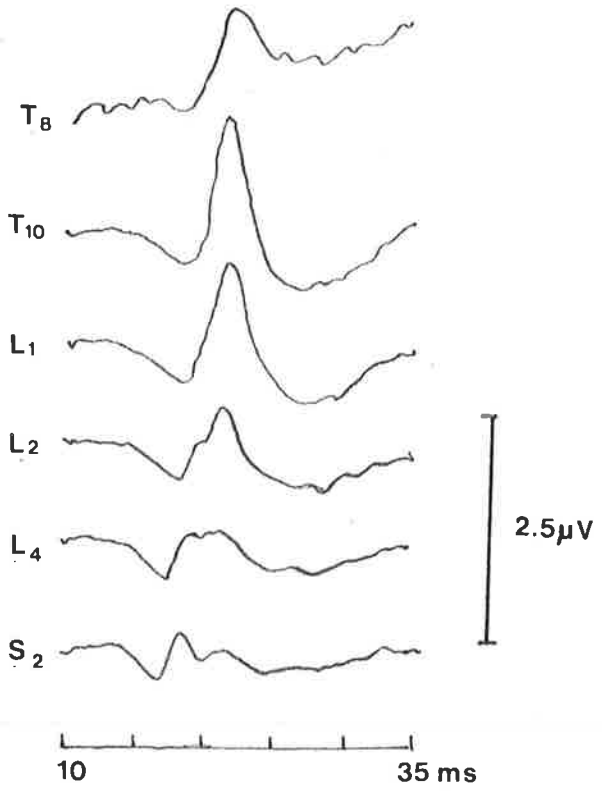


Fig 2.2 Surface recorded spinal SEPs, following tibial nerve stimulation, at different spinal levels. All recordings are referenced to the iliac crest (IC). Reproduced from Delbeke et al, 1978.

1982; Yamada et al, 1983, Iragui, 1984). The origin on the N13 potential, which may be recorded from the scalp (Fig 2.1) as a far field potential with phase reversal (P13) is believed to be from the nuclei gracilis and cuneatus or dorsal horn interneurones (El-Negamy and Sedgwick, 1978; Desmedt and Cheron, 1981; Hume et al, 1982; Chiappa and Ropper, 1982; Yamada et al, 1983; Iragui 1984; Emerson et al, 1984). The N13 potential has a duration and recovery cycle consistent with a post-synaptic generator. It is larger in the lower cervical segments than in the upper (Ganes, 1982; Iragui, 1984). Studies of the H reflex, the spinal evoked potential and the EMG response in the abductor pollis brevis have confirmed that N13 is generated essentially by sensory fibre activation with little contribution from the motor neurone pool (El-Negamy and Sedgwick, 1978).

The N14 potential is believed to represent either a non-synaptic brainstem potential from a group of relatively slow conducting high threshold fibres (Ganes et al, 1982; Mauguiere et al, 1983; Yamada et al, 1983) or a post-synaptic potential generated at the cervico-medullary junction (El-Negamy and Sedgwick, 1978).

2.3.iv Cortical SEP to major upper limb peripheral nerve stimulation

The morphology of the SEP waveform recorded at C3/4, referenced Fpz or Fz, to contralateral median or ulnar stimulation is similar to a distorted sine wave (Fig 2.1). There is a prominent N18-20 peak followed by a steep descending potential gradient to a trough at P22-25. Large amplitude long duration peaks and

troughs subsequently follow the short latency components.

It is generally agreed that the P22/25 component originates in the primary parietal somatosensory cortex since cortical damage abolishes this positivity (Chiappa and Ropper, 1982a), and direct pial recordings from Brodman areas 1 and 3 suggest a cortical origin for both the N18/20 and P22/25 components (Suzuki and Mayanagi, 1984). The generator of the N18/20 component remians contentious. Putative generator sites for this component include the primary post-rolandic sensory cortex (Allison et al, 1980; Desmedt and Cheron, 1980; Eisen et al, 1980; Hume et al, 1982; Suzuki and Mayanagi, 1984), specific thalamo-cortical projections (Stohr et al, 1983; Yamada et al, 1983) and the thalamus (Chiappa et al, 1980). Clinico-pathological material that suggests a particular generator is difficult to interpret because of the complex pattern of retrograde trans-synaptic neuronal atrophy between the specific thalamic relay nuclear groups and the somatosensory cortex (Chiappa and Ropper, 1982a).

2.3.v Spinal SEPs to Lower Limb Stimulation

The morphology of the surface recorded spinal SEP differs when a waveform obtained using a spinal electrode referenced to a distant silent electrode such as the iliac crest or scapula (Jones and Small, 1978; Dimitrijevic et al, 1978; El-Negamy and Sedgwick, 1978; Delbeke et al, 1978) is compared to a waveform recorded using a sequential bipolar spinal montage (Cracco R Q, 1973; Cracco J B et al, 1975 and 1979; Hashimoto et al, 1984). The differences are caused by phase reversals around the

generator sites of the evoked potential. Because of the methodology used in this treatise major consideration is given to surface spinal SEP waveforms recorded using a spinal electrode referenced to a distant non-spinal electrode.

2.3.vi The Cauda equina (S1) SEP

The cauda equina potential recorded over the posterior midline in the lumbosacral region has two negative peaks(Fig 2.2). The latency of the first peak (N1 component) has been variably labelled N9/10 or N18/19 depending on stimulus site at the popliteal fossa or ankle respectively (Delbeke et al, 1978; Phillips and Daube, 1980; Lastimosa et al, 1982). This N1 component represents the presynaptic ascending volley in the dorsal roots of the cauda equina (Dimitrijevic, 1978; Delbeke et al, 1978; Cracco et al, 1979; Jones and Small, 1978). The amplitude and latency of this component is also dependent upon patient height, patient age and lower limb temperature (Cracco et al, 1979; El-Negamy and Sedgwick, 1978; Phillips and Daube, 1980; Lastimosa et al, 1982; Hashimoto et al, 1984; Gilmore et al, 1985).

The characteristics of the cauda equina potential are also closely dependent upon stimulus intensity since the second component (N2) is attenuated at higher stimulus intensities (Delbeke et al, 1978), whilst the N1 component is maximal at stimulus intensities of approximately four times sensory threshold (Dimitrijevic et al, 1978; Tsuji et al, 1984b). From studies of the cauda equina evoked potential at different

stimulus intensities, EMG responses in the triceps surae and H reflexes, it has been postulated that the N2 component may represent the ventral root component of the H reflex (Delbeke et al, 1978; Dimitrijevic et al, 1978). However the latency of the N2 component is in some subjects identical to the peak negativity of the conus evoked potential and in some adults there is no relationship between N2 and the H reflex, these findings suggest that the N2 component may be a volume conducted potential (Delbeke et al, 1978; Lemkuhl et al, 1982; Small and Mathews, 1984).

2.3.vii The Conus Medullaris (L1) SEP

The conus medullaris potential recorded over the thoraco-lumbar junction has a prominent negative peak which has variable inflections on the ascending gradient (Fig 2.2). This N peak is referred to as N11 or N20/22 depending on stimulus site at the popliteal fossa or ankle respectively (Delbeke et al, 1978; Phillips and Daube, 1980; Lastimosa et al, 1982). The amplitude of the N wave is related to stimulus intensity, although not as closely as the cauda equina potential, being maximal at approximately four times sensory threshold (Dimitrijevic et al, 1978; El-Negamy and Sedgwick, 1978; Delbeke et al, 1978; Tsuji et al, 1984a). The same workers have also demonstrated that the amplitude of the spinal SEP from the conus medullaris can be augmented by bilateral lower limb stimulation.

The spinal evoked potential from the conus is due to post-synaptic activity following orthodromic transmission in low

threshold large diameter sensory fibres (Jones and Small, 1978; Delbeke et el, 1978; Dimitrijevic et al, 1978; El-Negamy and Sedgwick, 1978; Phillips and Daube, 1980). This has been confirmed by simultaneous recordings of the spinal evoked potential, EMG responses in the gastrocnemius and triceps surae and the H reflex at various stimulus intensities. As the stimulus increases, the H reflex increases and then wanes whilst the spinal evoked potential increases in amplitude with no change in waveform configuration. The amplitude of the spinal evoked potential then reaches a plateau even though further increases in stimulus intensity reveal that the EMG response from the triceps and gastrocnemius is sub-maximal (Delbeke et al, 1978; Dimitrijevic et al, 1978). The latter finding would suggest little contribution from the motor neurone pool to this spinal evoked potential and that increasing amplitude of the waveform is due to activation of increasing numbers of afferent fibres.

Further studies of the conus potential following stimulation of purely sensory cutaneous nerves of the lower limb, eg: saphenous nerve and sural nerve, have also reconfirmed these findings (Eisen et al, 1980). The amplitude of the conus potential decreases with distance from the thoraco-lumbar junction which also suggests a fixed generator site at this level (Jones and Small, 1978; Dimitrijevic et al, 1978; Phillips and Daube, 1980).

The small P-wave preceeding the major N component of the conus potential represents the propagation of the ascending volley into the spinal cord (Yates et al, 1982). This is a volume conducted potential that shows only minor latency changes with different

levels of recording (Nordwall et al, 1979). The variable inflections on the ascending gradient of N1 of the conus potential are felt to represent dorsal column components and interneurone activation within the dorsal horn (Delbeke et al, 1978). The long positivity following the N wave is felt to represent the process of primary afferent depolarisation (See review Yates et al, 1982).

Surface recorded spinal SEPs from the mid-thoracic and cervical spines following lower limb peripheral nerve stimulation are much less discrete waveforms than the cauda equina and conus segmental SEPs (Cracco et al, 1975; Happel et al, 1975; Ertekin, 1978). These waveforms are believed to represent volume conducted field potentials from both spinal neuronal activity and the ascending axonal volley. The latency of these SEPs become poorly defined and shorter with rostral recording (Happel et al, 1975). This, together with the relative non-specificity and low amplitude of the wave has led to the general opinion that these waveforms are not particularly useful as a clinical or research tool (Jones et al, 1982; Small and Mathews, 1984). Conversely Lueders and colleagues (1981 and 1983) report that the far-field potential that is recorded over the mid cervical spine referenced to the scalp following tibial nerve stimulation, is a particularly useful and stable SEP.

2.3.viii The Cortical SEP from lower Limb Stimulation

The morphology of the SEP recorded over Cz or Cz' referenced to Fpz, Fz or a non-cephalic electrode to peroneal or posterior

tibial nerve stimulation is polyphasic with stable short latency components (N-P1-N1) and variable long latency components (Fig 2.3). The short latency components consist of a variable peak negativity at 27 to 33 ms (N) followed by a steep descending potential gradient to a trough at around 35 to 39 ms (P1) and a prominent later negativite peak at 45-51 ms (N1) (Tsumoto et al, 1972; Small and Mathews, 1978; Eisen et al, 1980; Vas et al, 1981; Rossini et al, 1981; Worth et al, 1982; Feinsod et al, 1982; Lastimosa et al, 1982; Ertekin et al, 1984; Tsuji et al, 1984a and 1984b). These major short latency peaks (N-P1-N1) do not seem to be particularly altered by specific recording montage, or whether the SEP is generated from peroneal or tibial nerve stimulation (Small and Mathews, 1978; Feinsod et al, 1982; Tsuji et al, 1984b).

The origins of P1 and N1 components are felt to parallel the N18/20 and P22/25 components, respectively, of the cortical response from upper limb stimulation. Again however, whether these potentials represent activity generated in the primary leg somatosensory cortex or thalamo-cortical projections, is contentious (Chiappa and Ropper, 1982a; Eisen et al, 1980; Ebner et al, 1982). The origin of the later peaks are not particuarly well understood, although it is postulated from clinico-pathological studies that they arise in the cortex of the para-central lobule (Ebner et al, 1982).

Although the cortical response from unilateral lower limb peripheral nerve stimulation has been well documented some noted workers have reported difficulty in reproduction of this waveform

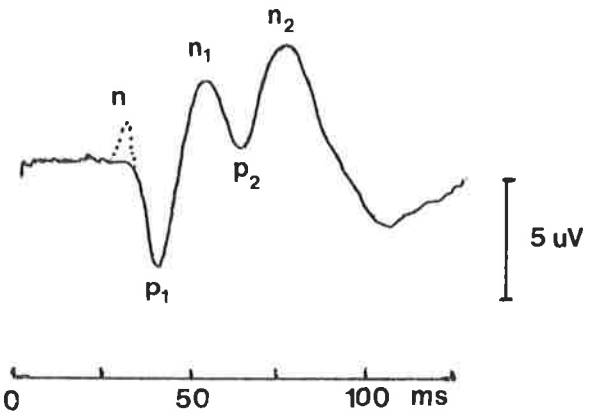


Fig 2.3 Representative scalp recorded cortical SEP (Cz referenced to linked ears) following tibial nerve stimulation. There is a variable initial n component (dotted line) followed by a P1-N1-P2-N2 complex. Reproduced from Dorfman et al, 1980.

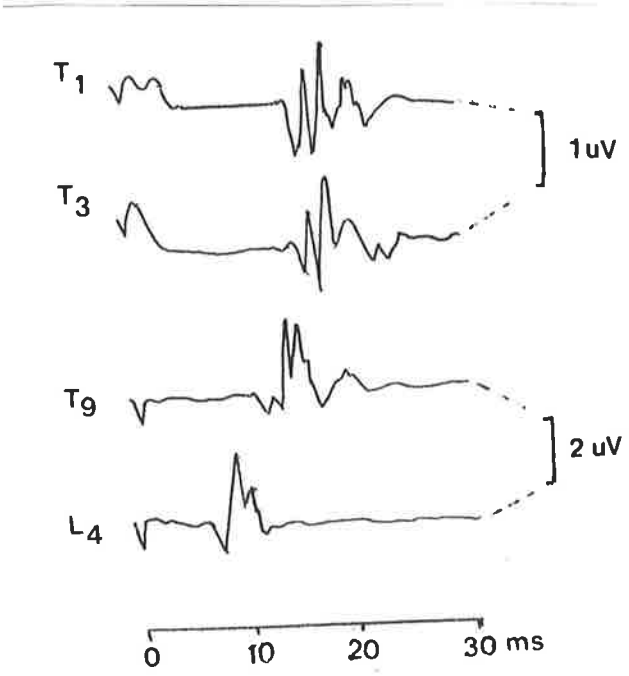


Fig 2.4 Examples of intraoperative spinal SEPs following tibial nerve stimulation recorded with an extradural spinal electrode referenced to an electrode in adjacent paraspinal muscle. The complexity of the waveform, and its latency increases with more rostral recordings. Reproduced from Jones et al, 1982.

in normal subjects (Jones and Small, 1978; Kimura et al, 1978; Chehrazi et al, 1981; Schiff et al, 1984). This difficulty in signal acquisition in normal subjects has important implications particularly when considering intra-operative monitoring using cortical SEPs to lower limb peripheral nerve stimulation and the use of this SEP as an index of spinal or cortical disease.

2.3.ix Intraoperative Cortical SEP Waveforms

The morphology of intraoperative scalp recorded cortical SEPs is, in general, similar to waveforms recorded without anaesthetic. The amplitude of the intraoperative acquired signal is however generally smaller (Engler et al, 1978; Worth et al, 1982; Nuwer and Dawson, 1984) and the latency and amplitude of the long latency peaks is variable (Allen et al, 1981; Raudzens, 1982). These factors make distortions and changes in the waveform difficult to interpret and have led to the more widespread adoption of spinal SEPs as a parameter of spinal cord function during spinal surgery. A study of the problems of cortical SEP recording during surgery has however recently been undertaken (Nuwer and Dawson, 1984). These workers, and Symon and colleagues (1984) noted that with the low bandpass set at 30Hz, close apposition of the recording scalp electrodes, stimulating at a frequency of 5Hz and the use of short preamplifier leads good quality cortical SEPs can be obtained intraoperatively.

2.3.x Intraoperative Spinal SEPs

Invasive modes both of neural stimulation and recordings have been repeatedly shown both experimentally and clinically to produce a short duration, polyphasic large amplitude spinal SEP (Magladery et al, 1951, Happel et al, 1975; Sarnowski et al, 1975; Ertekin, 1978; Shimoji et al, 1977; Shimizu et al 1981). The inconvenience, patient discomfort and some possible side effects of invasive recording methodologies are obviated by anesthesia and the surgical procedure.

The morphology of the spinal SEP obtained using invasive recording methodology depends upon the mode and locus of signal generation and acquisition, the spinal segment at which the SEP is being recorded, and associated spinal medullary pathology. Although numerous methods of intraoperative SEP recording have been described (See Section 2.2.iv), there is only one comprehensive study describing normal ascending spinal SEPs acquired intraoperatively in man (Jones et al, 1982). Several of these SEPs are illustrated in Fig 2.4.

Several reports have included illustrations of ascending spinal SEP waveforms recorded intraoperatively in humans to which the findings of Jones and colleagues (1982) can be compared (Lamont et al, 1983; Macon and Poletti, 1982). Comparative studies of ascending spinal SEPs recorded using invasive techniques in dogs, cats and sheep have revealed many similarities to those obtained in humans (Happel et al, 1975; Sarnowski et al, 1975; Nordwall et al, 1979; Synder and Halliday, 1984). These workers describe the

ascending spinal SEP as having three discrete components - an initial positive spike, a short, sharp polyphasic N potential, and a slower broader N wave. These different components show sequential activation as stimulus intensity increases and temporal dispersion at rostral levels of recording that probably represents transmission within different sized fibres of the dorsal columns and dorso-lateral funiculi (Jones et al, 1982; Snyder and Halliday, 1984).

The neural substrates represented by the 'tractus' or ascending spinal SEP are contentious when compared to the relatively well defined origin of segmental spinal SEPs. Much animal experimental work has addressed this subject, (Singer et al, 1970; Andersson et al, 1975; Sarnowski et al, 1975; Happel et al, 1975; Cusick et al, 1978; Cohen et al, 1981; Synder and Halliday, 1984). Most of these reports have suggested that the dorsal column ipsilateral to the side of stimulation primarily transmit the ascending SEP, although the spinocerebeller and spinocervial tract may contribute.

The transmission velocities of the ascending spinal SEP in man have been reported to range from 15 to 130 m/s. (Magladery et al, 1951; Cracco, 1973; Shimoji et al, 1977; Ertekin, 1978; Jones and Small, 1978; Jones et al, 1982). The reasons for these wide ranging results are mainly due to methodology, however ttransmission of the ascending volley in varying sized fibres in the dorsal funiculi, or through a slower multi-synaptic pathway may also contribute (Jones et al, 1982; Cracco et al, 1975, Cracco et al, 1979; Snyder and Halliday, 1984)

2.4 CLINICAL AND INTRAOPERATIVE APPLICATIONS OF SEPs IN NEUROSURGERY

2.4.i The Theoretical and Practical Utility of SEPs in Clinical Medicine

The use of evoked potential studies in clinical medicine is based upon the hypothesis that changes in afferent sensory pathway function will be reflected by alterations in evoked potential waveforms. The clinical application of this hypothesis rests upon the assumption that specific components of the evoked potential waveform correlate with specific neuroanatomical generators and pathways. Whilst the correlation between the five major short latency components of the BAER and their neural generators has been well established, allowing precise anatomical localisation of waveform changes (Chiappa and Ropper, 1982; Grundy, 1982), the situation with regard to some SEP waveform components and their neuro-anatomical correlates is, as has been previously discussed, considerably more complex.

As well as the problems of anatomical correlation there are other problems in SEP waveform interpretation since the amount of structural change in the somatosensory pathway that is required to cause distortion or attenuation in short latency peaks and troughs, the significance of changes in the longer latency cortical SEP components, and the effects of damage in other systems that modulate somatosensory input are poorly understood (Jones and Halliday, 1982). Furthermore, the relative importance of amplitude asymmetry and loss of certain component peaks as

parameters of afferent somatosensory neural dysfunction await clarification.

The accepted method of SEP waveform analysis utilises descriptions of peak and trough latencies. These parameters are more stable than amplitude variance of the waveform components (Jones and Halliday, 1982). The amplitude of waveform components has been shown to vary by as much as 25% in the same subject (Sances et al, 1978) and is also related to the state of awareness of the patient (Tsumoto et al, 1972) as well as parameters of stimulation and recording (Desmedt et al, 1974; Dorfmann et al, 1981; Tsuji et al, 1984b). When amplitude variance has been utilised there is the added problem of which peak to trough amplitude should be used for analysis, since often the largest amplitude components have the greatest latency variation.

Statistical analysis of waveform parameters is not always specific unless variables such as height, age and conditions of recording are stipulated. Furthermore the clinical relevance of statistical analysis of waveform data must be considered since the paradoxical situation has been reported in which changes in amplitude of the SEP were highly statistically significant and yet there was no clinical correlation in terms of neurological dysfunction (Worth et al, 1982). Conversely normal cauda equina and conus SEPs may be obtained in patients with complete para- or quadriplegia (Ertekin, 1978; Sedgwick et al, 1980; Ertekin et al,1984; Lemkuhl et al, 1984).

Recently questions have also arisen as to whether combined near and far field recording of SEPs allows acquisition of a more stable and useful signal than near field recording methods (Lueders et al, 1983; Small and Mathews, 1984), and whether multiple channel recording of field potential distribution (Jones and Halliday, 1982; Yamada et al, 1984) may be more specific than conventional recording techniques in indicating and localising neural abnormalities.

Regardless of methodology of signal acquisition, the contentious problem of interpretation of SEP waveform remains. This is particularly important in intraoperative monitoring when waveform changes may be induced by surgery, anaesthesia, and technical or methodological considerations. Understanding the significance of changes in intraoperative SEPs has important consequences. For this reason most intraoperative SEP monitoring during spinal surgery now utilises modes of recording spinal rather than cortical SEPs because of their specificity and superior stablity in both latency and amplitude (Bunch et al, 1983; Jones et al, 1983; Schramm et al, 1983).

Much experimental work has been performed on changes in the segmental and ascending spinal SEP and cortical SEP with spinal cord tractotomy, ischemia and compression (Singer et al, 1970; Brodkey et al, 1972; D'Angelo et al, 1973; Morrison et al, 1975; Cusick et al, 1979; Nordwall et al, 1979; Schramm et al, 1979; Guth et al, 1980; Yamada et al, 1981; Bennett, 1983; Schramm et al, 1983; Simpson et al, 1983). Extrapolation of this data, in terms of qualitative and quantative changes in waveform to

diseased humans is however often difficult. Many of these studies have demonstrated that changes in SEPs are not an early feature of spinal cord compression or ischemia, and that major structural damage to the spinal cord can occur with normal spinal or cortical SEPs.

2.4.ii Clinical Uses of SEPs in Neurosurgery

As with their use in clinical neurology, the reported use of SEP studies in neurosurgery has increased significantly in the last few years. SEP recordings are now used in a variety of conditions to facilitate and assess patient management, surgical decision making, and to provide information about the functioning of the nervous system when patients are under anaesthesia, or their conscious state precludes sensory examination (Greenberg and Ducker, 1982).

In order to correlate electrophysiological data to the functional status of the spinal cord, brain stem and diencephalic-cortical projections the concept of central conduction times was developed. Central somatosensory conduction time (CSCT) is the time taken for the afferent somatosensory volley to travel from the nuclei gracilis and cuneatus in the caudal medulla to the somatosensory cortex (Hume and Cant, 1978). This parameter is measured by subtracting the latency of the N1 cortical component (N18 equivalent) from the latency of peak negativity measured over Cv2 (N13 equivalent), following upper limb peripheral nerve stimulation. The variability of this conduction time has been shown to be very low and standard values have been documented for

adults aged fronm the second to eigth decades (Hume and Cant, 1978 and 1981; Hume et al, 1982). The importance of this parameter is that it potentially offers information concerning the status of vital brain stem pathways in patients who are in coma or undergoing surgery.

The other central conduction pathway that has been studied is the lumbar cord to scalp transit time (Rossini et al, 1982). This parameter is the difference between the negative peak latency of the thoracolumbar (L1) SEP (N14 or N22 equivalent) and the P1 latency of the cortical SEP (P28 or P37 equivalent) following lower limb peripheral nerve stimulation. Several studies in adults have documented mean transit times of 15.6 ms (Lastimosa et al, 1982), 16.6 ms (Ertekin et al, 1984; Tsuji et al, 1984). Some worker have noted no correlation between this transit time and lower limb length (Tsuji et al, 1984; Lastimosa et al, 1982), whilst others have correlated it with the length of the back (Small and Mathews, 1984). The clinical utility of this parameter is somewhat restricted by the wide range of values recorded in normal patients (Ertekin et al, 1984), and the fact that many patients with spinal cord disease will ahve loss of SEPs rather than delayed transit time (Small and Mathews, 1984).

The principle uses of SEPs and central conduction times in clinical neurosurgery have in the following disorders (Greenberg and Ducker, 1982).

1. Spinal Injury

Spinal and cortical SEPs have been used in the initial assessment of patients with spinal cord injuries, during surgical correction

of traumatic fracture dislocation and sequentially during rehabilitation of the patients. Perot (1972) initially correlated the level and severity of the spinal cord injury with cortical SEPs to median, peroneal and sural nerve stimulation. He found that with complete clinical cord transection there was no recordable SEP to lower limb stimulation, and that in approximately 50% of incomplete lesions, the cortical SEP was reproducible but had an increased latency and decreased amplitude. Cortical SEPs to median nerve stimulation were present if the lesion was below the C6/7 cervical cord segment but were impaired with lesions at more rostral levels.

Bricolo and colleagues (1976) also reported the use of cortical SEPs to assess the response of 11 patients with clinical cord transection to therapeutic cord hypothermia. Cortical SEPs were obtained in the 7 patients who made some clinical recovery whilst absence of SEP correlated with no clinical recovery. Similar findings have been reported in an experimental study in dogs (Kojima et al, 1979). However, reproducibility of cortical SEPs and improved waveform amplitude during spinal decompression and stabilisation after traumatic cord injury did not necessarily correlate with clinical outcome in another study (Spielholz et al, 1979).

Several other major studies have also suggested that there is no reliable predictive correlation between clinical and electrophysiological data in spinal cord injury (Chehrazi et al, 1981; Ertekin et al, 1984; Levy et al, 1984; York et al, 1984). The limitations of using the SEP as a parameter of spinal cord

function has also been demonstrated in experimental studies of spinal cord injury in the cat where normal Cortical SEPs may be recorded following compression and partial transection of the spinal cord (Schramm et al, 1979; Simpson et al, 1983).

2. Monitoring the Somatosensory Pathways During Surgery

Since the experimental work of Croft and associates (1973) and early clinical application of SEP monitoring during spinal surgery (Nash et al, 1972, 1974), several workers have described SEP monitoring during neurosurgical procedures (McCallum and Bennett, 1975; Owen et al, 1979; Hahn et al, 1981; Grundy et al, 1982; Macon et al, 1982b). Most clinical reports describing the use of intraoperative SEPs for monitoring spinal cord function have however, been from orthopaedic clinics (Nash et al, 1974; Engler et al, 1978; Spielholz, 1979; Uemato and Tolo, 1981; Maccabee et al, 1982; Worth et al, 1982; Jones et al, 1983; Lamont et al, 1983). These series describe monitoring during surgery for correction of kyphoscoliotic disorders. Some reports from orthopaedic clinics do however also contain anecdotal references to neurosurgical cases (Nash et al, 1977; Allen et al, 1981; Tamaki et al, 1981; Raudzens, 1982).

The value of SEP monitoring of spinal cord function during procedures such as correction of scoliosis with Harrington rod fixation is now well documented, although recently a case of paraplegia complicating correction of scoliosis was reported without change in intraoperative SEPs (Ginsburg et al, 1985). The role of SEP monitoring during spinal neurosurgery is less clear. Many of the neurosurgical and combined orthopaedic/neurosurgical series are lacking in detail of clinical data, SEP recording

methodology, SEP waveforms obtained and changes induced in these waveforms with surgery (McCallum and Bennett, 1975; Spielholz et al, 1979; Tamaki et al, 1981; Hahn et al, 1981; Raudzens, 1982; Grundy et al, 1982; Lueders et al, 1983). These facts together with the proliferation of methods of intraoperative monitoring described (see Section 2.2.iv) suggests that optimal methodology and utility of intra-operative monitoring in neurosurgery is not yet fully understood.

3. Head Injury

Prolonged coma following cranio-cerebral trauma, with or without intracranial haematoma, cerebral contusion and intracranial hypertension, form a large component of neurosurgical service requirements. Various therapeutic regimes used following neurotrauma together with depressed levels of consciousness make examination of some patients difficult. Studies of multimodality evoked potentials, SEPs and central conduction times have been used in an effort to quantitate and qualitate levels of brainstem and cortical dysfunction, and to predict outcome in these patients (Greenberg et al, 1977; Hume and Cant, 1978; Goldie et al, 1981; Greenberg et al, 1981; Lindsay et al, 1981; Narayan et al, 1981; Greenberg and Ducker, 1982; Lutschg et al, 1983). The results of these studies are reviewed in section 5.

4. Cerebral Ischaemia

In patients subjected to carotid endarterectomy and following subarachnoid haemorrhage from rupture of a cerebral aneurysm, serial monitoring of cortical SEPs and CSCT have been used to determine changes in neural function that occur with vasospasm and brainstem and cortical ischaemia. CSCT has been shown to be prolonged, and cortical SEPs abnormal in experimental

cerebrovascular ischaemia in baboons (Branston et al, 1974; Jakubowski et al, 1982), and in patients with deficits following aneurysm or carotid surgery (Moorthy et al, 1982; Fox and Williams, 1984; Symon et al, 1984). However the significance of these findings in terms of predictive value, when occurring in clinically stable patients awaits clarification (Symon et al, 1979; Fox and Williams, 1984).

5. Other Neurosurgical Disorders

(i) Assessment of pain related pathways.

Trigeminal SEPs have been recorded before and after rhizotomy for Tic douloureux (Bennett and Jannetta, 1980; Salar et al, 1982; Bennett and Lunsford, 1984). These studies have shown delayed trigeminal evoked potentials on the side of the tic douloureux and normalistion of these latencies following trigeminal rhizotomy.

Spinal and subcortical SEPs have also been recorded to assess somatosensory pathways following spinothalamic tractotomy and stereotactic mesencephalic tractotomy (Namerow, 1969; Colombo, 1984). The finding of normal SEPs in these patients is further evidence that the SEP is transmitted along the dorsal column-medial lemniscal pathway.

(ii) Myeloradicular Disorders .

Cortical, cervical and lumbar spinal SEPs have been recorded in discogenic and stenotic myeloradiculopathy both before and after surgery (Matsukado et al, 1976; Ganes, 1980; Feinsod et al, 1982; Stohr et al, 1982). These studies have shown prolonged latencies, attentuation of amplitude and disappearance of waveform components with myeloradiculopathy and variable improvement in waveform following surgical decompression. A very recent study has also shown that there is often little

correlation between myelographic and evoked potential abnormalities in cervical spondylitic myelopathy (Yu and Jones, 1985).

(iii) Brachial Plexopathy .

Erb's point and cervical SEPs to ulnar and median nerve stimulation have been used to localise brachial plexus lesions either proximal or distal to the dorsal root ganglion (Jones, 1979; Yiannicus and Walsh, 1983). hese studies have important implications for subsequent reconstructive surgery, since lesions proximal to the dorsal root ganglion are often associated with nerve root avulsion which will preclude successful reconstructive surgery (Landi et al, 1980).

(iv) Intracranial Neoplasms.

Several studies have correlated changes in cortical SEPs with intracranial tumours and CT scan findings in an effort to define the origin and contribution of various cortical areas to the cortical SEP (Williamson et al, 1970; Noel and Desmedt, 1980; Anziska and Cracco, 1980; Ebner et al, 1982; Stohr et al, 1983). The results of these studies are reviewed in section 6.

2.4.iii SEPs IN PAEDIATRIC NEUROSURGERY

Paediatric neurosurgery in general involves management of a different spectrum of pathological disorders than adult neurosurgery. Traumatic spinal injury, compressive myelopathy, discogenic radiculopathy, pain disorders, cerebrovascular ischaemia and subarachnoid haemorrhage due to aneurysm rupture, are uncommon in children. Although major head injury remains a frequent problem, the outcome in children is generally much more

satisfactory than in adults in terms of both morbidity and mortality (Bruce et al, 1978).

The application of SEP studies in paediatric neurosurgery is based upon practical and theoretical considerations. Clinical assessment of children is often difficult because of their age and the non-specific response of the infant or neonate to intracranial or spinal pathology. Furthermore, neuroradiological advances, particuarly the advent of CT scanning have facilitated diagnosis of structural abnormalities of the CNS. However, quantitative and qualitative assessment of neural dysfunction in sensory pathways remains difficult.

Although several recent studies have reported some normative SEP data for the paediatric age group (Blair et al, 1971; Cracco et al, 1975; Cullity et al, 1976; Desmedt et al, 1976; Laget et al, 1976; Willis et al, 1984; Hashimoto et al, 1984; Gilmore et al, 1985), there have been few studies of clinical application of SEPs in paediatric neurosurgical disorders (Riegel et al, 1976; Duckworth et al, 1976). Some of these papers are unsatisfactory from many viewpoints. Patient clinical status, methods of acquisition of SEP waveforms, analysis and description of normative SEP data, and quantitive and qualitative changes in SEP data with pathological disorders, and documentation of SEP data are invariably either omitted, poorly described, inadequately or merely anecdotal (Blair, 1971; Duckworth et al, 1976; Cullity et al, 1976; Riegel et al, 1976). In mitigation it should be noted that these papers were published prior to the pronouncement of criteria for publication of evoked potential

studies in humans (Donchin et al, 1977).

Subsequent sections of this thesis contain clinical and experimental work which was designed to examine and evaluate some of the clinical applications of SEP recording in paediatric neurosurgery. In particular these studies involved:

1. The application of standard methods of recording SEPs to children so that normative cortical and subcortical data could be documented following both upper and lower limb stimulation.
2. An examination of changes in SEP waveform that occur in the presence of known intracranial or spinal pathology. Three patient groups were studied. The first group consisted of 26 children with a variety of spinal disorders that were causing variable sensory, motor and sphincteric dysfunction. The second group consisted of 14 children who had suffered major head injuries. The third group consisted of seven patients with structural abnormalities of the brain. In all cases a correlation between clinical, radiological and electrophysical findings was attempted.
3. The development and application of methods of recording SEPs during paediatric spinal surgery that enabled an evaluation of this modality as a method of intraoperative monitoring of spinal cord function.
4 The application of one of these methods to an experimental model in sheep to examine the effects of stimulus intensity and halothane anaesthesia on spinal SEPs recorded intraoperatively. These experimental findings were correlated with intraoperative SEP data from 32 humans.

The results from these studies were evaluated so that the clinical utility and limitations of SEP recording in these aspects of paediatric neurosurgery could be objectively assessed and compared with published data from adult neurosurgical series. Although the major clinical and operative workload of the pediatric neurosurgeon concerns management of childhood hydrocephalus the use of SEP data in this area has not been included in this thesis. The complexities of differing etiology of the hydrocephalus, associated brain malformations and the effects of secondary insults complicating the hydrocephalus and its treatment would suggest that large serial studies would be required to obtain meaningful data.

SECTION 3

SHORT LATENCY SOMATOSENSORY EVOKED POTENTIALS FOLLOWING TIBIAL AND MEDIAN NERVE STIMULATION IN CHILDREN; NORMATIVE DATA

INTRODUCTION

Short latency SEPs following median, tibial and peroneal nerve stimulation in healthy adult humans have been well documented (Jones, 1977; Tsumoto et al, 1977; Delbeke et al, 1978; El-Negamy and Sedgwick, 1978; Dimitrijevic et al, 1978; Jones and Small, 1978; Phillips and Daube, 1980; Chehrazi et al, 1981; Kimura et al, 1981; Lastimosa et al, 1982; Schiff et al, 1984; Tsuji et al ,1984; Yamada et al, 1984). Although studies defining similar normative data for the paediatric population have been described (Hrbek et al, 1968, 1972; Cracco et al, 1975, 1979; Laget et al, 1976; Desmedt et al, 1976; Hashimoto et al, 1984; Willis et al, 1984; Gilmore et al, 1985) the documentation of spinal, subcortical and cortical SEPs in children to both upper and lower limb stimulation remains incomplete.

In this study of infants and children, SEPs were recorded from surface electrodes following either tibial or median nerve stimulation. The data obtained enabled analysis of normal SEP waveforms and peak latencies, correlation of this data with age and limb length, and a study of variations in central conduction

times in infancy and childhood. The study also allowed an assessment of methods and conditions required for satisfactory paediatric SEP recording and provided a normative data base against which SEPs recorded in children with central nervous system lesions could be compared.

PATIENTS AND METHODS

Forty five children and infants were included in a study of short latency SEPs following median nerve stimulation. The chronological ages of the subjects ranged from one month to 13 years 11 months. Twenty four subjects were male and 21 female.

Fifty six children and infants were included in the study of short latency SEPs to tibial nerve stimulation. The chronological ages of these subjects ranged from four weeks to 13 years 6 months. Thirty subjects were male and 26 were female.

All subjects were hospital inpatients free from neurological disease. They were studied under one of the following conditions: (a) SEP recording with no sedation, (b) SEP recording with sedation consisting of Vallergan (Trimeprazine) 3-4mgs/Kg orally, (c) SEP recording whilst under anaesthesia (Nitrous oxide/oxygen). The latter group consisted of 10 subjects who had previously been studied whilst awake. These subjects were premedicated with atropine 0.02 mg/Kg intramuscularly and were studied prior to minor non-neurosurgical operations. The first two groups had the studies performed in a sound attenuating room

with an ambient temperature of 22 degrees Celsius. In all cases, permission had been obtained from both the Hospital Ethics Committee and the parents to perform the recording procedure.

SEP Recording Methodology

SEPs were recorded in response to square-wave electrical pulses of 0.1ms duration and 25-100V amplitude delivered at 5Hz. This electric shock stimulus was delivered to the subject through saline soaked felt pad or pencil electrodes mounted in a saddle-shaped plastic mount. The cathode was placed proximal to the anode and inter-electrode distance varied between 1.0 and 2.0 cm, depending on the subjects' size. Satisfactory placement of the stimulating electrodes over the path of the tibial nerve in the popliteal fossa was confirmed by rhythmic foot plantar flexion. The median nerve was stimulated at the wrist and satisfactory electrode placement was confirmed by rhythmical muscle twitching in the thenar eminence. The effect of stimulus intensity on waveform amplitude was not studied.

Recording electrodes were commercial stick-on chlorided silver (Ag/AgCl) electrodes with additional conduction gel (Dracard, Dracard Ltd, Maidstone, Kent, England) smeared over the electrode face. The skin over the recording site was prepared with a mild dermabrasive (Omniprep, Weaver & Co, Denver, Colorado, USA) to decrease skin impedance. A 4 cm by 2.5cm metal alloy plate, which was used as an earth electrode, was taped over the chest or lower abdomen.

Scalp derived cortical SEPs utilised montages from the International 10/20 system (Jasper, 1958). SEPs to median nerve stimulation were recorded over Erb's point, Cv2 and the scalp corresponding to C3/C4. Fpz was used as a reference electrode.

Spinal SEPs to tibial nerve stimulation were recorded over the lumbosacral junction (S1), the thoracolumbar junction (L1) and the lower thoracic region (T8/9). These electrodes were referenced to an electrode positioned over the iliac crest (IC) contralateral to the side of tibial nerve stimulation. To record cortical SEPs to tibial nerve stimulation, an electrode was positioned over Cz' (2 cm posterior to a position midway between the nasion and inion), and referenced to Fpz.

The evoked potential was fed through a Medelec PA 89 pre-amplifier to a Medelec MS 91 signal averaging unit. To record spinal and subcortical SEPs the bandpass was set at 20-2,000Hz (-6dB rolloff), whilst for cortical SEPs the band width was 2Hz-10KHz. Amplifier sensitivity was set at either 20 or 50uV. Averaging was performed for either 10 or 30ms following stimulation when recording subcortical and spinal SEPs and 100-200ms when recording cortical SEPs. At least 64 stimuli were averaged for sub-cortical and spinal SEPs and up to 1,024 stimuli for cortical SEPs.

All records were duplicated and displayed on an oscilloscope screen which had facility for superimposition of the traces. Peak latencies were measured using a cursor with digital readout. Copies of all records were plotted onto graph paper from which

peak to trough amplitudes could be calculated. The patient's age and sex and the length from the stimulus site to either the thoracolumbar or Erb's point recording electrode was also documented.

Statistical and Waveform Analysis

Major waveform peaks were labelled negative (N) or positive (P) depending on either upward (N) or downward (P) deflection induced at the recording electrode from an iso-potential line. Conduction velocity in the upper limb and in the tibial nerve and cauda equina pathway was calculated by dividing the distance between stimulating and recording electrodes by the peak latency of the Erb's point and thoracolumbar SEPs respectively. The CSCT (Hume et al,1978) was calculated by subtraction of the N1 latency recorded at Cv2 (N13 equivalent) from the N1 latency recorded at C3/C4 (N18-20 equivalent). The lumbar spine to scalp transit time was the difference between peak negativity of the L1:IC spinal SEP and the latency of P1 of the cortical SEP following tibial nerve stimulation. An index of upper and lower limb length was obtained by measuring the distance from stimulating electrode to recording electrode at either Erb's point or at the thoracolumbar junction.

Statistical analysis was performed using the Statpro (Imhof et al, 1983) linear regression analyses programme. This programme used statistical methods and equations detailed by Snedecor and Cochrane (1967). Correlation coefficients (r), levels of significance (p) and 95% confidence estimates were obtained to

establish the relationships between subjects' age, limb length and SEP peak and interpeak latencies.

RESULTS

There was considerable variability in the childrens' tolerance to the studies. In general, older children tolerated the study well whilst younger children, even when sedated, often became agitated and difficult to study because of movement artefact. SEPs following median nerve stimulation were in general easier to record than tibial evoked potentials. Studies of thinner children were more readily obtained than comparable studies in fatter children. Some recordings in young infants were distorted by shift of the isopotential baseline. In many children under three months of age this distortion could not be rectified by revising electrode placement. This problem was probably attributable to the high skin impedance previously noted in infants (Blair et al, 1971).

A summary of the statistical correlations between peak latencies of particular SEPs and parameters such as age and limb length is given in Table 3.1

SUBCORTICAL, SPINAL AND CORTICAL SEPs TO MEDIAN NERVE STIMULATION

The Erb's Point SEP

This waveform (Fig.3.1) consisted of a negative wave with a prominent inflection prior to peak negativity (N1). Duration of

the negative wave was approximately 3 ms. The peak negativity fell sharply to a positive trough, which was followed by a smaller N wave (N2) similar in peak latency to the Cv2 SEP. The amplitude of the N1-P trough varied between 2-7uV. The latency of the N1 peak was directly related to limb length ($r = 0.91$, $p < 0.001$) and also the subjects'age (Fig.3.2). The relationship between N1 peak latency and age in the first two years of life was however not statistically significant ($r = 0.05$, $p > 0.05$). Median nerve conduction velocity increased directly with patient age during the first decade ($r = 0.78$, $p<0.001$). Nerve conduction velocities ranged from 31 m/s in a 15 week child to 69 m/s in a 13 year old boy.

Anaesthesia with nitrous oxide/oxygen did not alter the waveform of this SEP, and peak latencies were reproducible to within 0.4 ms of those recorded in awake subjects.

The Cervical (Cv2) SEP

This waveform consisted of a prominent negative wave of approximately 3-5ms duration with an inflection on the up slope (Fig.3.3). The proximity and amplitude of the inflection on the up slope often gave the negative peak a bifid character. Maximal negativity usually corresponded to the second peak (N2). There was a steep potential gradient from the N2 peak to a long positivity. The amplitude from peak negativity to positivity varied between 2-7 uV. During the first decade the latency of the peak negativity of the Cv2 SEP increased directly with patient limb length ($r = 0.86$, $p < 0.001$) and age (Fig.3.4). However for

subjects under 2 years of age there was no correlation between age and peak latency ($r = -0.72$, $p > 0.05$).

Anaesthesia with nitrous oxide and oxygen did not alter the cervical SEP waveform, and peak latency values were within 0.6 ms of those recorded in awake subjects (Fig.3.3).

The Cortical SEP

This SEP (Fig 3.5 and 3.6) was a polyphasic waveform consisting of an early negative peak (N1). The latency of the N1 cortical peak did not, in the age range seven months to 12 years, correlate with subjects' age ($r = 0.26$, $p > 0.1$). The mean latency for N1 in this age range was 16.7 $\pm$ 1.16 ms (Fig 3.7). Following N1 there was a variable potential gradient to a positive trough (P1). This trough usually had a bifid appearance with two discreet P waves separated by 3-5 ms (Fig 3.5 and 3.6). This doublet became more prominent with increasing age. Maximal positivity occurred at the second of these P waves in 74% of subjects. The time from peak negativity (N18 equivalent) to peak positivity (P22-25 eqivalent) ranged from 5.5 to 8.0 ms. This interpeak latency decreased with age over the first decade (Fig. 3.8). After P1, and in the first 100 ms post stimulus, there were three or four large amplitude peaks and troughs.

The morphology of the cortical SEP was altered by general anaesthesia wih nitrous oxide/oxygen. Although the latency of the N1 and P1 components did not vary by more than 0.9 ms the longer latency components were particularly variable both in

amplitude and latency (Fig.3.6).

Central somatosensory conduction time

CSCT showed an insignificant decrease over the first decade (Fig.3.9). The data is however biased by the low number of children less than three years of age included in this statistic. The mean CSCT over the age range 2-13 years, was 6.52 $\pm$ 1.02 ms (range 4.65 to 7.80ms). It was noted however that the mean CSCT in children less than 7 years (6.76$\pm$ 0.81 ms) was longer than that in children over 7 years of age (6.21$\pm$ 0.64 ms), although this difference was not statistically significant.

SPINAL AND CORTICAL SEPs FOLLOWING TIBIAL NERVE STIMULATION

Spinal SEPs

Two patterns of SEP were recorded at the lumbosacral junction. One SEP consisted of an initial P wave followed by a steep potential gradient to N1 which was followed by a slow N wave of 5 ms duration. This wave slowly fell back to the isopotential line. (Fig.3.10). The other SEP waveform recorded at the lumbosacral junction consisted of two separate negative peaks with an interpeak latency of approximately 3 ms. The second negative peak (N2), being smaller than the first (Fig.3.11). The latency of this N2 peak was in some subjects identical to the peak latency of the SEP recorded at the thoracolumbar junction, whilst in other subjects there was a distinct difference in latencies.

The spinal SEP recorded at the thoracolumbar junction consisted of a small early P wave followed by a prominent negative wave of approximately 3-5 ms duration and 1-5 uV amplitude. The N wave often had an inflection on the up slope and after peak negativity fell away to a long positivity. The latency of the peak negativity of the thoracolumbar SEP had a highly significant direct correlation with both limb length ($r = 0.92, p < 0.001$) and subject age (Fig.3.12). The spinal SEP recorded in the low thoracic region was similar in morphology although the inflection preceding maximal negativity was less remarkable and the amplitude was generally lower (Fig.3.10 and 3.11).

The transmission velocity in the tibial nerve and cauda equina increased directly with age until 30 months ($r = 0.50$, $p < 0.02$), thereafter the age related increase in conduction velocity was not significant ($r = 0.38$, $0.1 < p < 0.05$). The range for nerve conduction velocity was from 26 m/s in a four week old patient to 60 m/s in a three and a half year old patient.

Spinal SEP waveforms recorded in children under anaesthetic were identical to those recorded without anaesthetic, and the difference between peak latencies of the thoracolumbar SEP recorded in anesthetized and awake patients was always less than 0.6 ms.

Cortical SEPs

Cortical SEPs following tibial nerve stimulation were obtained in 24 subjects. The waveform consisted of a polyphasic P1 N1 P2 N2 complex (Fig.3.13). In some subjects a prominent N component

preceeded the P1 component by 5-7 ms. The range of peak latencies for P1 ranged from 26-42 ms. There was no correlation between subjects' age and latency of P1 (Fig.3.14). The N1 component followed between 6-12 ms after P1. Subsequently there were usually three to five other prominent peaks and troughs in the first 200ms post stimulus.

General anaesthetic did not alter the latency of the P1 component of this cortical SEP waveform by more than 1.9 ms from the value recorded in the awake subject. However as with the SEP from median nerve stimulation the subsequent components were variable both in latency and amplitude.

Data for lumbar cord to scalp transit time was available in 12 patients. This conduction time ranged from 14 to 24 ms (Fig.3.15). There was no difference in this parameter with age in the subjects studied ($r = 0.09$, $p > 0.1$).

DISCUSSION

The report of Willis and associates (1984), is the only account of subcortical SEPs following median nerve stimulation in neonates and infants. Their detailed study of children aged less than 13 months examined short latency SEP waveforms and presents data similar to that obtained in this study. Their finding that the peak latencies of the Erb's point and Cv2 potentials remained approximately 6.0 and 8.5 ms respectively, until 12 months of age, is complimented by this study which suggests that the latencies of these potentials remain stable until approximately 2

years. Thereafter there is a close relationship between both age and limb length and the peak latency of these potentials.

This study, together with the recently published report by Gilmore and associates (1985), is the first comprehensive report in children that uses a referential system to record spinal SEPs following lower limb peripheral nerve stimulation. The methodology used in these studies is almost identical to that used elsewhere in adults (Delbeke et al, 1978; Dimitrijevic et al, 1978; El-Negamy and Sedgwick, 1978; Phillips and Daube, 1980). A characteristic negative potential with variable inflections and maximal amplitude occuring over the thoracolumbar junction has been reported in all these studies.

The two different patterns of lumbosacral SEP recorded in this study may reflect the influence of different stimulus intensities and therefore differential augmentation of the neuroanatomical generators of the component N1 and N2 peaks. These peaks have been postulated to be the afferent and efferent limbs of the H reflex (Delbeke et al,1978; Lemkuhl et al, 1984). The N2 potential has also been attributed to volume conduction of the L1 potential, and posysynaptic activity in the sacral segments (Lemkuhl et al, 1984) Alternatively slight variations in the position of the recording electrode may also contribute to variations in waveform. Spinal SEPs recorded over L4 and S2 in adults (Delbeke et al,1978; Lemkuhl et al, 1984) correspond to the two waveforms elicited at the lumbosacral junction in this study. In children these spinal levels are much closer therefore the morphology of this SEP may be particularly dependent on exact

location of the recording electrode.

Recording spinal SEPs over the upper thoracic and cervico-thoracic junction from tibial nerve stimulation was possible in thin children. As the recording site was moved rostrally the negativity became broader whilst the early components (P1-N complex) of the waveform had latencies similar to that recorded in SEPs at the lumbosacral junction (Fig 6). It would therefore appear that the more rostral recordings are predominantly volume conducted potentials. This finding complements previous findings (Happel et al, 1975; Ertekin, 1978). Although some have used these potentials to calculate spinal conduction velocities (Cracco et al, 1975 and 1979; Hashimoto et al, 1984). our experience, and that of others (Schiff et al, 1984; Small and Mathews (1984), with these potentials would suggest that the problems in their acquisition as well as their non specificity limit their clinical utility.

Several studies in children have documented spinal SEPs using bipolar electrode montages (Cracco et al, 1975, 1979; Hashimoto et al, 1984). Direct comparison of SEP waveforms recorded using referential and sequential bipolar recording montages is difficult. Nonetheless, several findings have consistently been reproduced. The increase in conduction velocity in the tibial nerve and cauda equina is significantly related to age until approximately 30 to 36 months, thereafter conduction velocity attains adult values (Cracco et al,1975 and 1979; Hashimoto et al, 1984). There is also a highly significant relationship between the peak latency of the thoracolumbar SEP in the first

decade and both leg length and age (Gilmore et al, 1985). This finding together with the close relationship between both upper limb length and age and the peak latencies of the Cv2 and Erb's point potentials in children over two years of age suggest that increasing afferent pathway length is not offset by increasing fibre diameter and myelination. Afferent conduction in these pathways can therefore be explained by a simple cable model of a lengthening myelinated pathway (Gilmore et al, 1985).

Cortical SEPs folloowing median nerve stimulation in infancy and childhood have previously been described (Hrbek et al, 1968 and 1973; Blair et al, 1971; Cullity et al, 1976; Laget et al, 1976; Desmedt et al, 1976; Willis et al, 1984). Several of these studies have inadequate descriptions of recording methodology, the subjects studied and waveform analysis, and most have also emphasized long latency (150-350 ms) components rather than the short latency components (Hrbek et al, 1968 and 1973; Blair et al, 1971; Cullity et al ,1976).

In contrast the changing pattern and latencies of the cortical N1-P1 peaks following median nerve stimulation have been well documented with the latency of N1 decreasing from approximately 23 ms at birth to 16-20 ms at 12 months (Laget et al, 1976; Desmedt et al, 1976; Willis et al, 1984). There is conflicting data concerning the latency of N1 in children older than 12 months. Although there is agreement that the N1 latency does not changewith age during the first decade, its absolute value has been variably reported. The finding in this study of a mean of 16.5 ms lies between the values of 12.5 -16.0 ms (Desmedt et al,

1976) and 19.2 ms (Laget et al, 1976). This variability may be partly exlpained by shifts in N1 latency that occur with stimulus intensity and the position of the scalp recording electrode relative to the underlying neural generators (Desmedt et al, 1976; Tsumoto et al, 1972).

One change in the morphology of the cortical SEP following median nerve stimulation that occurs with age is shortening of the duration of the N1 peak (Desmedt et al, 1976; Laget et al, 1976). This maturational change occurs predominantly in the first two months of life , an age group not documented in this study. Laget and colleagues (1976) also noted that in children aged greater than six years the P1 trough could be recorded as either a single peak (38%) or a doublet (62%) with an interpeak latency of 15 ms. Although our experience is similar the interpeak latency of the doublet in this study, of approximately 6-8 ms, was much shorter. Variations in the latency of maximal positivity of the P1 trough will depend upon the morphology and the relative amplitudes of the peaks if it is a doublet. Under these circumstances the significance of changes in the N1-P1 interpeak latency with age may be difficult to interpret. This cortical interpeak latency decreased from approximately 8ms at one year of age to 6 ms at age 14 years, others have however reported values of 4-5 ms at age 12 months (Willis et al, 1984) and 9 ms from four months to 15 years (Laget et al, 1976).

In contrast to cortical SEPs following median nerve stimulation cortical SEP data from tibial nerve stimulation was difficult to obtain. There was often no apparent technical or methodological

cause for failure of signal duplication since most subjects had readily acquired spinal SEPs. Increasing stimulus intensity and the number of events averaged usually resulted in more muscle artefact and increased irritability even in the sedated patient. Furthermore this SEP was often difficult to obtain even with the patient under general anesthesia. Some difficulty with cortical SEP acquisition following unilateral lower limb peripheral nerve stimulation has previously been described (Jones and Small, 1978; Kimura et al, 1978; Chehrazi et al,1981; Nuwer and Dawson, 1984; Schiff et al, 1984) whilst others deny difficulty (Gilmore et al, 1985).Bilateral tibial nerve stimulation has been shown to increase the incidence of satisfactory signal acquisition (Kimura et al, 1981; Schiff et al, 1984). The tolerance of children to this technique may however be unacceptable, and this facility was not available for this study.

The lack of correlation between age and both P1 and N1 of the cortical SEPs following tibial and median nerve stimulation respectively, would suggest that myelination of the afferent pathways offsets increases in conduction pathway length. This explanation which utilises a model of a lengthening and widening, more rapidly conducting cable can be satisfactorily invoked for subcortical pathways (Gilmore et al, 1985). It is however an inadquate model for diencephalic-cortical projections since it neglects the plasticity of the developing brain. The increasing arborization and synaptic complexity of the dendrites of the receptor neurones of the primary somatosensory cortex, together with variable rates of maturation of the specific and nonspecific thalamocortical projections all contribute to changes in the

latency and complexity of the short latency components of the cortical SEPs (Yakolev et al, 1962; Macchi, 1983).

The variability of the N1-P1 component of the cortical SEP following median nerve stimulation, both in terms of interpeak latency and morphology of N1 and P1 are manifestations of brain maturation during the first decade (Desmedt et al, 1976; Laget et al, 1976; Willis et al, 1984). Gilmore and associates (1985) have also noted a similar variability in the morphology of the P1 component of the cortical SEP following tibial nerve stimulation. These workers also noted a poorer correlation between both subjects' age and height and onset latency of this SEP when compared to the close relationship between P1 and height in adults. Other groups have confirmed the latter finding (Lastimosa et al, 1982; Tsuji et al, 1984).

The age ranges and small number of subjects in whom lumbar cord to cortical transit times were recorded limit analysis of this parameter. There appeared however to be little change in cord to scalp conduction time with age during the first decade. This finding and the absolute values recorded for this parameter, complement the work of Gilmore and associates (1985) who recorded values between 10.8 and 19.7 ms in children aged from one to eight years. Interestingly this central conduction time in adults has been found to be independent of height and to have a mean value of 15-16 ms (Lastimosa et al, 1982; Tsuji et al, 1984).

Hume and colleagues (1982), have shown that CSCT does not alter significantly with age from 10 to 79 years, and that in the

decade 10-19 years, CSCT was 6.03 ± 0.23ms for males and 5.56 ± 0.38ms for females. The data from this study suggests that from age two to 13 years, a slow decrement in CSCT occurs with adult values obtained from approximately seven years of age.

In the subjects studied the latency and amplitude of subcortical and spinal SEPs were not influenced by atropine premedication and general anesthesia with nitrous oxide/oxygen. In contrast components of the cortical SEPs subsequent to N1-P1 were profoundly altered in both latency and amplitude. This effect parallels the influence of various stages of sleep on the SEP waveform in that the longer latency components are particularly influenced (Cracco et al, 1972; Tsumoto et al, 1972; Desmedt et al, 1980). Similar findings have also been reported with intraoperative recordings of SEPs (Raudzens,1982; Worth et al, 1982).

TABLE 3.1

SUMMARY OF CORRELATIONS BETWEEN PEAK LATENCIES AND TRANSIT TIMES AND SUBJECTS AGE AND LIMB LENGTH

SEP	n	Parameter	r	P
Erbs Pt	41	Age	0.84	$P < 0.001$
Erbs Pt	41	Arm Length	0.91	$P < 0.001$
Cv 2	36	Age	0.81	$P < 0.001$
Cv 2	36	Arm Length	0.86	$P < 0.001$
C3/C4:Fpz	26	Age	0.26	$P > 0.1$
Ll	44	Age	0.88	$P < 0.001$
Ll	44	Leg Length	0.92	$P < 0.001$
Cz':Fpz	24	Age	0.06	$P > 0.1$
CSCT	23	Age	-0.22	$P < 0.1$
Lumbar-Scalp	12	Age	0.09	$P > 0.1$

r = Correlation coefficient

P = probability valve

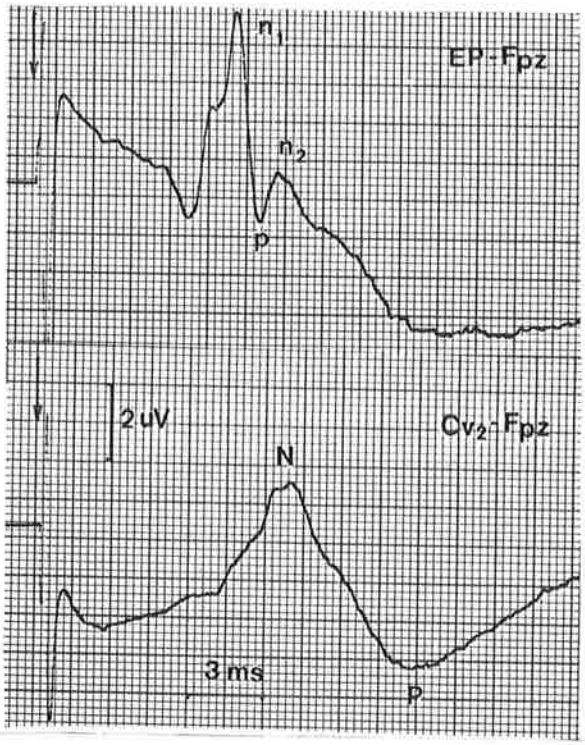


Fig 3.1 Erb's Point (EP) and cervical (Cv2) SEPs, referenced to Fpz, following median nerve stimulation in a 7 years old child. An inflection prior to N1 of the Erb's peak is well seen. The N2 peak corresponds closely to the latency of the N peak recorded at Cv2. (Arrows at left represent stimulus onset in this and subsequent diagrams)

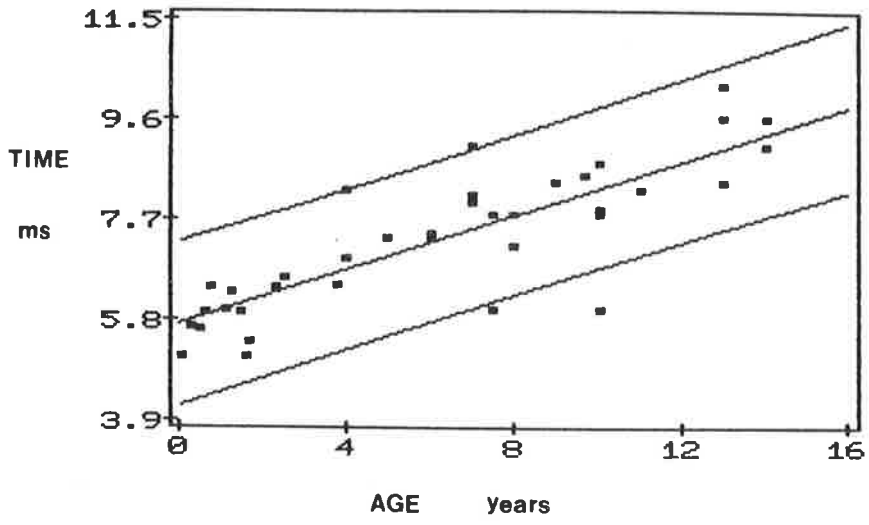


Fig 3.2 Scatter diagram showing the relationship between time of negative peak latency of the Erb's SEP (N9 equivalent) and subjects' age. This correlation is highly significant ($r = 0.84$, $p < 0.001$). In this and subsequent scatter diagrams the regression line is central and the upper and lower 95% data confidence limits parallel to it.

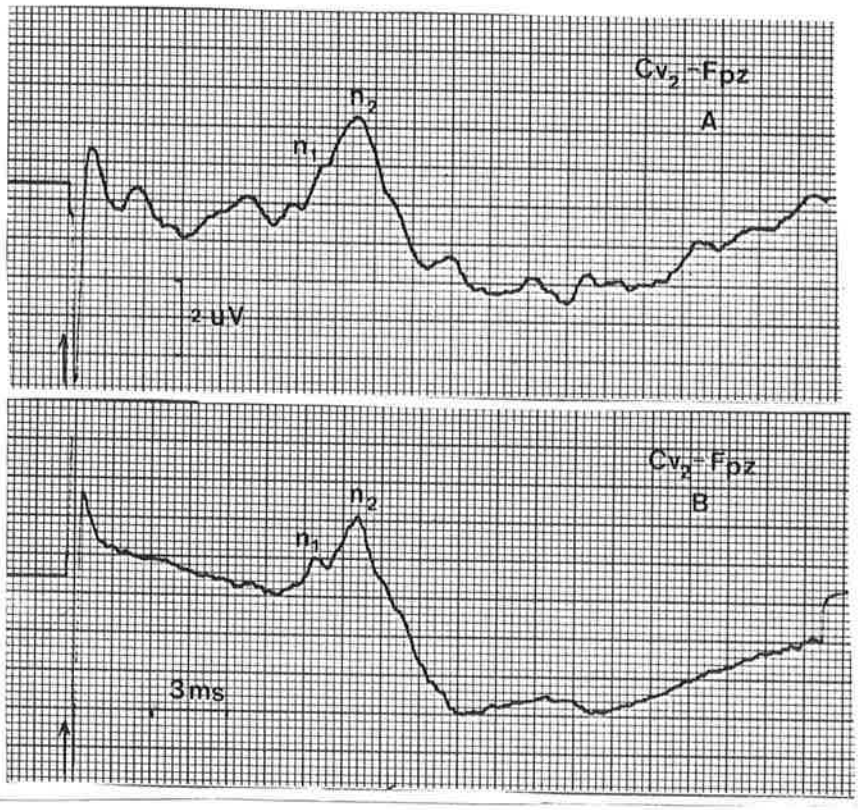


Fig 3.3 The cervical (Cv2) SEP referenced to Fpz following median nerve stimulation in a 10 year old child. The effect of general anaesthesia (Trace A) on the amplitude and latency on the waveform is minimal when compared to an outpatient recording (Trace B). The n1 and n2 peaks represent the equivalent of the adult N11, and N13 peaks.

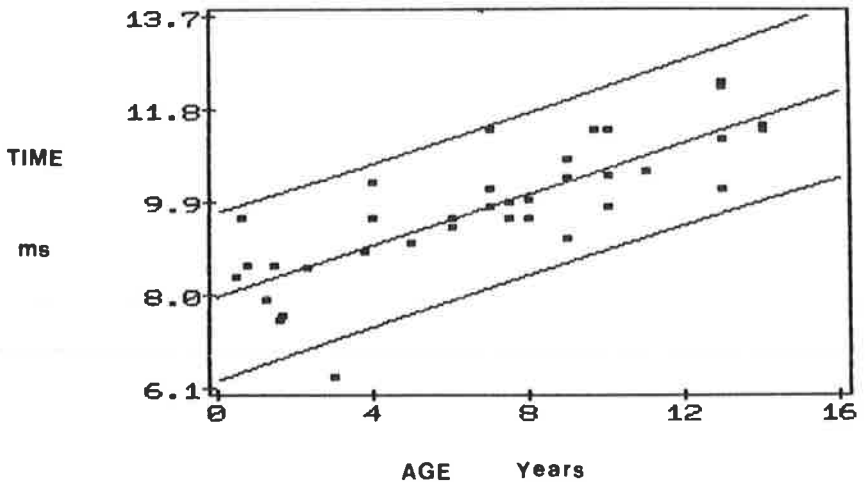


Fig 3.4 Scatter diagram showing relationship between negative peak latency of the Cv2 SEP (N13 equivalent) and subjects' age. This correlation is highly significant ($r = 0.81$, $p < 0.001$).

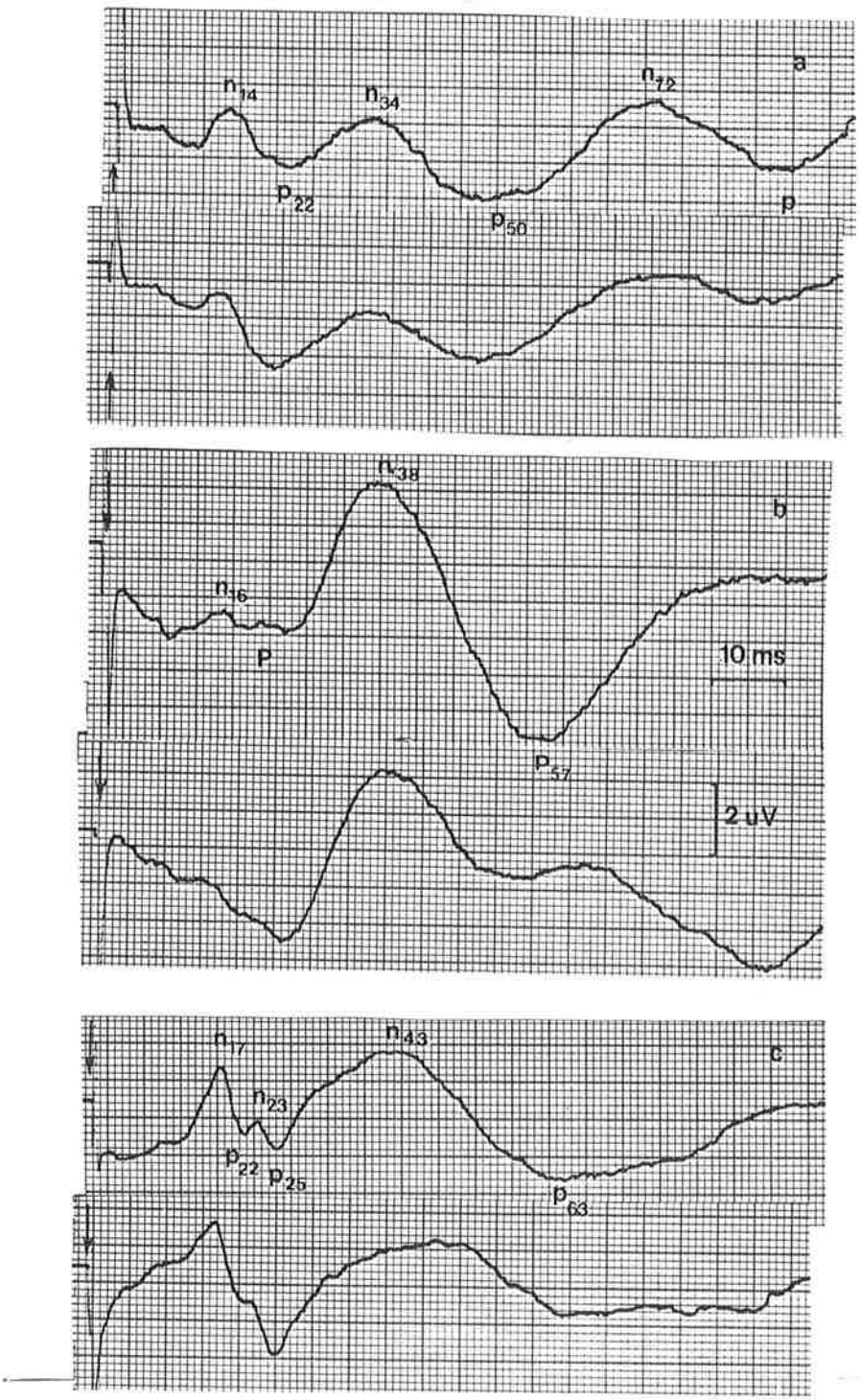


Fig 3.5 Cortical SEP (C3:Fpz) traces, recorded following median nerve stimulation in a 7 month (Traces a), a 4 year (Traces b) and an 8 year old child (Traces C). All SEPs are composed of an N1 peak that is followed by a positive, often bifid, trough. Latency variations reflect differences in limb length, peripheral and central nervous system myelination.

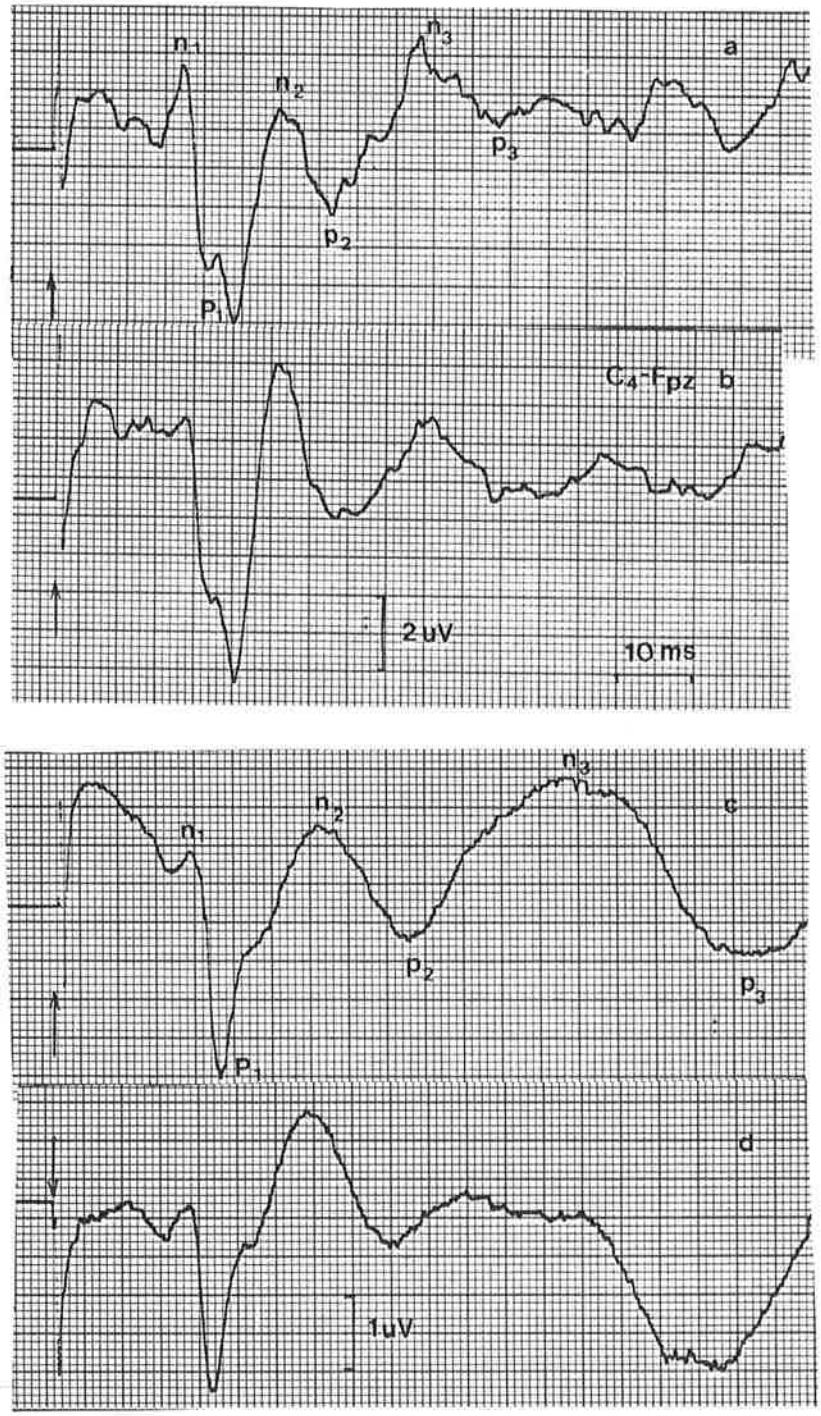


Fig 3.6 Cortical SEP (C4:Fpz) traces, recorded following median stimulation in a 10 year old child. Traces a,b were recorded as an outpatient and traces c,d under general anaesthetic. The short latency components (N1,P trough) are almost identical (16.8; 21.2; 23.6 ms as an outpatient and 17.2; 20.8, 25.1 ms with anaesthesic). The components following the P trough are reproducible but vary substantially in latency between studies. The bifid character of the P1 trough is not so well seen in the recording under anaesthetic.

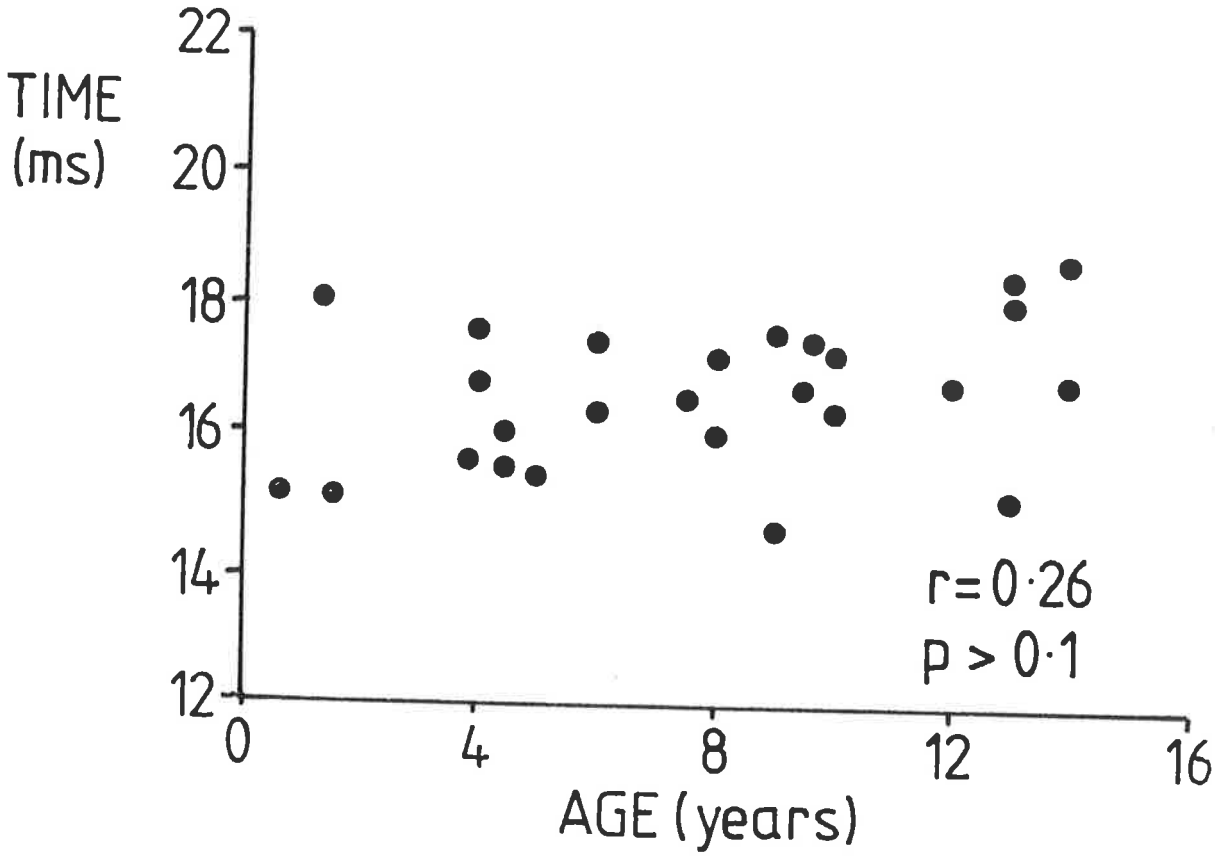


Fig 3.7 Scatter digram showing the relationship between latency of the cortical N1 component following median nerve stimulation (N18 equivalent) and subjects age. The correlation between these two parameters is not significant ($r = 0.26$, $p > 0.1$).

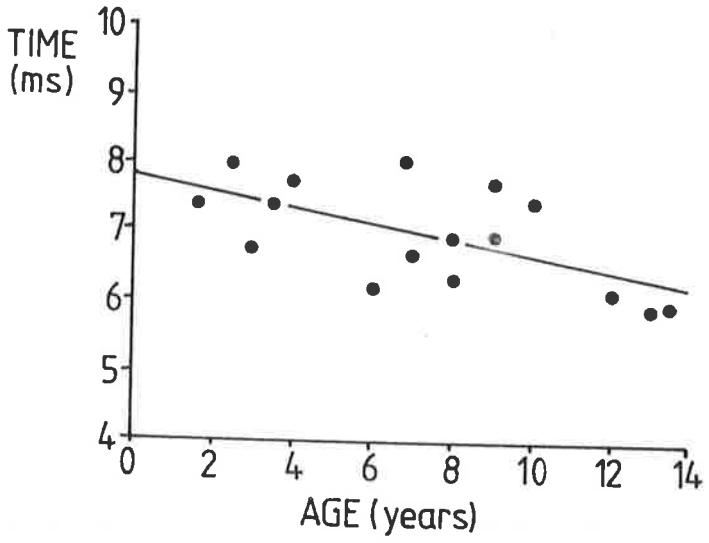


Fig 3.8 Scatter diagram showing the relationship between the median nerve evoked cortical N1-P1 interpeak latency and subjects age. This correlation is significant ($r = -0.59$, $p < 0.05$).

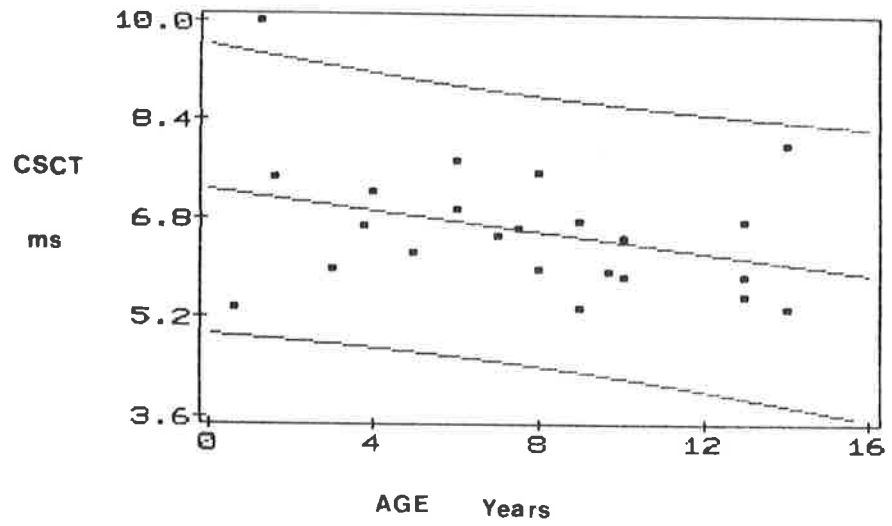


Fig 3.9 Scatter diagram showing the relationship between CSCT and subjects' age. Although there was a decrement in this conduction time with age in childhood it was not significant ($r = -0.22$, $p > 0.1$)

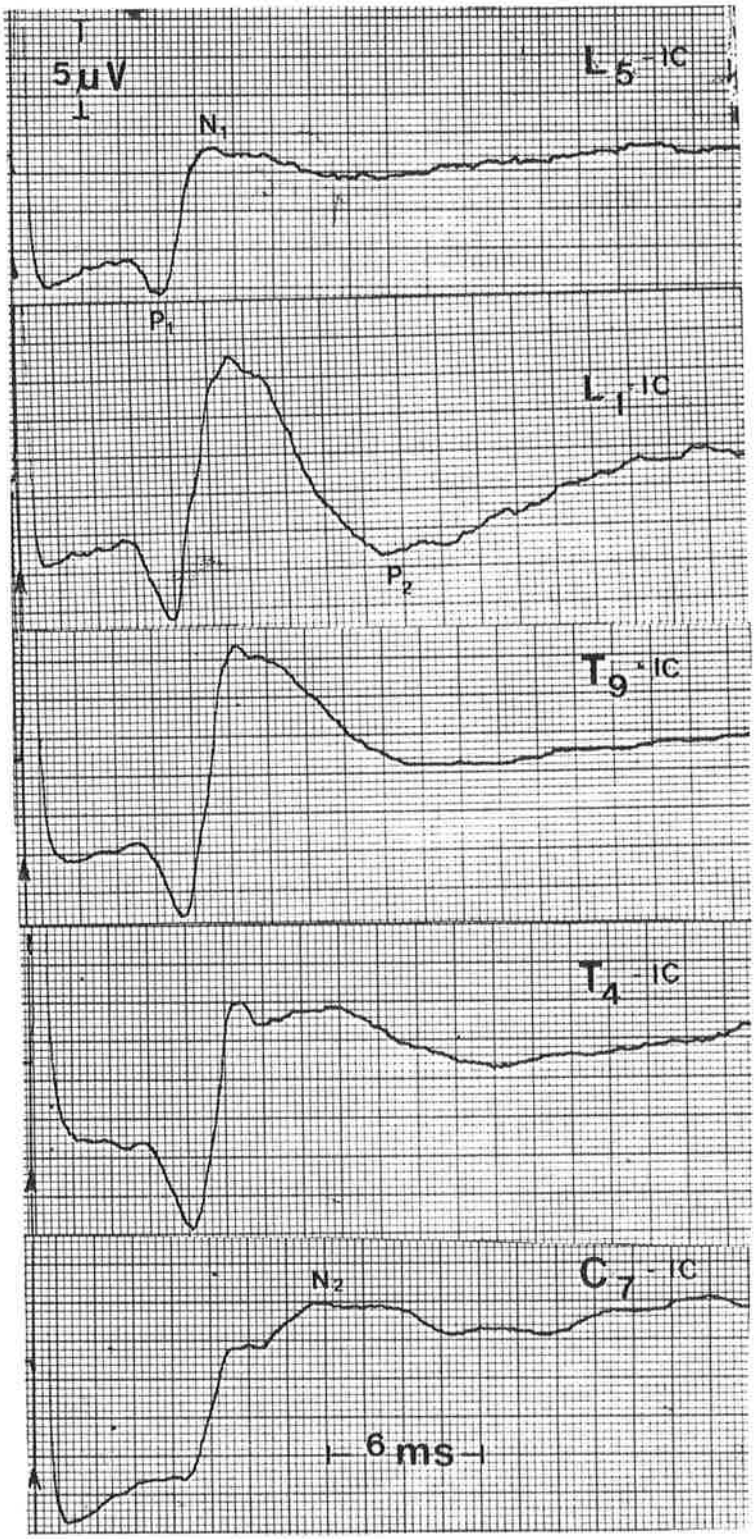


Fig 3.10 Surface recorded spinal SEPs referenced totheiliac crest (IC) following tibial nerve stimulation in a 2 year 6 month child. The latency of P1 is similar regardless of level of recording. The latency of N1 is almost identical from L4 to T4. These findings suggest that these surface recordings represent predominantly volume conducted potentials. The later N2 component may represent the ascending volley.

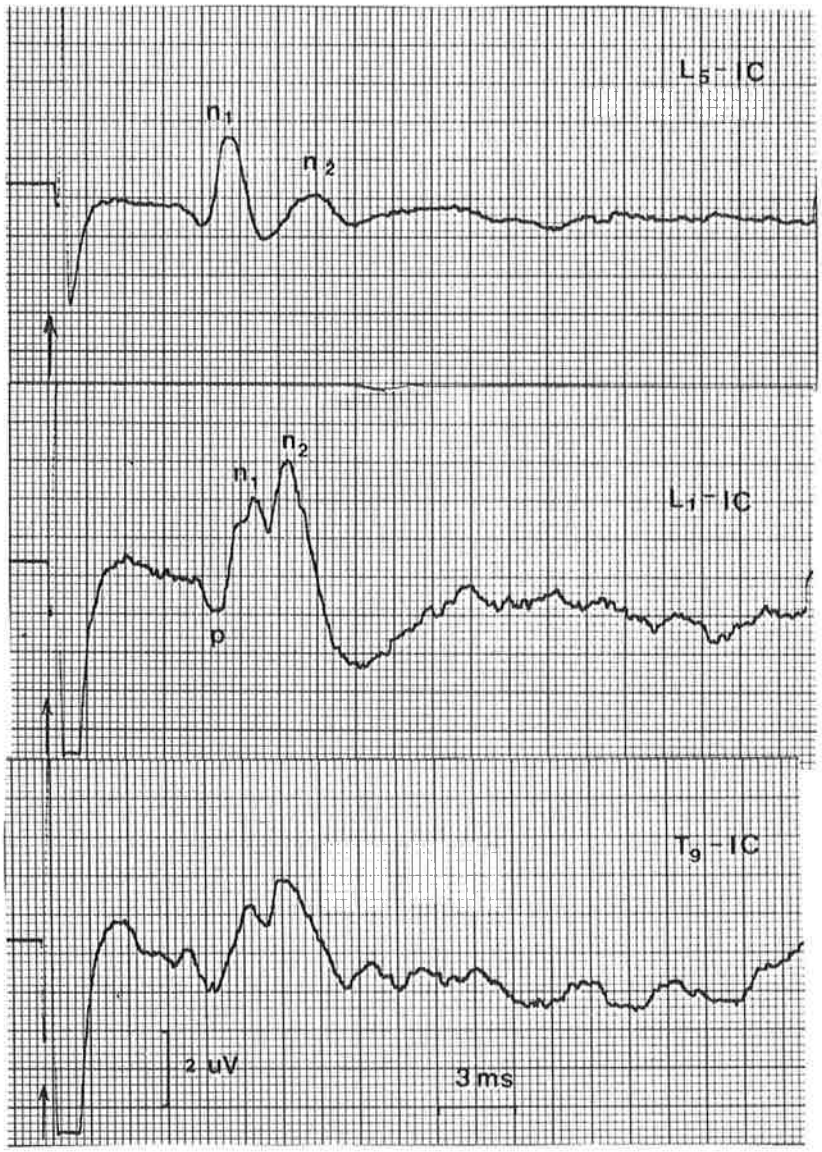


Fig 3.11 Surface recorded spinal SEPs referenced to the iliac crest (IC) following tibial nerve stimulation in a 6 year old. The lumbosacral SEP (L5:IC) is bifid (cf Fig 2.2). The latency of the lumbosacral n2 peak does not correspond with maximal peak latency of the L1:IC SEP. The n1 and n2 peaks recorded at L1:IC and T9:IC are almost identical at 8.16/8.20 ms (n1) and 8.40/8.36 ms (n2) respectively.

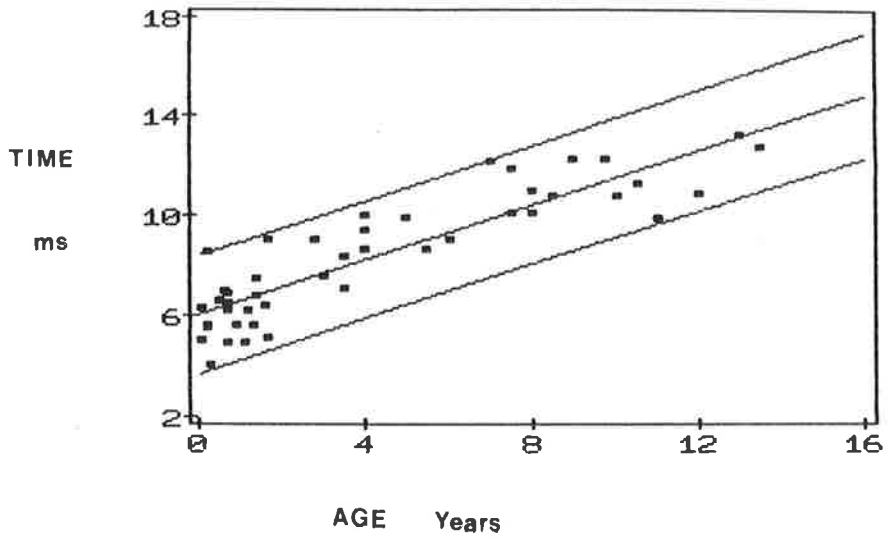


Fig 3.12 Scatter diagram showing the relationship between the peak negativity of the L1:IC SEP (N14 equivalent) and subjects age. This correlation is highly significant ($r = 0.88$. $p < 0.001$).

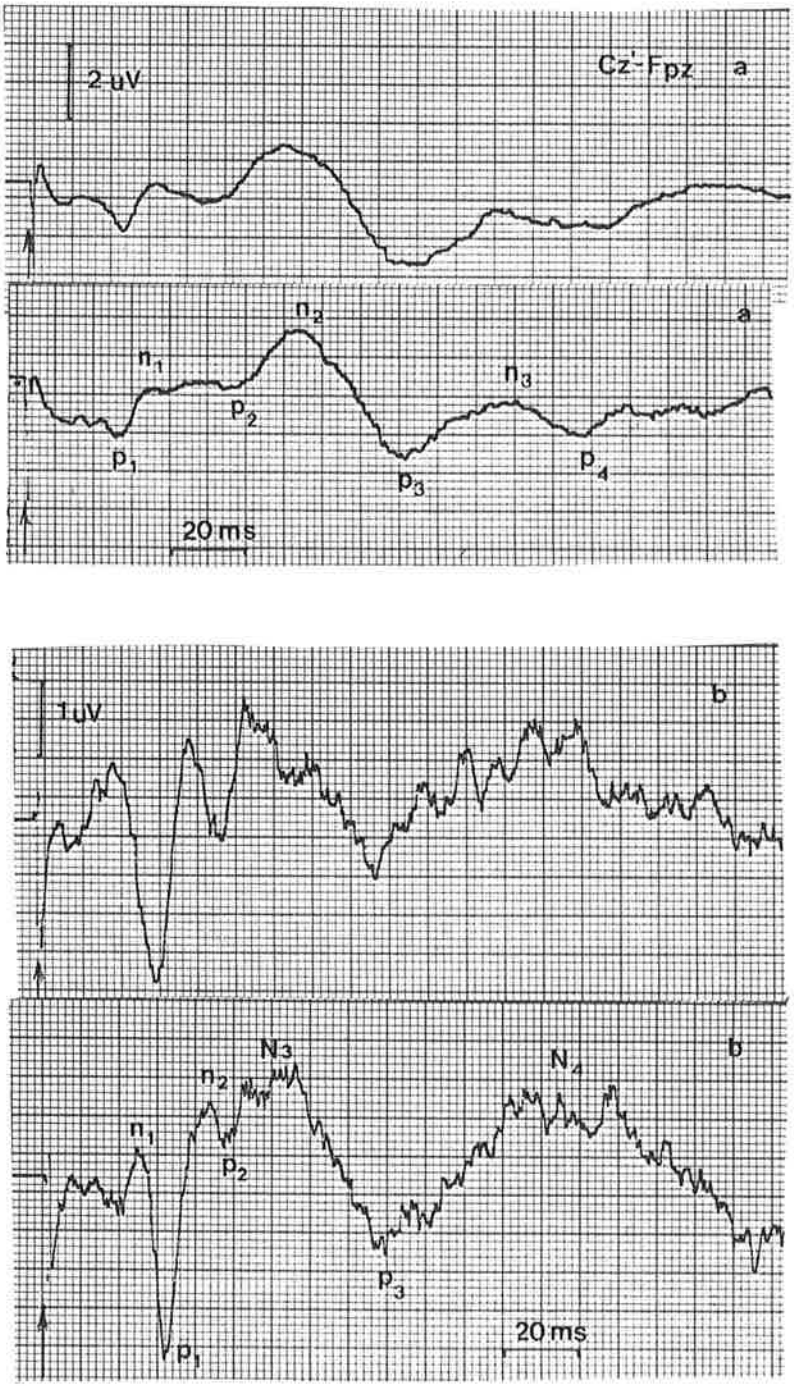


Fig 3.13 Cortical SEPs (Cz':Fpz) following tibial nerve stimulation in a 7 month (Traces a) and 10 year old child (Traces b). The initial N-P1 complex is not well defined in the 7 month child, however both waveforms are multicomponent in the first 200 ms post stimulus. Traces a with sedation, traces b nonsedated.

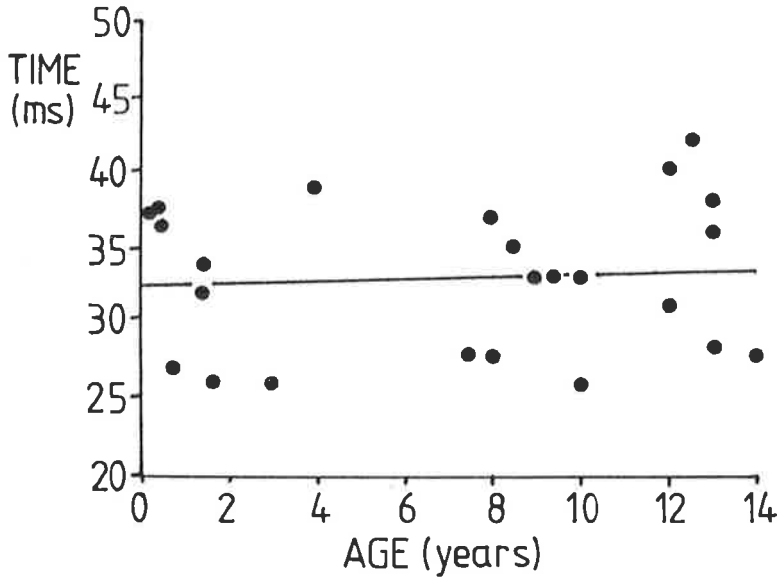


Fig 3.14 Scatter diagram showing the relationship between the latency of the tibial evoked cortical P1 component, recorded at Cz':Fpz, and subjects' age. This correlation is not significant ($r = 0.06$, $p > 0.1$).

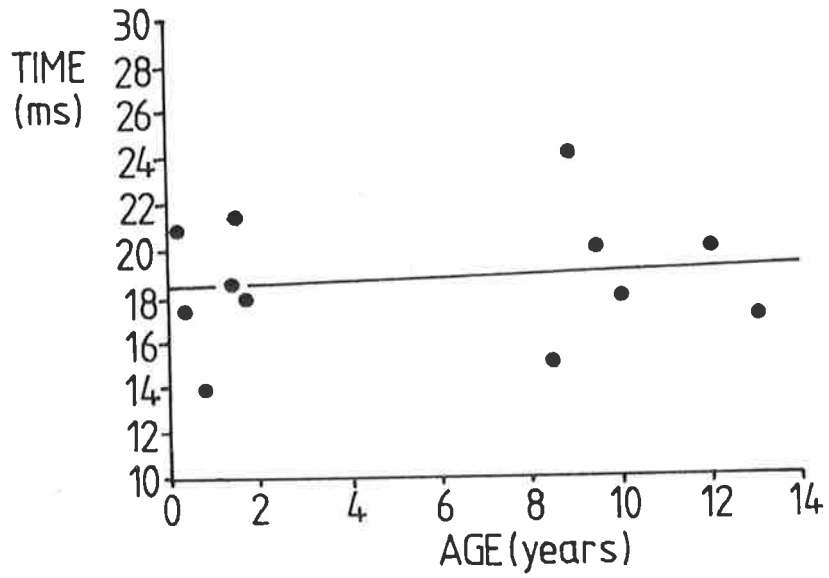


Fig 3.15 Scatter diagram showing the relationship between the lumbar cord to scalp transit time and subjects' age. This correlation is not significant ($r = 0.09$. $p > 0.1$).

SECTION 4

THE EFFECTS OF SPINOVERTEBRAL DISORDERS ON SHORT LATENCY SOMATOSENSORY EVOKED POTENTIALS IN CHILDREN

INTRODUCTION

The relationship between changes in SEPs and disorders of the spinovertebral axis have been reported in several adult studies (Caccia et al, 1976; Dorfmann et al, 1980; Sedgwick and El-Nagemy, 1980; Chehrazi et al, 1981; Lemkuhl et al, 1984; Stohr et al, 1982; Ertekin et al, 1984; Schiff et al, 1984). In contrast, there have been few studies investigating the utility and possible applications of SEPs in paediatric spinal neurosurgical disorders (Riegel et al, 1976; Duckworth et al, 1976). This omission may be partly explained by the time consuming and difficult nature of SEP recording in children (Desmedt et al,1976; see Section 3), and recent advances in neuroimaging techniques that allow increasingly more accurate localisation and classification of lesions.

The aim of this study was to investigate the utility of SEPs as an objective measure of the functional status of neural pathways in children with congenital and acquired spinal pathology. Congenital malformations of both mesenchymal and ectodermal elements of the spinal axis comprise a major cause of long term morbidity in children (French, 1982). Although most authorities are now agreed that appropriate surgery for myelodysplasia and tethered cord syndromes can prevent later neurological

deterioration, some of these children nonetheless later develop neurogenic scoliosis, bladder dysfunction and musculoskeletal deformities of the lower limbs (Lassman and James, 1967; French, 1982; Hoffman et al, 1985; Lapras et al, 1985).

The points to be determined were (i) could adquate SEPs a be obtained in children with myelopathy and congenital spinal malformations, (ii) The correlation between clinicopathological and electrophysiological findings in these patients and (iii) to evaluate the utility of SEP studies in the immediate and long term management of children with spino-vertebral disorders.

CLINICAL MATERIAL

Twenty six patients investigated by the Department of Neurosurgery, Royal Alexandra Hospital for Children, because of spinal disorders were studied. The clinical findings and diagnoses are summarised in Table 4.1. Fifteen patients were female and 11 were male. Ages ranged from 12 months to 13 years. The anatomical location of the spinal pathology was lumbosacral segments (11 patients), cervical segments (six patients), thoracic segments (four patients) and the conus medullaris in one patient. Four patients had more than one site of spinal pathology (Cases 5,12,13,16).

The largest diagnostic subgroup was composed of 14 patients with spinal dysraphism. All these patients were clinically stable although seven had longstanding lower limb and sphincteric dsyfunction and several also had deformities of the lower limbs,

scoliosis and short stature. Eight patients had previously undergone surgical treatment to release a tethered conus medullaris, to remove the lipomatous mass associated with a lipomeningocele or to repair a meningocele at variable periods prior to the SEP study. Five patients had SEP studies prior to surgery for tethered cord syndromes.

Twelve patients had nondysraphic congenital, neoplastic or other spinovertebral disorders. Two patients (Cases 8,10) with intramedullary astrocytomas, that had been subtotally removed several years previously, were reinvestigated for worsening myelopathy. Two Melanesian children (Cases 11,14) had progressive clinical syndromes suggestive of cervical syringomyelia, and three children (Cases 8, 12, 25) were investigated because of progressive spastic quadrapareses. One patient (Case 26) was investigated because of a 6 month history of back pain associated with progressive paresis in the right leg and one child (Case 1) presented with a subacute transverse myelopathy. Three patients (Cases 2,3,23) were studied whilst hospitalized because of other medical problems.

METHODS

SEP RECORDINGS

The data in this study was obtained using equipment and parameters of stimulation, averaging and recording previously described for surface SEPs in section 3 of this thesis. Percutaneous electrical stimulation was performed over the median

nerve at the wrist, the tibial nerve in the politeal fossa and on occasions the posterior tibial nerve at the ankle and ulnar nerve at the wrist. SEPs were recorded over the thoracolumbar region (L1), the vertebrae prominans (Cv7), the spine of the axis (Cv2), and the scalp overlying the somatosensory cortex (Positions C3,C4 and Cz' from the International 10/20 system). The L1 electrode was referenced to the iliac crest contralateral to site of stimulation, whilst the remaining electrodes were referenced to Fpz (Jasper, 1958). In most cases data was obtained both caudal and rostral to the diseased spinal segment, and following both right and left sided peripheral nerve stimulation.

SEP ANALYSIS

SEP peak latencies, waveform configurations and central conduction times were compared to the normal data previously described in section 3. In some cases data from a clinically normal limb was compared to that obtained from a neurologically impaired limb. When assessing the SEPs major consideration was given to abnormalities of the latencies equivalent to the adult N9 (Erb's point), N13 (Cv2), and N18 cortical potentials following median nerve stimulation and N14(L1) and P28 cortical potentials following tibial nerve stimulation.

CLINICAL, PATHOLOGICAL AND ELECTROPHYSIOLOGICAL CORRELATIONS

In each case the relationship between electrophysiological and both clinical and pathological findings was assessed. Because the spinal dysraphic group encompassed such a wide spectrum of

pathology it was suddivided into patients with complex (Lipomyelomeningocele or tethered cord syndrome associated with other spinal pathology rostral to the conus medullaris) and simple (lipomeningocele or meningocele with tethered cord syndrome, and uncomplicated diastematomyelia) congenital malformations. The pathological diagnosis and site of the lesion was in each case confirmed by metrizamide myelography, spinal CT scanning or operative findings (Fig.4.1).

RESULTS

SEP Data

Good quality SEP waveforms were recorded in 24 of the 26 patients (Table 4.1 and Fig 4.2). In 12 patients short latency SEP peak latencies, waveform configuration and symettry were normal. Normal lumbar cord to scalp transit time was recorded in nine of these patients and ranged between 14-20 ms. In 12 patients spinal or cortical SEP waveforms had either delayed peak latencies, were absent or asymmetrical. In two patients (Cases 8,9) no spinal or cortical SEP waveforms following tibial nerve stimulation were reproducible. Although 6 patients required several recording sessions to obtain satisfactory SEP data the overall aquistion rate for cortical and thoracolumbar SEPs following tibial nerve stimulation in the series was 63% and 85% respectively. Satisfactory and diagnostic cervical SEPs following median nerve stimulation were obtained in every patient studied.

Of the 12 patients with abnormal SEPs eight had abnormalities of

the L1 or cortical SEPs following tibial nerve stimulation. Two of these patients (Cases 15,16) had unilateral loss of the L1 SEP and bilateral loss of the cortical SEP, two (Cases 5,13) had normal L1 SEPs but absent cortical SEPs , one (Case 18) had normal cortical SEPs but unrecordable L1 SEPs and one patient (Case 7) had unilateral prolongation of the scalp P1 cortical potential with ipsilateral delay in lumbar cord to scalp transit time (30 ms). Two patients (Cases 3,11) had delayed L1 peak latencies.

Three patients (Cases 10, 11, 14) had unilateral loss or delayed peak latencies of their cervical SEPs. One patient (Case 1) had bilaterally normal Erb's point SEPs but no cervical SEPs to either median or ulnar nerve stimulation.

Clinical and Electrophysiological correlations

SEP studies were normal in all 7 patients who had no clinical deficit, and abnormal in 7 patients with proprioceptive dysfunction. In the latter subgroup (Cases 1,5,8,10,14-16) there was often excellent correlation between the clinical deficit, the spinal segments involved and distortion of the SEP at or rostral to the diseased level (Fig 4.3 and 4.4).

In 12 patients with permutations of upper and lower motor neurone deficits and sphincteric dysfunction, but clinically normal sensation, eight had normal and four abnormal SEPs (Cases 3,7,9,13). These abnormalities were often minor and could be partially attributable to technical problems (Table 4.1).

Electrophysiological and Pathological correlations

The incidence of abnormal SEP recordings within each pathogenic subgroup is listed in table 4.2.

Twelve of the 14 patients with caudal spinal dysraphic disorders had L1 SEPs with normal peak latencies (Fig.4.2). Two of these patients (Cases 15,16) with unilateral hypoplastic and atrophic limbs did however have ipsilateral absence of the L1 SEP. The two patients in whom no L1 SEP was recordable included one child (Case 9) in whom no SEP data was recorded and one (Case 18) with a normal cortical SEP to tibial nerve stimulation. Prior surgical treatment of a spinal dysraphic malformation did not influence L1 SEP findings, since of eight patients who had previous spinal surgery seven had normal studies on at least one side. Five patients studied preoperatively with both complex and simple dysraphic malformations all had normal L1 SEPs.

Failure to record a cortical SEP following tibial nerve stimulation occurred in four patients with complex spinal dysraphism (Cases 5,13,15,19). One patient with an uncomplicated tethered cord syndrome (Case 7) had unilateral delay in onset of the cortical SEP to tibial nerve stimulation despite being neurologically normal.

Two patients (Cases 8,10) with intramedullary astrocytomas had SEP findings consistant with the location of the lesion and clinical findings (Fig.4.3). A thoracolumbar SEP was not obtained caudal to the lesion in Case 8, and intraoperative spinal SEP

recording in this patient revealed major attenuation of the ascending waveform (See section 8). At operation this patient had cystic myelomalacia of the dorsal spinal cord. The two other patients (Caes 10,26) with intramedullary astrocytomas were found at operation to have diffuse cord widening. The patient with von Recklinghausen's disease (Case 3) and an extramedullary intra and extradural neurofibroma causing compression of the conus medullaris (Fig.4.1), had a delayed L1:IC SEP which was most likely attributable to concurrent peripheral neurofibromatosis.

The two patients (Cases 12,25) with spinal canal stenosis, and the one (Case 4) with paraparesis of unknown etiology (? A juvenile type of upper motor neurone disease with a normal myelogram) all had normal SEPs despite profound spasticity and an almost complete myelographic block in one case (Case 25). The patient with transverse myelitis and quadraplegia (C5 level) from Devics Disease had a complete conduction block bilaterally rostral to Erb's point. No cervical SEPs to either ulnar or median nerve stimulation were recorded.

The three patients with cervical syringomyelia showed variable changes in SEPs. One patient (Case 2) treated by syringostomy three years prior to the study had normal SEPs. One patient (Case 14) studied preoperatively had delayed and attenuated Cv2 SEPs ipsilateral to the side of lesion (Fig.4.4), whilst the third (Case 11) showed serial changes following syringopleural shunting. In the latter case the initial cervical SEPs were symmetrical, however following a right dorsal myelotomy (at the

C7-C8 level) and insertion of a syringopleural shunt there was ipsilateral delay in Cv2 SEP latency. Nonetheless six months later there was good clinical improvement following shunting, and both the Cv2 and L1 peak latencies improved.

DISCUSSION

The findings in this study demonstrate that in children with a large spectrum of disorders of the spinovertebral axis reproducible subcortical and cortical SEPs from median and tibial nerve stimulation can be obtained. There is a close correlation between SEP abnormalities and proprioceptive dysfunction. This finding is consistant with previous reports that have shown that the SEP is transmitted along the dorsal columns (Halliday and Wakefield, 1963; Cusick et al, 1979). Accurate clinical assessment of dorsal column mediated functions in young children is difficult and this may partly explain why some patients with mainly motor and sphincteric dysfunction also had abnormal SEPs. Adults with spinal pathology not causing deficits in proprioception or perception of vibration have however been reported (Anziska and Cracco, 1980; Chehrazi et al, 1981; Schiff et al, 1984).

The most precise correlation between clinical and SEP findings was seen in patients with cervical spinal lesions where the N13 equivalent peak was either delayed or abolished on the side of the lesion. Similar findings have been reported in adults with syringomyelia, myelitis, multiple sclerosis, vascular and traumatic cord lesions, and cervical spondylitic myelopathy

(Anziska and Cracco, 1980; Chehrazi et al, 1981; Jones and Halliday, 1982; Stohr et al, 1982; Schiff et al, 1984). Although it was shown in only one case in this series previous studies have shown that the SEP findings are related to the etiology and duration of the lesion and the effects of treatment (Dorfmann et al, 1980; Schiff et al, 1984).

The relatively poor correlation between abnormalities of the L1 SEP and caudal spinal cord and cauda equinae pathology can largely be attributed to the disparity between the anatomical pathways transmitting and generating the SEP and those diseased. Many of the patients with caudal spinal dysraphism in this series had normal lower limbs but impaired sphincteric function regardless of the exact pathology or influence of prior treatment. This would suggest that spinal segments caudal to S_2 are the most severely diseased. Dysfunction in these segments is unlikely to be demonstrable on the thoracolumbar SEP following tibial nerve stimulation even though this potential represents neuronal activity in segments from L_4-S_3 (Davies et al, 1965). The relative contributions of each of these segments to this potential make it unlikely that even total dorsal rhizotomy of S_{23} would cause a significant change in its amplitude and latency. This problem is a major limitation to the use of SEPs both intraoperatively (see Section 8) and as an outpatient mode of assessment in patients with tethered cord syndromes. This finding was also noted in a recent urological study examining various electrophysiological methods of assessing sphincteric function in myelodysplastic patients (Light et al, 1984). In an attempt to overcome these problems there have been studies

investigating cortically recorded bladder evoked potentials (Badr et al, 1982 and 1984), and rectal and vesical electromyograhy and manometry (James et al, 1980; Pang et al, 1983).

The recording of a normal thoracolumbar SEP in a patient with a rostral spinal lesion, a finding in five patients in this series, has previously been reported. In 24 patients with various spinal lesions between the levels T10 and C1, Lemkuhl and colleagues (1984) found that in only 15% was no lumbar SEP obtainable, similar findings were also demonstrated in another study of 29 comparable patients (Sedgwick and El-Negamy, 1980). The persistance of segmental spinal activity following a rostral cord insult, with slowing of the lumbar cord to scalp transit time or loss of the cortical SEP were also the findings in several other studies in heterogenous groups of patients with medical and surgical myelopathy (Dorfmann et al, 1980; Chehrazi et al, 1981; Ertekin et al, 1984; Small and Mathews, 1984; Schiff et al, 1984).

The clinical value of lumbar cord to scalp conduction time as an accurate parameter of spinal cord function is unclear. There is a wide range of overlap between normal values and values in patients with spinal disorders (Lastimosa et al, 1982; Ertekin et al, 1984; Small and Mathews, 1984; Tsuji et al, 1984). Major prolongation of this parameter has most commonly been reported in patients with multiple sclerosis and following extradural cord compression (Ertekin et al, 1979; Schiff et al, 1984). Slowing of conduction is probably due to focal demyelination or axonal dysfunction (Dorfmann et al, 1980). These patterns of spinal cord damage could be expected to accompany some of the segmental

lesions present in patients in this study, however this central conduction time was prolonged in only one of ten patients in this study.

The most common finding during intraoperative recordings of spinal SEPs from the dorsal surface of the spinal cord both rostral and caudal to segmental lesions, has been attenuation of the ascending spinal potential rostral to the lesion with failure to record a cortical potential (see Section 7 and 8). Loss of the cortical SEP, rather than prolonged conduction time was also the most common finding in a study of patients with medical myelopathies (Small and Mathews, 1984).

The results from this study suggest that the clinical application of spinal and cortical SEPs in the management of children with neurosurgical disorders of the spinovertebral axis is limited by technical, clinical, anatomical and pathological factors. In some patients several recording sessions may be required to obtain satisfactory electrophysiological data. Spinal disorders causing clinical syndromes related to anterior and anterolateral spinal cord dysfunction are not likely to cause major changes in SEPs since these are essentially dorsal column mediated. Therefore even if SEPs generated by pudendal nerve stimulation were used to assess the S_{2-4} radicles (and there are obvious practical problems that preclude the use of this nerve in children) it is possible that neurogenic bladder dysfunction would precede SEP changes.

The other problem that vitiates the use of spinal SEPs in the

long term monitoring of patients with tethered cord syndromes is that the clinical deficits encountered during growth spurts predominantly cause neuromuscular and sphincteric dysfunction rather than sensineural impairment (French, 1982; Hoffman et al, 1985; Lapras et al, 1985). From the experience with the more clinically disabled patients in this series, and the low perecentage of SEP abnormalities in patients with only motor or sphincteric dysfunction it is unlikely that SEP changes would precede clinical deterioration.

PATIENT	AGE	DIAGNOSIS	CLINICAL	SEP DATA (ms)	COMMENT
1 MC	11 yr	Subacute myelopathy	Quadraplegia below C5 & loss of all sensation	EP:Fpz (Ulnar) = 6.8 v =44 m/s EP:Fpz(Median) = 6.7 v =50 m/s	No cervical SEPs No recovery of function
2 LT	14 yr	Cervicothoracic syringomyelia	Scoliosis, mild spasticity R - l legs, normal sensation Shunted hydrocephalus	L1:IC = 13.2 v = 49 m/s Cz':Fpz = P27 N34 P43 N55	Normal SEPs in clinically stable patient
3 SD	10 yr	Neurofibromatosis R T11 neurofibroma	Multiple subcutaneous neurofibromata Hyporeflexia R leg	L1:IC = 15.0 v = 40 m/s Cz':Fpz = P33 N38 P48 N73	Delay peak latency to conus SEP
4 VT	8 yr	Spastic paraparesis etiology unknown	Spastic paraparesis with no sensory loss	L1:IC = 8.8 v = 57 m/s Cz':Fpz = P28 N37 P46 N56	Normal conus SEP and cord-scalp conduction
5 CW	2 yr	Chiari III syndrome Syringobulbia Thick filum & TCS	Bulbar pareses, hypotonia and paresis legs, unable to crawl properly	L1:IC = 5.3 ms v = 40 m/s Cz':Fpz not reproducible	Normal conus SEP Progressive bulbar pareses
6 DC	8 yr	Treated tethered cord syndrome (Leptomyelolipoma)	Neurologically normal	L1:IC = 7.0 (7 yr) v = 54 m/s L1:IC = 8.5 (8 yr) v = 51 m/s Cz':Fpz = P28 N37 P55	Normal SEPs

TABLE 4.1. CLINICAL, DIAGNOSTIC AND SEP DATA FOR THE 26 PATIENTS WITH SPINAL CORD DISORDERS. Abbreviations R = right; l = left LMN = Lower motor neurone; JPS = Joint position sense; TCS = Tethered cord syndrome; v = velocity

PATIENT	AGE	DIAGNOSIS	CLINICAL	SEP DATA (ms)	COMMENT
7 GD	4 yr	Treated tethered cord syndrome (Thick filum syndrome)	Bladder dysfunction, no focal deficit	L1:IC = 8.4 v = 50 m/s Cz':Fpz = N26 P39 Cz':Fpz = N38 P55 N68 P100	Cortical SEP asymmetry, slow cord-scalp conduction on left
8 VW	14 yr	Thoracic spinal astrocytoma	Scoliosis, mild spastic diplegia, bilateral loss JPS hallux	No SEPs recordable from tibial n stimulation	Low amplitude, distorted Spinal SEP recorded subdurally at operation
9 BT	2 yr	Partially treated tethered cord syndrome	Bladder dysfunction, no focal deficit	No SEPs recordable from tibial n stimulation	Myelography revealed residual tethering of spinal cord by lipomeningocele
10 SF	4 yr	Cervical spinal astrocytoma	Paresis forearm, wrist & fingers(R) with hypokinesis and loss JPS indicis	Cv7(Median n)= -(R) 9.8(L) Cv7(Ulnar n) = -(R) 10.3(L)	No SEPs recordable on side of lesion.
11 FP	5 yr	Cervical syrinx	Kyphoscoliosis, bilateral leg spasticity, LMN signs l ¯ R hand, JPS normal	L1:IC = 15.4 v = 35 m/s Cv2 = 10.2 (R + 1) Cx = 17.2(l)	Delayed R Cv2 SEP after R dorsal myelotomy for insertion of syringopleural shunt. Latencies of both Cv2 and L1 SEPs improved after shunting
	6 yr	Syringopleural shunt	Spasticity & gait improved LMN signs in hands resolve on l moreso than R side	Cv7 = 10.2(R) 10.2(l) Cv2 = 11.4(R) 10.4(l) C3:Fpz = 17.4 CSCT(R) = 6.0 C4:Fpz = 17.2 CSCT(l) = 6.8	
	61/2 yr	review	Clinically unchanged	Cv7 = 9.6(R) 9.6(l) Cv2 = 10.6(R) 9.9(l) L1:IC = 14.6 v = 40 m/s	

TABLE 4.1 (Cont)

PATIENT	AGE	DIAGNOSIS	CLINICAL	SEP DATA (ms)	COMMENT
12 AT	3 yr	Achondroplasia congenital canal stenosis	Mild spasticity all limbs No obvious sensory loss	L1:IC = 8.5 v = 45 m/s Cz':Fpz = P26 N32 P45 N57	Dwarf with myelopathy due to cervical spinal stenosis Conus SEP normal
13 KB	11 yr	Treated tethered cord syndrome & thoracolumbar diastematomyelia	Bladder dysfunction only	L1:IC = 12.5 v = 51 m/s L1:IC = 15.8 v = 48 m/s No Cz':Fpz recordable	Clinically stable girl with complex spinal dysraphic disorder
14 YM	7 yr	Cervical syrinx	Loss pain/temperature & atrophy/paresis R hand	Cv2 = 11.6 (R) Cv2 = 9.8 (1)	Delayed & attenuated SEPs on side of clinical deficit
15 AW	8 yr	Partially treated tethered cord syndrome	Sensory loss, atrophy & paresis below L5(R),bladder dysfunction, 1 leg normal	L1:IC = - (R) L1:IC = 7.2 (1) v = 44m/s Cz':Fpz not recordable	Clinically stable girl with residual tethering of cord by lipomeningocele
16 GK	13 yr	Treated lumbar diastematomyelia & tethered cord syndrome	Kyphoscoliosis, incontinence urine, motor & sensory loss below L4(1), S2(R). Short stature	L1:IC = 7.0(R) Cz':Fpz and L1:IC (1) not recordable	Clinically stable boy with complex spinal dysraphic condition
17 PG	13 yr	Partially treated tethered cord syndrome (Lipomeningocele)	Paresis 1 leg in L45 and S1 myotomes, no sensory loss	L1;IC = 20.1 v = 54 m/s Cz':Fpz = P38 N44 P54 N63 P70 Cz':Fpz = P36 N44 P53 N65 P70	Clinically stable boy with residual tethered cord on myelography. SEPs symmetrical

TABLE 4.1.(Cont)

PATIENT	AGE	DIAGNOSIS	CLINICAL	SEP DATA (ms)	COMMENT
18 DT	12 yr	Tethered cord syndrome	Neurologically normal, dimple and hypertrichosis low lumbar region	L1:IC not recordable Cz:Fpz = P42 N52 P68 N81	Clinically stable girl Normal cortical SEPs but no conus SEP
19 JB	11 yr	?Tethered cord syndrome	Neurologically normal, lumbo-sacral hpertrichosis	L1;IC = 8.3(R+l) v = 60 m/s Cz':Fpz = P27 N38	Normal SEPs
20 DG	9 yr	Thoracic diastematomyelia	Scoliosis, neurologically normal	L1:IC = 9.6(R+l) v = 52 m/s Cz':Fpz = P29 N39 P51	Normal SEPs
21 DL	1 yr	Repaired meningocele Residual tethering of cord with intradural epidermoid tumour	Neurologically normal Lumbosacral midline lipoma	L1:IC = 6.0(R+l) v = 42 m/s	Normal conus SEPs Preoperative study
22 EH	1 yr	Lipomeningocele & tethered cord	Neurologically normal Lumbosacral midline lipoma	L1:IC = 5.3 v = 34 m/s	Normal conus SEPs Preoperative study
23 MH	18 mth	Anterior sacral meningocele and caudal agenesis syndrome	Lower limbs normal, probable bisphincteric dysfunction (Chronic constipation)	L1:IC = 7.5 v = 51 m/s	Normal conus SEPs Preoperative study
24 JB	18 mth	Lipomeningocele & tethered cord	Neurologically normal Lumbosacral midline lipoma	L1:IC = 5.7 v = 47 m/s	Normal conus SEPs Preoperative study

TABLE 4.1 (Cont)

PATIENT	AGE	DIAGNOSIS	CLINICAL	SEP DATA (ms)	COMMENT
25 WH	7 yr	Congenital vertebral anomalies with cervical subluxation	Spastic tetraparesis with LMN signs both hands. No sensory deficit	Cv7 = 9.7 (R+1) v = 49 m/s	Symmetrical cervical SEPs
26 RS	10 yr	Conus astrocytoma	Paresis R thigh & foot with loss of KJ & AJ. No sensory loss	L1:IC = 8.3 (R+1) v = 48m/s Cz':Fpz = N21 P26 N31 P51 (R) Cz':Fpz = N21 P25 N27 P31 (1)	Asymettrical cortical SEPs on preoperative study

TABLE 4.1 (Cont)

TABLE 4.2

SEP FINDINGS RELATED TO SPINAL PATHOLOGY FOR THE 26 PATIENTS STUDIED IN SECTION 4.

PATHOLOGY	NORMAL SEP	ABNORMAL SEP
SPINAL DYSRAPHISM		
Simple	6	3
Complex	1	4
SPINAL TUMOUR	2	2
SYRINGOMYELIA	1*	2
SPINAL CANAL STENOSIS	2	-
MYELITIS/DEMYELINATION	1	1
CAUDAL AGENESIS SYNDROME	1	-
	14	12

* TREATED SYRINX

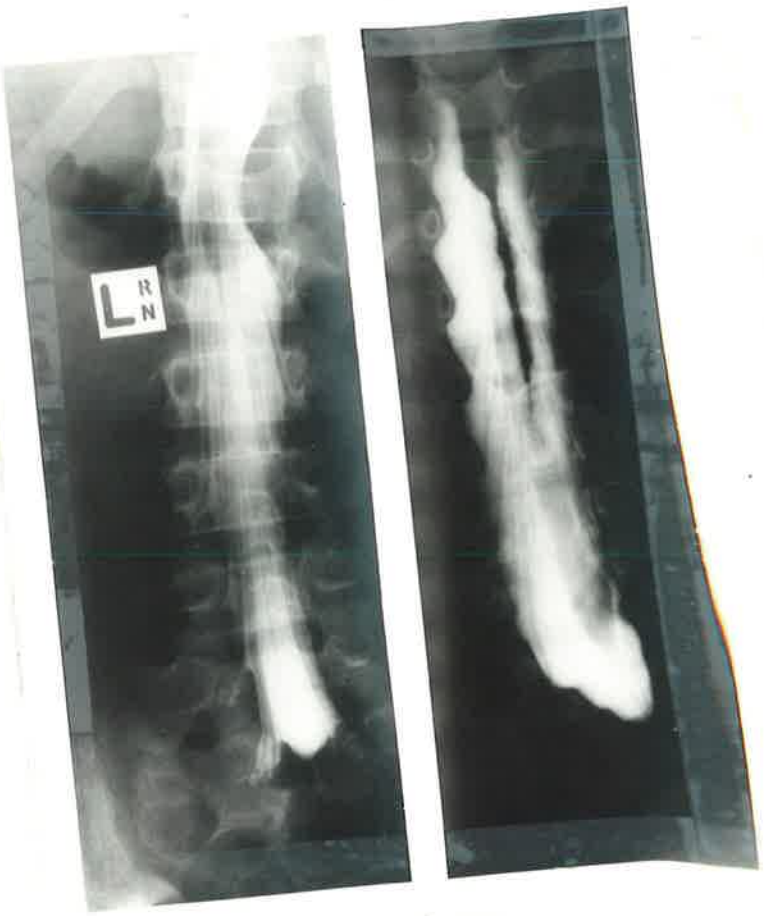


Fig 4.1 Metrizamide myelograms of cases 3 (left) and 5 (right). The right sided neurofibroma (left) and thick filum tethering an abnormal conus in a redundant sacral CSF cistern (right) are well seen. Except for delayed onset of the SEP in Case 3, both these patients had normal conus (L1:IC) SEPs despite major spinal pathology.

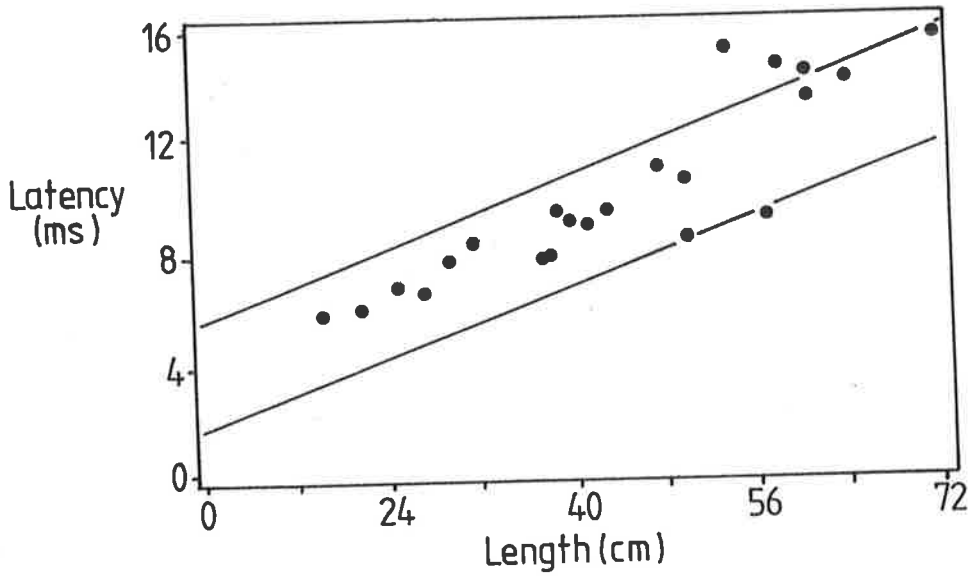


Fig 4.2 Scatter diagram showing relationship between the peak latency of the L1:IC (conus) SEP and leg length in patients with congenital, neoplastic and other spinal disorders. Most patients have SEPs with normal peak latencies (parallel lines mark values within $\pm$ 2 standard deviations of normal). Delayed peak latencies were seen in one patient with peripheral neurofibromata and one patient with a cervicothoracic syrinx.

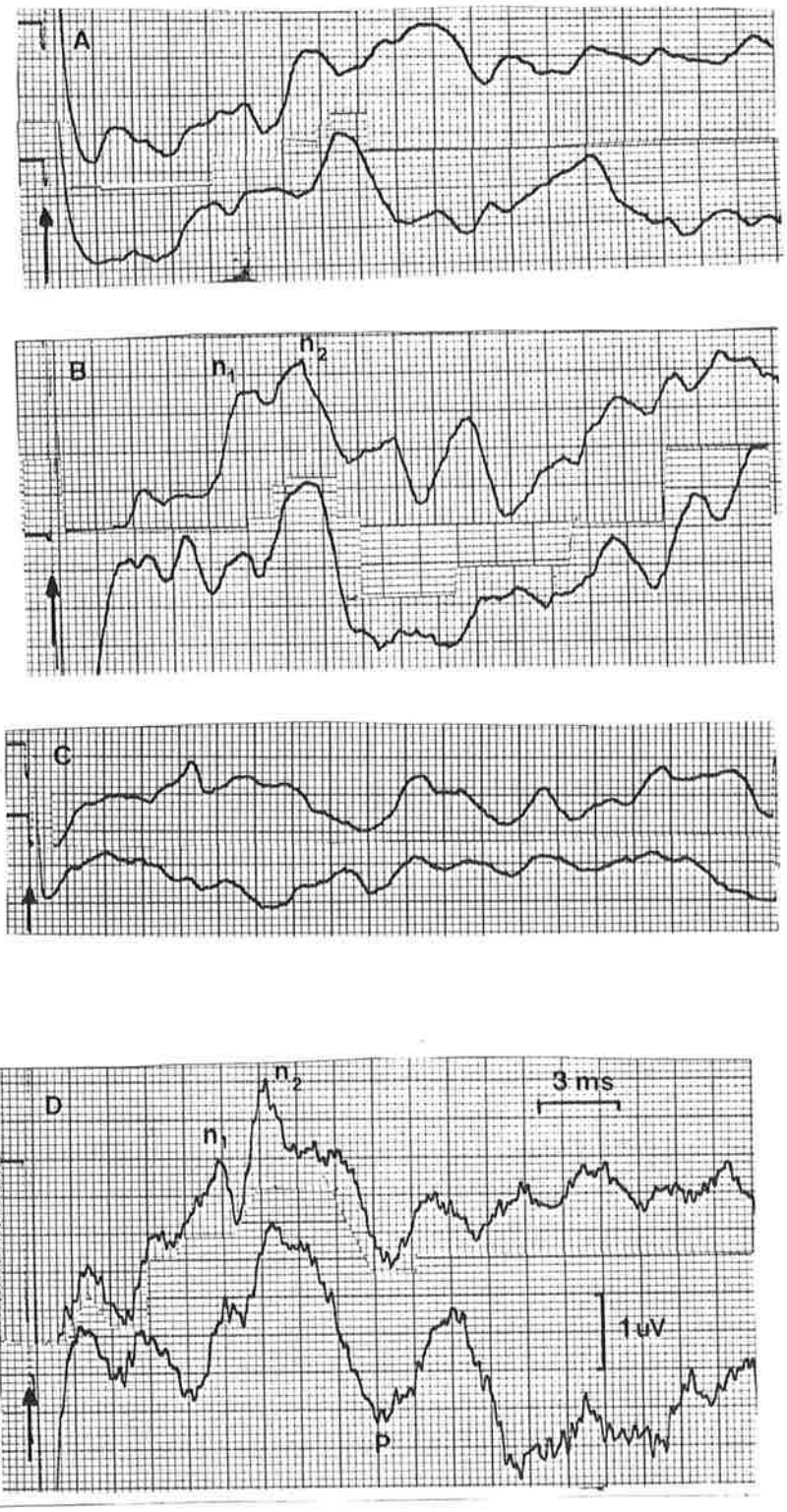


Fig 4.3 Cervical (Cv7) SEPs in a patient with right upper limb dysfunction caused by a low grade intramedullary astrocytoma (Case 10). There is ipsilateral loss of the SEP following ulnar (Traces A) and median (Traces C) nerve stimulation. The left sided SEPs are preserved with peak latencies (n2) of 10.3 ms following ulnar (Traces B) and 9,8 ms following median (Traces D) nerve stimulation.

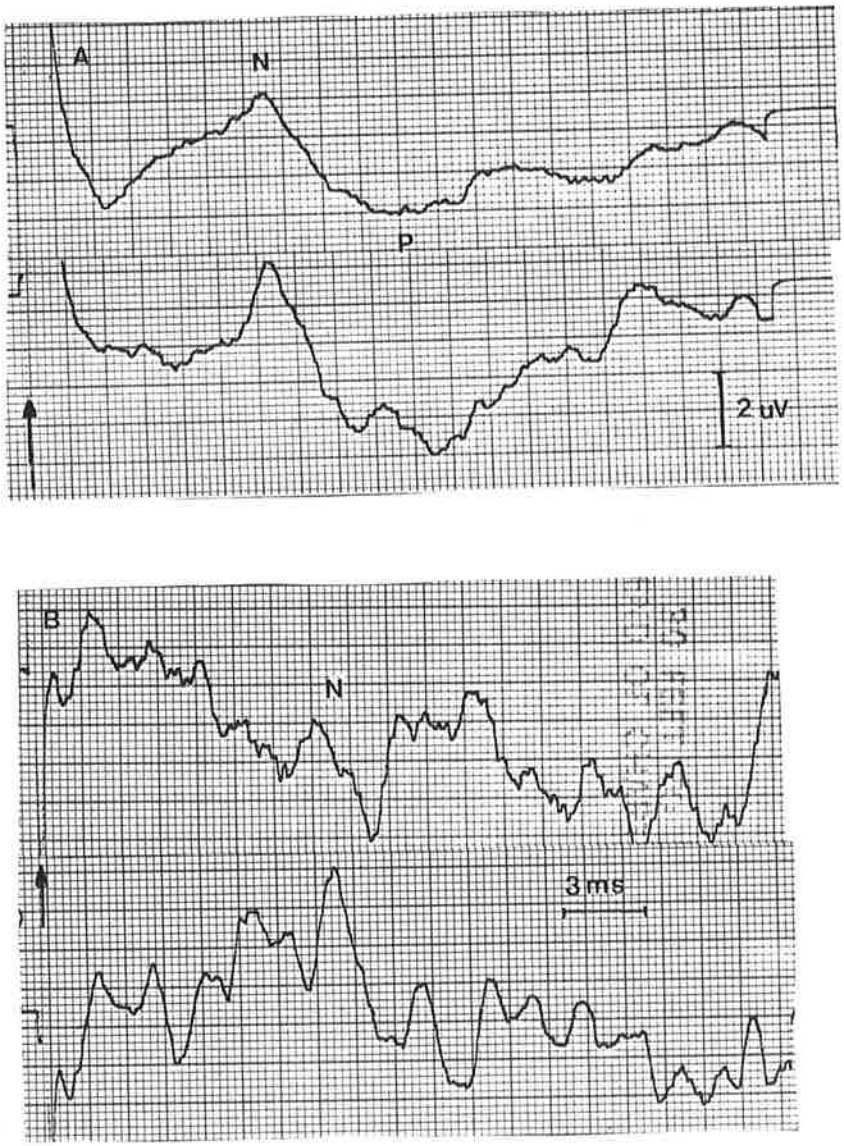


Fig 4.4 Cervical (Cv2) SEPs in a patient with syringomyelia causing right upper limb dysfunction (Case 14). There is a prominent N peak at 9.8 ms following left median nerve stimulation (Traces A). The right sided response, which is somewhat masked by "noise", is delayed with peak negativity at 11.6 ms (Traces B).

SECTION 5

SHORT LATENCY SOMATOSENSORY EVOKED POTENTIAL FINDINGS FOLLOWING MAJOR HEAD INJURY IN CHILDREN

INTRODUCTION

The etiology and outcome following head injury in children is different from that seen in adults (Bruce et al, 1978; Narayan et al, 1981; McLaurin and McLennan, 1982). Neurotrauma in adults is dominated by multitrauma victims from motor vehicle accidents. The pediatric neurotrauma group encompasse a wider spectrum of etiologies that reflect the increasing motor activity , inquisitiveness and indepedance of children. Despite the differences in etiology and the development and maturation of the brain at the time of injury, the principles of clinical assessment of the head injured adult and paediatric patient are similar (Teasdale and Jennett, 1974; McLaurin and McLennan, 1982).

Therapeutic protocols that include the use of muscle relaxants, intubation and mechanical ventilation, sedatives and barbiturates are now commonly used in the intensive management of neurotrauma patients (McLaurin and McLennan, 1982; Lutschg et al, 1983). Such therapy does however limit or preclude clinical assessment. It is in this category of patients that evoked potential studies have been employed to provide information about the functional integrity of afferent neural pathways (Larson et al, 1973; Greenberg et al, 1977; Hume et al, 1979; de la Torre et al, 1979;

Greenberg et al, 1981; Hume and Cant, 1981; Narayan et al, 1981; Lutschg et al, 1983; Strickbine-van Reet et al, 1984).

In this study the relationships between SEP findings and Glasgow coma score (Teasdale and Jennett, 1974) and neurological outcome (Jennett and Bond, 1975) have been assessed in a heterogenous group of head injured children. The results from this study are compared to the other studies that have utilised SEPs in the assessment of comatose and head injured patients.

CLINICAL MATERIAL

Fourteen patients, seven male and seven female, whose ages ranged from 3 to 11 years, were studied. All patients suffered from coma for at least six hours post injury. Coma was defined as an inability to obey commands, to speak or to open the eyes (Teasdale & Jennett, 1979). Eight patients suffered a closed head injury, two uncomplicated penetrating craniocerebral insults and four various injuries complicated by intracranial hematomas. The clinical and CT findings in these patients are summarised in Table 5.1.

Five patients required major craniocerebral surgery in the management of the head injury. These procedures included elevation and debridement of compound depressed craniocerebral injuries and evacuation of intracranial hematomas (Cases 1,2,7,11 and 13). Two of these patients (Cases 1,2) had such extensive calvarial fragmentation and cerebral swelling that bone fragments and craniotomy flaps were not replaced. Neither of these patients

had ICP monitoring despite prolonged paralysis and ventilation.

The intensive management of the patients included maintenance of normal hydration, and the use of phenytoin (loading dose 12 mg/kg, with 5 mg/kg/day maintenance dose) in those patients with intracranial hematomas or cortical lacerations complicating penetrating head injury. ICP monitoring with either a Richmond Bolt or an intraventricular catheter was performed in six patients. Nine patients required intermittent positive pressure ventilation for either control of ICP, or because of associated thoracopulmonary injury. As well as intermittent hyperventilation, 20% Mannitol solution was also used to control elevated ICP. In three patients (Cases 1,2 and 6), with refractory intracranial hypertension, high dose intravenous Pentobarbital therapy (5mg/kg/hr) was also used.

METHODS

SEP RECORDINGS

The equipment and methods of stimulation, recording and averaging described in Section 3 were used to obtain the SEP data. Only the Cv2:Fpz and C3/C4:Fpz SEPs following median nerve stimulation were studied. In some patients these SEPs were generated following median nerve stimulation at the elbow since venous and arterial monitoring lines precluded access to the median nerve at the wrist. In paralysed patients a stimulus intensity of 80 V was used to generate the SEP. In some patients with extensive scalp lacerations and bandaging platinum alloy sub dermal recording

electrodes were used rather than the stick on Ag/AgCl electrodes. All the initial recordings were made within 24 hours of injury and in the intensive care unit.

SEP ANALYSIS:

The CSCT, the peak latencies of the primary cortical complex (N1 and P1), the N1-P1 interpeak latency and cortical waveform configuration were compared to the normal data described in section 3. In those patients who had SEPs generated by stimulation of the median nerve at the elbow only the CSCT and waveform configuration were compared to normal. When cortical SEPs were asymettrical the CSCT was calculated using the N1 peak latency from the more normal hemisphere.

CLINICAL, RADIOLOGICAL AND ELECTROPHYSIOLOGICAL CORRELATIONS

The GCS at the time of recording the SEPs was documented in each case. In those patients paralysed and ventilated at the time of recording the GCS prior to intubation was used. In these patients the ICP was also documented. All patients were examined for focal neurological deficits. Clinical and electrophysiological findings were correlated with initial computerised tomographic (CT) findings.

Serial SEP studies were performed on those patients with prolonged hospitalization. The outcome using the Glasgow outcome scale of Jennett and Bond (1975) was based upon clinical assessment at time of discharge or last outpatient visit.

RESULTS

Although there were problems in recording patients in the intensive care environment because of electrical "noise" and access to sites of stimulation and recording satisfactory short latency SEP complexes were obtained in all 14 patients within 24 hours of injury. Seven patients had serial SEP studies during the course of recovery and rehabilitation. In some of these patients recording several sessions had to be abandoned because of patient irritability. Sixteen of the 27 recordings obtained were made whilst patients were paralysed (Pancuronium) and ventilated.

The peak latencies of the N1 component of the Cv2:Fpz SEP and N1 and P1 components of the cortical SEP following median nerve stimulation were bilaterally normal in 11 patients. The N1-P1 interpeak latency in eight patients ranged between 4.8 to 9.2 ms (Normal for children in first decade 7.3 $\pm$ 0.7ms). In three patients where the P1 trough was either single or a doublet with maximal positivity occurring at the first trough the N1-P1 interpeak latency ranged between 2.8 and 3.0 ms. In ten patients with normal thalamic and parietal regions on CT scans the interhemispheric variation in N1-P1 interpeak latency was 0 to 1.5 ms (mean 0.6 ms). One patient (Case 2) with gross unilateral cerebral swelling complicating a penetrating craniocerebral injury had a prolonged N1-P1 ipsilateral to the injury.

In three patients an abnormal cortical N1P1 complex was obtained. In one patient (Case 6) there was bilateral abscence of these

components (Fig.5.1) until three months post trauma. In another patient (Case 3) there was a bilateral asymettrical abnormality of N1P1 (Fig. 5.2), and in the third there was unilateral loss P1 ipsilateral to a large posterior parietal intracerebral hematoma (Fig 5.3). The latencies for the cervical SEP and N1 and P1 of the cortical SEPs are summarised in Table 5.1.

In 13 of the 14 patients the initial CSCT was within the normal range (Table 5.1 and Fig.5.4). In one patient (Case 6) it was not obtainable due to bilateral loss of the short latency cortical SEP components. In seven patients having multiple recordings, the range from fastest to slowest CSCT was less than 0.4ms in five patients and 1.0 and 1.2ms in the two others. Paradoxically in both the latter patients the prolongation in CSCT occurred as the patients were recovering.

There was no relationship between GCS at the time of recording and CSCT. Patients with GCS of less than 5, having a mean CSCT of 6.7 $\pm$ 0.5 ms, those with GCS between 6 and 8 inclusive had a mean CSCT of 6.8 $\pm$ 0.5 ms and those patients with GCS greater than 9 had a mean CSCT of 6.5 $\pm$ 0.4 ms. These differences are not significant (Paired t-test). The two patients in therapeutic barbiturate coma with normal short latency cortical SEPs both had normal CSCT.

The cortical SEP waveform appeared to become more polyphasic as the GCS increased. The mean number of reproducible peaks or troughs was 3 when GCS was 5 or less, four when GCS was between 6 and 8 inclusive, and 5 when GCS was greater than 9. It should be

pointed out however that three of the 14 patients with more severe injuries (and thus lower GCS) were receiving high dose intravenous barbiturates as part of their therapeutic protocol.

Of the 14 patients studied nine made a full recovery. Three patients (Cases 1,2 and 12) suffered from moderate disabilities and two patients (Cases 3,6) remained vegative. Both these patients sustained severe closed head injuries and had gross abnormalities on initial cortical SEP recordings, with neither having a normal N1-P1 complex (Fig 5.1 and 5.2). The course of one of these patients was complicated by uncontrollable intracranial hypertension (ICP intermittently greater than 60- 90 mm Hg for over 2 weeks). This patient regained a multicomponent cortical SEP several months after injury, with a normal CSCT, despite a clinical status resembling akinectic mutism. The other patient with a vegetative outcome had suffered a prolonged hypoxic insult prior to resuscitation at the scene of his accident.

DISCUSSION

The utility of SEPs in the evaluation of comatose patients with severe head injuries depends upon the correlation between the SEP findings and subsequent clinical course. In this series there was a correlation between good clinical outcome and both a normal CSCT and preservation of the N1 P1 cortical components on initial SEP. The importance of the cortical N1P1 complex , which represents function in the diencephalic-primary somatosensory cortical projection pathway (Goldie et al, 1981; Chiappa and

Ropper, 1982; Lutschg et al, 1983), in the prediction of both survival and focal neurological deficits has previously been reported. Loss of the short latency cortical SEP components has been associated with death or a vegetative outcome, whilst preservation of these components, even if abnormal, was associated with significantly better outcome (Greenberg et al, 1977; Goldie et al, 1981; Hume and Cant, 1981; Lutschg et al, 1983). Unilateral loss of these short latency cortical components has also been predictive of a focal neurological deficit (Greenberg et al, 1981; Hume and Cant, 1981; Narayan et al, 1981).

In contrast two earlier studies have found loss of long latency cortical components, even with preservation of short latency components was predictive of a bad outcome (de la Torre et al, 1978) and no relationship between SEPs and level of coma (Larson et al, 1973). The short latency cortical SEP components were not specifically studied in either of these reports and method of waveform analysis was not specifically stated. Comparison of results from different series is difficult because of differences in clinical criteria for admission to the studies, differences in the time of recording the SEPs after head injury, and differences in therapeutic regimes and age groups of the patients comprising the studies. Nonetheless the similarity in clinico-electrophysiological findings in this study and other recent series (Greenberg et al, 1977; Hume and Cant, 1979; Goldie et al, 1981; Greenberg et al, 1981; Narayan et al, 1981; Lutschg et al, 1983) would seem to justify the Greenberg classification of SEP abnormalities into Grades I-IV, with Grade I being normal and

Grade IV being complete abscence of the signal. Lindsay and colleagues (1981) have however reported that a simple count of the number of waves present in the evoked potential is the best guide to data analysis, since delays in peak latencies and low signal amplitude can make classification of the waveform difficult.

The number of components in the waveform was not necessarily related to eventual outcome but did seem related to the Glasgow Coma Score at the time of recording. Loss of the longer latency components was a frequent finding in patients with Glasgow Coma Scores of 5 or less. This relationship has previously been documented (Lindsay et al, 1981). Some of these findings may partly be explained by the use of opiates and barbiturates in the more severely injured patients, since it has been shown that these drugs influence the longer latency components of the cortical SEP but neither the CSCT nor N1 P1 components of the cortical SEP (Hume and Cant, 1979; Grundy et al, 1980; Hume and Cant, 1981; Lutschg et al, 1983; Newlon et al, 1983). The return of components to the SEP in the months following injury, as seen in Case 6, has also previousl been noted (Hume and Cant,1981; Lutschg et al, 1983).

The predictive value of the CSCT in comatose patients has been investigated in both children and adults (Hume and Cant, 1981; Lutschg et al, 1983). In a study o f 94 head injured patients Hume and Cant (1981) found that normalisation of CSCT in the first ten days following injury was associated with a favourable outcome, and that CSCT was not significantly altered by the pH,

blood gases or the use of barbiturate therapy. Some patients with normal CSCTs were severely disabled and there was also intertrial variability between patients. CSCT was found to be delayed in eight of 15 children suffering coma following head injury (Lutschg et al, 1983). However only one of these patients died and four fully recovered. Paradoxically, as in this study, some of the prolonged CSCTs were documented as the clinical condition of the patients improved.

The pathological prolongation of CSCT documented by Lutschg and colleagues (1983) suggest that there was major abnormality in the short latency components of the cortical SEP. Since the CSCT is dependent on the integrity of the N1 cortical component for its calculation, major distortion of the primary cortical complex (N1-P1) limits its use as an index of brain stem function. Most of the previous studies of SEPs in head injury have used the cortical SEP waveform to evaluate the functional status of the diencephalon and hemisphere and the BAER to evaluate rhombencephalic function (Greenberg et al, 1977 and 1981; Goldie et al, 1981; Lindsay et al, 1981; Narayan et al, 1981). The use of multi-modality evoked potentials (SEPs, VERs, BAERs) has been shown in several studies to evaluate central nervous system dysfunction much more comprehensively than any one of these modalities used alone and abnormalities in multi-modality evoked potentials also correlate well with outcome (Greenberg et al, 1977; Greenberg et al, 1981; Lindsey et al, 1981; Narayan et al, 1981; Lutschg et al, 1983; Strickbine-van Reet et al, 1984).

The relative infrequency of SEP abnormalities in this series, and

the generally good outcome of the patients could suggest that the sample was not representative of pediatric neurotrauma. In two comparable adult head injury studies 45-61% of the patients died or remained vegetative (Hume and Cant, 1981; Lindsay et al, 1981), similarly in the pediatric study of Lutschg and colleagues 62% of the patients died or were severely disabled. In contrast, and similar to our experience is the finding of Bruce and associates (1978) who found that 90% of 56 children suffering from coma for more than six hours had a good recovery or suffered moderate disabilities. This difference in outcome following head injuries in children and adults, also noted in the study of Narayan and colleagues (1981), may reflect differences in the pathophysiology of head injury in these two groups, and also explain the relative infrequency of SEP abnormalities in this study.

PATIENT	AGE	CLINICAL STATUS	CT SCAN	SEP DATA (ms)	R	L	COMMENT/GOS
1 PJ	7 yr	Decerebrate, GCS=4, post craniotomy. Paralysed & ventilated, barbiturate coma	R Intracerebral hematoma R Extradural hematoma Compound craniofacial fractures	Cv2	8.0	8.0	Passenger in car. MD (Resolving L hemiparesis, R oculomotor dysfunction)
				Cx N	19.0	14.0	
				P	-	21.6	
				CSCT	?	6.0	
2 AJ	9 yr	Post craniotomy, GCS=3, Paralysed & ventilated Barbiturate coma	Compound comminuted R frontal craniofacial injury R extradural hematoma	Cv2	11.4	9.0	Passenger in car. MD (Blind in R eye, mild L hemiparesis)
				Cx N	17.4	14.8	
				P	26/25	20.8	
				CSCT	6.0	5.8	
3 DB	9yr	Paralysed & ventilated GCS=4, ICP monitoring normal	Bilateral punctate capsular & thalamic hemorrhages	Cv2	11.4	7.9	Cyclist hit by car. Vegetative (Decerebrate, tube fed at 6 mth post trauma)
				Cx	16.2	-	
					30.5	47	
				CSCT	5.8	?	
4 LP	13 yr	Paralysed & ventilated ICP monitoring normal GCS=5	Slit ventricles, loss of basal CSF cisterns. Bilateral frontal contusions	Cv2	7.8	10.8	Pedestrian hit by car. Normal (Full recovery over 3 week period).
				Cx N	14.0	17.6	
				P	19.8	24.4	
				CSCT	6.2	6.8	
5 WF	7 yr	Paralysed & ventilated ICP monitoring normal GCS=5	Slit ventricles, loss of basal CSF cisterns, small L frontal contusion	Cv2	11.4	11.4	Passenger in car. Normal (No change in CSCT with recovery)
				Cx N	17.5	17.6	
				P	26.8	26.0	
				CSCT	6.1	6.2 (a)	
				CSCT	6.5	6.5 (b)	

TABLE 5.1. CLINICAL, RADIOLOGICAL AND SEP DATA FOR PATIENTS STUDIED FOLLOWING MAJOR HEAD INJURIES. THE GCS CORRESPONDS TO CLINICAL STATUS AT THE TIME OF INITIAL SEP STUDY. THE TIMES GIVEN FOR THE CORTICAL (Cx) SEP CORRESPOND TO THE N1 (N) AND P1 (P) COMPONENTS. SERIAL CSCT ARE INDICATED BY BRACKETS.
Abbreviation MD = Moderate Diasability

PATIENT AGE	CLINICAL STATUS	CT SCAN	SEP DATA (ms)	R	L		COMMENT/GOS
6 LR 12 yr	Paralysed & ventilated Sustained ICP 50 mmHg despite barbiturate coma for two weeks	Slit ventricles, loss of basal CSF cisterns	Cv2	9.8	9.8		Passenger in car. Vegetative (Akinectic mute at 12 mths) Cortical SEPs abnormal and reproducible only several months after injury
			Cx	-	-		
			CSCT	?	?	(a)	
			CSCT	?	6.5	(b)	
7 DH 4 yr	Paralysed & ventilated post craniotomy. GCS=3 Compound leg fractures	R Frontal hemorrhagic contusions, compound skull fracture (depressed)	Cv2	10.2	10.2		Run over by tractor. Normal (Full recovery over 6 weeks)
			Cx N	16.8	16.8		
			P	21.6	21.6		
			CSCT	6.6	6.6	(a)	
			CSCT	7.8	7.0	(b)	
			CSCT	7.8	7.6		
8 LH 9 yr	Drowsy with no focal neurological deficit GCS=11	Normal	Cv2	10.8	10.7		Thrown off horse. Normal (Full recovery over two days after injury)
			Cx N	17.6	17.5		
			P	24.4	24.4		
			CSCT	6.8	6.8		
9 BM 7 yrs	Paralysed & ventilated Multitrauma. ICP monitoring 25 mm Hg. Initial GCS=4	Slit ventricles, loss of basal CSF cisterns	Cv2	7.2	7.3		Pedestrian hit by car. Normal (Full recovery over 6 week period)
			Cx N	13.6	13.5		
			P	22.6	22.0		
			CSCT	6.4	6.2	(a)	
			CSCT	7.0	-	(b)	
			CSCT	6.6	-	(c)	
			CSCT	-	6.8	(d)	
			CSCT	6.7	-	(e)	
			CSCT	-	7.4	(f)	
10 LB 4 yr	Drowsy, no focal neurological deficit GCS=13	Normal brain, fractured frontal bone.	Cv2	8.9	8.9		Fall off wall (5 m). Normal (Full recovery over 3 day period).
			Cx N	15.6	16.0		
			P	24.8	24.0		
			CSCT	6.7	7.1		

TABLE 5.1 (Cont)

PATIENT	AGE	CLINICAL STATUS	CT SCAN	SEP DATA (ms)	R	L		COMMENT/GOS
11 AT	13 yr	Post craniotomy for fracture. Nil focal deficit. GCS=13	Depressed frontal fracture	Cv2	11.8	11.7		Sports injury. Normal (Full recovery over 2 day period).
				Cx N	18,0	18.0		
				P	20.8	21.0		
				CSCT	6.2	6.3		
12 SD	11 yr	Drowsy, dysphasic GCS=11. Mild right Hemiparesis	L Parietal contusion	Cv2	12.1	12.1		Cyclist hit by car. MD (Mild R hypertonia and hyper-reflexia at 12 months)
				Cx N	18.0	18.4		
				P	21.6	22.0		
				CSCT	6.1	6.3	(a)	
				CSCT	5.6	6.4	(b)	
				CSCT	-	6.6	(c)	
13 GS	10 yr	Post craniotomy for compound depressed fracture GCS=13. Nil focal deficit	Depressed frontal fracture	Cv2	10.3	10.3		Pedestrian hit by car. Normal (Full recovery within 72 hrs of trauma)
				Cx N	16.8	16.8		
				P	19.8	19.4		
				CSCT	6.5	6.5	(a)	
				CSCT	6.3	6.3	(b)	
14 NH	3 yr	Sedated & ventilated ICP monitoring normal GCS=7	Normal	Cv2	6.4	6.4		Passenger in car. Normal (Full recovery over one week).
				Cx N	12.5	12.4		
				P	19.2	18.5		
				CSCT	6.1	6.0	(a)	
				CSCT	6.0	6.0	(b)	

TABLE 5.1 (Cont)

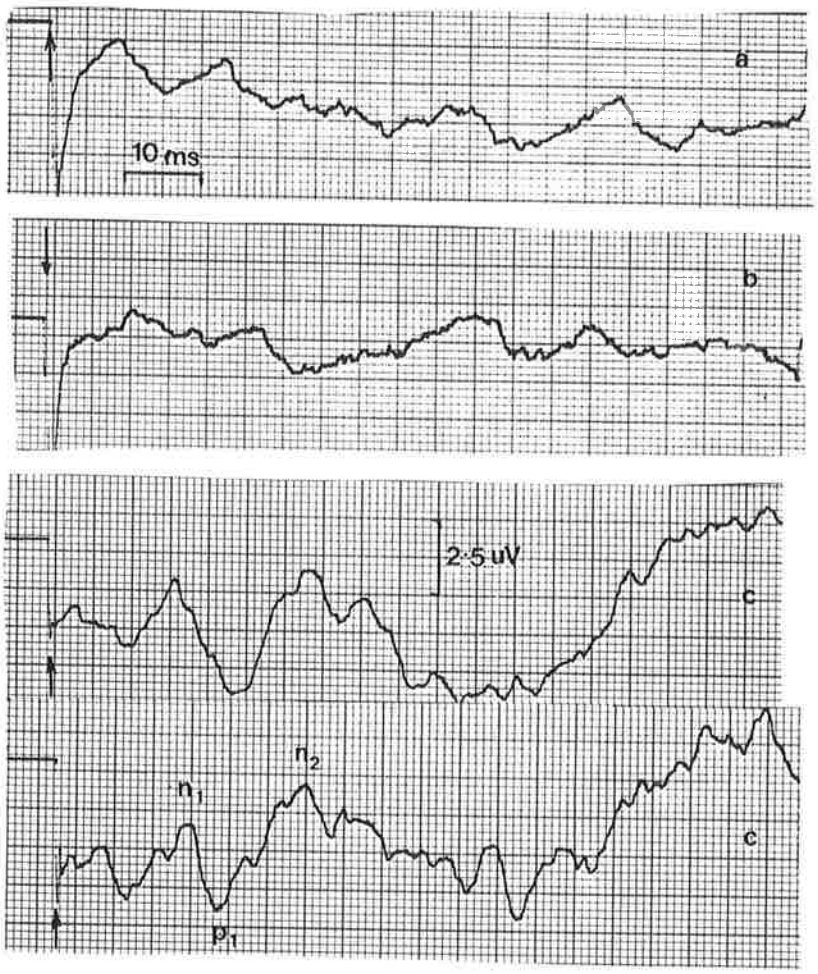


Fig 5.1 Sequential cortical (C3:Fpz) SEPs from trauma case 6. This 12 year old girl suffered a severe closed head injury. Persistant uncontrolled intracranial hypertension (>50 mmHg) marked the first 2 weeks of admission. The patient survived in a vegetative/ akinectic mute state. Trace a (21.6.83) shows no recognisable waveform, ICP = 40 mmHg, GCS = 4. Trace b (5.7.83) reveals similar findings although GCS was 9. Traces c (18.8.83) shows remarkable recovery of waveform despite a clinical state of akinectic mutism.

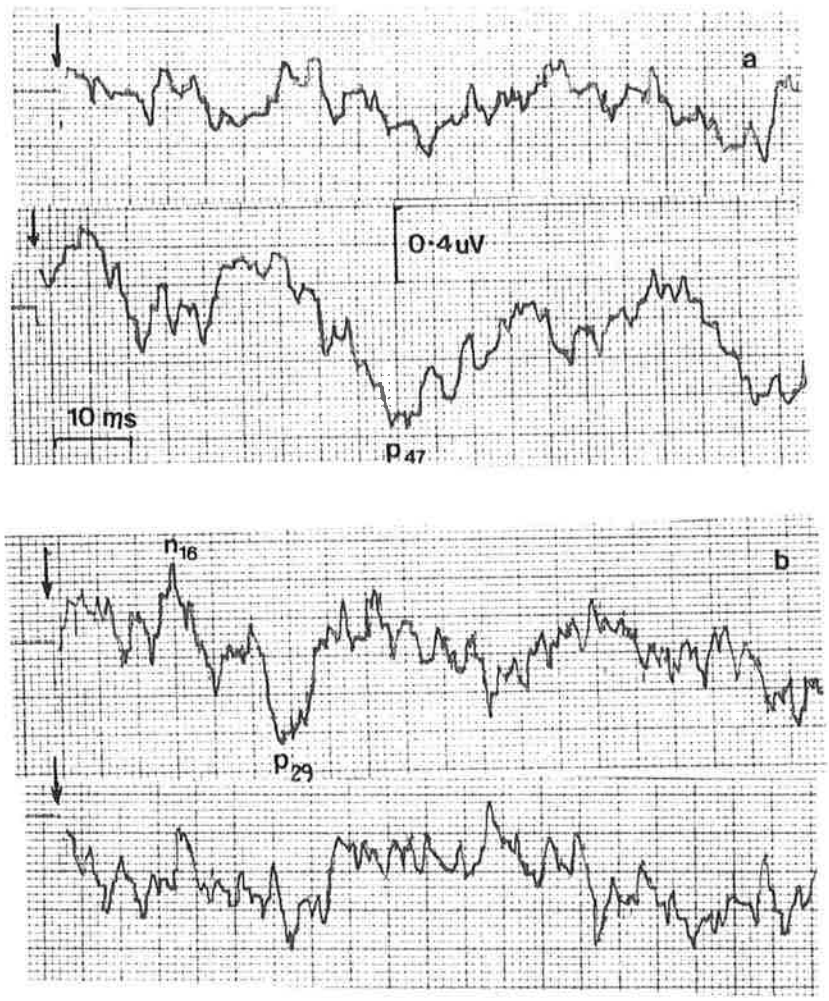


Fig 5.2 Initial cortical SEP recordings in trauma case 3. This patient suffered a closed head injury complicated by hypoxia at the scene of accident. His CT scan showed bilateral thalamic and capsular hemorrhages with the left side being more severely damaged (Fig 5.2b). ICP monitoring was normal. Recordings from both hemispheres are grossly abnormal. On the left (Traces a) there is only a reproducible P47 component, whilst on the right (Traces b) there are N16 and P29 components. This patient survived in a vegetative state.

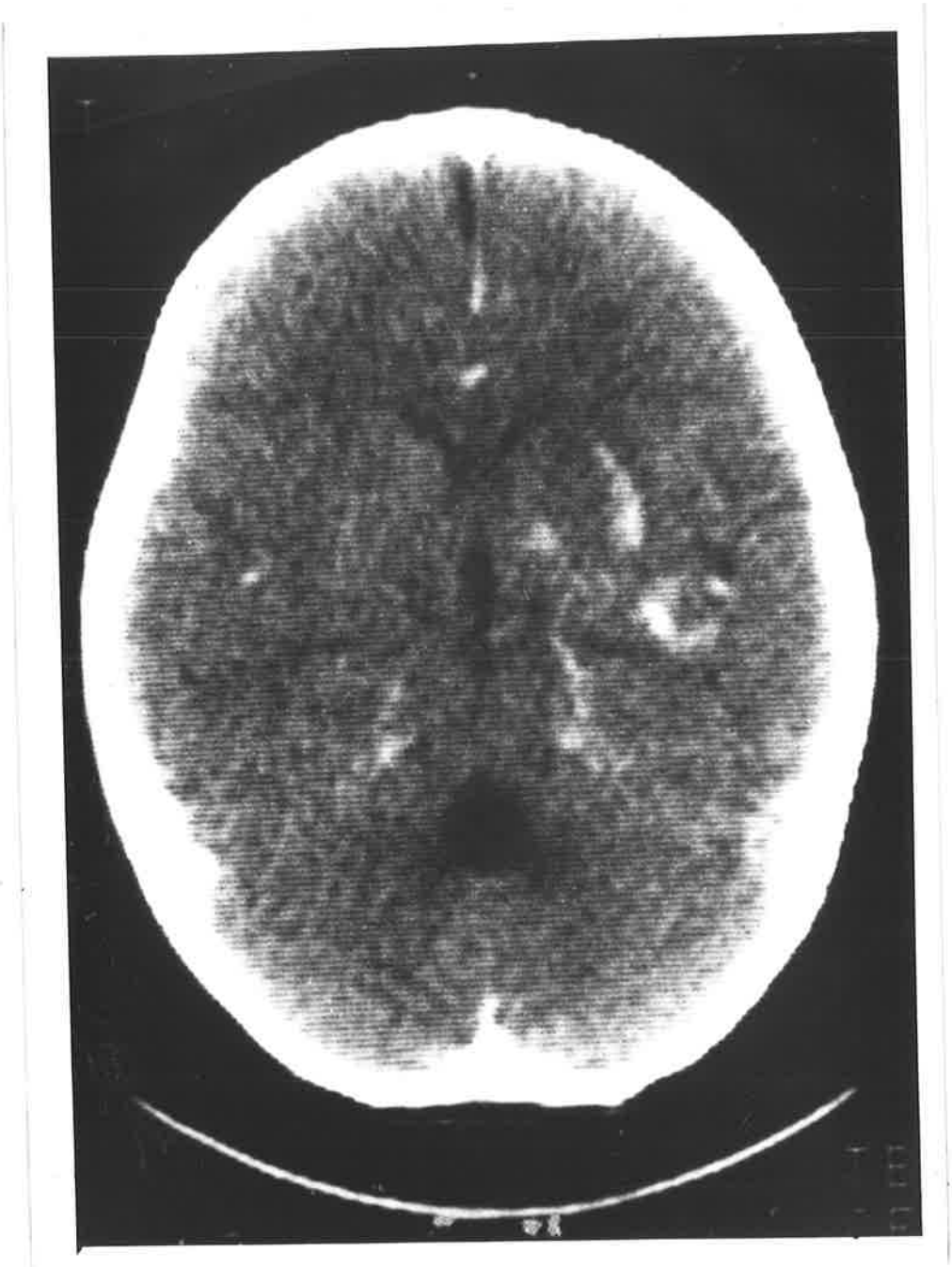


Fig 5.2b Unenhanced axial CT scan performed 16 hours after head injury in a 9 year old boy who was knocked off his bicycle by a motor car (case 3). He suffered a major head injury that was complicated by apnoea and cyanosis. The major features of the scan are bilateral, but predominantly left sided, thalamo-capsular hemorrhages.

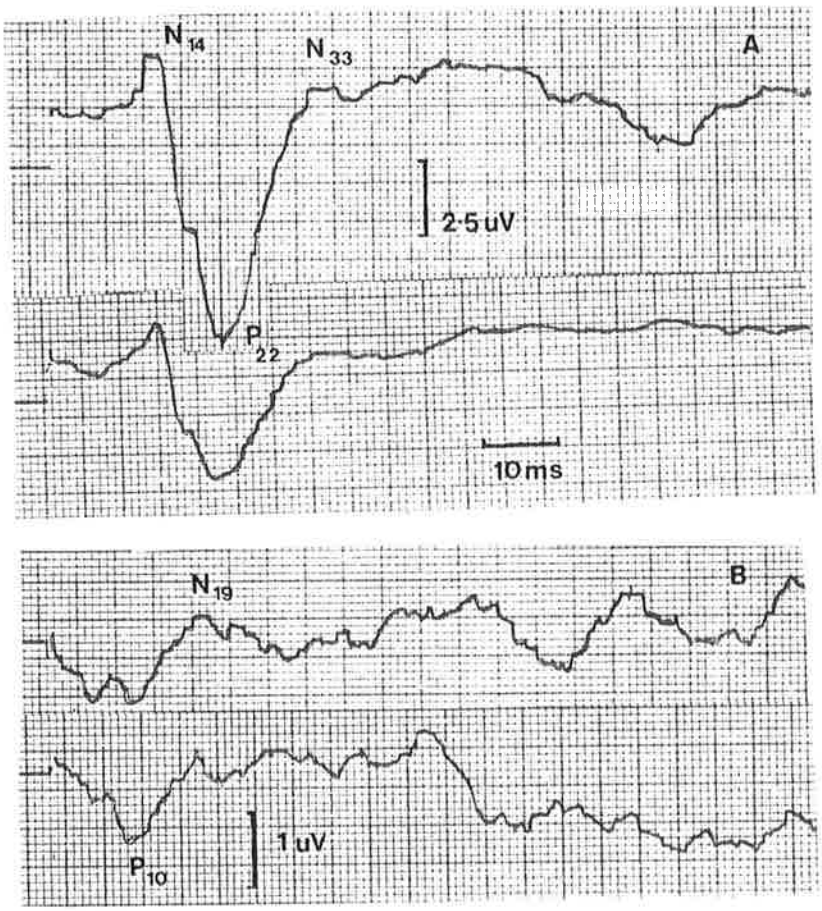


Fig 5.3. Initial cortical SEP study from case 1. This patient had a large right parietal intracerebral hematoma and profound ipsilateral cerebral swelling. The left sided cortical response to stimulation of the median nerve in the cubital fossa shows a normal N14-P22-N33 complex (Traces A). The right cortical response (Traces B) to median nerve stimulation at the wrist shows only a N19 peak with abscence of later components. The P10 wave is probably a far field subcortical response.

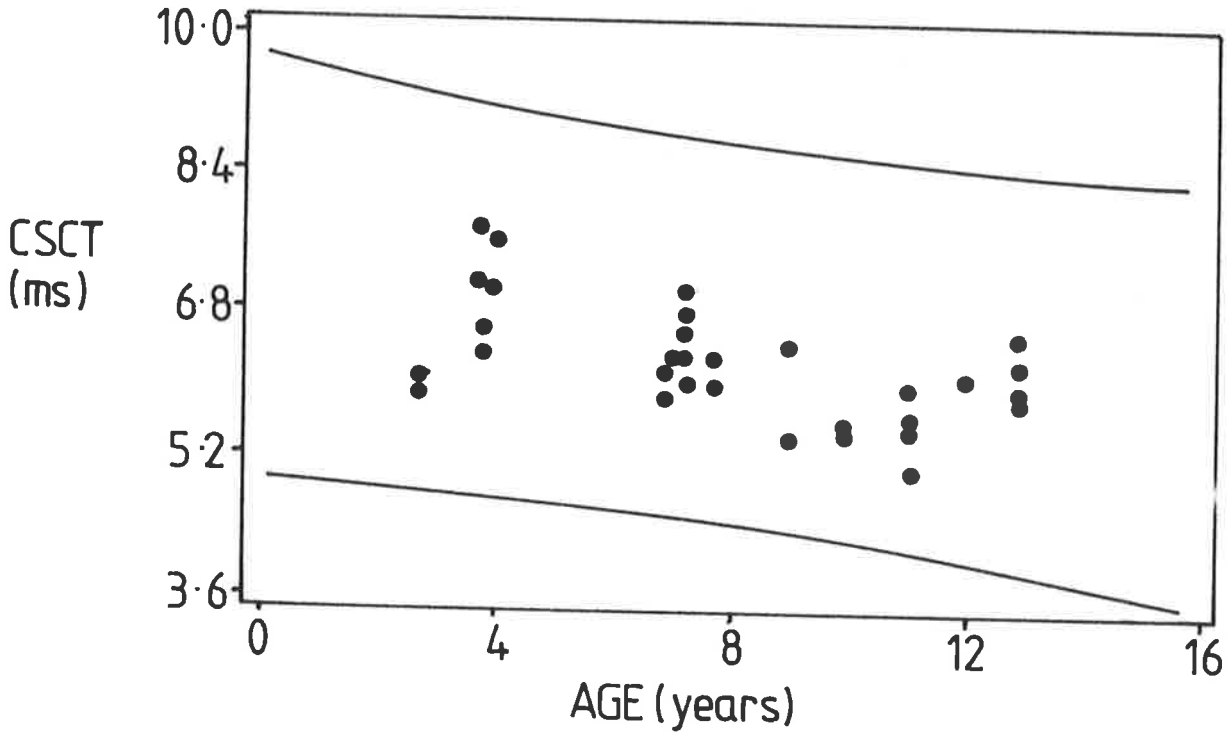


Fig 5.4 Scatter diagram showing the relationship between CSCT and age in patients suffering major head injury. The parallel lines mark the 95% limits from normality. All except one patient had a normal CSCT on at least one side on initial study. The CSCT was not related to the Glasgow coma score at the time of recording.

SECTION 6

THE EFFECT OF CEREBRAL SPACE OCCUPYING LESIONS ON SOMATOSENSORY EVOKED POTENTIALS IN CHILDREN

INTRODUCTION

Much of the current knowledge concerning the anatomical generators of subcortical and cortical SEPs has been derived by comparing normative SEP data with that from patients with neurological disorders. These SEP findings have then been correlated with clinical, radiological and pathological findings and particuler generators attributed to waveform components (Halliday and Wakefield, 1963; Williamson et al, 1970; Okazaki et al ,1971; Noel and Desmedt, 1975; Shibasaki et al, 1977; Yamada et al, 1977; Nakanishi et al, 1978; Obeso et al, 1980; Anziska and Cracco, 1980; Jones and Halliday, 1982; Mauguiere et al, 1982; Ebner et al, 1982; Stohr et al, 1983; Yamada et al, 1983).Some of these studies have employed cephalic and non-cephalic references, and simultaneous multiple site recordings of both long and short latency components that have further contributed to the understanding of the neuroelectric generators.

Some limitations are inherent when comparing SEP data from a diseased cerebral hemisphere to that obtained from the contralateral hemisphere or matched normal data. These limitations arise because of the well documented phenomena of trans-synaptic neuronal degeneration (Chiappa and Ropper, 1982; Duchen, 1984), callosally mediated facilitation or inhibition of

neuronal pools (Kempinsky, 1958), and the diffuse effects of a focal cerebral lesion on cortical blood flow, glucose metabolism and oxygen utilisation (Beaney et al, 1985). In children these problems may be compounded by the plasticity of the developing brain. Despite these problems several patterns of SEP abnormality have been documented in adults with cerebral space occupying lesions (Williamson et al, 1970; Mauguiere et al, 1982; Stohr et al, 1983), however there have been no comparable studies in children.

In this study bilateral cortical SEP studies have been performed in seven children with cerebral space occupying lesions of varying etiology. The patterns of abnormality have been correlated with clinical, pathological and radiological findings, and compared to similar studies in adults. The clinical utility of these investigations in children and their contribution to the origins of the cerebral generators of various waveform components is also assessed.

CLINICAL MATERIAL

Seven patients with a spectrum of intracerebral space occupying lesions were studied. The location of the lesions was parietal lobe (2), thalamic/internal capsule (3), frontal lobe (1) and posterior temporal (1). The pathogenesis of the lesions was neoplastic (3), vascular (3) and traumatic (1). Four patients were male and three female. The age, diagnosis, clinical features and radiological findings are summarised in Table 6.1.

All children were studied within several days of hospital admission, although the duration of the disease process was quite variable (Table 6.2). In six of the seven cases the recordings were performed prior to neurosurgical management of the intracranial condition. Open biopsy in all these cases confirmed or classified exact diagnosis.

Consciousness was impaired in 2 patients (Cases 1,5) both of whom had clinical and radiological evidence of intracranial hypertension. These two patients required intubation, hyperventilation and intermittent 20% Mannitol solution. The other five patients were alert and speaking although two (Cases 2,6) had clinical and radiological evidence of intracranial hypertension. Several patients were taking dexamethasone (Cases 4,6), carbemazepine (Case 4) and phenytoin (Case 1) at the time of study.

METHODS

SEP ACQUISITION AND ANALYSIS

The equipment used and methods of stimulation, recording and averaging the SEP data were as previously described in Section 3.Cortical SEPs following median (six patients) and tibial nerve (two patients) stimulation were studied using C3/C4:Fpz and Cz':Fpz montages respectively. These SEP waveforms were compared to age matched controls, and also to data obtained from the contralateral hemisphere. The components of the SEP studied included the latencies of the primary cortical response (N1 arm,

P1 leg) and the N1-P1 interpeak latency of the SEP following median nerve stimulation. The number of waveform components in the first 100 (median nerve) or 200 (tibial nerve) ms post stimulation was also assessed. The amplitude of the SEP was measured from the maximal N peak to P trough in the first 100 ms post stimulation.

CLINICAL AND RADIOLOGICAL STUDIES

The location of the intracerebral lesion was documented from studies of CT scans performed on a GE 8800 scanner. In three patients the anatomical status of the cerebral vasculature was assessed from angiographic studies. All patients were clinically examined prior to the SEP study.

RESULTS

SEP DATA

The cortical SEPs recorded from the scalp over the hemisphere contralateral to the side of the lesion were normal in six patients. Criteria for normality included normal N1 latency and N1-P1 interpeak latency for SEPs following median nerve stimulation, and normal P1 latency for SEPs following tibial nerve stimulation, together with a total of five or more waveform components in the first 100 or 200 ms post stimulus. One patient (Case 5) had abnormal SEPs contralateral to the lesion.

Ipsilateral to the lesion the cortical SEPs were abnormal in six

cases (Fig 6.1, 6.2, 6.3). The abnormalities included abscence or distortion of the primary cortical complex (N1-P1) in three patients (Cases 1,2,5) and loss of intermediate and long latency components with normal short latency components in three patients (Cases 4,6,7). There was also attenuation of the SEP amplitude on the side of the lesion in each of these six cases. In one patient (Case 3) SEPs to both tibial and median nerve stimulation were symmetrical. The peak latencies, number of waveform components and amplitude of the SEPs from both hemispheres are summarised in Table 6.2.

CLINICOPATHOLOGICAL AND ELECTROPHYSIOLOGICAL CORRELATIONS

The two patients (Cases 2,5) with abscence of a primary cortical response on the side of the lesion had large intracranial hematomas (posterior parasaggital and frontal, respectively) with clinical and angiographic evidence of more widespread cerebrovascular dysautoregulation (Fig 6.1). One of these patients (Case 5), and another with an abnormal primary cortical complex (Case 1), had clinical evidence of ipsilateral uncal herniation and radiological evidence of diffuse cerebral swelling. None of these three patients had mass lesions directly involving the primary thalamo-cortical projections.

The two patients (Cases 6,7) with loss of SEP components occurring 30-40 ms after an initially normal primary cortical response had lesions directly involving the posterior thalamo-capsular region. One of these patients had clinical evidence of raised intracranial pressure and peritumoral edema that extended

centrally into the posterior thalamic region. Another patient (Case 4) with ipsilateral loss of a long latency (N75) component had a posterior temporal oligdendroglioma that was not causing any mass effect nor involving primary thalamocortical projections (Fig 6.2). The patient (Case 3) with normal SEPs to both upper and lower limb stimulation had a low grade subependymal astrocytoma of the anterior thalamic region that was not causing any mass effect.

Impairment of somatosensory function and the presence of other neurological deficits correlated well with SEP abnormalities. The three patients (Cases 1,2,5) with the largest intracerebral lesions all had major sensorimotor dysfunction and variable impairment of their conscious state with significant abnormalities of the SEP. The two patients (Case 3,4) who were clinical normal had minimal asymmetry on their SEPs. One patient with a homonymous hemianopia, but no apparent somatosensory deficit (Case 6), and another (Case 7) with profound hemiplegia and sensory loss both had normal primary cortical responses but loss of later components.

DISCUSSION

The advent of CT scanning has enabled a more precise correlation between structural abnormalities of the brain and attendant changes in cortical SEPs (Shibasaki et al, 1977; Yamada et al, 1977; Nakanishi et al, 1978; Ebner et al, 1982; Mauguiere et al, 1982; Yamada et al, 1983). Several patterns of change in cortical SEPs have now been reported with supratentorial lesions

(Williamson et al, 1970; Mauguiere et al, 1982; Stohr et al, 1983). These abnormalities may be summarised as Type I (bilaterally normal), Type II (Normal primary cortical response with loss of longer latency components) and Type III (No reproducible SEP on the side of the lesion). Attenuation of the amplitude of the SEP ipsilateral to the lesion has also been another common finding accompanying these changes (Noel and Desmedt, 1975; Shibasaki et al, 1977; Jones and Halliday, 1982). Amplitude attenuation on the side of the lesion has also been related directly to the proximity of the lesion to the primary somatosensory pathways (Obeso et al, 1980).

Several workers have correlated patterns of change in the cortical SEP with the anatomical sites of lesions and postulated the generators of particular waveform components. Thus the N18 component has been found to be effected in disorders of the thalamus or thalamocortical projections and the P25 in disorders of the primary somatosensory cortex (Domino et al, 1965; Anziska and Cracco, 1980; Mauguiere et al, 1982; Yamada et al, 1983). The components following the primary cortical response (the N18-P25 complex) are felt to represent activity in the parietal association cortex (Okazaki et al, 1971; Yamada et al, 1977; Obeso et al, 1980; Ebner et al, 1982; Stohr et al, 1983; Yamada et al, 1983), the reticulocortical pathways (Yamada et al, 1977; Obeso et al, 1980) and other nonspecific cortical projection pathways (Williamson et al, 1970; Okazaki et al, 1977).

The results from this study complement many of these findings but also demonstrate that a simple correlation between SEP changes

and the location of a lesion neglects some important pathophysiological phenomena occurring around the lesion. Large intracerebral hemorrhages complicated by severe cerebral swelling and uncal herniation were associated with almost complete loss of the primary cortical response in two patients despite abscence of a structural lesion in the midbrain or thalamus. Focal edema and vascular dysautoregulation from a contiguous arteriovenous malformation may also explain the absence of the primary cortical response in another patient with no CT evidence of a thalamic lesion. Conversely, as occurred in one patient in this study, previous series have described the apparently incongruous finding of normal cortical SEPs in patients with lesions of the thalamus, and subcortical areas of the parietal lobe (Nakanishi et al, 1978; Mauguiere et al, 1982; Yamada et al, 1983).

Secondary effects from the mass lesion can modify the cortical SEP by physiological disconnection, desynchronization and disorganization of the thalamocortical projection and cortico-cortical association fibres and neuronal pools. Changes in local cerebral blood flow, white matter tissue water content and duration of the lesion as well as the precise location of the lesion all influence the exact pattern of waveform distortion or attenuation (Larson et al, 1966; Branston et al, 1974; Ebner et al,1982;Stohr et al,1983; Tanaka et al,1983; Yamada et al,1983). The clinical utility of cortical SEP studies in patients with intracerebral space occupying lesions would appear limited since in all cases with asymmetrical short latency cortical SEPs there was an obvious sensorimotor clinical deficit. In these patients an accurate radiological diagnosis can readily be made with CT

scanning. Other studies have also correlated major clinical deficits with disorganization of the short latency SEP components (Okazaki et al, 1971; Mauguiere et al, 1982; Yamada et al, 1983), and minor clinical deficits with alterations in the longer latency components (Okazaki et al, 1971; Shibasaki et al, 1977; Ebner et al, 1982; Mauguiere et al, 1982).

The location, etiology and duration of the lesion are also important when considering clinico-electrophysiological correlations. Following cerebral insults due to cerebrovascular disease and head injury the cortical SEP may recover after several weeks although the clinical lesion may remain profound (Larson et al, 1966; Hume and Cant, 1981; Lutschg et al, 1983; See Section 5). These parameters also would seem to influence the correlation between abnormalities of the SEP and impairment of both joint position sense and stereognosis (Williamson et al, 1970; Anziska and Cracco, 1980; Stohr et al, 1983). Ebner and associates (1982) noted minimal clinical and electrophysiological dysfunction with slow growing tumors. The unusual radiological features of the oligodendroglioma in Case 4 suggest that it had been present for several years, this patient also had no clinical deficit and minimal asymmetry of cortical SEPs despite the size of the lesion.

PATIENT	AGE	DIAGNOSIS/CT SCAN	CLINICAL STATUS
1	7 yr	Large R frontoparietal intracerebral hematoma (post traumatic).	Unconscious with evidence of ICP. Later Left hemiplegia, homonymous hemianopia impaired JPS and stereognosis left.
2	10 yr	Right posterior parietal intracerebral hematoma from ruptured arteriovenous malformation	Left foot drop and dysesthesia lower left leg with impaired JPS at the hallux
3	10 yr	Left thalamic subependymal astrocytoma	Neurologically normal. Shunted hydrocephalus
4	7 yr	Left posterior temporal oligodendroglioma	Neurologically normal. Complex partial siezures 3/12
5	12 mths	Massive spontaneous right frontal intracerebral hemorrhage	Left hemiplegia, moribund with marked intracranial hypertension.
6	3 yr	Left intraventricular and posterior thalamic glioblastoma multiforme	Right homonymous hemianopia, behavioural change. Gait ataxia
7	8 yr	Left striatal/capsular intracerebral hemorrhage. ? thrombosed arteriovenous malformation	Right hemiplegia with sensory inattention, impaired JPS but preserved pain perception

TABLE 6.1. CLINICAL AND PATHOLOGICAL DATA FOR THE SEVEN PATIENTS WITH INTRACRANIAL SPACE OCCUPYING LESIONS

PATIENT		SEP N1 P1 N2	WAVEFORM COMPONENTS	AMPLITUDE (uV)	DISEASE DURATION	COMMENT
1	R	N19	2	0.5	24 hr	Abnormal primary cortical response
	L	N14 P18 N35	5	4.7		
2	R	Nil	0	-	6 days	No waveform on side of lesion
	L	N22 P26 N35	5	1.7		
3	R	N16 P20 N28	6	2.5	6 wks	Essentially symmetrical short latency SEP waveform
	L	N16 P19 N28	7	3.2		
4	R	N17 P25 N38	6	3.1	3 mths	Loss of N73 on side of lesion
	L	N18 P24 N38	4	3.0		
5	R	Nil	0	-	? 2 wks	Abnormal SEP from left hemisphere, no waveform on right
	L	N17 P24	2	5.0		
6	R	N15 P22 N35	6	4.5	? 2 wks	No reproducible components after 30ms on side of lesion
	L	N16 P24	3	1.9		
7	R	N15 P20 N31	6	7.5	8 days	No reproducible components after 40ms on side of lesion
	L	N16 P23 N34	3	3.5		

TABLE 6.2. THE EFFECT OF INTRACEREBRAL SPACE OCCUPYING LESIONS ON THE AMPLITUDE AND SYMMETRY OF THE CORTICAL SEP IN CHILDREN

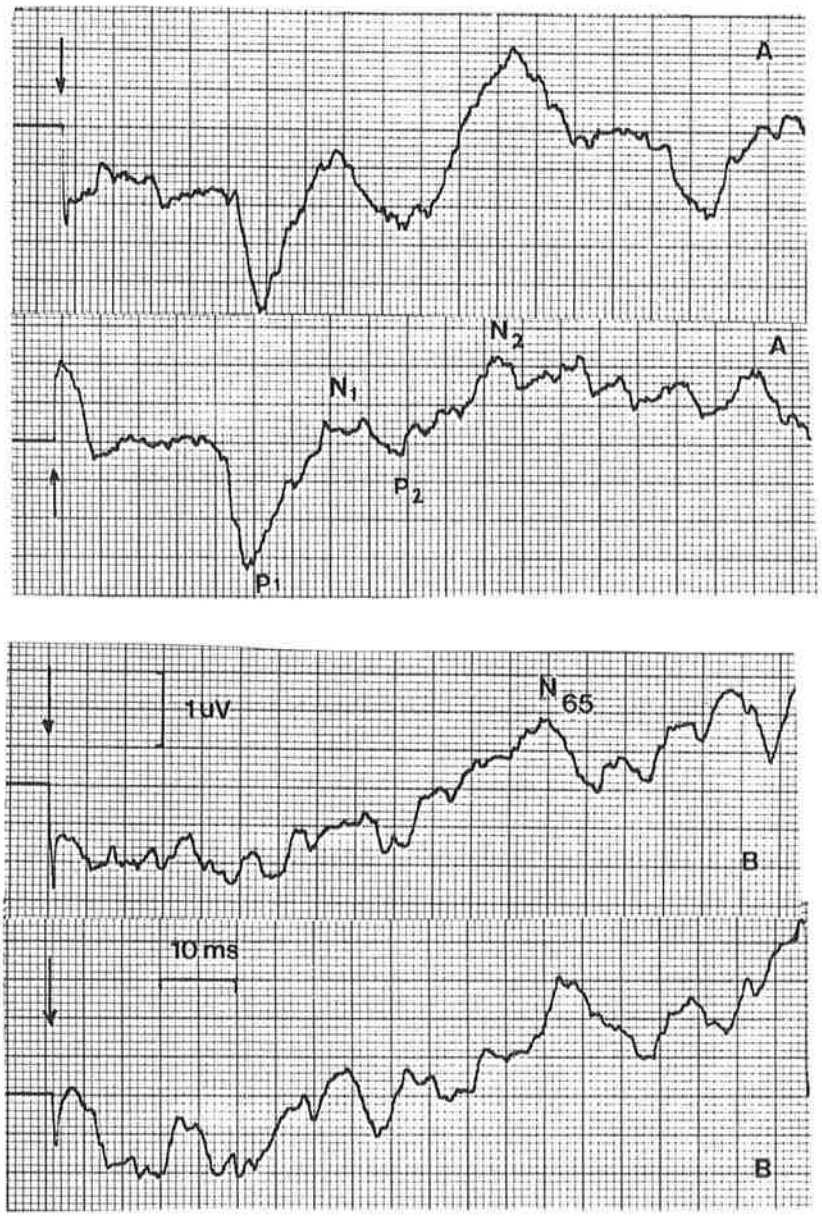


Fig 6.1 Cortical SEPs following tibial nerve stimulation from case 2. This 10 year girl, with a right posterior parasagittal intracerebral hematoma from a ruptured arteriovenous malformation (Fig 6.1B), had a left sided foot and knee paresis with dysesthesia and loss of JPS in the hallux. The right hemispheric response has no short latency components and there is only one peak (N65) in the first 100 ms post stimulus (Traces B). The left cortical response is multicomponent with a prominent P1 at 26 ms (Traces A).

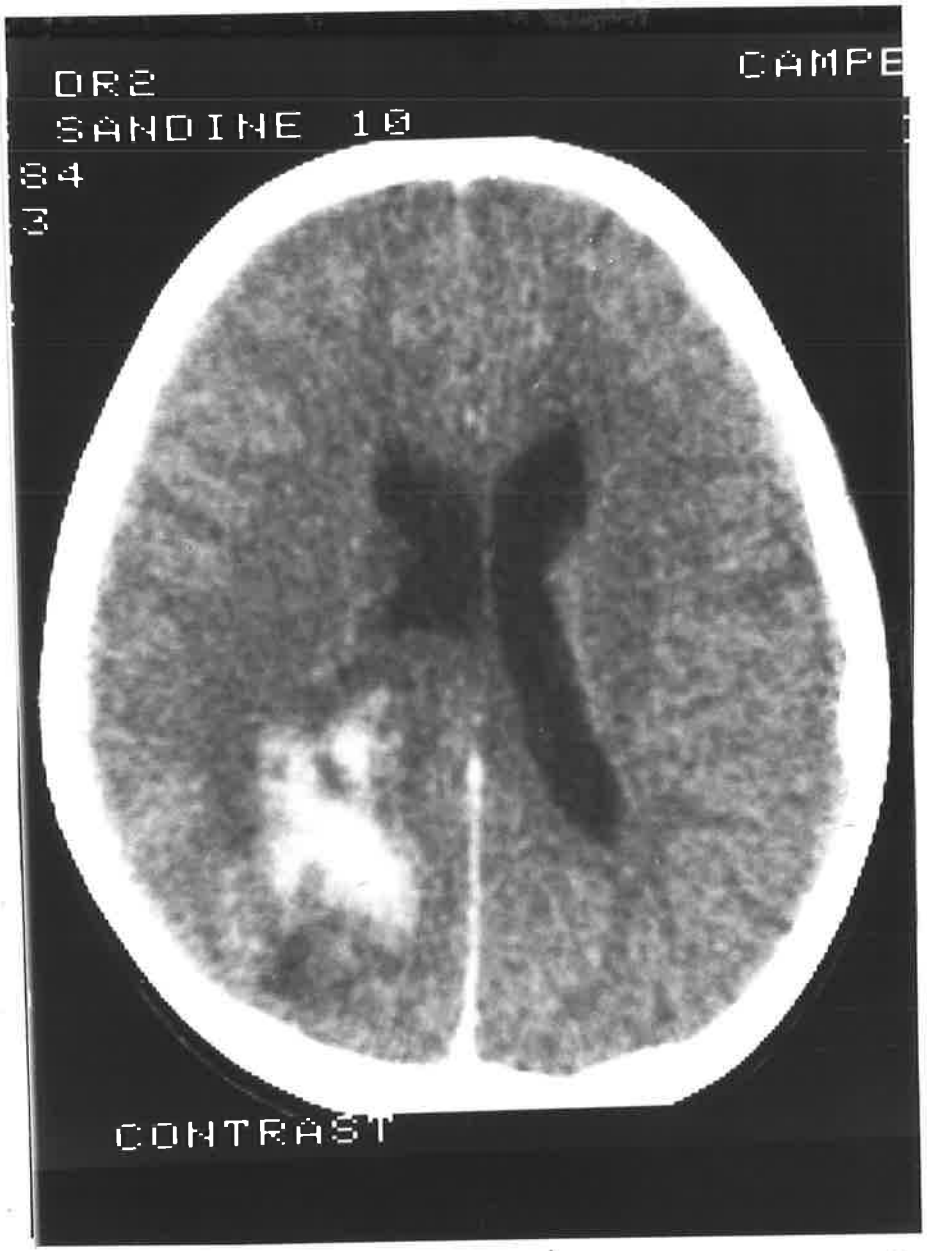


Fig 6.1b Axial CT scan of the brain of a 10 year old girl who presented with sudden onset of severe headache and left lower limb paresis (Case 2). Angiography confirmed that the right posterior parietal hematoma was due to a ruptured arteriovenous malformation.

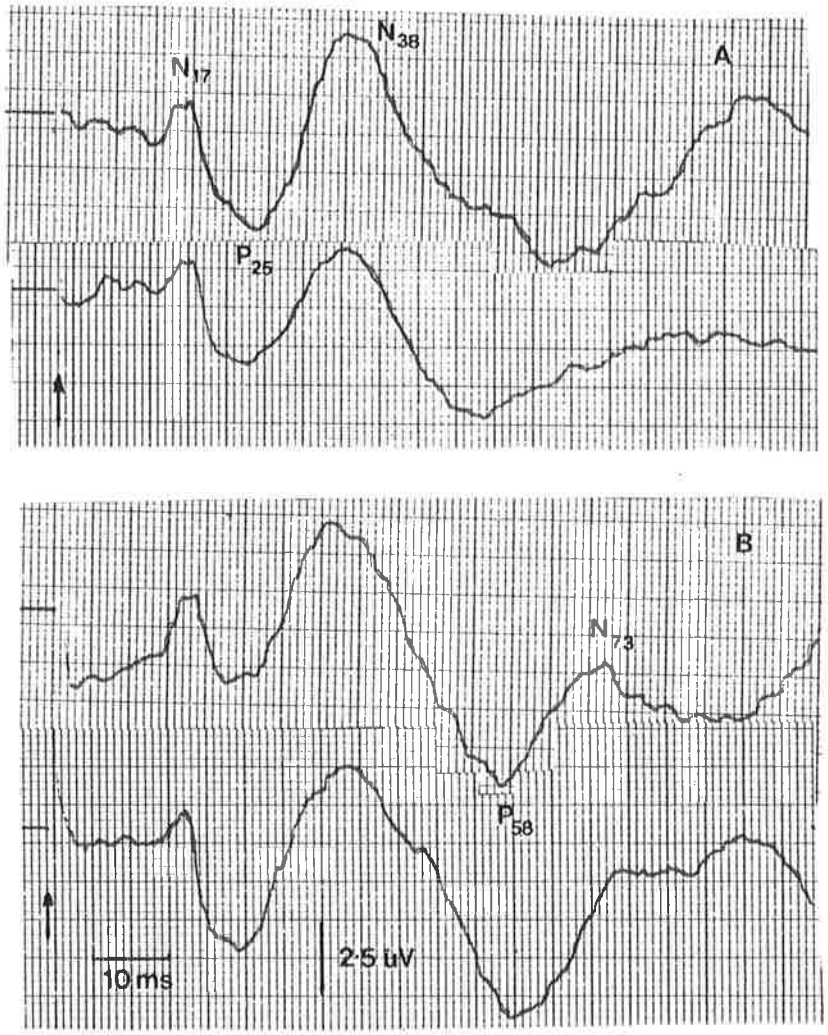


Fig 6.2 Cortical SEPs following median nerve stimulation from case 4. This seven year old boy presented with focal seizures. His CT scan showed a heavily calcified left posterior temporal lesion that had caused deformation of the skull (Fig 6.2b). Despite the presence of this oligodendroglioma the only difference between left (traces A) and right (Traces B) SEPs is ipsilateral loss of the N73 component.

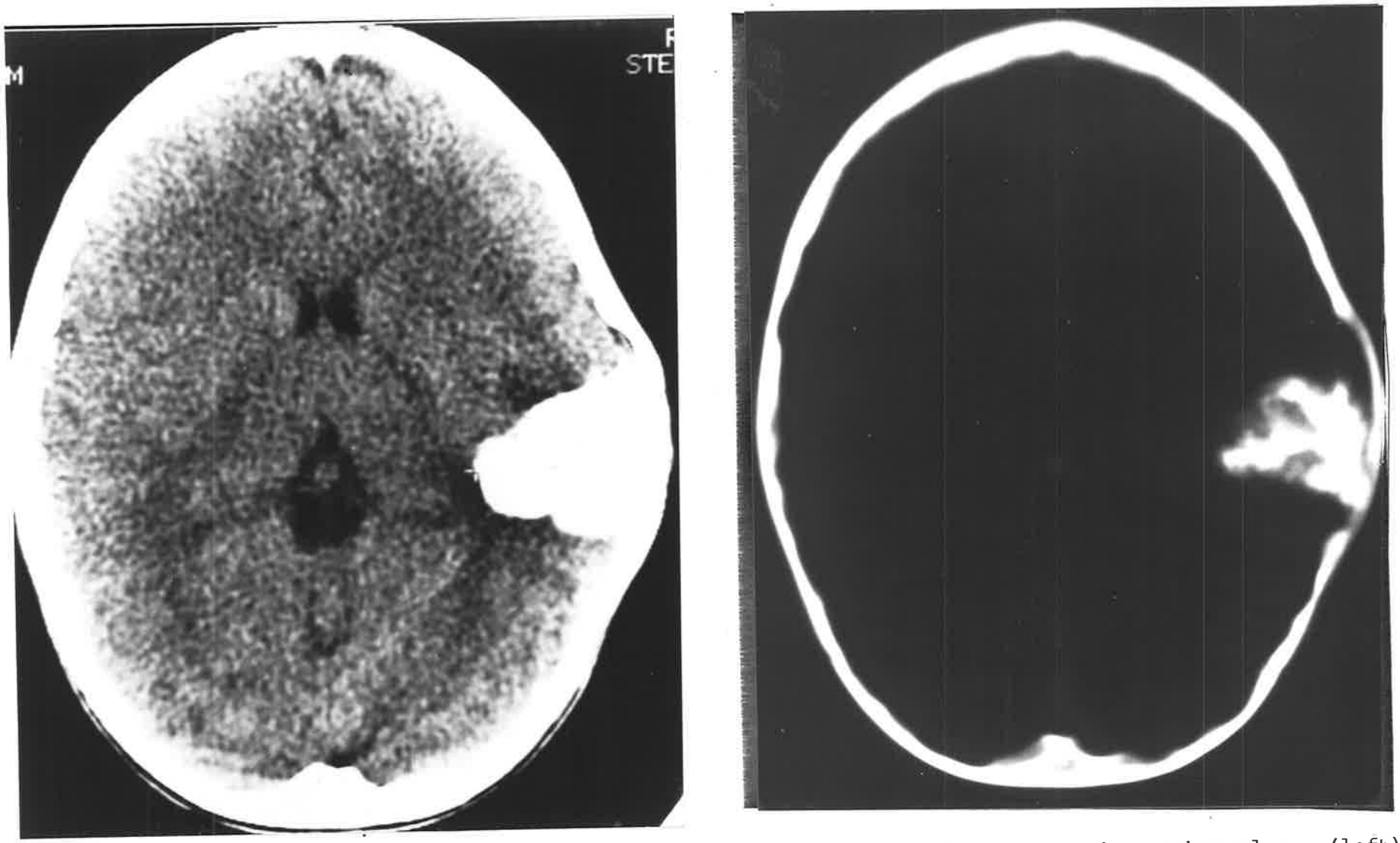


Fig 6.2b Axial CT scans of the brain of a 10 year old boy who presented with focal siezures. The unenhanced scan (left) shows a conical shaped , calcified lesion projecting into the posterior temporal lobe. There is minimal contiguous edema and no shift of the midline. Bone window settings revealed that the lesion was heavily calcified and had caused distortion of the overlying calvarium (Right). Histology revealed the lesion to be an oligodendroglioma.

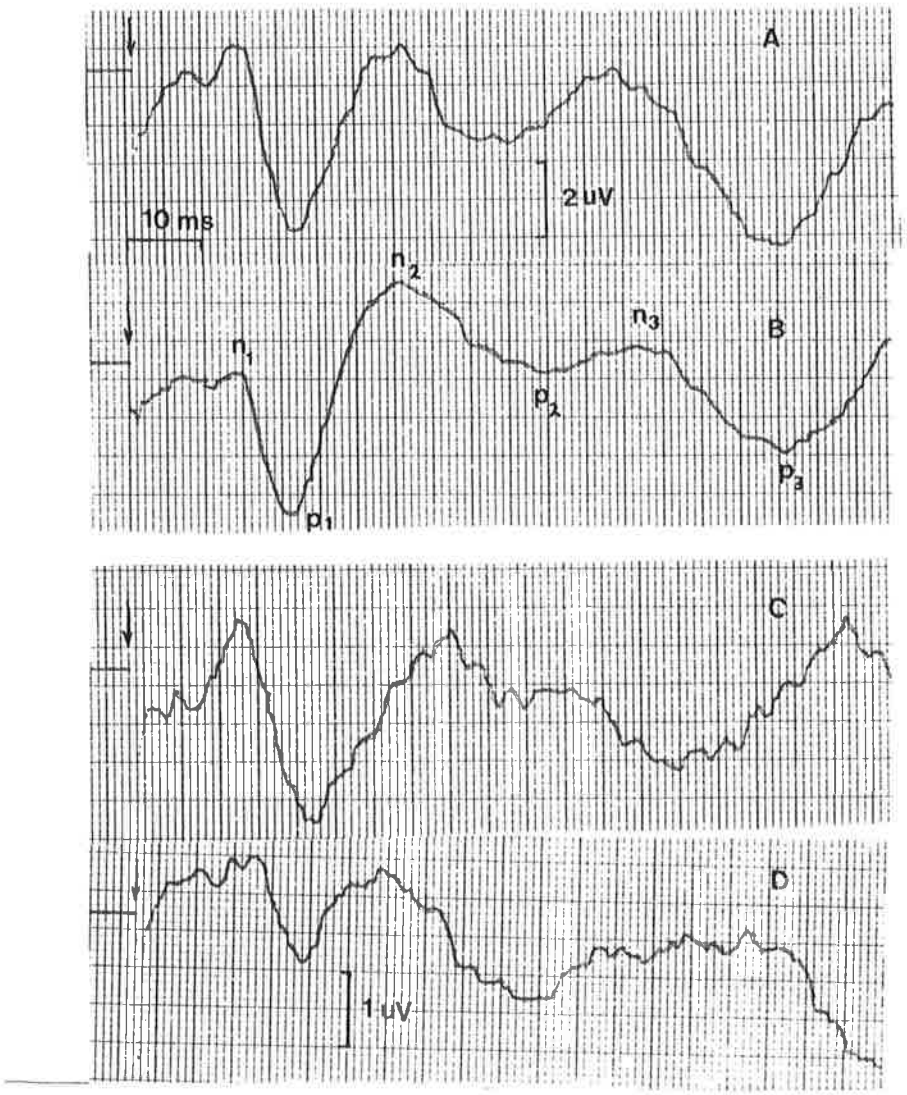


Fig 6.3 Cortical SEPs following median nerve stimulation from case 6. This three year girl had a left posterior thalamic and lateral intraventricular glioblastoma. The right cortical response shows six discrete waveform components (Traces A and B). The left sided cortical response shows an N1-P1-N2 complex but no reproducible components after 30 ms (Traces C and D). There is also amplitude reduction on the side of the lesion.

SECTION 7

EXPERIENCE WITH INTRAOPERATIVE RECORDING OF CORTICAL SOMATOSENSORY EVOKED POTENTIALS AS A METHOD OF SPINAL CORD MONITORING DURING SPINAL SURGERY

INTRODUCTION

Several large series describing the use of intraoperative cortical SEP recording to monitor spinal cord function during correction of kyphoscoliotic disorders have been reported (Nash et al, 1977; Engler et al, 1978; Uemato and Tolo, 1981; Maccabee et al, 1982; Worth et al, 1982; Nuwer and Dawson, 1984). In these reports, stability of various components of the cortical SEP from tibial or peroneal nerve stimulation was associated with preservation of neural function post operatively. Similarly, in two reports of cortical SEP monitoring during neurological surgery, stability of the SEP also correlated with a satisfactory post operative neurological status (McCallum and Bennett, 1975; Grundy, 1982).

Several other series reporting cortical SEP monitoring during both orthopaedic and neurological surgery have however described many technical and methodological problems (Allen et al, 1981; Uemato and Tolo, 1981; Raudzens, 1982; Jones et al, 1983). Large fluctuations in both amplitude and latencies of the SEP waveform components and "technically unsatisfactory" signal acquisition in between 20-59% of cases have suggested that the practical utility

of this mode of monitoring was questionable. Little consideration has been given however to the relationship between patients' neurological deficits, their causative pathology and intraoperative cortical SEP findings. It would appear that there is a fundamental difference in monitoring SEPs in orthopaedic and neurological cases. In general, many orthopaedic cases have an intact, albeit mechanically deformed spinal axis, whereas neurosurgical patients in general have a spinal medulla that is already compromised by congenital malformations, secondary compression or primary infiltration and destruction. "Technical problems" with intraoperative signal acquisition could therefore be expected more frequently in neurosurgical than orthopaedic patients.

The aim of this study was to assess the utility of cortical SEP monitoring as an adjunct to spinal neurological surgery. Particular consideration was given to:

(1) The effect of preoperative neurological deficits and their pathogenesis on the generation and intraoperative recording of cortical SEPs.

(2) The stability of the cortical SEP waveform during general anaesthesia and spinal neurosurgery, and factors that alter the amplitude and latency of the cortical SEP waveform.

(3) The particular contribution of cortical SEP compared to spinal SEP recording as a means of intraoperative assessment of spinal cord function.

CLINICAL MATERIAL

A series of 13 patients undergoing spinal surgery had cortical SEP recordings as a routine part of intraoperative physiological monitoring. The patients' spinal pathology included congenital vertebral and neural dysplasias, intra and extra medullary spinal cord tumours, and degenerative conditions with secondary spinal cord involvement. Patient diagnosis and clinical neurological deficits are summarised in Table 7.1. Diagnosis was confirmed by metrizamide myelography, surgery or biopsy in all patients.

METHODS

Anaesthesia for all procedures consisted of muscle relaxation with curare analogues (pancuronium or alloferin), endotracheal intubation and intermittent positive pressure ventilation with 66% nitrous oxide, 33% oxygen, supplemented by either intermittent halothane or fentanyl (0.003 mg/kg).

Platinum alloy subdermal needles or stick-on chlorided silver (Ag/AgCl) disc recording electrodes were placed on the scalp using standard and modified positions from the International 10/20 system (Jasper, 1958). The electrode for recording cortical SEPs from lower limb stimulation was positioned 2cm behind Cz and termed Cz'. Positions C3/C4 were used for recording cortical SEPs from median or ulnar nerve stimulation. All recordings were referenced to Fpz. An earth electrode was taped either to the buttock or the sternum.

SEPs were generated by unilateral electrical stimulation of the median, or in one case ulnar, nerve at the wrist or tibial and peroneal nerves in the popliteal fossa. Electrical shocks were delivered as square wave pulses of 0.1ms duration, 50-70V amplitude at a rate of 5-10Hz. This stimulation was applied percutaneously through saline soaked felt pads mounted in a saddle shaped plastic unit. The cathode was proximal to the anode and inter-electrode distance was 2cm. Satisfactory electrode position was confirmed by generation of a good muscle twitch after an initial dose of succinyl choline given for muscle relaxation during endotracheal intubation had worn off. The stimulating unit was then taped in position over the path of the nerves. In some cases, stimulating electrodes were positioned on both legs, thus allowing recording of SEPs to both left and right sided stimulation during surgery.

Signals were fed through a Medelec PA89 preamplifier connected to a Medelec MS91 signal averaging unit. The preamplifier had been taped onto the operating table close to the patient's head. The frequency bandpass was set at 20-2000Hz (-6dB roll off). Amplifier sensitivity was set at 20 or 50uV. Signals containing large amptitude interference were therefore automatically rejected. At least 256 events were averaged over epochs of 50-200ms depending on site of peripheral nerve stimulation.

A baseline cortical SEP was obtained prior to any surgical manipulation. This record was stored on one channel. Further SEPs were recorded throughout the surgical procedure. All traces were reproduced onto an oscilloscope screen where peak latencies

could be obtained using a cursor with digital readout. Major peaks were termed negative 1 (N1), positive 1 (P1), N2, etc, and their latencies documented. Direct superimposition facility of the two channels on the oscilloscope screen enabled the baseline recording to be compared with the on-going trace throughout the procedure. SEP recordings were reproduced onto heat sensitive paper, prior to erasure from memory. Peak to trough amplitudes were quantitated from these records.

In several cases SEPs were also recorded from a subdural or extradural spinal electrode (See section 8). Arterial blood pressure, end tidal carbon dioxide, pulse rate and manipulation of anaesthetic agents were documented with each cortical SEP recording.

RESULTS

The surgical procedures performed in the 13 cases are summarised on Table 7.1.Cortical SEPs were recorded intraoperatively in 8 of the 13 patients. In 7 of these cases the SEP waveform was reproducible throughout the procedure. None of these 7 patients sustained a neurological deficit as a result of the surgery. In 5 patients no cortical SEP could be recorded at any stage of the surgical procedure. There was transient attenuation of the N1-P1 component of the cortical SEP with cervical laminectomy in 2 cases of cervical spondlytic myelopathy (Fig 7.1). Neither of these patients sustained an iatrogenic neurological insult. In the one case in which the cortical SEP was grossly distorted following removal of an intramedullary astrocytoma, the patient

sustained a major bilateral neurological deficit (Fig 7.2).

The cortical SEPs recorded intraoperatively had amplitudes and short latency components similar to those recorded without anaesthetic (Fig 7.3). The most stable component of the wave form was the initial triphasic short latency component whether it was an N1-P1-N2 or P1-N1-P2 complex. The peak latencies of these complexes were stable , with one exception, to within $\pm$ 1-2 ms of baseline (Table 7.2 and Fig 7.1, 7.2, 7.4, 7.5). In one patient there were also stable long latency components. In this patient attenuation of the spinal SEP, which was also being monitored, was found to be due to migration of the spinal electrode since the cortical SEP remained stable (Fig 7.4).

There was variability in amplitude of both the early and late components of the SEP. The peak to trough amplitude of the short latency components ($<$ 40 ms) ranged between 0.8 and 6.0 uV, whilst the longer latency components ranged from 1.1 to 8.4 uV. In most cases, random amplitude fluctuations of up to 50% of baseline value were commonly seen in the short latency SEP components (Table 7.2). This variability did not appear related to the use or manipulation of particular anaesthetic agents, or fluctuations in blood pressure or end tidal carbon dioxide since both the latter parameters were in all cases stable throughout the surgical procedures.

There was a significant correlation between major preoperative neurological deficits involving the dorsal column modalities and failure to record a cortical SEP intraoperatively (Fisher exact

probability, $p < 0.02$). None of the 8 patients in whom cortical SEPs were recorded had a clinical dorsal column or other somatosensory deficit preoperatively, although 2 did have pyramidal tract dysfunction and one a lower motor neurone syndrome due to focal tumour infiltration in the conus. All except one of the patients in whom intraoperative cortical SEPs were recorded had extramedullary compressive myeloradiculopathies.

Of the 5 patients in whom no intraoperative cortical SEP could be recorded, 4 had documented deficits in dorsal column function preoperatively. All 4 of these patients (cases 3, 4, 5 and 10) had intramedullary spinal tumours that were also causing variable motor and sphincteric deficits (Table 7.1). One patient (case 13) whose age precluded formal assessment of dorsal column function appeared to be neurologically intact, even though cortical SEPs were not recorded. In all 5 patients in whom no cortical SEP could be obtained spinal SEPs were recorded from the surface of the cord rostral to the level of the spinal pathology (See section 8).

In this series, there did not appear to be any relationship between the use of halothane anaesthesia or intraoperative fentanyl analgesia and the reproducibility of cortical SEPs (Table7.2). The inspired volume per cent of halothane administered during the anaesthetics was low (0.3 - 0.5%). However in some cases the inspired halothane concentration was intermittently raised up to 1.5%, but since it takes approximately 30 minutes for halothane levels to equilibriate within the body (See section 9) the actual end expired halothane

levels would have been much lower (Fig 7.5).

The results of recording cortical SEPs with the platinum alloy subdermal needles and stick-on Ag/AgCl disc electrodes revealed that there was little difference between the two electrodes in signal acquisition. Despite the theoretically superior recording characteristics of the Ag/AgCl electrodes (Cooper et al, 1980) the use of these electrodes was offset by the practical problems of maintenance of good skin/electrode contact throughout sometimes lengthy surgical procedures. Patients sweating beneath the drapes and manipulation of the head and surrounding monitoring lines by the anaesthetist tended to cause dislodgement of the stick on electrodes - a problem that did not occur with the subdermal needle electrode.

DISCUSSION

This study has attempted to evaluate the utility of intraoperative recording of cortical SEPs to monitor spinal cord function during neurosurgical procedures on the spinal axis. Although the series is small, the application of this method of monitoring to a wide range of spinal pathologies and surgical procedures has enabled several useful points to be made. Intraoperative recording of cortical SEPs to monitor spinal cord function is associated with several well documented problems (Nash et al, 1977; Nuwer and Dawson, 1984; Symon et al, 1984). These are related to the problems of recording the cortical SEP in the theatre environment, the limitations of monitoring somatosensory function as an index of spinal cord function and

most importantly problems in interpreting the cortical SEP recorded under anaesthesia and during surgery.

Cortical SEPs can at the best be regarded as a very indirect and limited method of assessing spinal cord function since the cortical SEP is the electrophysiological end point of the dorsal column-medial lemniscal somatosensory pathways (Sarnowski et al, 1975; Cusick et al, 1979; Cohen et al, 1981; Snyder and Halliday, 1984). The particular component of the cortical SEP that most accurately reflects spinal cord function, if in fact any particular component serves this purpose, is speculative. Histological studies have demonstrated that normal cortical SEPs can be recorded even with hemorrhagic necrosis of the spinal cord gray matter (Schramm et al, 1979), and experimental cordotomy (Simpson, 1983).

Reports that have described the utilisation of cortical SEPs as a parameter of spinal cord function during surgery have emphasised different aspects of the waveform as being an index of stable functioning within the caudal somatosensory pathways. Some workers have emphasised the stability of the short latency components (Maccabee et al, 1982; Jones et al, 1983; Nuwer and Dawson, 1984) and others the stability of the long latency components (Allen et al, 1981; Worth et al, 1982). The amplitude of these, or other unstated components have also been variably used as an index of spinal cord function (McCallum and Bennett, 1975; Grundy, 1982), whilst in another study the normal side was used as a control and a guide to electrophysiological dysfunction on the operative side (Raudzens, 1982).

Theproblems with acquisition and interpretation of the cortical SEP recorded during surgery are compounded by random fluctuations in waveform amplitudes and latencies under anaesthesia. Some of this variability may be due to sampling error since acquisition of a stable cortical SEP may require averaging of up to 1024 impulses which at stimulating rates of 5Hz requires a recording time of over 3 minutes. During this period random changes in spinal and cerebral blood flow, blood pH, arterial oxygen, arterial carbon dioxide and blood pressure may all significantly influence cerebral functioning.

Failure to record a cortical SEP to lower limb stimulation in apparently neurologically intact patients such as our case 13 has previously been documented (Nash et al, 1977; Allen et al, 1981; Uemato and Tolo, 1981; Raudzens, 1982; Nuwer and Dawson, 1984) Some of these cases have been labelled "technically unsatisfactory" whilst no reason has been given in others. Experience gained in attempting to obtain normal cortical SEP data following unilateral lower limb stimulation under anaesthesia (See section 3) would suggest that this is not an infrequent occurence. Simultaneous bilateral tibial or peroneal nerve stimulation has been reported to minimise this problem (Lueders et al, 1981; Schiff et al, 1984).

As in this study previous neurological and orthopaedic series that have described intraoperative cortical SEP monitoring in over 600 patients have shown a correlation between preservation of the general waveform configuration and the clinical neurological status quo. Although Levy (1983) and Ginsburg and

colleagues (1985) have recently alluded to several patients who had stable intraoperative SEPs but awoke with lower limb neurological deficits all other reports have invariably associated a post operative neurological deficit with permanent loss of the SEP waveform (McCallum and Bennett, 1975; Nash et al, 1977; Engler et al, 1978; Owen et al, 1979; Allen et al, 1981; Uematо and Tolo, 1981; Grundy, 1982; Maccabee et al, 1982; Raudzens, 1982; Worth et al, 1982; Nuwer and Dawson, 1984).

The value of cortical SEPs as an intraoperative index of spinal function may therefore appear unequivocal. Scrutiny of the literature however reveals that his conclusion must be accepted with several reservations from the neurosurgical viewpoint. The success of this modality of monitoring in orthopaedic surgery is largely related to the fact that the surgery is extradural, and most patients with juvenile and idiopathic scolioses do not have associated neurological deficits in the lower limbs. Thus if during a Harrington procedure for correction of scoliosis, the cortical SEP signal attenuates, it could reasonably be expected if anaesthetic and other monitored physiological variables are stable that release of compression and distraction rods would rectify any distortion of the signals. In these cases, the spinal funiculae, glia and neurones are usually normal preoperatively and are not directly damaged by surgery. Impairment of spinal cord function in these cases is believed to be caused by mechanical traction on the spinal and radicular blood vessels (Dolan et al, 1980; Jones et al, 1983) and also on the spinal medulla through the tethering effect of arachnoid adhesions (Yamada et al, 1981).

In neurosurgical patients, the highest risk of iatrogenic surgical insult usually occurs in those with major preoperative clinical deficits and intradural pathology. However it is in this group of patients that cortical SEPs are extremely difficult to obtain. Intraoperative monitoring of spinal SEPs in these cases is usually possible even though the signal amplitude is less than that recorded in patients free of neurological disease (See section 8). Spinal SEP monitoring also has the added advantages of simplicity of recording, and rapidity of acquisition of a signal that is a more specific index of spinal cord function, and which is easier to interpret (Schramm et al, 1983; Worth et al, 1982; Jones et al, 1983).

Specific situations in which cortical SEP monitoring may be used to assess spinal cord function in neurosurgical operations are in extradural procedures in the upper cervical region. During operations such as a odontoidectomy, posterior atlanto-axial fusions, cervical laminectomy for spondylitic myelopathy and removal of cervical extradural tumours, satisfactory placement of an extradural electrode for spinal SEP monitoring may be difficult because of either spatial considerations or adhesions from prior surgery. Cortical SEP monitoring in these cases also has the advantage of giving some objective evidence of somatosensory function whilst the patient is positioned for the procedure. This is an important consideration in some cases of upper cervical spinal instability and spinal canal stenoses.

The other use of cortical SEP monitoring is as an adjunct to spinal SEP monitoring. Some workers have utilised multi-channel

monitoring of cortical and spinal SEPs to gain a greater understanding of how the two signals are inter-related and the significance of changes that occur during surgery (Maccabee et al, 1982). Simultaneous monitoring of cortical and spinal SEPs in animals undergoing compressive and ischaemic spinal cord insults have however produced conflicting results, although these differences may be partly due to experimental methodology (Schramm et al, 1979; Bennett, 1983). Multi-channel SEP monitoring would also demonstrate the differential effect of various anaesthetic agents on the cortical and spinal SEP.

The results in this study suggest that neither low inspired volumes of halothane nor fentanyl analgesia have a significant effect on the general configuration and amplitude of the cortical SEP recorded intraoperatively. These findings are at variance with previous reports (Clark and Rosner, 1973; Engler et al, 1978; Grundy, 1980; Worth et al, 1982). None of these studies however have specified the levels of anaesthetic agents required to attenuate the cortical SEP. Another study has reported findings similar to this series, and in particular noted that cortical SEPs were recordable with halogenated inhalation anaesthetics delivered at less than 0.5% inspired volumes. At levels greater than 0.75% no cortical response was reproducible (Nuwer and Dawson, 1984).

PATIENT	DIAGNOSIS	CLINICAL STATUS	PROCEDURE	DURATION	SEP TRACE
1 TR	Osteochondroma C2	Neurologically normal	C1/2 Laminectomy tumor removal	2 hr	Reproducible
2 LP	Congenital scoliosis Cervical rib	Neurologically normal Scoliosis	Harrington rod insertion and spinal fusion	1 hr	Reproducible
3 VW	Intramedullary astrocytoma (T5-7)	Spastic gait, impaired JPS hallux, spinothalamic sensation and	Reexploration, biopsy of tumor	1/2 hr	No waveform recordable
4 GL	Intramedullary neurofibroma (C2-5)	Atrophy, fasciculation, paresis left greater than right arm and hand. Ataxic with spastic paraparesis and bisphincteric incontinence	Excision of tumor	5 hr	No waveform recordable
5 AA	Intramedullary (T9conus) hemangioblastoma	Saddle anesthesia, bisphincteric dysfunction. Muscle atrophy and sensory loss from L4 on left.	Myelotomy, drainage syrinx	1 hr	No waveform recordable
6 GG	Cervical arachnoiditis	Neurologically normal	Laminectomy, division adhesions	1 hr	Reproducible

TABLE 7.1 CLINICAL, PATHOLOGICAL AND SURGICAL DATA FOR THE 13 PATIENTS HAVING INTRAOPERATIVE CORTICAL SEP MONITORING DURING SPINAL SURGERY

PATIENT	DIAGNOSIS	CLINICAL STATUS	PROCEDURE	DURATION	SEP TRACE
7 FD	Dsychondroplasia with spinal canal stenosis	Mild spastic quadraparesis Sphincters and JPS intact	Decompressive cervical laminectomy	1 hr	Reproducible
8 AL	Cervical spondylitic myelopathy	Mild spastic quadraparesis Sphincters and JPS intact	Decompressive cervical laminectomy	1 hr	Reproducible
9 VS	Lumbar lipomeningocele with tethered cord	Muscle atrophy, paresis and sensory loss below L4 on left. Impotent	Transection lipomeningocele	2 hr	Reproducible
10 HP	Ependymoma of conus & cauda equina	Spastic paraplegia with neurogenic bladder and sensory lelel at L2. Impaired JPS below hip joint	Excision of tumor	4 hr	Waveform not recordable
11 SD	T11 Neurofibroma	Neurologically normal except (R) hyperactive KJ and extensor plantar	Excision of neurofibroma	2 hr	Reproducible
12 RS	Intramedullary (T10-conus) astrocytoma	Paresis R hip flexion,foot eversion & dorsiflexion, with depressed R KJ and AJ. Normal sensation	Laminectomy, excision tumor	2 hr	Waveform not recordable after myelotomy
13 BW	Lumbar diastematomyelia with low conus	Neurologically normal	Laminectomy, exploration lumbar theca	1/2hr	Waveform not reproducible

TABLE 7.1 (Cont)

PATIENT	STIMULUS/ RECORDING SITE	PEAK LATENCIES(ms) BASELINE RANGE	SEP AMPLITUDE(uV) BASELINE RANGE	ANESTHETIC	COMMENT
1 TR	L/R Median nerve C3/C4:Fpz	N1 = 17.2(16.4-17.8) P1 = 21.0(20.4-21.2) N2 = 38 (29-39)	N1P1 = 2.3(2.0-3.0)	Nitrous oxide/oxygen Intermittent halothane to 1%	Stable SEPs No postop deficit
2 LP	L ulnar nerve C4:Fpz	P1 = 15.5(15.0-16.0) N1 = 17.0(16.5-17.5) P2 = 21.5(20.0-21.3) N2 = 50 (47-56)	N1P2 = 2.5(2.0-3.0) P2N2 = 2.5(1.5-3.2)	Nitrous oxide/oxygen Intermittent halothane to 1.5%	Stable SEP. No postop deficit
3 VW	L/R Tibial nerve Cz':Fpz	No cortical SEP		Nitrous oxide/oxygen halothane to 0.5%	Abnormal spinal SEP recorded
4 GL	L/R Median nerve C3/C4:Fpz	No cortical SEP		Nitrous oxide/oxygen halothane to 0.3% Fentanyl	Abnormal spinal SEP recorded
5 AA	L/R Tibial nerve Cz':Fpz	No cortical SEP		Nitrous oxide/oxygen Fentanyl	Abnormal spinal SEP recorded

TABLE 7.2 DETAILS OF STIMULATING AND RECORDING SITES, TOGETHER WITH MAJOR PEAK LATENCIES AND THEIR AMPLITUDES OF THE CORTICAL SEPs RECORDED INTRAOPERATIVELY IN 13 PATIENTS UNDERGOING SPINAL SURGERY.

PATIENT	STIMULUS/ RECORDING SITE	PEAK LATENCIES(ms) BASELINE RANGE	SEP AMPLITUDE(uV) BASELINE RANGE	ANESTHETIC	COMMENT
6 GG	R Median nerve C3:Fpz	P1 = 17.6(16.0-18.6) N1 = 20.5(20.4-21.6) P2 = 26.7(25.8-30.6) N2 = 43 (42-47)	N1P2 = 2.9(1.7-3.6) P2N2 = 7.0(6.5-7.0)	Nitrous oxide/oxygen Fentanyl	Stable SEP. No postop deficit
7 FD	L Median nerve C4:Fpz	P1 = 16.8(16.4-17.0) N1 = 20.4(19.6-20.8) P2 = 29.8(27.8-30.8) N2 = 42 (40-47)	N1P2 = 2.5(0.8-2.6) P2N2 = 2.3(2.3-3.2)	Nitrous oxide/oxygen Fentanyl	Stable SEP. No postop deficit
8 AL	L Median nerve C3:Fpz	P1 = 17.2(16.8-17.8) N1 = 21.3(20.8-21.6) P2 = 30.5(28.8-31.2) N2 = 47 (42-48)	N1P2 = 3.9(2.4-6.0) P2N2 = 7.0(3.0-8.4)	Nitrous oxide/oxygen Halothane 0.5%	Stable SEP. No postop deficit
9 VS	R Tibial nerve Cz':Fpz	N1 = 40 (37-40) P1 = 52 (52-55) N2 = 69 (69-72) P2 = 154(150-180) N3 = 172(170-180)	P1N2 = 1.7(1.7-2.5) P2N3 = 2.1(1.1-2.1)	Nitrous oxide/oxygen Halothane 0.5%	Stable SEP. No postop deficit
10 HP	L/R Tibial nerve Cz':Fpz	No cortical SEP		Nitrous oxide/oxygen halothane 0.3% Fentanyl	Abnormal spinal SEP recorded

TABLE 7.2 (Cont)

PATIENT	STIMULUS/ RECORDING SITE	PEAK LATENCIES(ms) BASELINE RANGE	SEP AMPLITUDE(uV) BASELINE RANGE	ANESTHETIC	COMMENT
11 SD	R Tibial nerve Cz':Fpz	P1 = 33 (29-44) N1 = 41 (30-41) P2 = 51 (51-63) N2 = 62 (43-62)	P1N1 = 2.0(1.8-2.5) P2N2 = 2.1(2.0-4.0)	Nitrous oxide/oxygen halothane 0.5%	Reproducible but variable SEP. No postop deficit
12 RS	L/R Tibial nerve Cz':Fpz	N1 = 20 (20-22) P1 = 25 (25-27) N2 = 31 (31-34) P2 = 51 (51-56) N3 = 69 (69-76)	P1N3 = 3.0(2.5-5.0)	Nitrous oxide/oxygen halothane 0.5%	SEP stable until midline dorsal myelotomy performed major postop deficit
13 BW	L/R Tibial nerve Cz':Fpz	No cortical SEP		Nitrous oxide/oxygen halothane 0.5%	Spinal SEP recorded

TABLE 7.2 (Cont)

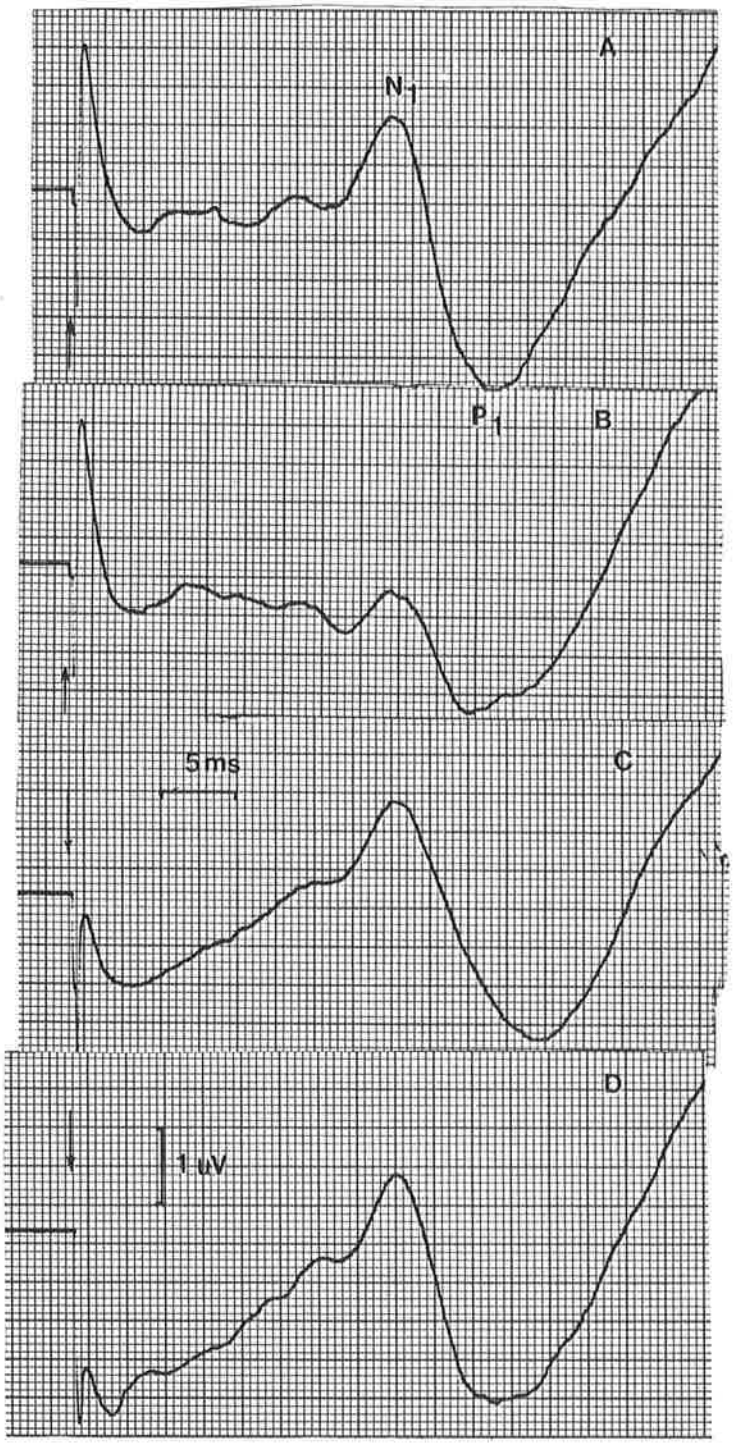


Fig 7.1 Sequential intraoperative cortical (C3:Fpz) SEPs from median nerve stimulation in case 6. The baseline N1-P1 component (trace a) has an amplitude of 3.5 uV. With cervical laminectomy amplitude falls to 1.5 uV (trace b). The amplitude recovers following laminectomy despite dural opening (trace c) and division of pial adhesions (trace d).

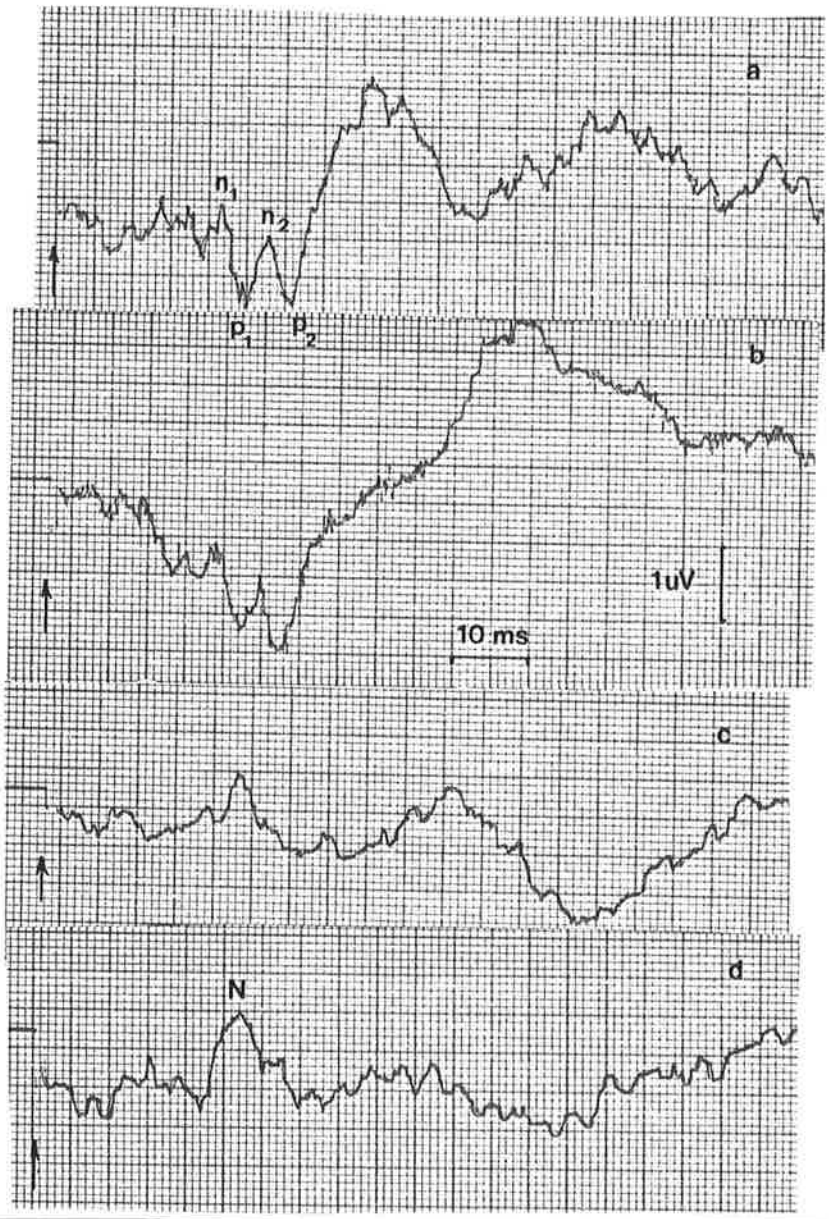


Fig 7.2 Sequential intraoperative cortical (Cz':Fpz) SEPs from tibial nerve stimulation in case 12. The waveforms prior to dural opening have a reproducible N1-P1-N2-P2 complex (trace a,b). Following myelotomy (trace c) there is loss of this complex. Later recordings performed after resection of an intramedullary astrocytoma show persistant loss of this complex and its replacement by a broad N component. There was also gross disturbance of the intraoperative spinal SEP (Fig 8.10). This patient had an intramedullary astrocytoma extending from T9 to L1 that is well seen on the myelogram (Fig 7.2b).

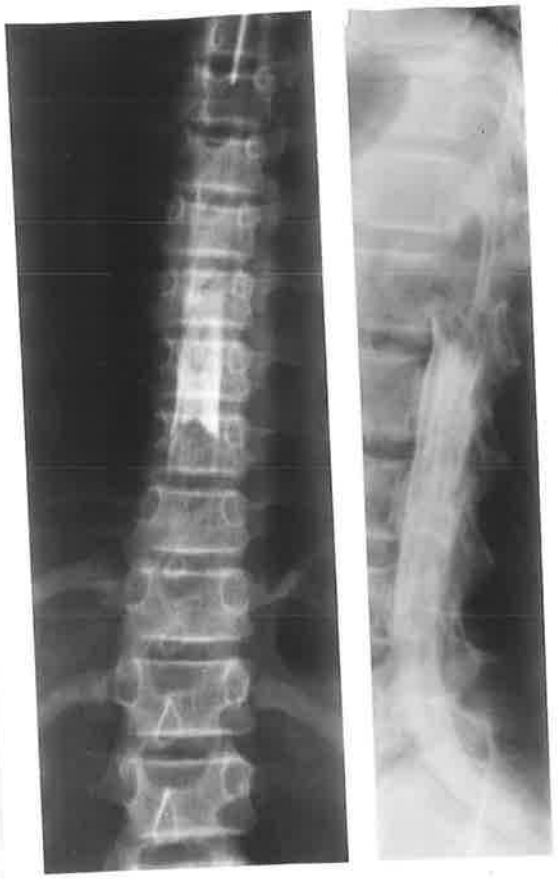


Fig 7.2b Metrizamide myelogram of case 12. There is complete obstruction to flow of the contrast agent at the T9 level rostrally (left) and the L1 level caudally (right) by an intrinsic spinal cord lesion. Biopsy revealed the lesion to be a low grade intramedullary astrocytoma.

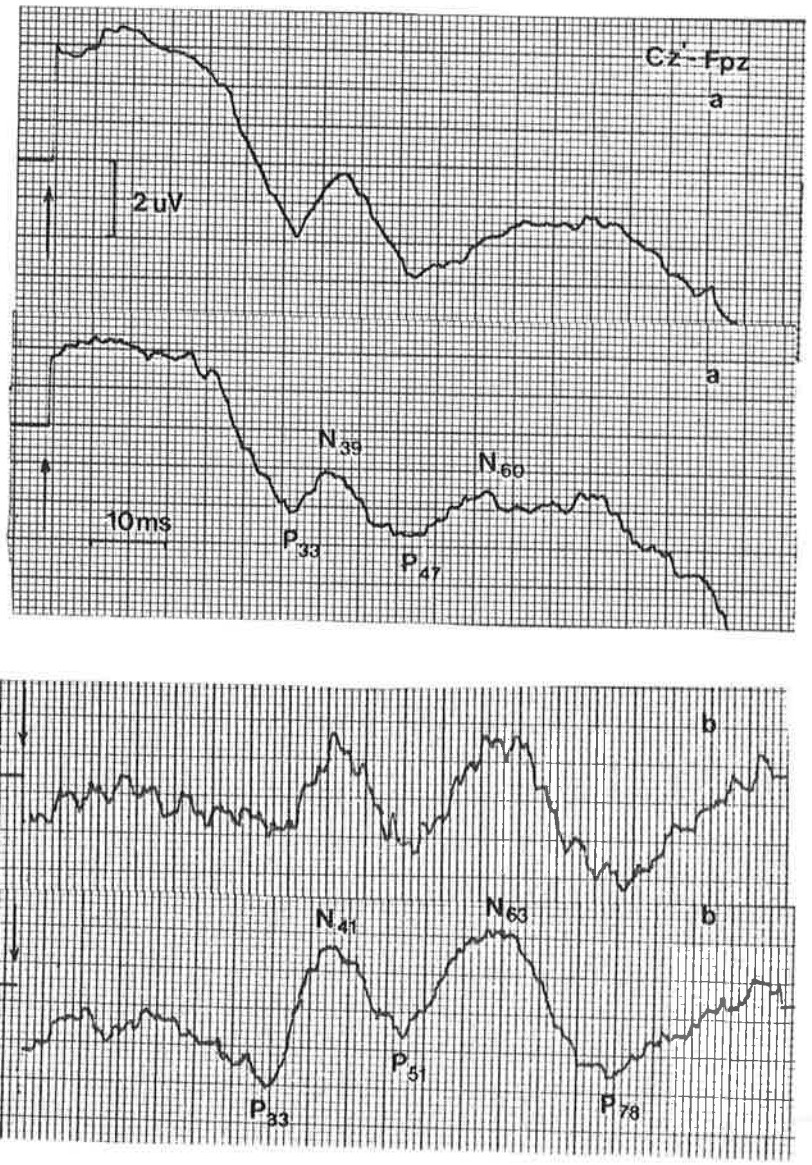


Fig 7.3 Preoperative (traces a) and intraoperative (traces b) Cortical SEPs (Cz':Fpz) following tibial nerve stimulation in case 11. The amplitude is larger, and the P2 (P51) and N2 (N63) components better defined with the intraoperative recording. The P1-N1 components of both recordings have similar latencies.

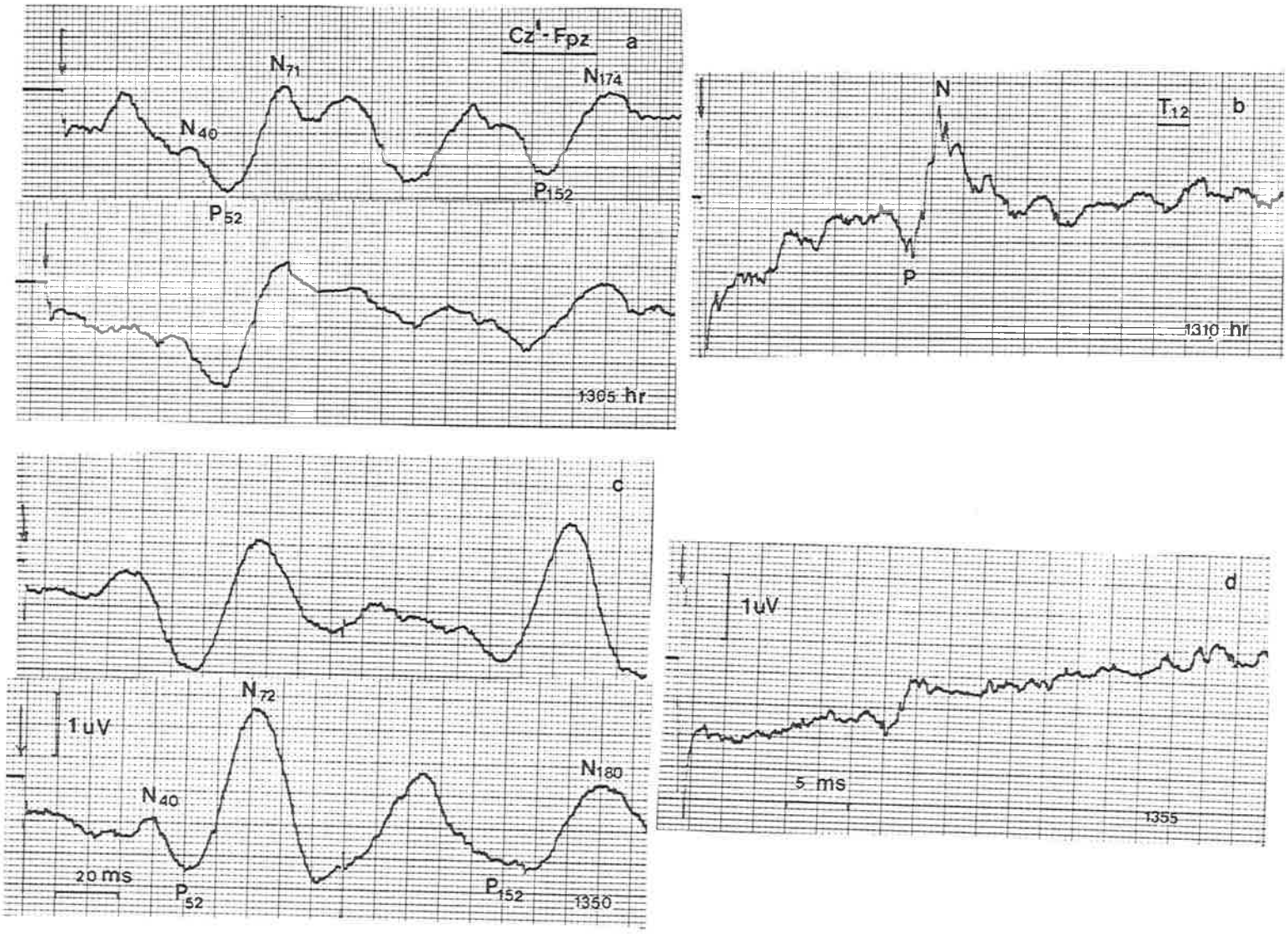


FIG 7.4 (Legend overleaf)

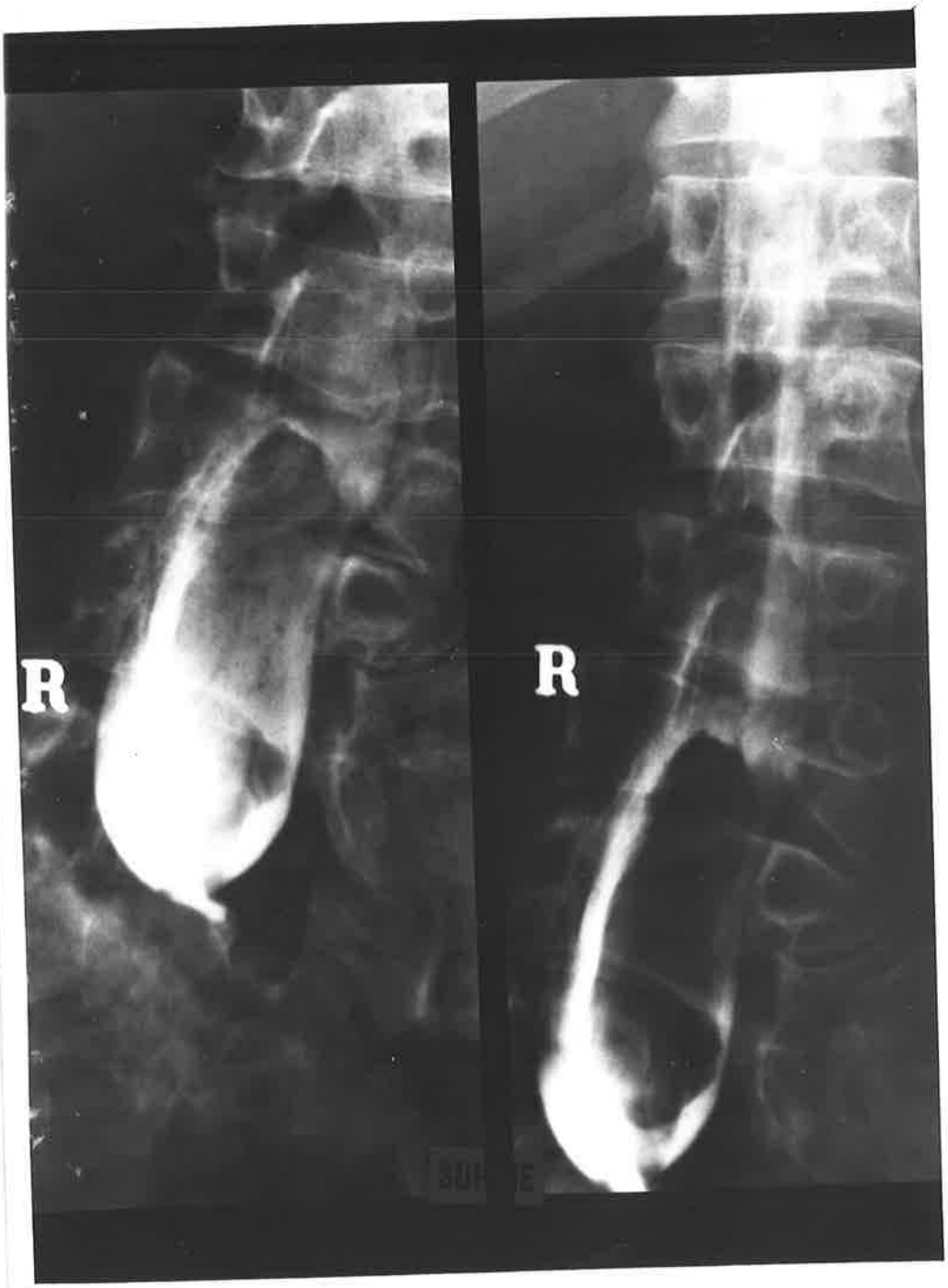


Fig 7.4 Intraoperative cortical (Cz':Fpz) and spinal (extradural T12; referenced adjacent paraspinal muscle) SEPs following tibial nerve stimulation in case 9. This patient had a large lumbosacral lipomeningocele and tethered cord syndrome that are well shown on the myelogram (above). Intraoperative SEP waveforms revealed reproducible cortical components at N22, N40, P52, P152 and N174/180 (traces a) and a good spinal SEP waveform (traces b). During transection of the lipomeningocele the spinal SEP amplitude attenuated considerably (trace d) however the cortical SEP waveform (traces c) remained stable. The spinal recording electrode had been distracted from the extradural plane.

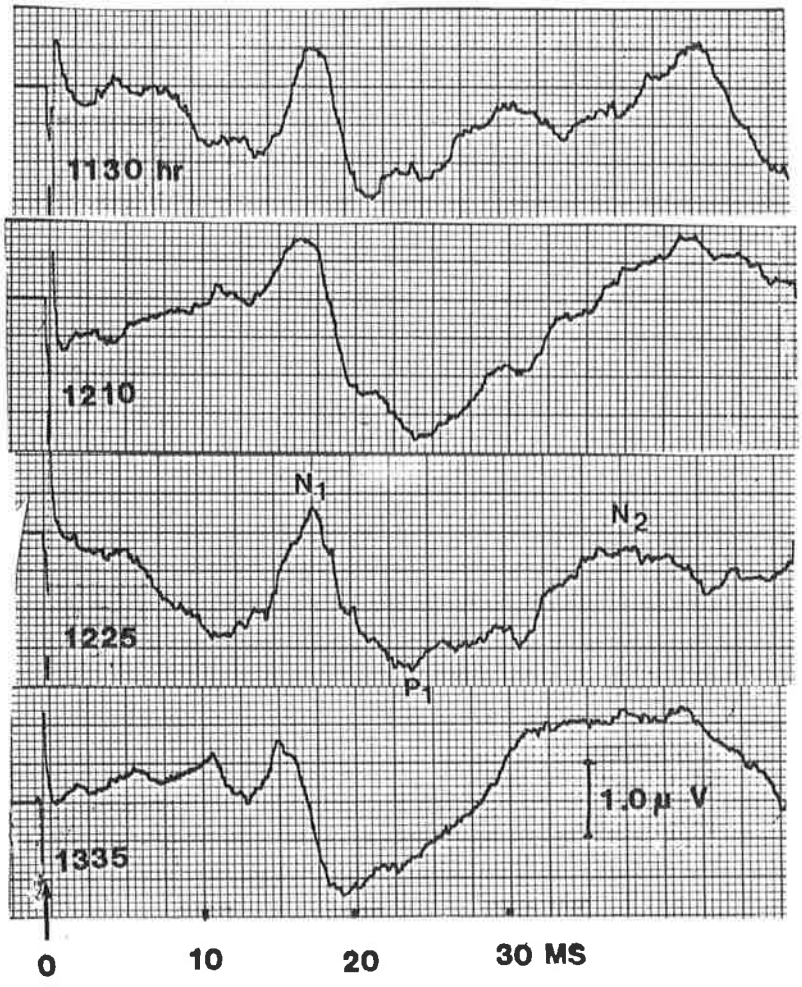


Fig 7.5 Intraoperative cortical SEPs (C4:Fpz) following median nerve stimulation in case 1. The latency of the N1 component is beyween 15.5 and 17.8 ms in all recordings. Recordings at 1130 hr and 1335 hr were with halothane off, 1210 with 0.5% halothane and 1225 with 1.0% halothane. The amplitude of N1-P1 and latency of N2 are variable although the waveform remained stable.

SECTION 8

EXPERIENCE WITH INTRAOPERATIVE RECORDING OF SPINAL SOMATOSENSORY EVOKED POTENTIALS AS A METHOD OF SPINAL CORD MONITORING DURING SPINAL SURGERY

INTRODUCTION

As experience with intraoperative cortical SEP recording to monitor spinal cord function during surgery has increased, many problems and limitations with this mode of monitoring have become apparent (Bunch et al, 1983; Grundy, 1983; Jones et al,1983). Experimental data has suggested that the spinal SEP is a more specific index of spinal cord function and that acquisition and interpretation of this signal is considerably simpler than its cortical equivalent (Bennett, 1983; Nordwall et al, 1979; Raudzens, 1982; Schramm et al, 1983). Subsequently new methods of intraoperative recording of spinal SEPs have been developed and described (Hahn et al, 1981; Tamaki et al, 1981; Lueders et al, 1982; Maccabee et al, 1982; Macon et al, 1982; Jones et al, 1983; Kaschner et al, 1984; Nashold et al, 1985).

Clinical experience with the various methods of monitoring spinal SEPs is now accumulating and several series have demonstrated their undoubted utility in surgery for the correction of kyphoscoliosis (Tamaki et al, 1981; Maccabee et al, 1982; Jones et al, 1983; Lamont et al, 1983). Reports describing spinal SEP monitoring during neurological spinal surgery are much less frequent. Except for the series of 15 patients reported by Macon

and colleagues (1982) reports are mainly technical with anecdotal references to case reports (Hahn et al, 1981; Lueders et al, 1982). Although the techniques of recording spinal SEPs intraoperatively are established, their utility in spinal neurological surgery remains unknown.

In this study a new technique of monitoring spinal SEPs intraoperatively is described together with its clinical application. The same equipment is also applied to monitor epidural spinal SEPs during scoliosis surgery. Experience obtained with these cases has enabled an evaluation of the utility of these modes of spinal cord function monitoring from both the surgical and neurophysiological viewpoints. Particular areas addressed included;

(1) Technical problems in acquisition of the spinal SEP
(2) The stability of the spinal SEP under anaesthesia
(3) Patterns of spinal SEP at different levels of the spinal cord
(4) Changes in the spinal SEP waveform with intramedullary spinal disorders
(5) The stability of the SEP during intra and extradural spinal surgery
(6) Factors attenuating the spinal SEP intraoperatively.
(7) Correlation between changes in spinal SEPs recorded intraoperatively and post-operative clinical outcome.

The results from this study were compared to the literature and some general conclusions made concerning the utility of this mode of monitoring during neurological and orthopaedic surgery.

CLINICAL MATERIAL

A series of 26 patients undergoing spinal surgery had spinal SEP monitoring as a routine part of intraoperative physiological monitoring. These patients were divided into two groups.

Group 1 consisted of nine patients having Harrington procedures for the correction of scoliosis. The mean age of these patients was 11 years six months (range 8 to 13 years). None of these patients had preoperative neurological deficits in the lower limbs. One patient was deaf and another with the Praeder-Willi Syndrome was mentally retarded (Table 8.1).

Group 2 consisted of 17 patients who underwent a variety of spinal neurosurgical procedures. The mean age of patients, excluding three adults aged 23, 25 and 53 years, was 5 years three months (range 6 months to 16 years). Many of these patients had neurological deficits in the lower limbs and urinary or anal sphincter dysfunction (Table 8.2). These patients had either developmental abnormalities of the spinal axis or intradural spinal neoplasms. The diagnosis in all cases had been confirmed by combinations of metrizamide myelography, CT Scanning of the spine and biopsy of the lesion (Table 8.2).

METHODS

The patient was anaesthetised and an endotracheal tube positioned using succinyl choline muscle relaxation. When there was evidence that the depolarising muscle blockade was waning,

saline-soaked felt pad stimulating electrodes were placed in both popliteal fossa with the cathode being proximal to the anode. The electrodes were positioned over the tibial or common peroneal nerves so that a 50-100V electrical stimulus elicited a strong muscle twitch in the foot. After correct placement of the stimulating electrode was verified, they were strapped or taped in situ and an earth plate taped to one buttock.

After paraspinal muscle clearance and exposure of the vertebral laminae a reference needle electrode was placed in the muscle and subcutaneous tissues at the level of surgery. In patients having orthopaedic correction of scoliosis using Harrington Instrumentation the non-polarisable platinum recording electrode (Fig 8.1 * - Spinal electrode recording kit ELK-7, manufactured by Ceramic Substates Ltd, the Broadway, Farnham Common, Buckingshire SL2 2PQ England) was placed in the epidural space through a small fenestration in the ligamentum flavum rostral to the upper limit of spinal instrumentation. In patients having intradural spinal neurological surgery the electrode was placed after laminectomy under direct vision in the subdural plane, rostral to the level of pathology (Fig 8.2 #). The spinal recording electrode was in either case stablised in situ with one of more 3.0 silk sutures to the dura or surrounding soft tissues. The recording and reference electrode leads were then covered with a sterile drape and directed from the surgical field. The were plugged into an extension cable which was handed to the recording physician and connected to the preamplifier.

Electrical shock stimulation was applied transcutaneously to the

* See Appendix 1, Fig 1. # See Appendix 2, Fig 2.

popliteal fossa stimulating electrode through a stimulus isolation unit. Square wave pulses of 0.1ms duration and 50-100V were administered at 5Hz. Signals were fed through a Medelec PA89 preamplifier connected to a Medelec MS91 signal averaging unit. The frequency bandpass was set at 20-2000Hz (-6dB rolloff) and amplifier sensitivity set at 50uV. Epochs of 10-30ms were averaged. SEP waveforms could be obtained after averaging 30-128 stimuli. The time, mean blood pressure during the period of each recording, surgical manoevres and any variations in anaesthetic technique were also tabulated with the recordings.

A baseline SEP waveform was recorded prior to any vertebral column distraction, compression or spinal manipulation. This record was stored on one channel so that immediate waveform or latency changes could be detected by comparison with subsequent records. Waveforms obtained during the surgical procedure were directly compared with a baseline waveform by superimposition of the two channels on the oscilloscope screen. Peak and interpeak latencies could be measured directly from the oscilloscope trace by a cursor with a digital readout. Spinal SEPs recorded throughout the procedure were intermittently plotted onto a paper chart recorder prior to a erasure. Peak to trough amplitudes of various components of the waveform were quantitated from these recordings.

Because of the large amplitude and short duration of the spinal SEP, a discreet waveform could be elicited, from the thoracolumbar region, after as few as 30 stimuli were averaged. This allowed almost continuous monitoring of spinal cord function

during potentially hazardous surgical manoevres such as the distracting process when correcting a scoliotic deformity and during spinal cord manipulation or dissection. After maximal vertebral distraction and compression had been obtained during orthopaedic surgery to correct scoliosis, further recordings were performed whilst fusion of the facet joints proceeded to ensure that any delayed impairment of spinal cord function was recognised. If in these cases the spinal SEP monitoring was suggestive of impaired cord function vertebral distraction was decreased or a "wake-up" test (Vauzelle et al, 1974) was performed.

Anaesthetics for the surgical procedures consisted of muscle relaxation with curare analogues (pancuronium, alloferin) and intermittent positive pressure ventilation with 60% nitrous oxide and 40% oxygen supplemented by either Halothane or Fentanyl. During the orthopaedic correction of scoliosis using Harrington's Instrumentation, induced hypotension during the period of spinal fixation and fusion was achieved using nitroprusside infusion and titrated levels of Halothane. Mean blood pressures during this period of the operation were around 60 mm Hg.

RESULTS

The operations performed on the twenty six patients in this series included nine Harrington procedures with spinal fusion (Group 1) and 17 neurosurgical procedures (Group 2). These neurosurgical operations are detailed in Table 8.3. There were no anaesthetic complications and, except for one case described

later, blood pressure and end tidal carbon dioxide levels remained within physiological limits throughout the operation. There were no complications because of the spinal monitoring.

Intraoperative Spinal SEP Acquisition and Morphology

Reproducible spinal SEPs were obtained throughout the desired period of spinal monitoring in 24 of the 26 cases. Despite the range of spinal cord pathology in these 24 patients some patterns of spinal SEP were characteristic of different segments of the spinal cord. At the thoracolumbar, or low lumbar levels in those patients with tethered cord syndromes, a large amplitude sinuous or triphasic potential was recorded (Fig 8.3). In the low thoracic region, the spinal SEP had either an early negative (N) or positive (P) wave followed by a sharp di or triphasic complex (Figure 8.4). Spinal SEPs recorded from the mid thoracic to cervicothoracic regions in patients with normal spinal function (all the Group 1 patients) had a variable initial P wave that was followed by a large amplitude polyphasic P-N-P-N complex, and a later longer duration negative complex (Fig 8.5). As the level of recording moved rostrally the latency of the P-N-P-N complex increased and the negative complex became longer in duration and more polyphasic. To simplify characterisation and analysis of these polyphasic waveforms the peak latencies of the initial P or N wave and the last major component of the negative complex, and/or interpeak latencies were studied (Tables 8.1 and 8.3).

The amplitude of the spinal SEP decreased from approximately 10-20uV in the thoracolumbar region to 1-2uV in the cervicothoracic

region. It was possible to demonstrate a direct relationship between amplitude of the segmental spinal SEP recorded over the conus and stimulus intensity. The complexity of this lumbar segmental SEP did not significantly change with stimulus intensity (Fig 8.6). A relationship between stimulus intensity and the ascending spinal SEP recorded over the upper thoracic region was not satisfactorily demonstrated.

In the two patients in whom spinal SEP acquisition was unsatisfactory, one (case 1, Group 1) was the first to have spinal monitoring during a Harrington procedure. In this case the spinal SEP was acquired only during the initial period of mechanical distraction of the spine. This failure was probably due to inexperience with intraoperative recording. A "wake up" test (Vauzelle et al, 1973) revealed no neurological deficit following correction of the scoliosis. The other patient (case 9, Group 2) had attenuation of the spinal SEP during transection of a lipomeningocele. Cortical SEPs which were also monitored in this patient remained stable and therefore it was assumed the spinal electrode had been distracted from the correct recording plane . The surgeon continued the procedure without repositioning the spinal electrode.

Changes in Baseline Spinal SEP Waveforms

Major distortion, asymmetry or attenuation of the baseline spinal SEP waveform was seen in five patients (cases 8, 10, 11, 14, 15 Group 2). All these patients had intramedullary spinal cord tumours that were causing significant neurological deficits

variably involving the dorsal column and spinothalamic modalities, the corticospinal tracts and dysfunction of the urinary and anal sphincters (Table 8.2). The spinal SEPs in these patients, when compared to SEPs recorded in patients without neurological deficits showed distortion of the initial polyphasic P-N-P or N-P-N complex, and its relacement by a shallow P wave. Flattening and loss of definition of the N complex was another prominent change (Fig 8.7). These changes were highlighted by multiple site recordings of spinal SEPs before surgical manipulation of the cord (Fig 8.8,8.9 and 8.10).

The changes from normality in the lumbar spinal SEPs caused by tethered cord syndromes of variable aetiology were difficult to quantitate since normative data for conus SEPs covering the ages of patients recorded in this series is unknown. The spinal SEP recorded in these patients was a sinuous generally non specific waveform, despite the variable clinical deficits and other congenital abnormalities associated with the tethered cord syndrome (Fig 8.3). In two patients with tethered cord syndromes due to a lipomeningocele (cases 7, 16, Group 2), there was major amplitude attenuation of the spinal SEP when considering the low level of monitoring. The amplitude of the SEPs in these cases varied between 0.4 and 1.3uV. Although one patient (case 16) had a major bilateral neurological deficit that would have involved the rootlets of the tibial and peroneal nerves, the other patient was apparently neurologically normal and there was no obvious cause for the low signal amplitude.

Stability of Intraoperative Spinal SEPs

The spinal SEP waveforms were stable under general anaesthesia with nitrous oxide and oxygen, supplemented by either fentanyl (0.003 mg/kg) or halothane. The components of the spinal SEP waveform remained stable for periods up to several hours. Peak, and interpeak latencies were stable to within $\pm$ 0.36 ms in all except two cases where the variability was $\pm$ 0.48 ms. Halothane concentrations of up to 2.5% inspired volume during Harrington procedures for scoliosis did not alter the spinal SEP configuration or peak latencies, although there was some variable amplitude attenuation.

Therapeutic hypotension to mean levels of 55mmHg during Harrington's procedures did not alter the spinal SEP waveform or peak latencies (Fig 8.11). However, profound hypotension (approximately 30mmHg mean BP) recorded in one patient (case 1, Group 2) following haemorrhage from the base of a bony diastematomyelic septum was associated with loss of waveform components (Fig 8.12).

Over zealous correction of a major scoliotic deformity by Harrington distraction rods resulted in the loss of the polyphasic P-N-P component and loss of definition of the slower late negative wave in one patient. This electrophysiological dysfunction was not rectified by elevating the blood pressure but did show partial recovery following release of several ratchets of the distraction rods (Fig 8.13).

Dorsal midline myelotomy, placement of pial retraction sutures and resection of an intramedullary astrocytoma was associated with loss of P1 and attentuation and loss of definition of the subsequent N complex of the spinal SEP in one case (Fig 8.10). Spinal cord manipulation with microdissectors and intramedullary tumour removal or biopsy was associated with distortion of baseline SEP waveforms in two other patients. In one of these patients (case 10, Group 2) there was transient recovery of the waveform component following cessation of the surgery for 15 minutes. Whilst in the other (Case 15, Group 2), the waveform remained distorted.

In four patients in whom distortion of the spinal SEP was seen intraoperatively, a prominent early P wave appeared or was augmented coincidentally with electrophysiological spinal dysfunction. This P wave had a latency to suggest it represented the afferent volley as it entered the spinal cord and was thus a volume conducted potential. This wave has been previously termed the S or standing wave (Nordwall et al, 1979).

In two patients (cases 2, 11, Group 2) the waveform amplitude and complexity increased following surgery (Fig 8.14). One of these patients had aspiration of a low thoracic syrinx associated with an intramedullary haemangioblastoma and the other freeing of a tethered spinal cord from adhesions at the site of closure of a dorsal lumbosacral meningocele. This electrophysiological "improvement" was not associated with clinical improvement in the patient with major neurological deficit.

In six of the nine patients monitored whilst undergoing Harrington's procedure, there was greater than 30% attentuation of the spinal SEP when compared to baseline amplitude. This attenuation was not associated with changes in SEP peak latencies, and appeared independent of surgical and anaesthetic manipulations (Fig 8.15 and Table 8.1).

When correlating post operative outcome with changes in spinal SEPs from the baseline intraoperative recording, preservation of waveform components was much more important than fluctuations in the amplitude of the waveform. Persistent amplitude loss of up to 70% of baseline value was not associated with acquired post operative neurological deficits. Persistent loss of waveform subcomponents was however correlated with an intraoperatively acquired neurological deficit. This phenomenon was seen in three cases (cases 10,14,15, Group 2). In contrast no patient with stable intraoperative spinal SEPs waveforms sustained a neurological deficit as a result of surgery.

DISCUSSION

The best method of recording spinal SEPs intraoperatively is unknown. The advantages of subdural and epidural recording electrodes have previously been described (Hahn et al, 1981; Tamaki et al, 1981; Macon et al, 1982; Bunch et al, 1983; Jones et al, 1983; Lamont et al, 1983; Kaschner et al, 1984; Nashold et al, 1985) and from the diversity of methodologies reported in the literature it seems important only that the method utilised is simple, safe, reliable and does not unnecessarily prolong the

procedure.

The use of a small light-weight flexible subdural recording electrode in neurosurgical procedures has particular advantages.It provides the ultimate in non-invasive near field spinal recording since it is separated from the neural generators of the ascending or segmental SEP only by the leptomeninges. Placement of the recording electrode by the surgeon is simple and atraumatic, and the electrode is unobtrusive once placed in the surgical field. This mode of recording also affords the facility for recording SEPs at different spinal segments both caudal and rostral to the level of the lesion. Such neurophysiological investigation can facilitate study of the 'killed end' or 'spinal cord evoked injury potential' in patients with segmental spinal lesions (Schramm et al, 1983).Furthermore, spinal SEPs were recorded rostral to the level of the lesion in all cases in which monitoring was attempted. In the other neurosurgical series reporting the use of spinal SEP monitoring, three of fifteen patients had no recordable SEP rostral to the lesion when recording was performed with epidural electrodes (Macon et al, 1982b).

Near Field Spinal SEP Waveforms

Neurophysiological data acquired using intraoperative spinal SEP monitoring techniques offers a unique opportunity to correlate electrophysiological with clinical and pathological findings. The spinal SEP waveforms obtained in many cases with minimal or no neurological deficit were almost identical to those described

by Jones and colleagues (1982). The waveform complexity, duration and amplitude all varied with the spinal segment at which recordings were performed. Attenuation of amplitude of the spinal SEP, a prolonged initial P wave and loss of definition of the later N wave components were characteristics of patients with major neurological deficits. Such waveforms correspond closely to the spinal cord evoked injury potential (Schramm et al, 1983) and waveforms recorded from cat spinal cords rostral to experimental myelotomies (Snyder and Halliday, 1984). In the one patient with an intramedullary tumour and no preoperative deficit in dorsal column function, the waveform was essentially normal until a dorsal midline myelotomy was performed (Fig. 8.10).

The ease and rapidity of acquisition of the spinal SEP even when the recordings were from the high thoracic region and in the presence of major intramedullary pathology contrasts with the difficulties in acquisition of the cortical SEP.It has previously been shown that cortical SEPs may be difficult to record intraoperatively, particularly when there is a significant neurological deficit involving the dorsal column modalities (Tamaki et al, 1981; Raudzens, 1982; Bunch et al, 1983; Jones et al, 1983). The stability of the peak latencies of the ascending spinal SEP also contrast with the variable stability of the short latency and particularly longer latency components of the cortical SEP (Uemoto and Tolo, 1981; Raudzens, 1982; Bunch et al, 1983; Jones et al, 1983). Furthermore, the spinal SEP waveform would from the results in this study and others, appear to be stable to most anaesthetic agents as well as moderate hypotension (Nordwall et al, 1979; Jones et al, 1983; Lamont et al, 1983; See

Section 9). These features suggest that given satisfactory systemic physiological parameters under anaesthesia changes in spinal SEPs reflect specific spinal cord dysfunction.

The Rationale of intraoperative Spinal SEP Monitoring

Spinal SEP monitoring as a method of intraoperative assessment of spinal cord function relies for its utility upon the hypothesis that (1) changes in spinal SEPs specifically represent spinal cord dysfunction, (2) changes in spinal cord function do not occur independently of changes in spinal SEPs, and (3) changes in spinal SEPs can alert the anaesthetist and surgeon to the possibility of spinal medullary damage, so that manoevres to rectify or minimise this damage may be undertaken. This latter point is particularly important since the particular raison d'etre of intraoperative spinal cord monitoring must be evidence that it decreases the morbidity associated with spinal surgery.

Variations in Intraoperative SEPs

The spinal SEP signal may change by fluctuations in amplitude, variations in peak latencies of components or the loss of waveform components. Changes in the latency of SEP components of greater than 0.48 ms were not seen in this series. The stability of the spinal SEP peak latencies has also been reported by others (Macon et al, 1982a and 1982b; Jones et al, 1983). In contrast variability in the amplitude of the waveform component was common and in five of the twenty-four cases (21%) fully monitored, amplitude fluctuations represented signal attentuation of greater

than 50% of baseline value. None of these cases had loss of waveform components and none of the patients had post-operative neurological deficits. It would appear therefore that transient signal attentuation not associated with loss of waveform components represents electrophysiological variance under operative conditions. It has recently been demonstrated that halothane anesthesia in inspired volumes of 2% or more can attenuate the spinal SEP recorded subdurally by 30-40% (See section 9).

Experience with monitoring spinal SEPs in other centres reflects similar findings. Amplitude variations of greater than 30% from baseline to final SEP recording during orthopaedic surgery for Harrington's procedure occurred in at least 14% of two large series (Jones et al, 1983; Lamont et al, 1983), and Macon and colleagues (1982a) found in a group of patients having spinal SEP monitoring during surgery in which the integrity of the spinal cord was not threatened, variations of up to 40% of baseline value were common. In an experimental study in cats however permanent SEP amplitude attentuation of greater than 70% following spinal compression was commonly associated with paresis in the hind limbs (Nordwall et al, 1979). It would appear therefore that one must be wary of major attenuation of the spinal SEP but that this change alone is not predictive of significant spinal cord dysfunction.

Loss of a waveform component during operative spinal SEP monitoring is a much less frequent occurence than amplitude fluctuation (Tamaki et al, 1981; Macon et al, 1982b; Jones et al,

1983; Lamont et al, 1983). It would appear that loss of a waveform component represents impairment of spinal cord function, either due to a physiological or anatomical insult, and that persistence of the waveform abnormality will correlate with a post operative deficit (Tamaki et al, 1981; Macon et al, 1982b; Jones et al, 1983; Lamont et al, 1983). The experience with both transient and permanent loss of waveform components in five patients in this series supports these conclusions. However all of the three patients who had neurological deficits aggravated by surgery (myelotomy and intramedullary tumor removal) did improve significantly with post operative rehabilitation.

A particular finding in this series was that if components of the SEP were transiently lost, when they returned they had latencies almost identical to baseline values. This feature has previously been noted in an experimental study of spinal SEPs in cats (Nordwall et al, 1979). It would appear therefore that with intraoperative impairment of spinal cord function there is an "all or none" conduction of the ascending volley and prolongation of peak latency does not preceed later loss of a waveform component. These findings, although not specifically mentioned in previous reports would also appear to be substantiated by analysis of the published SEP waveforms in illustrative cases from other studies (Tamaki et al, 1981; Jones et al, 1983; Lamont et al, 1983).

The appearance of a prominent early P wave coincidentally with the loss of waveform subcomponents suggests that this P wave is normally inhibited or modulated by descending spinal tract

activity. Its appearance or augmentation may therefore be an additional sign of spinal axonal dysfunction. This phenomena of augmentation of the segmental SEP below the level of cord transection has previously been described in cats (Morrison et al, 1975). Although the appearance of this P or S (Nordwall et al, 1979) wave was not specifically commented upon, it has previously been shown to appear coincidentally with electrophysiological spinal dysfunction and also to attenuate as the waveform recovered (Jones and colleagues 1983 cases 2 and 3; Lamont and colleagues, 1983 figures 4 and 5). The significance of this wave is however difficult to interpret since in this series, and the cases alluded to above, clinical deficits associated with persistence of this S wave have been variable.Furthermore some patients free of neurological deficits had a prominent P or S wave on their baseline recording (Fig 8.5).

The Utility of Spinal SEP Monitoring

The question of whether spinal monitoring with SEPs makes spinal surgery safer is difficult to answer. In the case of orthopaedic spinal surgery for kyphoscoliosis the reported incidence of neurological insults aquired intraoperatively was less than 1% (Macewan et al, 1975). In the decade since this report, the incidence could be expected to have increased because more powerful instrumentation has led to more radical correction of deformities and selection of patients for surgery who previously would have been considered unsuitable candidates (Taylor T K, personal communication, 1984). Since this type of orthopaedic surgery exposes the cord to an insult that involves mechanical

distortion of the thecal sac and its contents, it would be reasonable to expect that spinal SEP monitoring may be particularly useful in these patients. These cases need some type of functional spinal monitoring intraoperatively and spinal SEP monitoring has many advantages over the "wake up" test (Tamaki et al, 1981; Maccabee et al, 1982; Jones et al, 1983).

The role and potential for spinal SEP monitoring in extramedullary spinal neurological surgery is analagous to that of monitoring during orthopaedic surgery. In such cases as removal of an extramedullary neurofibroma, or a diastematomyelic spur, attentuation of the spinal SEP could indicate inappropriate focal traction or distortion of the spinal cord. In these cases, modification of surgical technique could be appropriately guided by SEP signals.

The utility of spinal SEP monitoring during intradural intramedullary spinal surgery is more difficult to assess because of the diversity of pathological conditions encountered and the variability in each case of the potential threat to spinal cord function. Following myelotomy alterations in spinal SEPs may occur. However although electrophysiological change is indicative of induced cord dysfunction it is not evidence of irreparable damage to the spinal cord since some patients recover remarkably well from surgically acquired spinal deficits.

An electrophysiological analogy between neurones in parts of the brain exposed to blood flow levels below 18ml/100g of brain/min and the function of spinal axons and neurones retracted to

exposed intramedullary lesions may be appropriate. In the former situation the neurones remain viable but are "idling" because the ischaemia restricts synaptic function. Similarly, spinal axons and neurones in the region of a tumour or myelotomy may be viable but because of local environmental factors may not be able to transmit the SEP through a physiological block. Therefore spinal SEP monitoring in its current state has limitations during intramedullary spinal tumour surgery.

The possibility of an anterolateral cord insult occuring independently of a change in spinal SEP is theoretically possible because of the different anatomical localisation and blood supplies of the dorsal columns and pyramidal tracts. This problem has stimulated the development of methods of monitoring motor evoked potentials intraoperatively (Levy et al, 1984). Experimental evidence in cats has indeed suggested that differential damage of these two long tract systems can occur, particularly with induced spinal cord ischaemia. Furthermore, the insult to the anterolateral cord may not be reflected in changes in spinal SEPs (Bennett, 1983). There are anecodotal (Levy, 1983) and well documented case reports (Ginsburg et al, 1985) of several patients having surgery with intraoperative SEP monitoring who post operatively had neurological deficits that had occurred without changes in intraoperative SEPs. These are to date the only reports of this complication in over 430 cases of intraoperative spinal SEP monitoring reported in the literature (Hahn et al, 1981; Tamaki et al, 1981; Maccabee et al, 1982; Macon et al, 1982b; Jones et al, 1983; Lamont et al, 1983; Levy, 1983).

PATIENT	AGE	OPERATION LENGTH	PEAK LATENCY(ms) BASELINE	RANGE	AMPLITUDE(uV) BASELINE	RANGE	COMMENT/ANESTHESIA
1 LP	13 yr	1 hr	P1 = 20.1 N2 = 24.8 P2 = 27.2	-	N2-P2 = 1.6	-	Technical problems with monitoring after 10 minutes. Halothane 0.5-1.5% Mean BP 70 mm Hg
2 PC	12 yr	1 hr	P1 = 11.16(11.04-11.28) N2 = 16.92(16.80-13.16)		N-P1 = 2.0(2.5-3.4) P2-N2 = 3.0(2.8-3.2)		Waveform stable. Halothane 0.5-1.5% Mean BP 55-73 mm Hg
3 GA	11 yr	2 hr	P1 = 12.96(12.96-13.20) N1 = 13.56(13.68-13.92) N2 = 15.36(15.36-15.60)		P1-N1 = 4.0(0-4.0) N1-P2 = 3.0(0-3.4)		Loss of P1-N1-P2 complex with spinal distraction. Recovery with release instrumentation. Halothane to 2%. Prominent S wave at 9.24ms. Mean BP range 60-75 mm Hg.
4 MW	12 yr	11/2 hr	P1 = 15.36(15.12-15.48) N1 = 17.76(17,76-17.88) N2 = 19.68(19.44-19.80)		N-P1 = 1.1(0.5-1.1) P1-N2 = 2.4(1.0-1.9)		Waveform stable. Halothane to 2% Mean BP range 60-67 mm Hg
5 HF	10 yr	11/2 hr	P1 = 12.96(12.84-13.08) N1 = 13.68(13.56-13.92)		N-P1 = 1.2(0.8-1.5) N1-P2 = 1.5(1.4-3.2)		Waveform stable. Halothane to 1.5% Mean BP range 57-100 mm Hg
6 FM	12 yr	11/2 hr	P1 = 13.80(13.80-14.28) N1 = 14.40(14.40-14.76) N2 = 15.84(15.84-16.20)		N-P1 = 1.2(0.5-1.2) N2-P2 = 1.5(0.3-1.3)		Waveform stable. Halothane to 1.5% Mean BP range 60-71 mm Hg

TABLE 8.1 PATIENT AND SEP DATA FOR THE 9 PATIENTS HAVING HARRINGTON INSTRUMENTATION FOR SCOLIOSIS. ALL RECORDINGS WERE EXTRADURAL AND AT THE CERVICOTHORACIC REGION. MEAN BLOOD PRESSURE (BP) REFERS TO LEVEL DURING ROD FIXATION AND FUSION.

PATIENT	AGE	OPERATION LENGTH	PEAK LATENCY(ms) BASELINE RANGE	AMPLITUDE(uV) BASELINE RANGE	COMMENT/ANESTHESIA
7 NM	13 yr	11/2 hr	P1 = 9.96(9.72-9.96) N1 = 13.56(13.20-13.56) N2 = 14.04(13.80-14.16)	P1-N1 = 2.5(1.3-2.5) P2-N2 = 2.5(1.5-3.5)	Waveform stable. Halothane to 1.5% Mean BP range 55-63 mm Hg
8 CO	11 yr	1 hr	P1 = 12.76(12.64-13.00) N1 = 13.32(13.20-13.56) N2 = 14.88(14.76-15.12)	P1-N1 = 1.5(1.0-1.5) P2-N2 = 3.7(3.3-3.7)	Waveform stable. Halothane to 2% Mean BP range 60-73 mm Hg
9 DG	8 yr	1 hr	P1 = 11.28(11.04-11.28) N1 = 11.72(11.64-11.88) N2 = 13.08(12.84-13.08)	P1-N1 = 3.0(2.0-3.7) P2-N2 = 2.0(1.8-3.7)	Waveform stable. Halothane to 1.5% Mean BP range 65-72 mm Hg

TABLE 8.1 (Cont)

PATIENT	AGE	DIAGNOSIS	MYELOGRAM/SPINAL CT	CLINICAL STATUS
1 DG	8 yr	Thoracic diastematomyelia	Thoracic diastematomyelia with focal diplomyelia	Neurologically normal Scoliosis
2 EH	1 yr	Lipomeningocele	Lumbosacral lipomeningocele with low conus medullaris	Neurologically normal. Posterior midline lumbosacral lipoma
3 MH	18 mths	Caudal agenesis syndrome Sacral meningocele	Sacral meningocele. Hemisacral agenesis. Gross rectal dilatation	Chronic constipation. ? Urinary sphincteric dysfunction
4 DL	1 yr	Tethered cord syndrome (Meningocele closed at 1 week)	Dorsal lumbar meningocele with low conus medullaris	Neurologically normal
5 CM	2 yr	Double diastematomyelia, thick filum and tethered cord syndrome	Thoracic and lumbar diastematomyelia, thoracic diplomyelia, thick filum, low conus medullaris. Multiple vertebral abnormalities	Paresis R leg. Extensor plantars. Sphincters and sensation ?normal. Scoliosis. Hypertrichotic skin lumbothoracic region
6 SD	7 mths	Lipomeningocele	Lumbosacral lipomeningocele with low conus medullaris	Neurologically normal. Posterior midline lumbosacral lipoma
7 JB	6 mths	Lipomeningocele	Lumbosacral lipomeningocele with low conus medullaris	Neurologically normal. Posterior midline lumbosacral lipoma

TABLE 8.2 CLINICAL, PATHOLOGICAL AND RADIOLOGICAL DATA FOR THE 17 NEUROSURGICAL PATIENTS HAVING SPINAL SEP MONITORING

PATIENT	AGE	DIAGNOSIS	MYELOGRAM/SPINAL CT	CLINICAL STATUS
14 RS	10 yr	Intramedullary astrocytoma (T10-conus)	Myelographic block caused by widened cord at T10. Conus distorted	Paresis hip flexion; eversion & dorsiflexion foot (R). Absent R knee & ankle jerks. Sensation and sphincters norma
15 HP	58 yr	Ependymoma of the conus and cauda equinae	Myelographic block low thoracic region caused by intramedullary mass	Spastic paraparesis with urinary retention Sensory loss below L2, marked muscle atrophy, power grade 2 (MRC), JPS lost below hips bilaterally
16 IM	3 yr	Lipomeningocele Tethered cord syndrome	Lumbosacral lipomeningocele with low conus medullaris tethered to dorsal lipoma	Bilateral saddle anesthesia. Sensory & motor loss below L3/4 (R) and L5 (L). Overflow urinary incontinence. Large dorsal midline lumbosacral lipoma
17 CW	2 yr	Chiari III syndrome, with syringobulbia and tethered cord syndrome. Hydrocephalus and encephalocele treated as neonate	Low conus medullaris with thick filum. Widened upper cervical cord with syringobulbia. Residual cervico-occipital encephalocele	Multiple cranial nerve palsies, shunted hydrocephalus. Paretic lower limbs despit normal tonus and reflexs. Sensation to pin prick seemed normal in legs

TABLE 8.2 (Cont)

PATIENT	AGE	DIAGNOSIS	MYELOGRAM/SPINAL CT	CLINICAL STATUS
8 VW	14 yr	Intramedullary astrocytoma (T5 -7)	Focal widening of thoracic spinal cord	Scoliosis. Spastic gait, bilateral impairment JPS at hallux. Sphincters normal
9 VS	23 yr	Lipomeningocele	Lumbosacral lipomeningocele with low conus medullaris, lumbar vertebral dysplasia	Muscle atrophy & paralysis/ sensory loss below L4 (R). Impotent. Left leg normal
10 GL	25 yr	Intramedullary neurofibroma (C2-5)	Widening of upper cervical spinal cord	Atrophy, paresis, fasciculation & sensory loss left brachium and hand. Spastic paraparesis legs, bisphincteric incontinence
11 AA	16 yr	Intramedullary hemangioblastoma (von Hippel-Lindau syndrome)	Myelographic block by intramedullary mass at T9. Large draining viens	Bilateral saddle anesthesia. Poor sphincteri function. Muscle atrophy, paresis & sensory loss from L4 caudally (L). Blind from retina hemorrhage from angiomata
12 BW	2 yr	? Diastematomyelia Tethered cord syndrome	? Diastematomyelia, low conus medullaris	Neurologically normal. Sacral dermal sinus, hypertrichotic skin lumbothoracic region
13 SD	8 yr	Extramedullary neurofibroma T11 (R). von Recklinghausen's disease	Extradural mass compressing conus medullaris. Enlarged foramin & paravertebral extension on CT	Neurologically normal except ? R knee jerk hyperreflexia and extensor R plantar

Table 8.2 (Cont)

PATIENT	LEVEL AND DURATION OF SPINAL MONITORING	SURGICAL PROCEDURE	INTRAOPERATIVE SPINAL SEP Complex, Latency, Amplitude	COMMENT	POST-OP STATUS
1 DG	High thoracic 2hr	Laminectomy and excision of bony spur	S-P1,N1,P2,N2 P1-N2 = 1.80 (1.80-1.92)ms P1-N2 = 6.25 (6.0 -0)V	Loss of waveform subcomponent with hypotension. Recovery with transfusion	Unchanged
2 EH	Thoracolumbar 1/2hr	Laminectomy, transection, partial resection lipomeningocele	P1,N1,P2,N2 P1-N2 = 1.24(1.20-1.28)ms P1-N2 = 12.5(7.5-12.5)uV	Waveform stable. Amplitude fluctuation	Unchanged
3 MH	Thoracolumbar 1/2hr	Laminectomy, exploration sacral meningocele	P1,N1,P2,N2 P1,N1 = 1.80(1.70-1.82)ms P1-N1 = 13(11-18)uV	Waveform stable. Amplitude fluctuation	Unchanged
4 DL	Thoracolumbar 1hr	Laminectomy,division thick filum. Excision extramedullary dermoid	P1,N1 P1-N1 = 1.20(0.96-1.20)ms P1-N1 = 7.2(6.0-9.2)uV	Waveform stable	Unchanged
5 CM	High thoracic 1hr	Laminectomy,excision bony spur	P1,N1,P2,N2,P3,N3 P1-N3 = 3.00(2.92-3.12)ms P1-N3 = 7.0(3.0-5.0)uV	Waveform stable	Unchanged
6 SD	Thoracolumbar 1hr	Laminectomy,transection partial resection lipomeningocele	P1,N1,P2,N2 P1-N2 = 1.24(1.18-1.30)ms P1-N2 = 20(19-22)uV	Waveform stable	Unchanged

TABLE 8.3 SURGICAL PROCEDURES, RECORDING SITES, DURATION OF MONITORING AND SEP DATA FOR THE 17 NEUROSURGICAL PATIENTS

7	JB	Thoracolumbar	2hr	Laminectomy, transection partial resection lipomeningocele	S-P1,N1,P2,N2 P1-N2 = 1.50(1.42-1.60)ms P1-N1 = 0.7(0.6-1.3)uV	Latency variation due to electrode movement Interpeak latencies stable	Unchanged
8	VW	Mid-thoracic	1hr	Re-exploration, biopsy intramedullary cystic astrocytoma	Shallow P wave P1 = 14.0 ms N-P1 = 0.3(0.3-0.4)uV	Low amplitude poorly defined SEP	Unchanged
9	VS	Thoracolumbar	1hr	Laminectomy, partial resection of lipomeningocele	P-N1 P = 17.5 N = 18.0ms P-N1 = 2.4uV	Waveform lost during surgery. Cortical SEP stable	Unchanged
10	GL	Cervicomedullary junction	5hr	Re-exploration, excision intramedullary (C2-5) neurofibroma	P1,N1,P2,N2 P1-N2 = 2.24(2.16-2.42)ms P1-N1 = 0.5(0-1.1)uV	Poorly defined SEP signal lost during cord dissection	Poor respiratory and arm function postoperatively
11	AA	Low thoracic region	1hr	Rexploration, drainage of syrinx & biopsy of spinal hemangioblastoma	N1P1 N1-P1 = 2.88(2.56-3.00)ms N1-P1 = 1.25(1.1-2.0)uV	Different waveforms from L & R tibial stimulation. Amplitude increased after aspiration of syrinx	Unchanged
12	BW	Thoracolumbar junction	1/2hr	Laminectomy, excision of bony septum from neural arch, intradural exploration.	P1,N1,P2 P1-N1 = 3.00(3.00-3.24)ms P1-N1 amplitude stimulus dependant (12-35uV)	Classical conus potential.	Unchanged

TABLE 8.3 (Cont)

13 SD	Mid thoracic region	2hr	Laminectomy, excision of intra and extradural neurofibroma	N1,P1,N2,P2,N3 P1-N3 = 2.00(1.80-2.08)ms P1-N2 = 3.0(1.0-3.2)	Stable polyphasic waveform	Unchanged
14 RS	Mid thoracic region	90 min	Laminectomy, excision of intramedullary astrocytoma	P-P1,N P1-N1 = 1.26(1.26-2.08)ms P1-N1 = 3.0(0-3.0)uV	Loss of polyphasic component following myelotomy	Bilateral foot drop, impaired JPS hallux and L2-3 hyperpathia. Areflexic legs.
15 HP	Low thoracic region	2hr	Re-exploration, resection conus & cauda equina ependymoma	N,P,N P1-N2 = 3.00(2.64-3.48)ms P1-N1 = 1.2(0-1.6)uV	Shallow abnormal baseline waveform. Distortion with tumor resection.	Unchanged
16 IM	Thoracolumbar junction	1hr	Laminectomy, partial resection of lipomeningocele	P1,N1,P2,N2 P1-N2 = 0.92(0.92-1.04)ms P1-N1 = 1.0(0.4-1.0)uV	Stable waveform	Unchanged
17 CW	Thoracolumbar junction	45 min	Laminectomy division of thick filum terminale	P1,N1,P2 P1 = 3.96(3.88-4.08)ms N1 = 5.00(4.90-5.16)ms P1-N1 = 10(9-10)uV	Stable waveform	Unchanged. ? More leg movement

TABLE 8.3 (Cont)

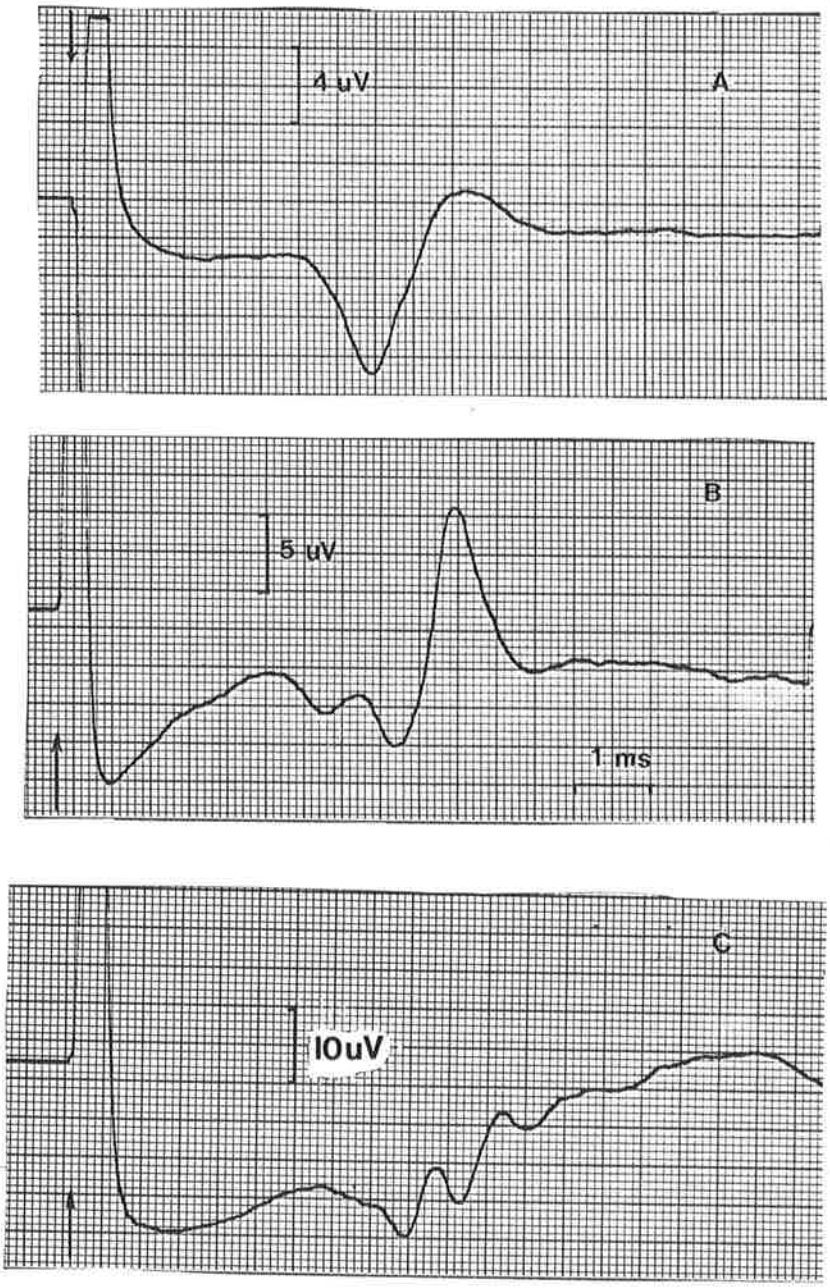
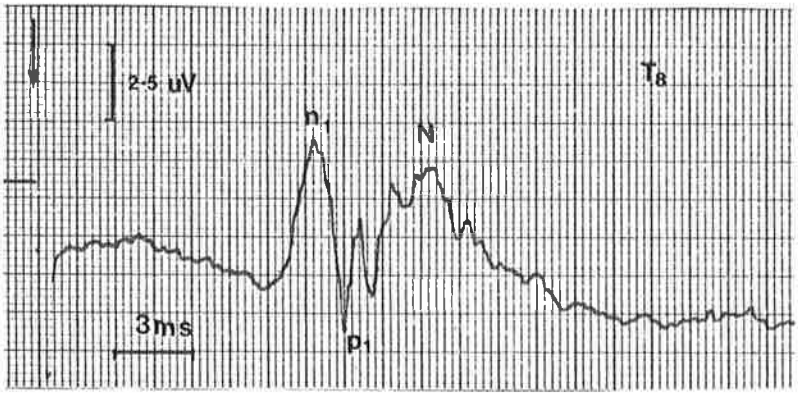


Fig 8.3 Intraoperative recordings of spinal SEPs recorded subdurally from the lumbar region following tibial nerve stimulation in three patients with tethered cord syndromes. Despite differences in caudal spinal pathology all waveforms are of large amplitude and short duration with discrete subcomponents (A = Case 17; B = Case 3; C = Case 6, all patients Group 2).



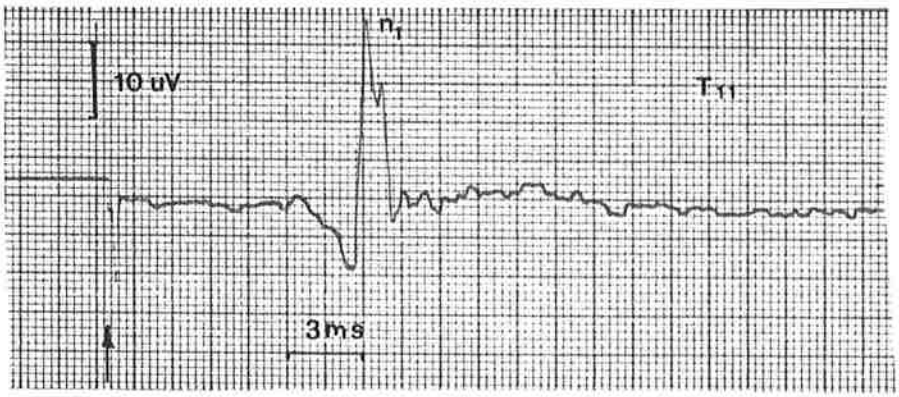


Fig 8.4 Intraoperative spinal SEPs recorded subdurally from the low thoracic region following tibial nerve stimulation. These SEPs were obtained from clinically normal legs in cases 13 (T8) and 14 (T11), Group 2.

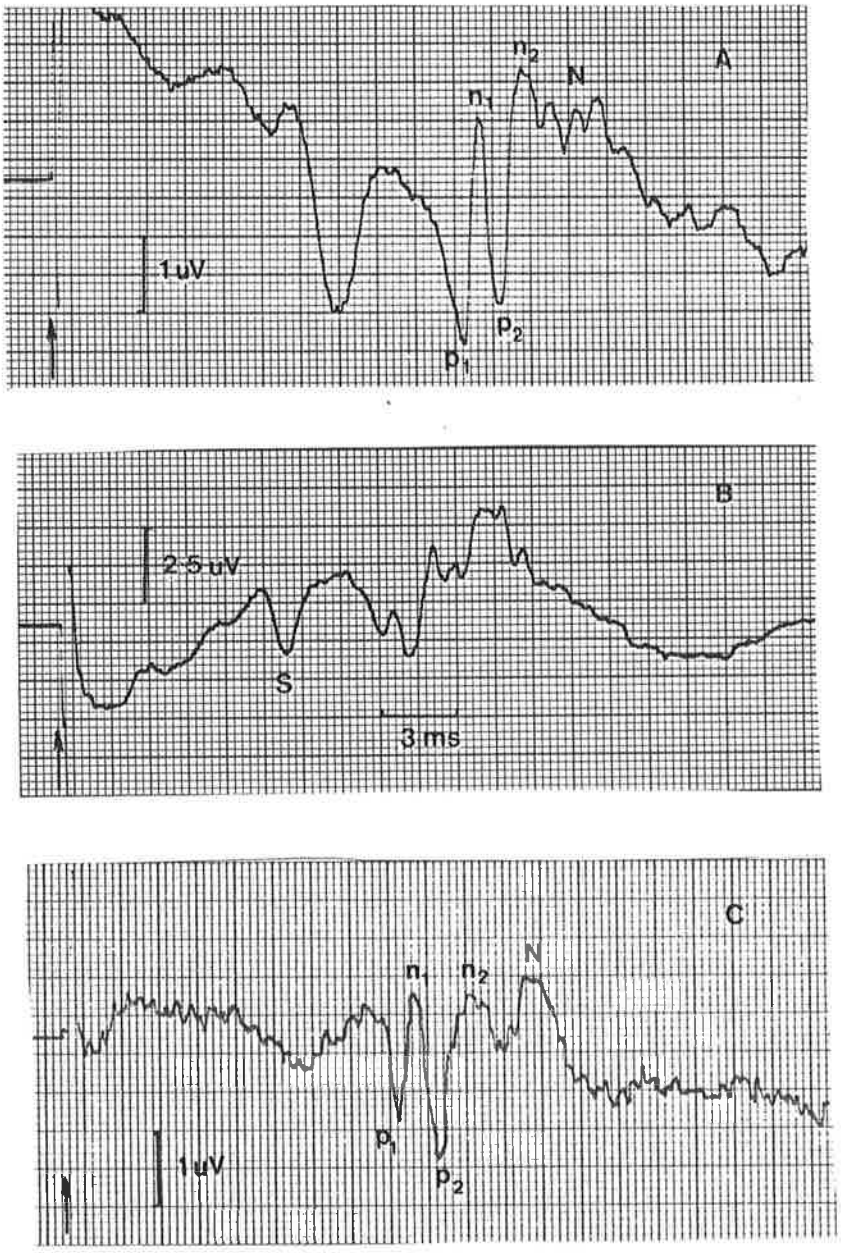


Fig 8.5 Baseline intraoperative spinal SEPs recorded extradurally at the cervicothoracic junction following tibial nerve stimulation in neurolgically normal patients having Harrington's procedure for scoliosis. A prominent S wave is seen in trace A and B. The polyphasic P1-N1-P2-N2 complex followed by later N components are well seen in all patients. (Trace a = Case 2, trace b = Case 8; trace c = Case 5).

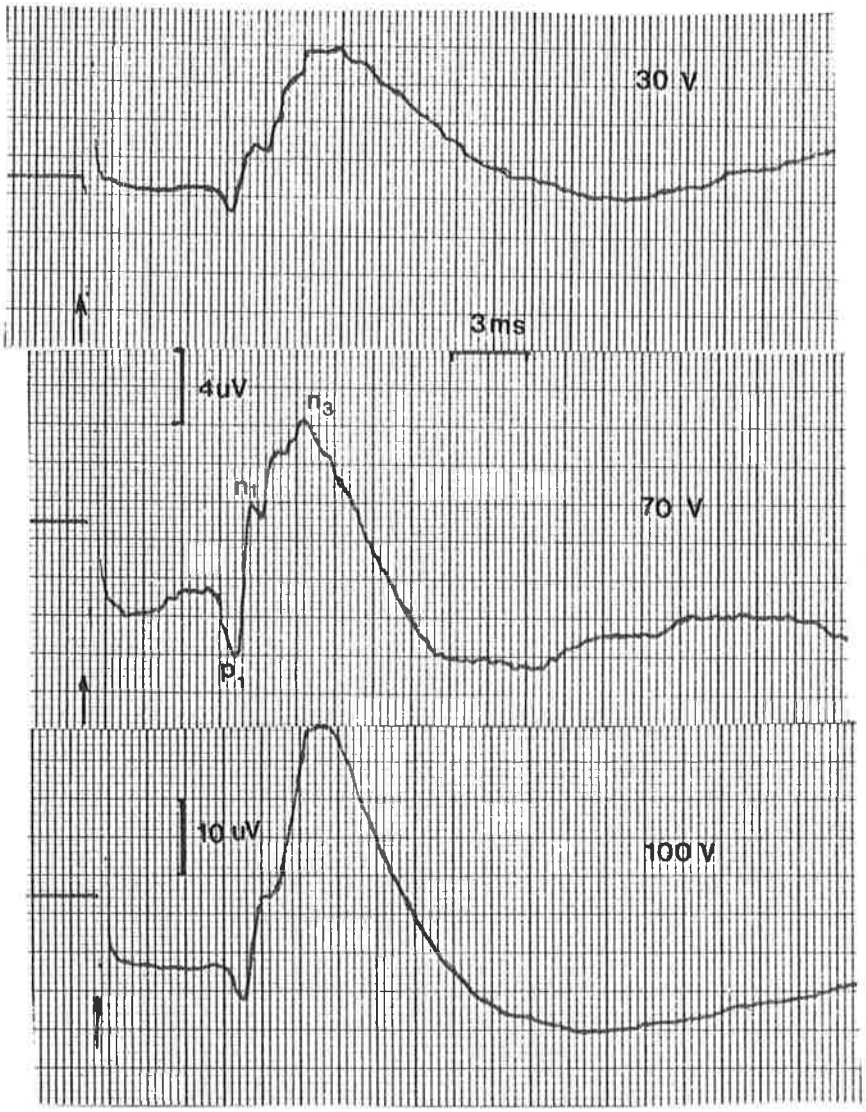


Fig 8.6 Sequential intraoperative spinal SEPs recorded from directly over the conus medullaris following tibial nerve stimulation in a clinically normal child being explored for diastematomyelia. At stimulus 30 V intensity a P-N complex of 8 uV is elicited. At 70 V a 12 uV complex is elicited, and at 100V a 35 uV complex is recorded (Case 12).

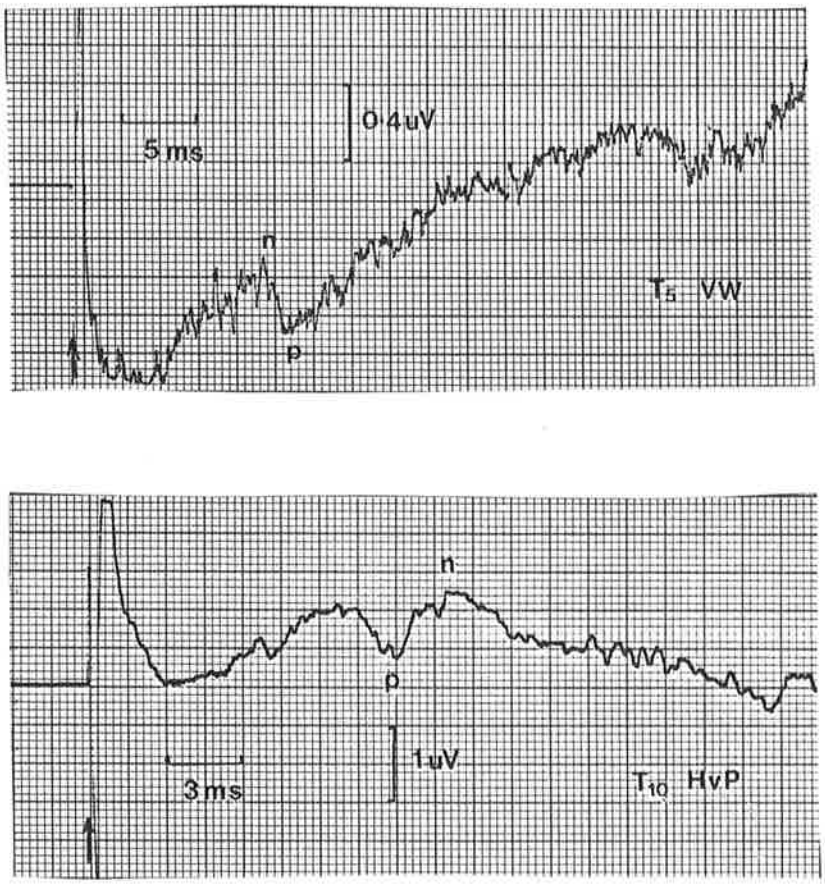


Fig 8.7 Baseline intraoperative spinal SEPs recorded subdurally and rostral to intramedullary neoplasms (Cases 8 [T5] and 15 [T10], Group 2). The signal is composed of a shallow P wave that lacks definition, and an ill defined n component (cf Fig 8.5). Both these patients had major neurological deficits.

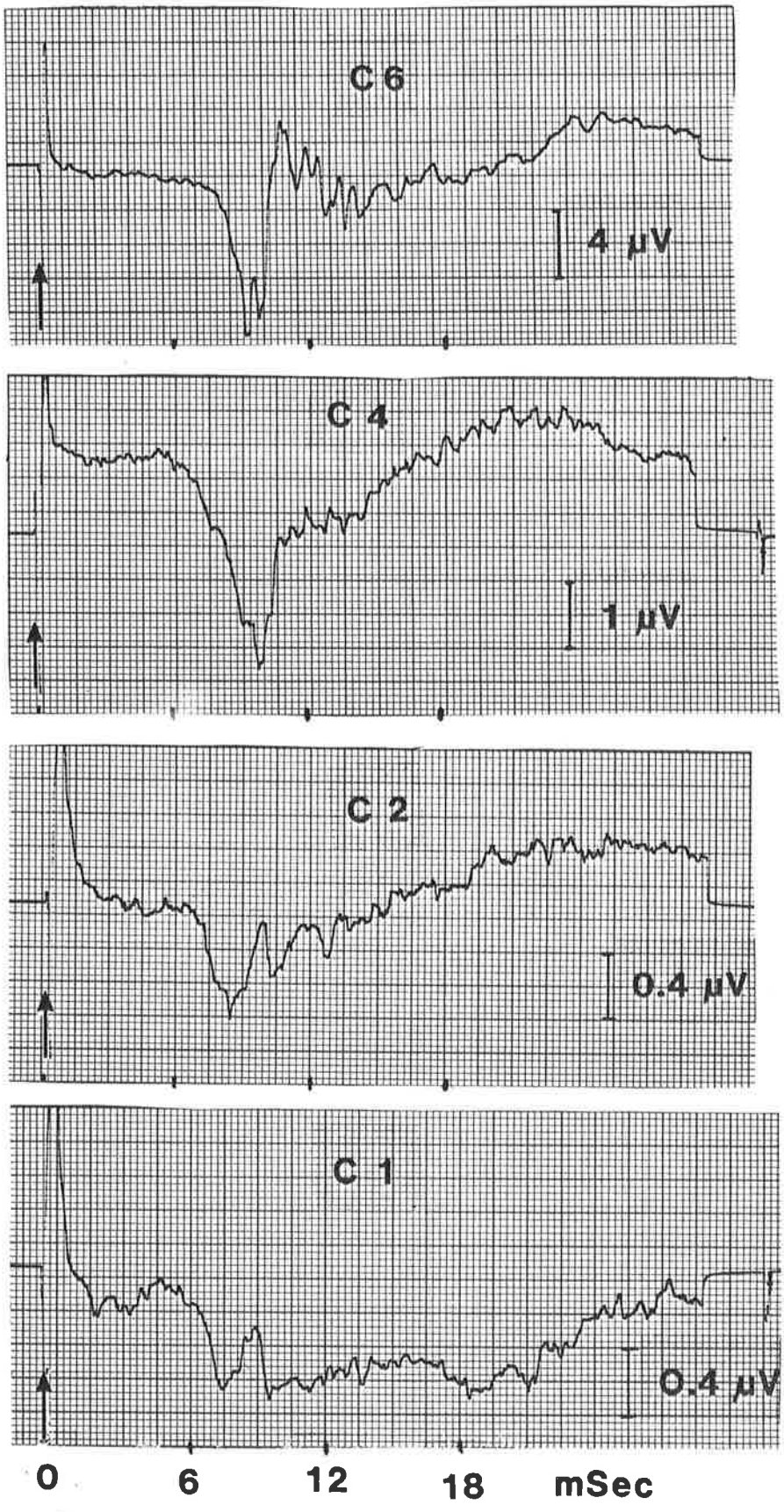


Fig 8.8 Baseline intraoperative spinal SEPs recorded from the different levels of the dorsal surface of the cord following median nerve stimulation in a patient with an intramedullary neurofibroma that extended from C2-C5. There is attenuation of the signal between C6 and C4, and further attentuation and also loss of waveform definition over C2 and C1. The low amplitude and latencies of the waveforms recorded at C1 and C2 suggest these are volume conducted potentials (Case 10, Group 2).

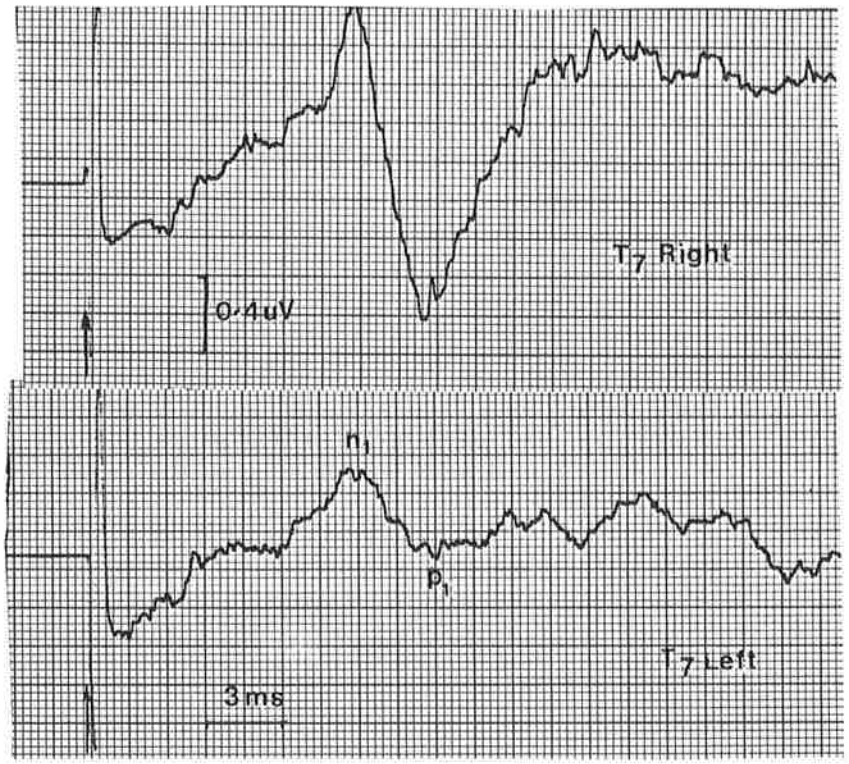


Fig 8.9 Baseline intraoperative spinal SEPs recorded directly from the surface of the cord in a patient with an intramedullary hemangioblastoma. There is marked attentuation and loss of waveform definition following left tibial nerve stimulation (the clinically diseased side) when compared to the response from right tibial nerve stimulation (Case 11, Group 2).

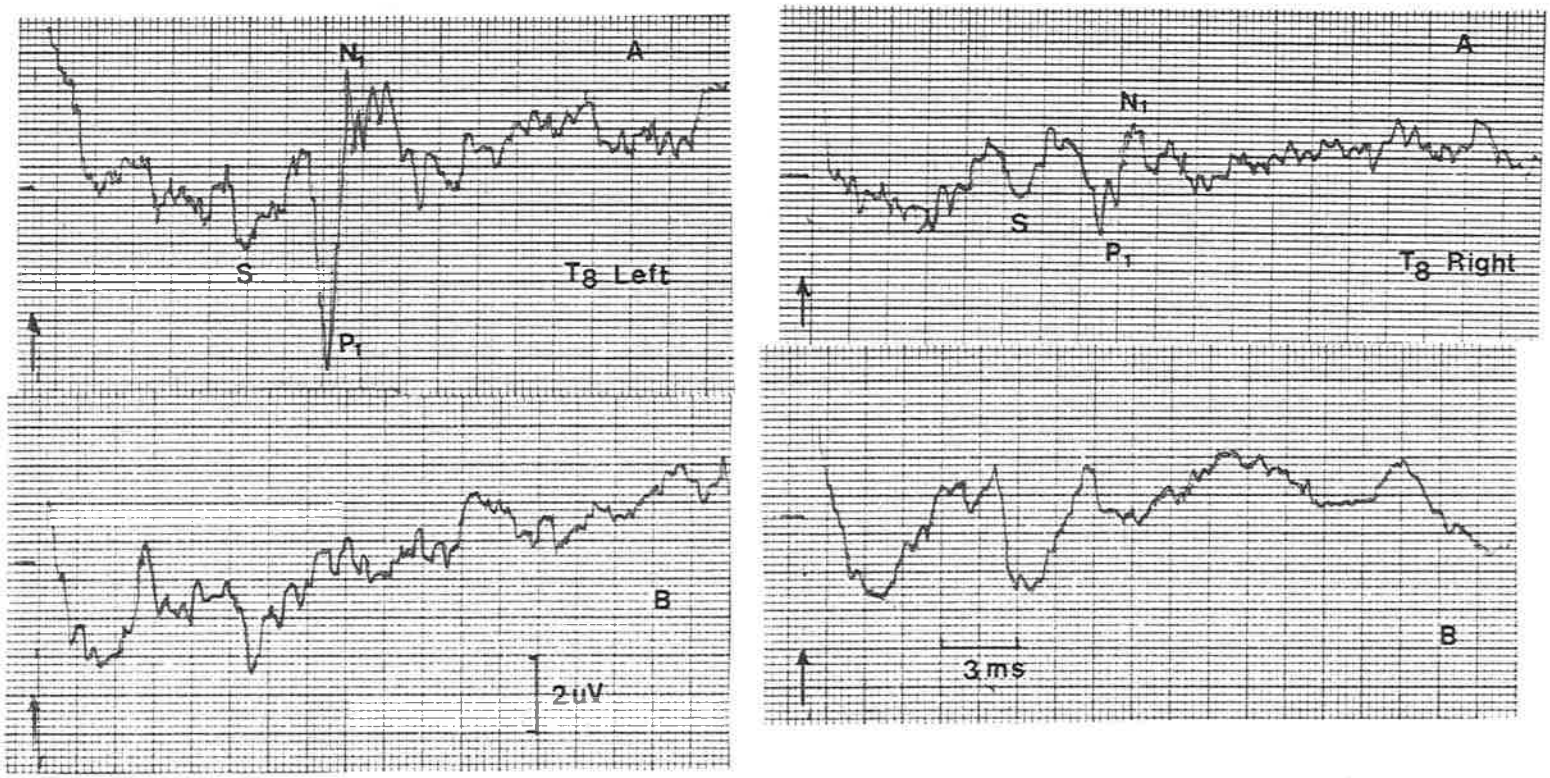


Fig 8.10 Intraoperative spinal SEPs recorded subdurally following right and left tibial nerve stimulation in a patient with an intramedullary astrocytoma involving the conus-T10 region. The baseline recordings are asymmetrical with lower amplitude, poorer waveform definition and an S wave on the neurologically impaired side (right). Following resection of the lesion there is gross distortion of the SEP bilaterally. This patient sustained a major bilateral neurological deficit following surgery (Case 14, Group 2).

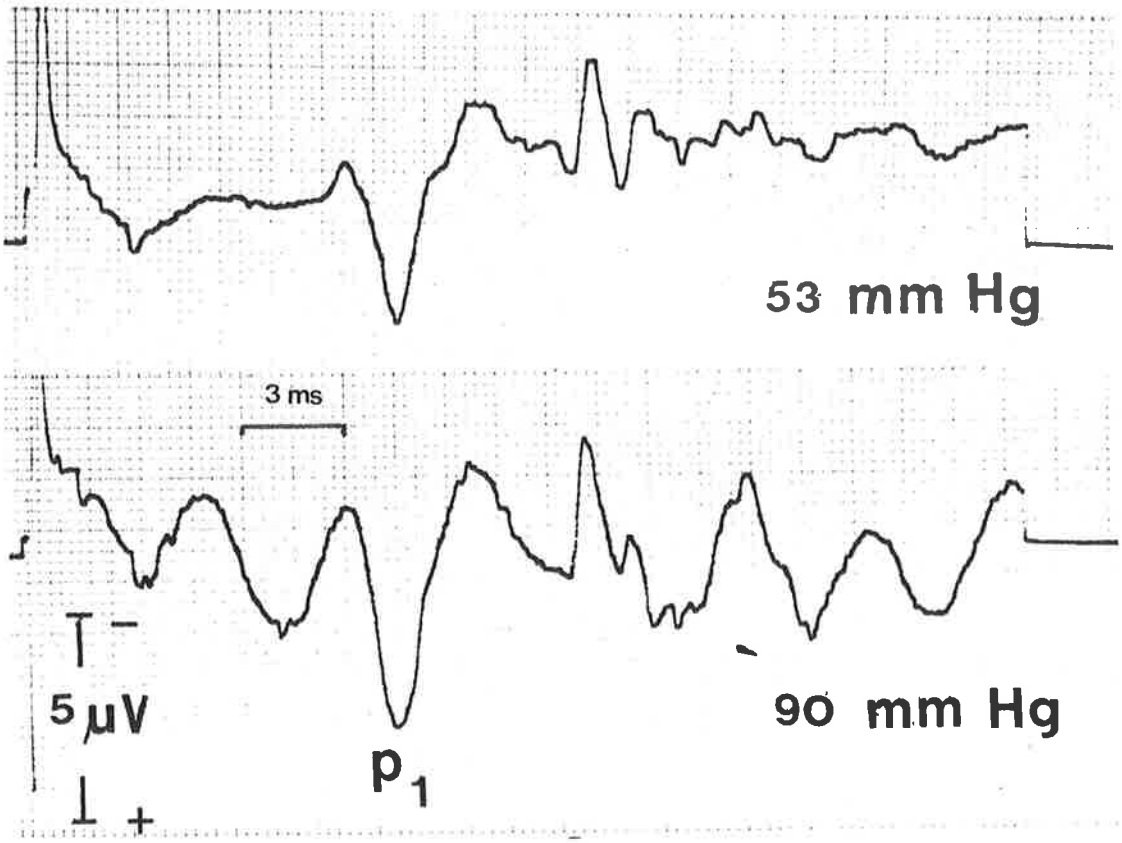


Fig 8.11 Intraoperative SEP waveforms demonstrating the effect of nitroprusside induced hypotension during a Harrington procedure for scoliosis (Case 2). The spinal SEP shows some variation in amplitude of the P1 component however the latency of this and later N components remain identical at mean arterial pressures of 53 and 90 mmHg. There is less amplitude variation in the more slowly conducted N component.

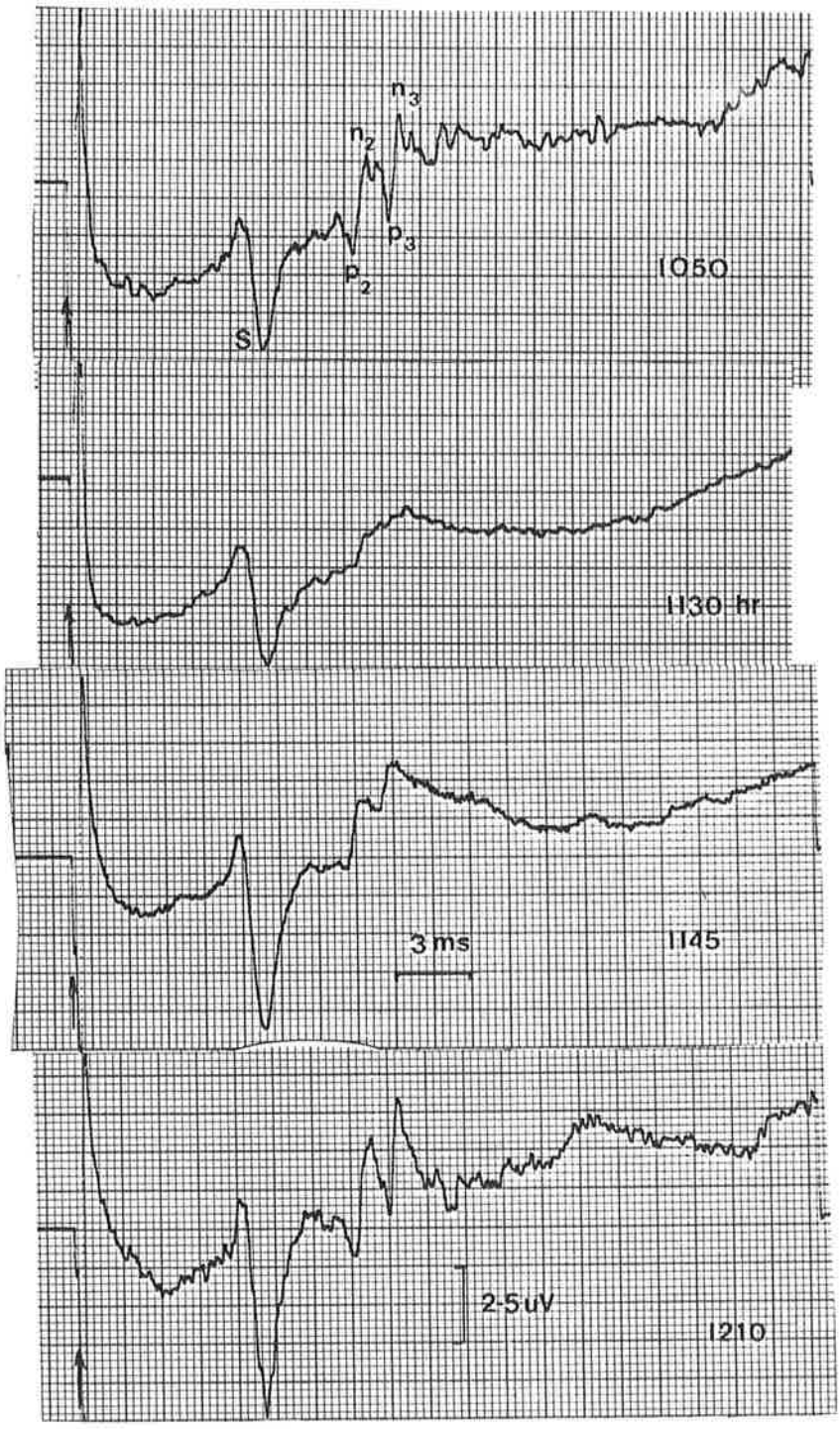


Fig 8.12 Intraoperative spinal SEPs recorded extradurally at the upper thoracic region following tibial nerve stimulation in a patient having a bony septum causing diastematomyelia resected. The baseline signal (1050 hrs) shows an S wave and P2-N2-P3-N3 complex. During resection of the septum 1.5 L blood loss occured over 30 minutes. Blood pressure registered 30-40 mm Hg on an arterial line. At this level of hypotension the P-N-P-N complex is lost (1130 hrs). Over the ensuing 40 minutes this complex returns following transfusion. The patient was neurologically intact following operation (Case 1, Group 2).

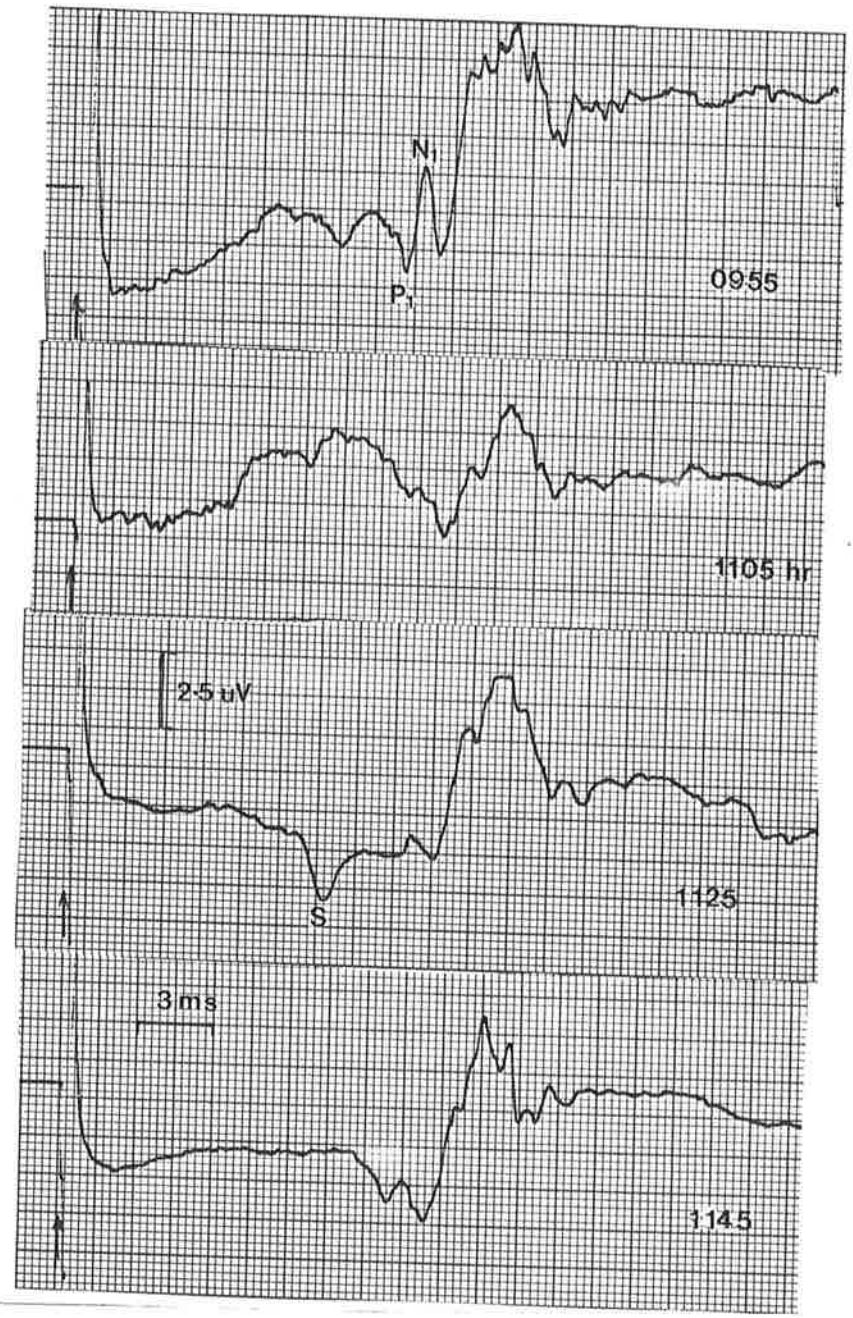


Fig 8.13 Intraoperative spinal SEPs recorded extradurally following tibial nerve stimulation in a patient undergoing a Harrington procedure for scoliosis. The baseline waveform (0955) has a prominent P1-N1 complex. Following maximal vertebral distraction this complex is lost (1105). The instrumentation was released with partial recovery of this complex (1125 & 1145). There was also a prominent S wave during the initial recovery period (1125). The patient was neurologically intact postoperatively (Case 3, Group 1).

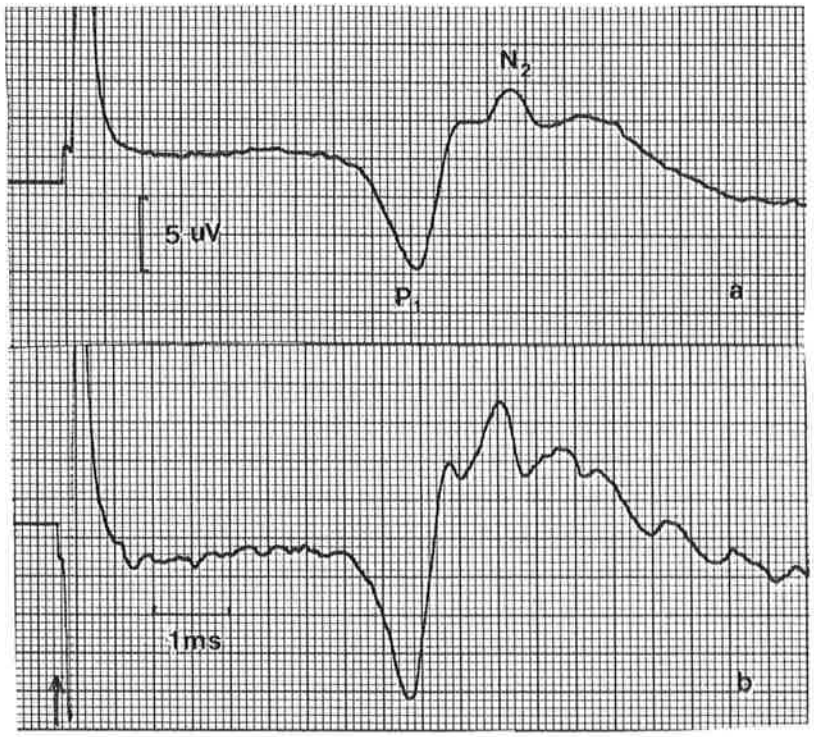


Fig 8.14 Baseline (a) and final (b) intraoperative spinal SEP traces for case 2 (Group 2). There has been augmentation of the P1-N2 amplitude following transection of a lipomeningocele that was causing a tethered cord syndrome. The patient was neurologically normal both before and after operation.

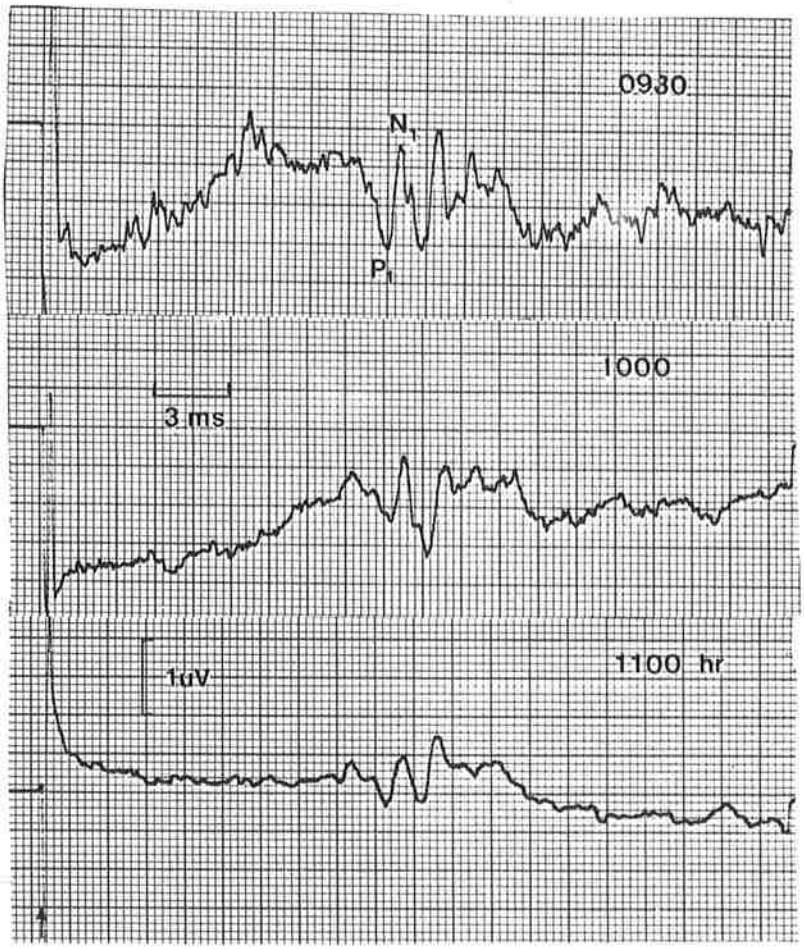


Fig 8.15 Sequential intraoperative spinal SEPs recorded extradurally at the cervicothoracic junction following tibial nerve stimulation in a patient having a Harrington procedure for scoliosis. The P1-N1 waveform is stable throughout the procedure. However baseline (0930) amplitude is 1.4 uV whilst the final (1100) amplitude is 0.7uV. The patient was neurologically intact after operation (Case 5, Group 1).

SECTION 9

THE EFFECT OF HALOTHANE AND STIMULUS INTENSITY ON SPINAL SEPs RECORDED INTRAOPERATIVELY IN SHEEP

AN EXPERIMENTAL STUDY

Clinical and experimental reports have demonstrated that the spinal SEP waveform has been shown to attenuate with spinal cord hypoxia, ischaemia, compression, distraction and cordotomy (Nordwall et al, 1979; Kojima et al, 1979; Schramm et al, 1979; Bennett, 1983; Jones et al, 1983; Schramm et al, 1981; Snyder and Halliday, 1984). There has however been no systematic study on the effect of particular anaesthetic agents on the spinal SEP waveform, except for the brief account of the effect of halothane on the ascending spinal SEP described by Nordwall and colleagues (1979). The effect of halothane on spinal SEPs is important since during procedures such as Harrington instrumentation of the spine, inspired halothane concentration may be titrated to levels of 2.5% to obtain a desired level of therapeutic hypotension. Furthermore it has been shown that halothane attenuates both cortical SEPs and BAERs (Clark and Rosner, 1973; Raudzens, 1982; Worth et al, 1982; Thornton et al, 1984).

This section describes an experimental study that was designed to examine and define the effects of varied inspired concentrations of halothane on the morphology of the ascending spinal SEP. Changes in both peak latency and amplitude of the SEP recorded subdurally at the thoracolumbar junction, were studied. Using

the same experimental model, additional studies were also performed to determine the effect of stimulus intensity on the morphology of the ascending spinal SEP. The relevance of this study to intraoperative monitoring of spinal cord function and its clinical implications are discussed.

METHODS

Seven adult cross-bred sheep were anaesthetised with thiopentone sodium (8-10mg/Kg), intubated with a cuffed orotracheal tube, paralysed with pancuronium bromide (0.1mg/Kg) and ventilated via a modified T-piece circuit with nitrous oxide in 40% oxygen. Central venous access was secured via the external jugular vein and the femoral artery was cannulated to enable continuous monitoring of the mean arterial blood pressure (Telectronics HS12-Medtel). The electrocardiogram was monitored via subcutaneous needle electrodes and the end-tidal carbon dioxide concentration was continuously recorded with an infra-red analyser (Datex carbon dioxide analyser-CD2300). Core temperature was monitored via a rectal thermistor probe.

The sciatic nerve was exposed in the thigh between the hamstring muscles. A laminectomy was then performed at the thoracolumbar junction and the dura opened to expose the spinal cord.

Spinal SEPs were generated by direct electrical stimulation of the sciatic nerve. This was applied through a unit consisting of parallel, curved, blunt metal rods mounted in perspex. Inter-electrode distance was 1.5cm and the cathode was proximal to the

anode. This unit was hooked beneath the segment of exposed sciatic nerve and electric shock stimulation, delivered as a square-wave pulse of 0.1ms duration and 20V amplitude, was applied via a stimulus isolation unit at a rate of 5Hz. Stimulation voltage was constant throughout each experiment.

Spinal SEPs were recorded directly from the spinal cord using a non polarisable platinum electrode (spinal electrode kit ELK-7) referenced to a needle electrode in the adjacent para-spinal muscles. Recording electrodes, and a large earth plate electrode, which was sutured to the thorax, were connected to a pre-amplifier (Medelec PA89), and the signal fed to a signal averaging unit (Medelec MS91). The frequency band was 20Hz to 2 KHz (-6dB rolloff). At least 120 events were averaged over epochs of 30-50ms. Amplifier sensitivity was set at 20 uV, thus signals containing large amplitude interference were automatically rejected. SEP waveforms were reproduced on an oscilloscope where peak latency could be measured using a cursor with a digital readout. All recordings were duplicated and then printed onto heat-sensitive paper.

A baseline SEP waveform was obtained and then halothane introduced into the anaesthetic circuit from a recently calibrated Fluotec Mk2 vapouriser. Inspired halothane concentrations were sequentially increased by 1% each 30 minutes up to 3%. At the end of each 30 minute period, inspired gas samples and end-tidal gas samples were taken for analysis by gas chromatography to confirm that equilibrium of halothane had occurred. Blood pressure was maintained within physiological

limits with an infusion of lactated Ringer's solution and, if necessary, a dopamine infusion. Spinal SEPs were recorded at the end of each 30 minute period. Blood pressure, heart rate, core temperature and end-tidal CO2 concentrations were documented with each SEP recoding.

To assess the stability of spinal SEPs recorded using this experimental model, with time, another sheep was prepared as per protocol but anaesthetic maintained only with nitrous oxide and oxygen. Spinal SEPs were then recorded sequentially over a 90 minute period.

When quantitating the effect of halothane on the amplitude of the spinal SEP, the amplitude of the baseline recording from maximum positive deflection to maximum negative deflection was given an arbitrary value of 1.0 (Fig.9.1). The amplitude of subsequent SEPs obtained with varying levels of inspired halothane were then calculated as a ratio of this baseline value.

Following completion of the study of the effects of halothane anaesthesia on the ascending spinal SEP, further SEPs were recorded in two sheep using stimulus intensities of 20, 35, 60, and 80V. These SEPs were recorded at the site of laminectomy (thoraco-lumbar junction), and both rostral and caudal to this site. The latter recordings were made by advancing the spinal subdural recording electrode 5-6cms in the appropriate direction. At the end of the experiment the sheep were sacrificied with an overdose of barbiturates.

RESULTS

The stability of the spinal SEP recorded using this technique was confirmed by the study of one sheep anaesthetised with nitrous oxide. There was no significant change in waveform peak latency (+0.12msec) or amplitude over the 90 minute period of this study.

A summary of the physiological parameters measured for the seven sheep anaesthetised with halothane is shown in Table 9.1. Gas chromotography confirmed that in all cases the end-tidal Halothane concentration was greater than or equal to 87% of the inspired concentration.

Good amplitude (3-15uV) spinal SEPs were obtained in each of the 7 sheep. The waveform consisted of an initial positive wave, (P1) followed by a broader polyphasic negative segment of approximately 5 ms duration (Figure 9.1). This waveform was very similar in morphology to the ascending spinal SEPs described for dogs, (Snyder and Halliday, 1984), cats (Happel et al, 1975) and humans (Jones et al, 1982). The latency of the P1 wave and early polyphasic components were not altered ($\pm$0.24ms) by inspired halothane concentrations of up to 3% (Figure 9.2).

The amplitude of the waveform with 1% halothane was similar to base-line values (mean ratio 1.03 $\pm$ 0.15) (Figure 9.2). In several cases 1% halothane appeared to augment the SEP amplitude. At inspired halothane concentrations of 2% and 3% there was significant attenuation of the SEP amplitude compared to baseline and 1% values (mean ratios 0.67 $\pm$ 0.09, and 0.62 $\pm$ 0.05 at 2% and

3% halothane respectively). This difference was significant ($p < 0.005$) using the paired t-test (Table 9.2). The difference in amplitude of the waveforms at halothane concentrations of 2% compared to that of 3% did not reach statistical significance.

Increasing the stimulus intensity from 20-80V had little effect on latency ($\pm$ 0.12 ms) of spinal SEPs recorded from the conus to the low thoracic region (Fig 9.3). There was a small increase in the amplitude of P1 of the spinal SEPs recorded from conus and thoraco lumbar regions with increasing stimulus intensity (Table 9.3). This augmentation was not seen in the low thoracic region. The later negative polyphasic component of the spinal SEP did not appear directly related to stimulus intensity at any region (Table 9.4 and Fig 9.3).

DISCUSSION

The successful use of evoked potentials to monitor the integrity of various afferent neural pathways during neurological and orthopaedic surgery, depends upon accurate interpretation of changes in the evoked potential waveform. Since the work of Clark and Rosner (1973), on the effects of anaesthesia on evoked potentials, major advances have occurred in microprocessor based computer technology that have enabled acquisition of evoked potential signals in increasingly adverse recording conditions. These advances have necessitated a re-evaluation of the effect of anaesthetic agents on evoked potential waveform, and indeed a recent study has systematically studied the effects of halothane and enflurane on the BAER (Thornton et al, 1984).

A decrease in spinal SEP amplitude of greater than 10% during cases monitored for scoliosis surgery has been noted in 30-34% of patients (Jones et al, 1983; Lamont et al, 1983). The cause of these random fluctuations is uncertain since they frequently occur independently of mechanical distraction of the vertebral column. Since high levels of Halothane may be used in these cases to facilitate therapeutic hypotension it is possible that some of this attenuation in spinal SEP is induced by anaesthesia or stimulus intensity fluctuation. The latter factor must be considered when saline-soaked felt pad electrodes are used for percutaneous peripheral nerve stimulation during surgery, since with long operative procedures, some of their water content may evaporate, causing attendant changes in both skin and electrode conductivity and resistance

Results from our study indeed suggest that halothane administered at levels of 2% or greater will significantly alter the amplitude of the spinal SEP. The changes in physiological parameters measured in each sheep during the experiments were deemed insufficient to cause this attenuation of spinal SEP. Similarly the experimental model was shown to produce stable amplitude SEPs over the 90 minute period, a finding which has recently been reconfirmed in another study (Hitchon et al, 1984). Peak latencies and the general configuration of the waveform however remain unaltered by inspired halothane concentration of 3%.

The augmentation of the spinal SEPs seen in some cases with 1% halothane may be due to chemical de-afferentation of segmental spinal generators from inhibitory supra-segmental controls.

Presumably higher concentrations of halothane inhibit both supra-segmental and local spinal neuronal activity, causing attenuation of near field signal potentials.

The finding of a reduction of spinal SEP amplitude with inspired halothane concentrations of 2% or greater is at variance with the work of Nordwall and associates (1979). These workers noted no significant change in SEP amplitudes or peak latencies with up to 4% halothane using a feline model. This finding may be due to differences in experimental model and protocol. Recently, Thornton and associates (1984), have shown attenuation of auditory evoked potentials with halothane anaesthesia. Although there are differences in the generation and transmission of BAERs and ascending spinal SEPs, the former having multiple fixed site neural generators, whilst the latter is an ascending axonal volley, it would appear that these potentials react in a similar fashion to the neural depressive effects of halothane.

The conclusions that may be inferred from this study of stimulus intensity on the ascending spinal SEP recorded under anaesthesia are unfortunately limited because of a technical problem and weakness in experimental design. The voltage calibration on the stimulating unit of the MS91 was found to be incorrect when tested. Stimulating voltages thought to be 1, 10, 30 and 50V were in fact 20, 35, 60 and 80V respectively. Therefore, the initial stimulating voltage (20V) was already supra-maximal (Hitchon et al, 1984), and since the animal was paralysed this error was not noticed. Nonetheless, it may be concluded that up to four times the supra-maximal stimulus intensity does not

induce a significant change in SEP waveform. Jones and associates (1982), have shown that the morphology of the ascending spinal SEP is related to stimulus intensity until a maximal amplitude and complete waveform complex is obtained. This finding, and the result of this study would suggest that if the morphology of the spinal SEP changed during a surgical procedure, and this change could not be rectified by increasing stimulus intensity, the cause of the waveform distortion may be related to the surgical procedure or anaesthetic.

SHEEP NO	MEAN BP (kPa)	END-TIDAL CO_2 (kPa)	TEMPERATURE (^{o}C)
1	10.7 - 11.3	5.6 - 5.8	38.0 - 38.5
2	13.3 - 15.9	5.9 - 6.3	37.3 - 37.7
3	11.3 - 13.9	4.6 - 5.5	38.3 - 39.0
4	11.5 - 13.8	5.1 - 5.9	38.1 - 38.3
5	11.6 - 13.9	4.9 - 5.6	38.4 - 38.7
6	12.7 - 15.3	4.3 - 5.1	37.6 - 38.0
7	11.9 - 12.8	4.8 - 6.0	37.1 - 38.0

TABLE 9.1. RANGE OF PHYSIOLOGICAL PARAMETERS RECORDED IN THE SEVEN SHEEP DURING THE EXPERIMENTS

SHEEP NO	INSPIRED HALOTHANE CONCENTRATION 1%	2%	3%
1	1.25	-	-
2	1.11	0.67	0.61
3	1.08	0.79	0.69
4	1.00	0.62	0.62
5	1.00	0.60	0.53
6	0.77	0.59	0.64
7	1.03	0.76	0.66
MEAN $\pm$ SD	**1.03 $\pm$ 0.15**	**0.67 $\pm$ 0.09**	**0.62 $\pm$ 0.05**

TABLE 9.2. AMPLITUDE OF THE SHEEP SPINAL SEP, RELATIVE TO VALUE RECORDED WITH NO HALOTHANE, AT VARIOUS LEVELS OF HALOTHANE ANESTHESIA.

SHEEP NO	RECORDING SITE	STIMULUS INTENSITY (V)			
		20	35	60	80
1	Mid lumbar	16	17	-	19
2	Mid lumbar	11	11	13	14
1	Thoracolumbar	6.5	7.0	8.0	8.0
2	Thoracolumbar	2.6	2.8	3.8	5.6
1	Low thoracic	4.8	4.6	-	4.8
2	Low thoracic	2.1	2.2	2.2	-

TABLE 9.3. AMPLITUDE (uV) OF P1 COMPONENT OF THE SHEEP SPINAL SEP RECORDED SUBDURALLY FOLLOWING SCIATIC NERVE STIMULATION AT VARIOUS INTENSITIES.

SHEEP NO	RECORDING SITE	STIMULUS INTENSITY (V)			
		20	35	60	80
1	Mid lumbar	7.0	-	7.0	7.0
2	Mid lumbar	11	12	11	12
1	Thoracolumbar	3.4	4.0	4.0	2.8
2	Thoracolumbar	7.0	6.0	6.5	6.0
1	Low thoracic	2.3	-	1.8	1.9
2	Low thoracic	3.8	3.5	-	3.5

TABLE 9.4. AMPLITUDE (uV) OF THE NEGATIVITE COMPONENT OF THE SHEEP SPINAL SEP RECORDED SUBDURALLY FOLLOWING SCIATIC NERVE STIMULATION AT VARIOUS INTENSITIES.

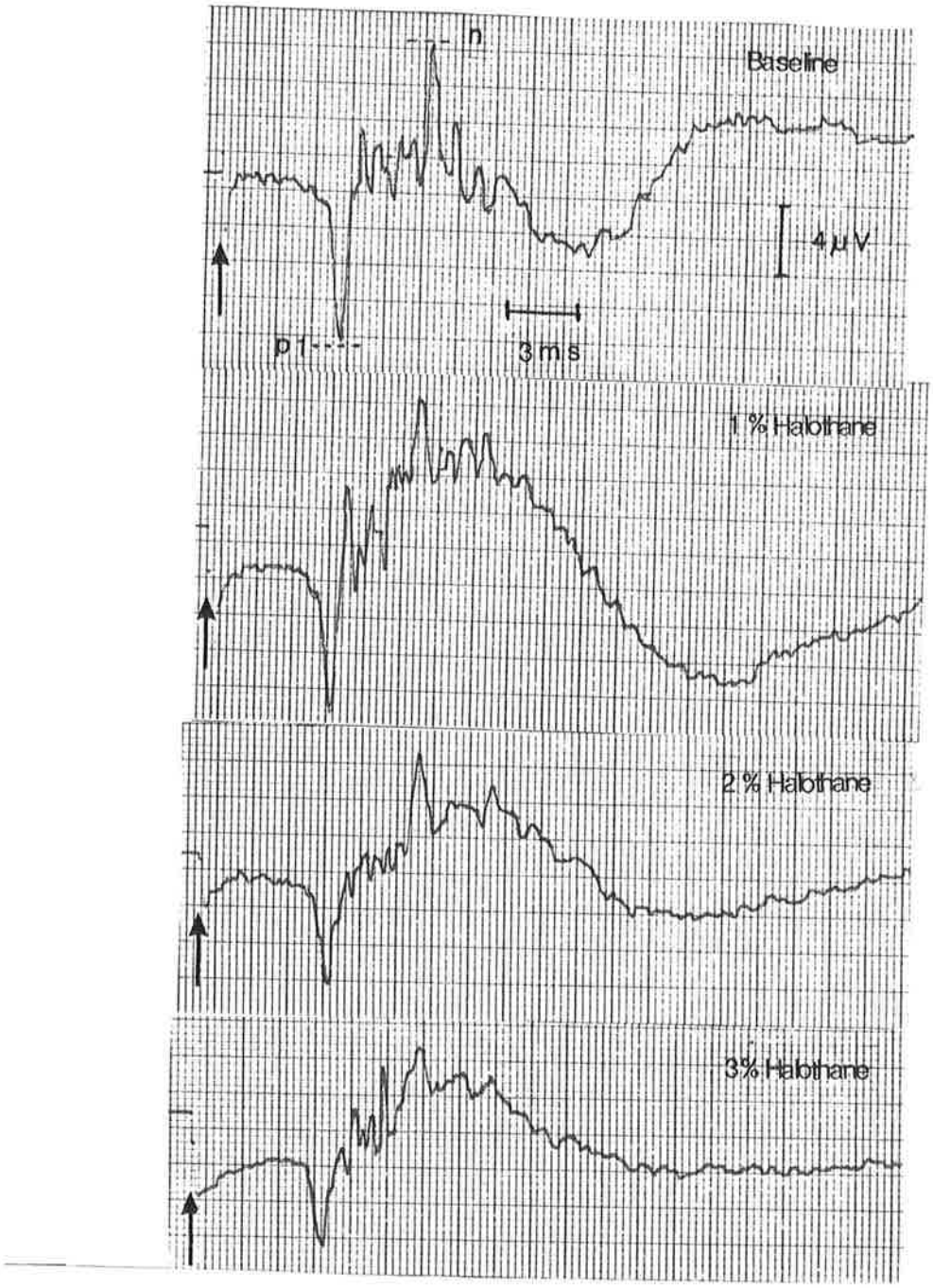


Fig 9.1 The effect of increasing the inspired concentration of halothane on the sheep spinal SEP recorded subdurally at the lumbothoracic junction following sciatic nerve stimulation. The latency of the subcomponent peaks remained stable with up to 3% halothane. However the amplitude of the waveform (measured from P1 trough to n peak) is attenuated by inspired concentrations of halothane greater than 1%.

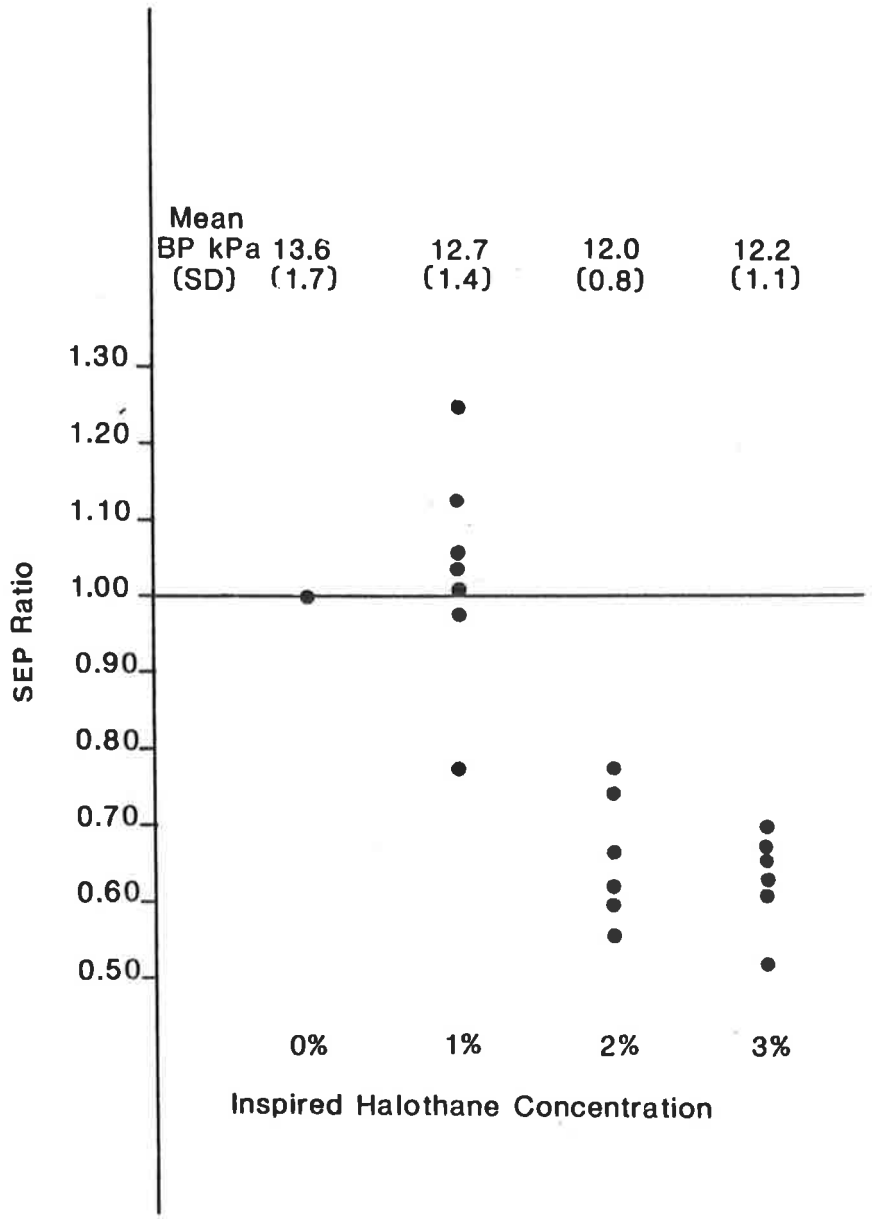


Fig 9.2 The effect of increasing the inspired concentration of halothane on spinal SEP amplitude. The amplitude of the baseline (0% halothane) SEP was given a value of 1.00 in each sheep. Amplitudes of subsequent SEPs, recorded with varying concentrations of halothane, were then expressed as a ratio of baseline value. There was significant attenuation in amplitude with inspired halothane concentrations of 2% and 3%. The mean blood pressure (BP, kPa) at time of recording the SEPs was almost identical at each halothane level.

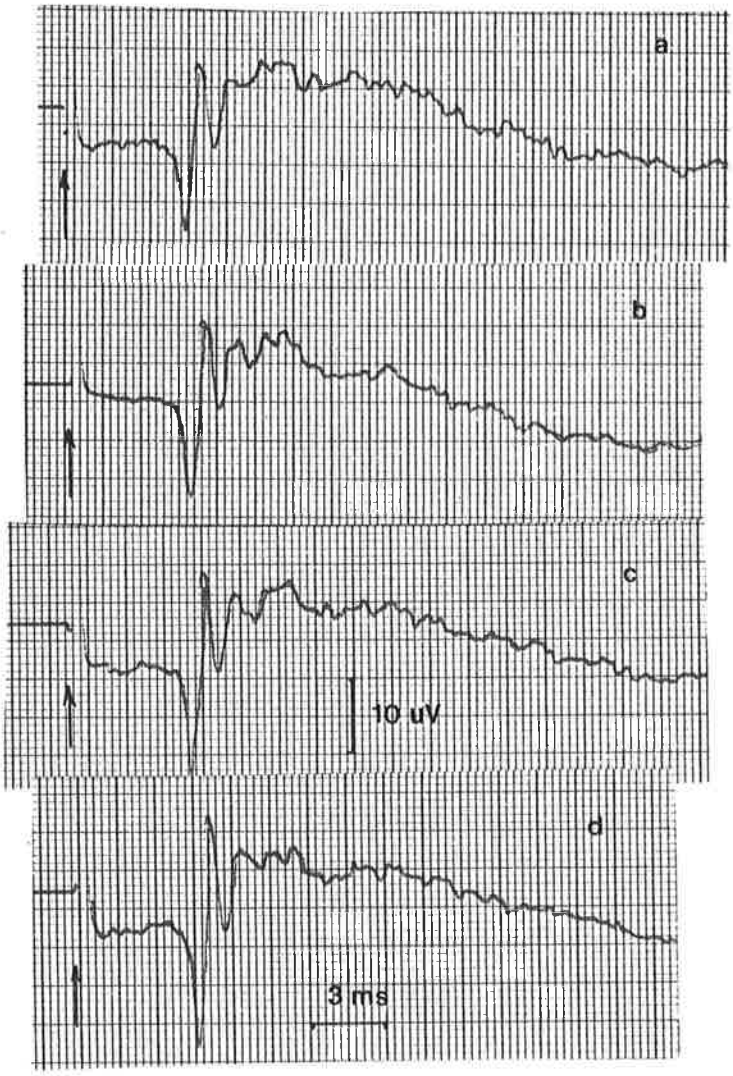


Fig 9.3 The effect of increasing stimulus intensity on the sheep spinal SEP recorded in the lumbar region. The waveform remains stable and there is a small increase in amplitude of P1-N1 with higher stimulus intensity. Trace a, stimulus = 20V, amplitude = 24uV; b, 35V, 24uV; c, 60V, 25uV and d, 80V, 30uV.

SECTION 10

FINAL DISCUSSION AND CONCLUSIONS

This thesis has examined the clinical utility of SEP studies in several areas of pediatric neurosurgical practice. During the period of these studies other publications, many of which have been specifically alluded to in the discussions, have made further contributions as to their diagnostic and clinical utility in different areas of adult and operative neurosurgery (Chabot et al, 1985; Nashold et al, 1985; Yu et al, 1985). Considering the widespread use of SEPs in adult neurology their application to pediatric practice has been slow. Several studies have evaluated SEP findings in degenerative disorders of the central nervous system in children (Cracco et al, 1980), whilst some other tentative studies evaluated cortical SEP findings in neonates and infants with myelodysplasia (Duckworth et al, 1976; Riegel et al, 1976). There has however been no comprehensive study of SEPs in children, althought it would appear that Gilmore and associates at the University of Kentucky have undertaken some studies and are now presenting their findings (Gilmore et al, 1985).

The specific conclusions about SEPs in children that can be made from this thesis are;

1. The morphology of the SEPs in children is similar to that obtained in adults, however the initial components of the cortical SEP following median nerve stimulation do show maturational changes in both interpeak latencies and

morphology. The negative peak latencies recorded over Erb's point (N9 equivalent) and the second cervical vertebra (N13 equivalent) following median nerve stimulation, and over the lumbothoracic junction (N14 equivalent) following tibial nerve stimulation are directly related to patient age and limb length. There is no correlation between age and the latencies of either the initial negativity (N18 equivalent) or the initial positivity (P39 equivalent) of the cortical SEPs following respective median and tibial nerve stimulation. The central somatosensory conduction time (CSCT) decreases slowly during the first decade and attains adult values after eight years of age. The lumbar spine to scalp transit time showed no direct relationship to age. Comparison of SEPs recorded in the same subject when awake and under general anesthesia showed that the latencies of the subcortical, spinal and N1P1 complex of the cortical SEP are identical, however the later components of the cortical SEP vary both in latency and amplitude with anesthesia.

2. In children with neurosurgical disorders of the spinovertebral axis SEP abnormalities were found in 50% of patients. Loss of an SEP at or rostral to the site of spinal pathology was the most common finding whilst prolongation of central conduction times or delay in SEP peak latencies was much less common. Correlations between clinical and electrophysiological findings revealed that all patients with sensory dysfunction had abnormal SEPs, whilst patients with motor or sphincteric dysfunction, but intact sensation, usually had normal SEPs. These relationships between SEP and

clinical findings were constant irrespective of the etiology and location of the spinal pathology. These results suggest that although SEP studies provide information regarding the somatosensory pathways their clinical utility as a method of assessing spinal cord function in children with spinal disorders is limited by technical, clinical and anatomical factors.

3. In children who have suffered from coma for at least six hours after head injury, the majority (70%) will have symmetrical short latency cortical SEPs. Patients with hemorrhagic cerebral contusions and associated cerebral edema will have ipsilateral abnormalities of the cortical SEP. These patients all had normal CSCTs and all made satisfactory neurological recoveries, although the patients with abnormal cortical SEPs had focal neurological deficits. Two patients whose head injuries were complicated by hypoxia and refractory intracranial hypertension had bilaterally abnormal cortical SEPs, and both had vegetative outcomes. There was no correlation between the Glasgow Coma Score (GCS) and CSCT, however the number of components to the cortical SEP was related to the GCS. The results suggest that the recording of SEPs can provide useful information about the functional status of central and cortical somatosensory pathways in children following major head injury.

4. In children with unilateral cerebral lesions involving either directly or indirectly the thalamo-capsular or

perirolandic regions of the brain, patterns of SEP asymmetry include abnormality of the primary cortical complex (N1-P1), loss of intermediate and later waveform components, and attenuation of the response on the side of the lesion. The most abnormal SEPs were seen in patients with major clinical deficits whilst patients with minimal neurological signs had minor SEP changes. The location, etiology and duration of the lesion all appeared to influence the SEP waveform. The value of SEPs studies in the diagnosis and localization of cerebral lesions would seem to be limited because of pathophysiological phenomena related to the primary cerebral lesion.

5. Experience with cortical SEP recording as a method of spinal cord monitoring during spinal neurological surgery revealed that good quality cortical SEPs can be obtained in patients with a variety of intradural and extradural spinal disorders as long as function in the dorsal column mediated sensory modalities is preserved. The short latency components of the cortical SEP were stable during anesthesia with nitrous oxide, 0.5% halothane and fentanyl. However fluctuations in signal amplitude were common. In the one patient in whom the cortical SEP waveform was distorted intraoperatively there was an increased neurological deficit postoperatively. Monitoring spinal cord function using cortical SEPs can provide useful neurophysiological information, however there are limitations to its utility. These relate to difficulties in signal acquisition, the low signal amplitude, attenuation of the signal during

intramedullary surgery and uncertainties in signal interpretation. All these problems are exacerbated if the patient has a preoperative clinical somatosensory deficit.

6. Experience with intradural and epidural recording of spinal SEPs as a method of monitoring spinal cord function during spinal surgery revealed that these techniques provided good quality SEP waveforms in patients both with and without neurological deficits. The SEP configuration and peak latencies remained stable for up to 5 hours during anesthesia with nitrous oxide, halothane and fentanyl. Patterns of baseline spinal SEPs were characteristic of different spinal segments. Distortion and asymmetry of these baseline patterns were seen in several patients with spinal neoplasms. Loss of waveform components during surgery occurred with profound hypotension, overdistraction of the vertebral axis, dorsal midline myelotomy and removal of intramedullary tumours. Persistant loss of waveform components was associated with a surgically acquired neurological deficit. Fluctuations in the amplitude of the spinal SEP were common, but were not associated with postoperative neurological deficits. Experimental studies in sheep suggest that this attenuation is related to the use if halothane anesthesia in concentrations greater than 1%.

Although spinal SEP monitoring of spinal cord function is superior to cortical SEP monitoring and would appear to be particularly useful during extradural surgery, intraoperative SEP recordings are not an infallible guide to

spinal cord integrity since they reflect the functional status of only the dorsal column-medial lemniscus pathway. Abnormalities in the baseline signal, and distortion of the baseline waveform following myelotomy suggest that spinal SEP monitoring will have particular limitations during intramedullary spinal surgery. Disadvantages also arise with SEP monitoring during surgery for the tethered cord syndrome and other caudal spinal abnormalities since SEPs generated by tibial nerve stimulation do not adequately monitor the vital S_2-S_4 radicals.

The results from this thesis also enable several general conclusions to be made. A major factor that will limit the use of outpatient SEP studies relates to problems that may be encountered with acquisition of the SEP data. Patient noncompliance may necessitate several recording sessions to obtain reproducible data. Demands on the time of trained personel and other uses for the electrophysiological equipment may limit the number of studies that can be performed. These factors are of obvious importance when considering intraoperative monitoring since checking and setting up equipment together with actual monitoring time will occupy highly trained personel for the whole day.

Given these considerations one must question whether the cost and effort of SEP data acquisition contributes usefully to patient management. The findings from this work suggest that SEP studies can provide prognostic information following pediatric neurotrauma, and are superior to the use of the "wake up" test

during Harrington instrumentation of the spine. The use of SEPs to evaluate patients with intracerebral lesions and spinovertebral disorders, and to monitor spinal function during intramedullary or caudal spinal surgery would appear limited by technical, anatomical, clinical and pathological factors.

APPENDIX NO 1

Reprint

Whittle, I R., et al.

Spinal cord monitoring during surgery by direct recording of somatosensory evoked potentials. Technical note.

J Neurosurg **60**; 440-443, 1984

Whittle, I. R., Johnston, I. H., & Besser, M. (1984). Spinal cord monitoring during surgery by direct recording of somatosensory evoked potentials. Technical note. *Journal of Neurosurgery, 60*(2), 440-443.

APPENDIX NO 2

Reprint

Whittle, I R., et al.

Intraoperative monitoring of spinal somatosensory evoked potentials during surgery for the tethered cord syndrome.

Riv Neurosc Ped (J Ped Neurosc) **1**; 178-186, 1985

Controllo intraoperatorio dei potenziali evocati somatosensoriali durante la correzione chirurgica del «tethered cord»

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Riassunto

Viene descritta l'utilizzazione di registrazioni dirette di potenziali evocati somatosensoriali (SEPs), ottenuti con stimolazione del nervo tibiale e del nervo peroneo, durante la correzione di una malformazione spinale disrafica in nove casi di «tethered cord» ed in due pazienti con diastematomielia toracica. SEPs di buona qualità, con caratteristiche tipiche dei vari segmenti spinali, al livello dei quali le registrazioni sono state effettuate, sono stati ottenuti in tutti i casi. I SEPs sono rimasti stabili per tutta la durata della procedura chirurgica in tutti gli undici soggetti esaminati, sebbene in un caso sia stata registrata una transitoria attenuazione del segnale a seguito di un'ipovolemia. In nessun paziente è stato osservato un deficit neurologico come risultato dell'intervento chirurgico. Sebbene questo metodo di controllo della funzione spinale abbia i vantaggi della semplicità della tecnica, della rapidità di esecuzione, della facile acquisizione ed interpretazione del segnale e sebbene il segnale stesso rimanga stabile in condizioni di anestesia generale, si devono segnalare alcuni problemi potenziali che possono sorgere durante la sua utilizzazione in caso d'intervento chirurgico a livello spino-caudale. Infatti, a tale livello, il metodo appare limitato per fattori anatomici ed elettrofisiologici, che determinano un controllo inadeguato della funzione delle radici S_2-S_4. Vengono pertanto discusse alcune tecniche di controllo elettrofisiologico, che possono portare ad una valutazione più completa della integrità neurale durante la correzione chirurgica degli stati disrafici lombosacrali.

Parole chiave:
«tethered cord», controllo intraoperatorio, stato disrafico spinale, diastematomielia, potenziali evocati somatosensoriali spinali.

Intraoperative monitoring of spinal somatosensory evoked potentials during surgery for the tethered cord syndrome

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Summary

The use of direct recording of spinal somatosensory evoked potentials (SEPs), following tibial and peroneal nerve stimulation, during surgery for spinal dysraphism is described in 9 cases of the tethered cord syndrome and 2 cases of thoracic diastematomyelia. Good quality SEPs were obtained in all cases. Patterns of SEP were characteristic of the spinal segment at which recordings were made. The SEPs remained stable throughout the surgical procedure in all 11 cases, although in one patient there was transient signal attenuation due to hypovolemia. No patient suffered a neurological deficit as a result of the surgery. Although this method of spinal monitoring has the advantages of simplicity of technique, rapidity and ease of signal acquisition and interpretation, and the signal is stable under general anesthesia there are potential problems with its utilization during caudal spinal surgery. These disadvantages arise from anatomical and electrophysiological factors which lead to inadequate monitoring of the vital S_2-S_4 radicles. Electrophysiological monitoring techniques that may lead to more comprehensive assessment of neural integrity during surgery for lumbosacral dyraphism are discussed.

Key words:
tethered cord syndrome, intraoperative monitoring, spinal dysraphism, diastematomyelia, spinal SEP

Introduzione

La correzione chirurgica delle malformazioni spinali quali la fissazione abnormente bassa del cono midollare, con stiramento del midollo spinale («tethered cord») e la diastematomielia, è accompagnata da un rischio potenziale di danno della funzione neurologica a seguito dello stesso atto operatorio.

Per minimizzare tale rischio di danno iatrogeno sono stati utilizzati per il controllo della funzione spinale i potenziali evocati somatosensoriali (SEPs) (8,11), l'elettromiografia anale (5) o la manometria evocata anale (4,10). Ogni singolo caso, tuttavia, richiede una valutazione specifica circa la scelta più appropriata della tecnica di controllo da utilizzare, a causa della variabilità della patologia spinale e del differente livelo della malformazione congenita. Notevoli informazioni circa la funzione neurale possono essere ottenute con registrazioni elettrofisiologiche intraoperatorie; tuttavia, l'atto chirurgico può essere reso relativamente più sicuro solo da una tecnica di controllo che permetta di verificare la funzione neurologica a livello del campo operatorio stesso.

Questo studio descrive la nostra esperienza in 11 pazienti con malformazione spinale congenita in cui è stato applicato un controllo diretto intraoperatorio della funzione neurologica, utilizzando SEPs spinali.

I vantaggi e i limiti di questo metodo vengono discussi, insieme alle possibili ulteriori applicazioni dei potenziali evocati spinali, soprattutto in relazione alla correzione chirurgica di malformazioni spinali complesse della regione lombosacrale.

Materiale clinico

Sono stati studiati 11 pazienti, con una varietà di malformazioni congenite spinali. Le età, le sindromi cliniche ed i risultati degli esami mielografici relativi a tali pazienti sono riassunti nella tabella 1.

La maggior parte dei soggetti era portatrice di uno stato disrafico spinale associato ad una serie di lesioni intradurali della regione lombosacrale.

Otto pazienti (di cui uno con una doppia diastematomielia toracica e lombare) presentavano una sindrome da «tethered cord», uno una sindrome da agenesia caudale, uno una diastematomielia toracica con scoliosi di grave entità ed uno ancora un cono midollare «basso» con una sospetta diastematomielia lombare e malformazione di Chiari, tipo III (Fig. 1).

Sei soggetti (Casi n. 1, 2, 4, 6, 7 e 9) erano neurologicamente indenni. Due pazienti con lipomielomeningocele presentavano deficit asimmetrici a carico degli arti inferiori (paresi, areflessia, disestesie ed ulcerazioni trofiche), associati ad incontinenza ed impotenza. Il paziente (Caso n. 5) con sindrome da agenesia caudale era normale neurologicamente ma era afflitto da una grave costipazione. Il paziente (Caso n. 3) con diastematomielia e sindrome da «tethered cord», dovuta ad un filum terminale ispessito, presentava una scoliosis major, paresi ed accorciamento di un arto inferiore, mentre il paziente (Caso n. 11) con malformazione di Chiari tipo III mostrava una serie di deficit neurologici in relazione ad un meningoencefalocele cervico-occipitale e siringobulbia. Nella tabella 1 vengono riportate le procedure chirurgiche utilizzate nei vari pazienti. Lo scopo dell'intervento, a seconda dei casi, era di liberare il midollo spinale, fissato in una posizione anormale, per un ancoramento basso del cono o per la presenza

Introduction

Surgery for congenital malformations of the spinal axis such as the tethered cord syndrome and diastematomyelia is associated with hazards that may potentially impair neurological function. Spinal monitoring using spinal somatosensory evoked potentials (SEPs) (8,11), anal electromyography (5) or evoked anal manometry (4,10) has been utilised in an attempt to minimise iatrogenic morbidity. Because of the variability in the spinal pathology and the level of the congenital malformation each case requires particular consideration as to the optimal method of spinal monitoring. Much information about neural functioning can be obtained by intraoperative electrophysiological recordings, however, surgery is only made safer by a monitoring technique that audits neural functioning in the surgical field.

This study describes the experience with direct intraoperative monitoring using spinal SEPs in 11 patients with congenital spinal malformations. The utility and limitation of this method are discussed together with further possible applications of spinal evoked potentials that are particularly relevant during surgery for complex spinal malformations in the lumbosacral region.

Clinical material

Eleven patients with a variety of congenital spinal malformations provide the cohort for this study. The ages, clinical syndromes and myelographic findings in these patients are summarised in Table 1. Most patients had spinal dysraphism associated with a multiplicity of intradural pathology in the lumbosacral region. Eight patients had tethered cord syndromes, one the caudal agenesis syndrome, one thoracic diastematomyelia and a major scoliosis and one patient a low conus medullaris with a suspected lumbar diastematomyelia and Chiari type III malformation (Fig. 1).

Six patients (Cases 1,2,4,6,7 and 9) were neurologically normal. The two patients with lipomyelomeningoceles had asymmetric neurological deficits in the lower limbs (paresis, areflexia, dysesthesiae and trophic ulceration) associated with incontinence and impotence. The patient (Case 5) with the caudal agenesis syndrome was neurologically normal but suffered from severe constipation. The patient (Case 3) with diastematomyelia and tethered cord syndrome due to a thick filum had a major scoliosis, shortening and paresis of one leg, whilst the patient (Case 11) with the Chiari III syndrome had a multiplicity of neurological deficits due to the cervico-occipital meningoencephalocele and syringobulbia.

The surgical procedures undertaken in the patients are summarised in Table 1. These operations were undertaken to untether the conus medullaris or lower spinal cord, to free a transfixed spinal cord and reduce the mass effect of subcutaneous lipomata. In most patients the indication for the operation was to prevent damage to neural tissue during growth spurts, however in two patients (Cases 3 and 8) it was aimed at arresting documented neurological deterioration. One patient (Case 7) had resection of an upper thoracic diastematomyelia prior to placement of Harrington instrumentation and spinal fusion. Lyophilized dural grafts were used when primary dural closure could not be attained. All operations were performed using microsurgical technique.

Tabella 1

Diagnosi, età, reperti radiologici e descrizione dell'intervento nei pazienti con stato disrafico nei quali è stato effettuato il controllo intraoperatorio dei SEPs spinali

Paziente	Età	Diagnosi	Mielografia	Intervento
1	6/12	Lipomeningocele con T.C.S.	Lipomeningocele dorsale lombosacrale contiguo ad un cono midollare basso	Transezione, resezione parziale del lipoma, plastica durale
2	7/12	Lipomeningocele con T.C.S.	Lipona intraspinale adiacente al midollo spinale lombare	Transezione e parziale resezione del lipoma, plastica durale
3	2	Stato disrafico spinale complesso con T.C.S.	Filum terminale ispessito con cono «basso». Diastematomielia toracica e lombare	Asportazione di una spina ossea toracica (diastematomielia), plastica durale
4	1	Meningocele e T.C.S.	Cono abnormente «basso», ancorato dorsalmente	Sezione delle aderenze aracnoidee, escissione di una cisti epidermoide intradurale
5	18/12	Agenesia caudale. Meningocele sacrale	Meningocele sacrale. Agenesia dell'emisacro	Esplorazione del meningocele sacrale
6	12/12	Lipomeningocele con T.C.S.	Lipomeningocele lombosacrale, aderente al midollo lombare	Transezione, resezione parziale del lipoma, plastica durale
7	8	Scoliosis major	Diastematomielia toracica	Asportazione della spina ossea
8	23	Lipomielomeningocele	Lipoma intraspinale aderente al midollo lombare, grande sacco durale lombosacrale con meningocele dorsale	Transezione ed asportazione parziale del lipoma
9	2	? T.C.S.	Cono midollare abnormemente basso, diastematomielia lombare	Esplorazione della regione lombare spinale (intraspinale)
10	3	Lipomielomeningocele con T.C.S.	Lipoma intraspinale aderente al midollo lombare, meningocele dorsolombare	Transezione parziale e resezione del lipoma, plastica durale
11	2	Malformazione di Chiari tipo III (Siringobulbia, encefalocele occipito-cervicale, idrocefalo) e T.C.S.	Filum terminale ispessito con cono espanso	Resezione del filum terminale ispessito

T.C.S.: Sindrome del «Tethered Cord».

di una spina ossea, o di ridurre l'effetto massa di un lipoma sottocutaneo. Nella maggior parte dei pazienti l'indicazione chirurgica s'identificava nella prevenzione del danno neurale che si può verificare durante le fasi di rapida crescita dell'organismo; tuttavia, in due soggetti (Casi n. 3 e 8) l'intervento era diretto principalmente ad arrestare un deterioramento neurologico già documentato. In un paziente (Caso n. 7) era stata asportata una spina ossea che

Methods

Intraoperative spinal SEPs were recorded directly from the dorsal surface of the spinal cord using the technique and parameters previously described by Whittle and colleagues (11). This method requires placement, by the surgeon, of a small platinum tipped recording electrode in the subdural plane rostral to the site of surgery. A reference needle elec-

Table 1

Diagnosis, age, radiographic findings and operative description in patients with spinal dysraphism in whom intraoperative spinal SEP monitoring was performed

Patient	*Age*	*Diagnosis*	*Myelogram*	*Operation*
1	*6/12*	*Lipomeningocele with T.C.S.*	*Dorsal lumbosacral lipomeningocele contiguous with low cord*	*Transection, partial resection of lipoma, dural graft*
2	*7/12*	*Lipomeningocele with T.C.S.*	*Intraspinal lipoma contiguos with lumbar cord*	*Transection and partial resection of lipoma, dural graft*
3	*2*	*Complex spinal dysraphism with T.C.S.*	*Thick filum with low conus, thoracic and lumbar diastematomyelia*	*Resection of thoracic bone spur, duroplasty*
4	*1*	*Meningocele and T.C.S.*	*Low conus tethered dorsally*	*Division of aracnoidal adhesions, excision of intradural epidermoid cyst*
5	*18/12*	*Caudal agenesis. Sacral meningocele*	*Sacral meningocele, hemisacral agenesis*	*Exploration of sacral meningocele*
6	*12/12*	*Lipomeningocele with T.C.S.*	*Lumbosacral lipomeningocele contiguous with lumbar cord*	*Transection, partial resection of lipoma, dural graft*
7	*8*	*Major Scoliosis*	*Thoracic diastematomyelia*	*Resection of bone spur*
8	*23*	*Lipomyelomeningocele with T.C.S.*	*Intraspinal lipoma contiguous with lumbar cord, capacious lumbo-sacral thecal sac with dorsal meningocele*	*Partial transection and resection of lipoma*
9	*2*	*? T.C.S.*	*Low conus medullaris, lumbar diastematomyelia*	*Exploration lumbar spinal cord*
10	*3*	*Lipomyelomeningocele with T.C.S.*	*Intraspinal lipoma contiguos with lumbar cord, dorsal lumbar meningocele*	*Partial transection and resection of lipoma, dural graft*
11	*2*	*Chiari III Syndrome (Syringobulbia, occipitocervical encephalocele, hydrocephalus) and T.C.S.*	*Thick filum, low expanded conus*	*Resection of thick filum*

T.C.S.: Tethered Cord Syndrome

determinava una diastematomielia a livello toracico superiore, prima dell'applicazione delle barre di Harrington e di una fusione spinale. Una plastica durale, con l'uso di innesti di dura liofilizzata, è stata eseguita quando non era possibile ottenere una chiusura durale primaria. Tutti gli interventi sono stati eseguiti con tecnica microchirurgica.

trode is placed in adjacent paraspinal muscle (Fig. 2). Spinal SEPs are generated by electrical stimulation of the tibial and peroneal nerves in the popliteal fossa. The stimulation unit consisted of saline soaked felt pad electrodes positioned in a saddle shaped plastic mount. This unit is taped over the popliteal fossa with waterproof strapping to prevent dehydration of the electrodes.

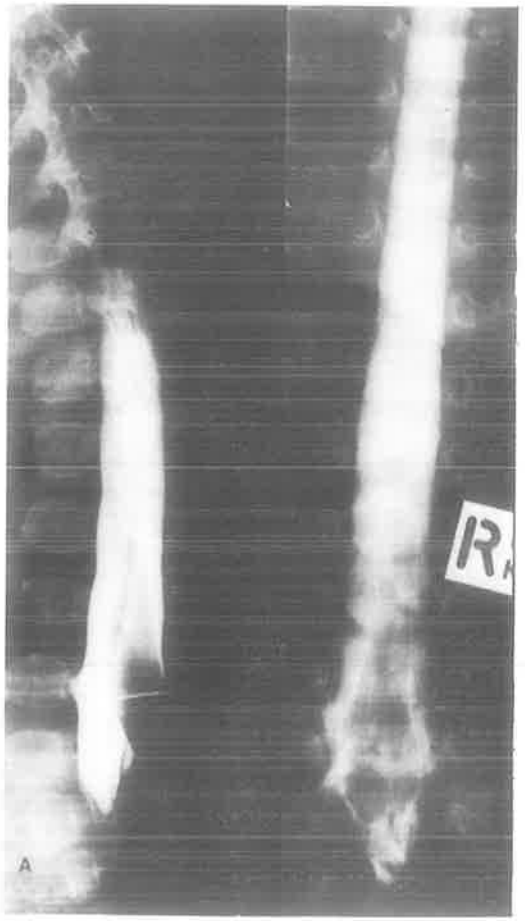

Figura 1. Mielografia con metrizamide dei Casi n. 5 (A) e n. 4 (B). Sono ben evidenti il leptomielolipoma (A) e l'ancoramento dorsale con stiramento del midollo spinale a livello della riparazione del meningocele sacrale (B). Nonostante le differenze della patologia spinale caudale, i SEPs lombari spinali sono molto simili a quelli mostrati nella Figura 3.

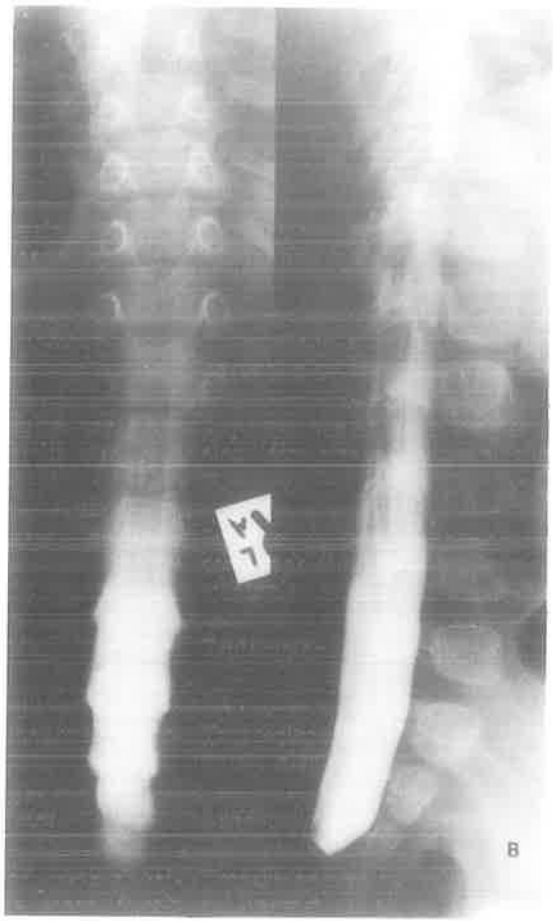

Figure 1 Metrizamide myelograms of Case 5 (A) and Case 4 (B). The leptomyelolipoma (A) and dorsal tethering of the spinal cord at site of closure of a sacral meningocele (B) are well demonstrated. Despite the variability of caudal spinal pathology the lumbar spinal SEPs were very similar as is shown in Figure 3.

Metodi

I SEPs intraoperatori spinali sono stati registrati direttamente dalla superficie dorsale del midollo spinale utilizzando le tecniche ed i parametri descritti in precedenza da Whittle e coll. (11). Il metodo richiede l'applicazione da parte del chirurgo di un piccolo elettrodo registrante a punta di platino nel piano subdurale, in una localizzazione rostrale rispetto al campo operatorio.

Un elettrodo ad ago di riferimento viene situato nel muscolo paraspinale adiacente (Fig. 2) I SEPs spinali sono generati con la stimolazione elettrica dei nervi tibiale e peroneo nella fossa poplitea. Come unità di stimolazione sono stati utilizzati degli elettrodi con tamponi imbevuti di soluzione fisiologica mantenuti in posizione nella fossa poplitea da un contenitore in plastica a forma di sella, applicato con un rivestimento a prova d'acqua per prevenire la deidratazione degli elettrodi stessi.

Il segnale, raccolto con un preamplificatore (Medelec PA 89), viene quindi mediato attraverso un'unità di averaging (Medelec MS 91), con una banda di frequenza stabilita tra i 20 ed i 2000 Hz.

Signals are fed through a preamplifier (Medelec PA 89) and averaged on a Medelec MS 91 Unit, with the frequency bandpass set at 20-2000 Hz. A baseline spinal SEP is recorded prior to any manipulation of neural structures. Because of the large amplitude (10-40 μV) of the spinal SEP in the lumbar region a reproducible waveform can be obtained after averaging as few as 30 stimuli. The SEP waveform appears on the oscilloscope screen as it is being averaged, and because of the superimposition facility available on the MS 91 it may be directly compared to the stored baseline signal. Traces can be copied onto heat sensitive paper as desired. These records have the timebase, amplitude, number of stimuli averaged and frequency bandpass automatically encoded.

Results

Good quality SEPs were obtained in all cases and there were no complications due to spinal monitoring. In 9 patients the level of spinal SEP recording was in the upper

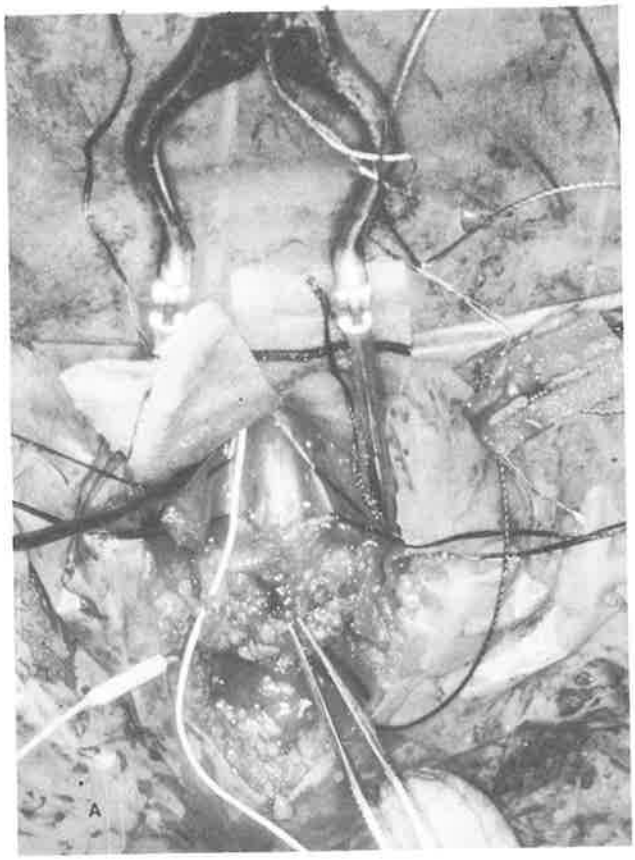

Figura 2. Riprese fotografiche intraoperatorie che dimostrano il posizionamento degli elettrodi di registrazione spinale e di riferimento paraspinale. Il leptomielolipoma, adiacente al midollo lombare ancorato, è ben evidente in A (Caso n. 1), mentre l'emimidollo, stirato attorno ad un setto osseo (S), è mostrato in B (Caso n. 3).

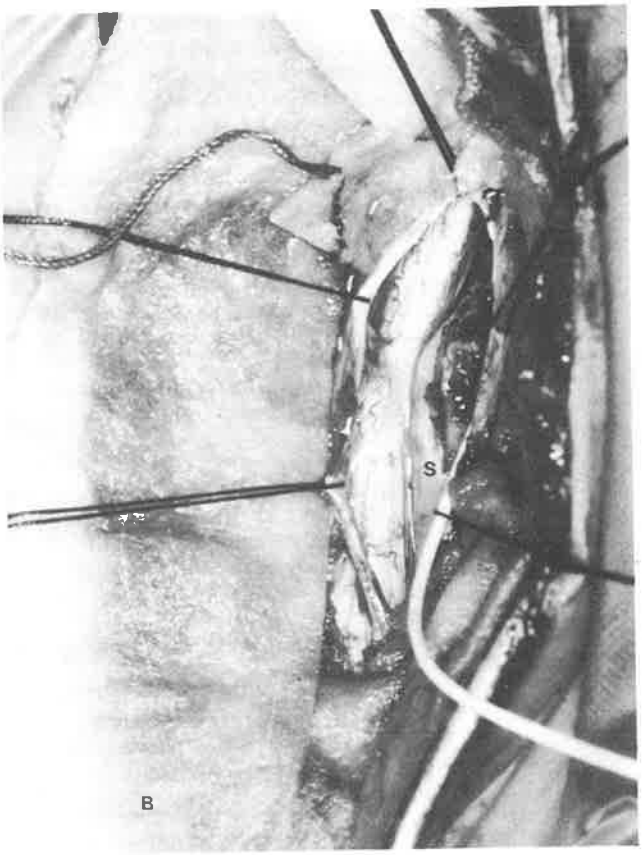

Figure 2 Intraoperative photographs demonstrating placement of spinal recording and paraspinal reference electrodes. The leptomyelolipoma contiguous with the tethered lumbar cord is well shown in A (Case 1), whilst the hemicord stretched around a bony septum (S) is shown in B (Case 3).

Un SEP spinale di base viene registrato prima di qualsiasi manipolazione delle strutture nervose. A causa della grande ampiezza (10-40μV) dei SEPs spinali nella regione lombare, un'onda riproducibile può essere ottenuta mediando solo 30 risposte allo stimolo. L'onda del SEP appare sullo schermo dell'oscilloscopio dopo il processo di mediazione e può essere direttamente paragonata al segnale di base registrato, utilizzando le possibilità di superimposizione dei due segnali offerte dal MS 91. Le immagini, qualora lo si desideri, possono essere infine trasferite su carta sensibile al calore. I segnali registrati riportano automaticamente i codici relativi alla base di tempo, all'ampiezza, al numero di stimoli mediati ed alla frequenza di banda.

Risultati

SEPs di buona qualità sono stati ottenuti in tutti i casi, senza alcuna complicazione. In 9 pazienti il livello della registrazione dei SEPs spinali era la regione lombare superiore, mentre nei rimanenti due soggetti la giunzione cervicotoracica. I SEP spinali a questi livelli differivano in ampiezza, latenza e complessità della forma dell'onda.

lumbar region, whilst in two it was from the cervicothoracic junction. The pattern of spinal SEP at these levels was different in amplitude, latency and complexity of waveform.

The SEP waveforms recorded from the lumbar region were all of short latency (4-9 ms) with between two and five components (Fig. 3). Except for one patient (Case 1) these spinal SEPs were of large amplitude (8-30 μV), and had a prominent initial positivity (P1 component) that marked the onset of the waveform. The pattern of the waveform between P1 and maximal negativity (N) varied from a small inflection (Fig. 3b) to an additional N-P complex (Fig. 3a, c).

The SEP waveforms recorded from the cervicothoracic junction were polyphasic, with a large early P component (Fig. 4). The latency between the early P and polyphasic component increased with spinal length. The amplitude of the waveforms was between 2-4 μV and averaging of at least 100 stimuli was required to produce a reproducible waveform.

The spinal SEPs had latencies that were stable to within +0.36 ms under general anesthesia using curariform

La forma d'onda dei SEPs registrati dalla regione lombare era caratterizzata sempre da una latenza breve (4-9 msec.) e da 2 a 5 componenti (Fig. 3). Ad eccezione di un paziente (Caso n. 1), questi SEPs spinali presentavano una grande ampiezza (8-30 μV) ed una componente iniziale positiva (P1) molto evidente che marcava l'inizio della risposta. Il pattern dell'onda tra P1 e la massima negatività (N) variava da una deflessione di bassa ampiezza (Fig. 3b) ad un complesso addizionale N-P (Fig. 3a, c).

Le forme d'onda dei SEPs registrati dalla giunzione cervico-toracica erano polifasiche, con una componente iniziale P precoce di grande ampiezza (Fig. 4). La latenza tra l'iniziale P e la componente polifasica aumentava con la lunghezza del segmento spinale. L'ampiezza delle forme d'onda era di 2-4 μV; una media di almeno 100 risposte allo stimolo era richiesta per ottenere una forma d'onda riproducibile.

In condizioni di anestesia generale, con rilassanti muscolari curarosimili, fentanyl (0,003 mg/kg), alotano (fino a 1,5% del volume inspirato) e ossido d'azoto al 70%, i SEPs spinali avevano latenze stabili, con margini di 0,36 msec. In un paziente è stata osservata una scomparsa di componenti della forma d'onda a seguito di un'ipotensione marcata (pressione ematica media 30-40 mmHg); tuttavia, la risposta ritornava nei limiti di base dopo trasfusione.

In tutti gli altri soggetti, la forma d'onda del SEP restava stabile per tutta la procedura chirurgica, nonostante le manipolazioni chirurgiche delle strutture nervose caudali. In nessun caso è stata osservata la comparsa di un deficit neurologico come risultato del trattamento chirurgico.

Discussione

Il controllo elettrofisiologico peroperatorio della funzione neurale dovrebbe rendere l'atto operatorio più sicuro, senza complicare o prolungare eccessivamente la procedura chirurgica. I rischi specifici associati alla chirurgia degli stati disrafici spinali complessi della regione lombosacrale s'identificano nella possibilità di danneggiare il cono midollare stirato ed ancorato in basso e le radici spinali lombosacrali durante le manovre di asportazione di un lipoma intraspinale e di sezione di adesioni aracnoidee o di un filum terminale ispessito. Il riconoscimento delle strutture nervose e dei piani di dissezione durante tali procedure può essere infatti assai difficile e le radici S_2-S_4, che innervano i muscoli anali, vescicali e del pavimento pelvico ancora funzionanti appaiono particolarmente a rischio.

La situazione anatomica della maggior parte delle anomalie intradurali associate ad uno stato disrafico spinale caudale o ad una sindrome da «tethered cord» suggerisce che la valutazione dei SEPs spinali generati dalla stimolazione dei nervi tibiale e peroneo costituisce un metodo sub-ottimale per stabilire l'integrità delle strutture nervose durante la correzione chirurgica di queste malformazioni. Il segmento più caudale che contribuisce ad uno o l'altro dei due nervi è S_3 (3), ma il contributo probabilmente è solo una piccola frazione delle fibre afferenti contenute in questi nervi. Ne deriva che strutture neurali distali possono essere irreparabilmente danneggiate senza modificazioni apparenti della forma d'onda del SEP spinale. Un altro fattore che limita il valore del controllo della funzione neurale spinale con SEPs generati dalla stimolazione dei nervi tibiale e peroneo è che il segnale nella regione lombare (Fig. 3) è essenzialmente un potenziale del «cono»

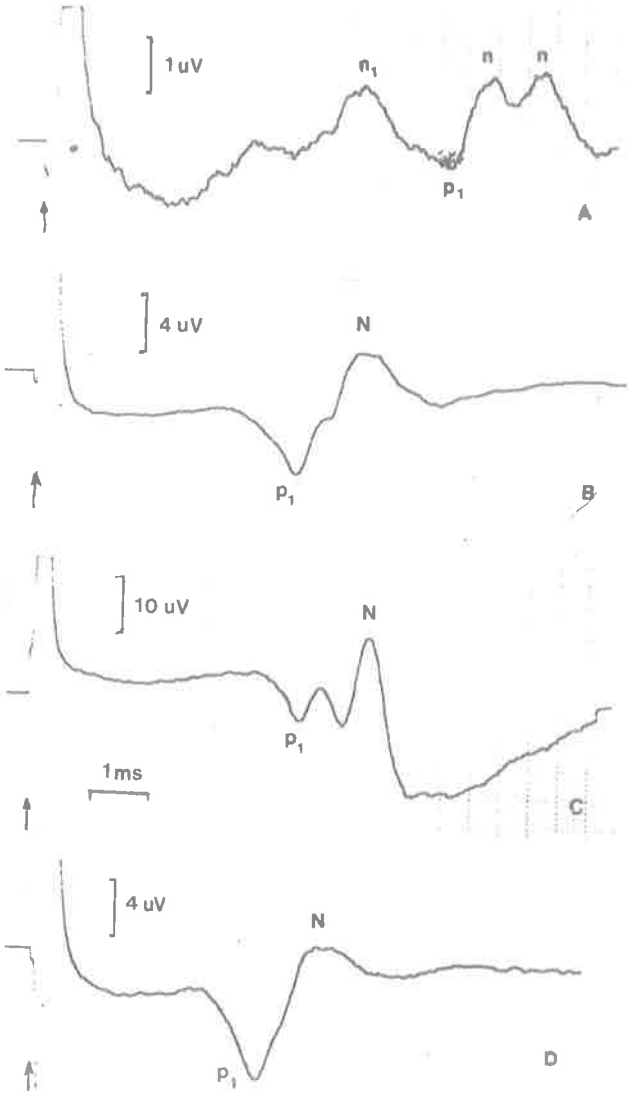

Figura 3. Registrazioni intraoperatorie di base dei SEPs spinali in diversi soggetti (Caso n. 1, A; n. 4, B; n. 11, D). Queste registrazioni a livello della regione lombare superiore dimostrano la grande ampiezza e la latenza breve della forma d'onda che rappresenta il potenziale di «cono».

Figure 3 Intraoperative baseline spinal SEP records from Cases 1 (A), 4 (B), 5 (C) and 11 (D). These recordings from the upper lumbar region demonstrate the large amplitude, short latency waveform that represents the «conus» potential.

muscle relaxant, fentanyl (0.003 mg/kg), halothane (up to 1.5% inspired volume) and 70% nitrous oxide. In one patient there was loss of waveform components with significant hypotension (mean blood pressure 30-40 mmHg), however, following transfusion the waveform returned to baseline pattern. In all the other patients the SEP waveform

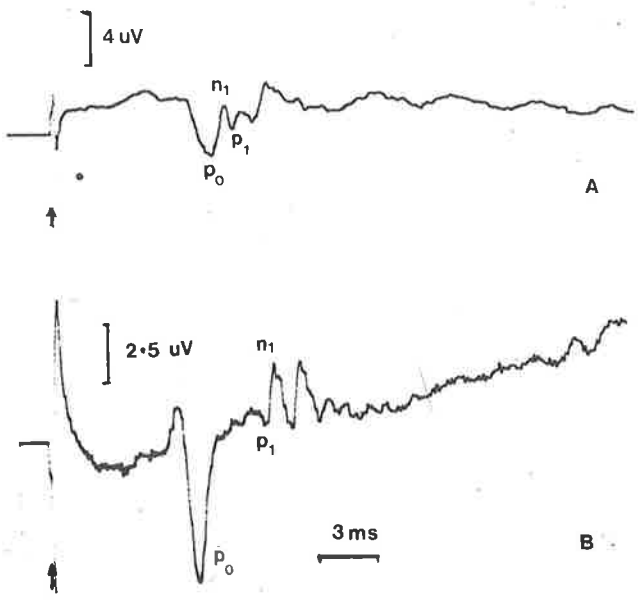

Figura 4. Registrazioni intraoperatorie di base dei SEPs spinali della regione toracica (Caso n. 3, A e Caso n. 7, B). È ben evidente la natura polifasica delle registrazioni che seguono P1 N1. La positività precoce Po rappresenta un potenziale di campo distante (attività del cono midollare).

Figure 4 Intraoperative baseline spinal SEP recordings from the thoracic region in Cases 3 (A) and 7 (B). The polyphasic nature of the recordings following P1 N1 is well shown. The early positivity Po represents a far field potential from activity in the conus medullaris.

(12). Ciò rappresenta il campo potenziale generato nelle corna dorsali dei segmenti spinali da L_4 a S_3.

A causa di queste estese connessioni sinaptiche è molto verosimile che anche qualora le radici S_2 e S_3 venissero amputate ci sarebbe una modificazione insignificante del SEP spinale, poichè il loro contributo al potenziale del «cono» è assai ridotto.

Nondimeno, pur considerando questi limiti anatomici ed elettrofisiologici del controllo della funzione neurale con i SEPs spinali durante la correzione chirurgica degli stati disrafici spinali, vorremmo sottolineare che nella nostra casistica non sono state notate modificazioni dei SEPs e neppure si sono verificati danni iatrogeni.

Metodi di controllo elettrofisiologico che permettono di superare i limiti appena ricordati possono essere identificati nelle tecniche di controllo del tono anale (4,10) e nell'elettromiogramma anale (5). Entrambi questi metodi utilizzano stimoli generati dalla stimolazione diretta del tessuto nervoso da parte del chirurgo; essi, tuttavia, presentano il grave limite di non fornire una valutazione obiettiva dell'integrità dell'innervazione neurale degli arti inferiori. Quest'ultimo problema può essere superato, in parte, attraverso l'utilizzazione di tecniche anestesiologiche che non comportino l'uso di miorilassanti. In tal caso, una valutazione soggettiva può essere ottenuta osservando la scossa dei gruppi muscolari degli arti inferiori, a seguito di una stimolazione diretta spinale o radicolare.

remained stable throughout the procedures despite manipulation of caudal neural structures. No patient suffered a neurological deficit as a result of surgery.

Discussion

Intraoperative electrophysiological monitoring of neural function should make surgery safer without unduly complicating or prolonging the operative procedure. The particular risks associated with surgery for complex spinal dysraphism in the lumbosacral region are damage to a tethered conus medullaris and lumbosacral spinal rootlets during transection and resection of intraspinal lipomata, thick arachnoidal adhesions or a thick filum terminale. Identification of neural tissues and safe planes of dissection during these procedures can be extremely difficult and the vital S_2-S_4 radicles, which innervate the anal, vesical and pelvic floor musculature are at particular risk.

The anatomical location of most intradural abnormalities associated with caudal spinal dysraphism and the tethered cord syndrome means that monitoring spinal SEPs generated by tibial and peroneal nerve stimulation is a suboptimal method of assessing neural integrity during surgery for these conditions. The most caudal spinal segment contributing to either of these nerves is S_3 (3) and this contribution is probably a small fraction of afferent fibres contained within these nerves. Therefore neural structures distal to this segment may be irreparably damaged with no change in spinal SEP waveform. Another factor that vitiates against spinal monitoring of SEPs generated by tibial and peroneal nerve stimulation is that the signal in the lumbar region (Fig. 3) is essentially a «conus» potential (12). This represents the potential field generated in the dorsal horns of spinal segments from L_4 to S_3. Because of these extensive synaptic connections it is highly likely that even if the S_2 and S_3 radicles were amputated there would be an insignificant change in the spinal SEP since their contribution to this «conus» potential is so small. Despite these anatomical and electrophysiological shortcomings of spinal SEP monitoring during surgery for caudal spinal dysraphism we should add that in our series there were no changes in SEPs and nor were there any iatrogenic deficits.

Methods of electrophysiological monitoring that overcome some of the aforementioned shortcomings are the techniques that monitor anal tone (4, 10) and the anal electromyogram (5). Both these methods utilise stimuli that are generated by direct electrical stimulation of neural tissues by the surgeon. These methods do, however, have the limitation of not providing an objective assessment of the integrity of neural innervation to the lower limbs. The latter problem can be overcome to some extent by using an anesthetic technique that does not incorporate muscle relaxation. A subjective assessment can then be made by observing lower limb twitching to direct spinal or radicular stimulation.

An optimal method of neural monitoring for caudal spinal surgery would combine the technique of direct recording of spinal evoked potentials together with a method of assessing anal or vesical muscle function in response to electrical stimulation of neural tissues by the surgeon. This would enable documentation of the contribution of different radicles to both the «conus» potential and sphincteric function. Badr and associates (1) have demonstrated a cortical evoked potential following electrical stimulation of

Un metodo ottimale di controllo della funzione neurale nella chirurgia della spina caudale dovrebbe combinare insieme una tecnica di registrazione diretta dei potenziali evocati spinali e un metodo per stabilire la funzione dei muscoli anali e vescicali in risposta alla stimolazione elettrica delle strutture nervose da parte del chirurgo. Ciò infatti permetterebbe di documentare il contributo delle diverse radici sia al potenziale di «cono» che alla funzione sfinterica. Badr e coll. (1) hanno dimostrato la presenza di un potenziale evocato corticale dopo stimolazione elettrica con elettrodo intravescicale. Dovrebbe pertanto essere possibile registrare sia le risposte afferenti che quelle efferenti dopo stimolazione di S_2-S_4. Le risposte intraoperatorie dovrebbero quindi essere correlate agli studi urodinamici per un'ulteriore conoscenza della neurofisiologia della vescica.

L'utilizzazione dei SEPs spinali nella correzione di una diastematomielia al di sopra della regione del cono non presenta i problemi associati con la chirurgia della regione lombosacrale. La componente polifasica PNPN (Fig. 4) della forma d'onda rappresenta la scarica spinale afferente, che passa al di sotto dell'elettrodo di registrazione (6). Questa componente è molto sensibile agli insulti indiretti che possono danneggiare la funzione del midollo spinale, quali l'ipossia, l'ischemia e l'eccessivo stiramento dell'asse spinale (2,6,9). Poiché la rimozione chirurgica di spine ossee o cartilaginee non deve necessariamente mettere a repentaglio la funzione dell'emi-midollo contiguo, ogni attenuazione della forma d'onda del SEP spinale dovrebbe essere dovuta ad una causa iatrogena. Esiste, tuttavia, la possibilità di una danno della parte anteriore del midollo spinale che può realizzarsi senza evidenti modificazioni dei SEPs spinali, poiché questi ultimi sono in relazione alle colonne dorsali. Proprio per questa possibile evenienza, Levy e coll. (7) hanno recentemente proposto la valutazione dei potenziali motori, dopo stimolazione percutanea della corteccia motoria.

an intravesical electrode. It should therefore be possible to record both afferent and efferent responses following stimulation of S_{2-4}. These intraoperative responses could then be correlated with urodynamic studies to further knowledge of the neurophysiology of the bladder.

The utilization of spinal SEP monitoring during surgery for diastematomyelia above the conus region is free of the problems associated with surgery in the lumbosacral region. The polyphasic PNPN component (Fig. 4) of the waveform represents the afferent spinal volley as it passes beneath the recording electrode (6). This component is particularly sensitive to indirect insults that may impair spinal cord function such as hypoxia, ischemia and overdistraction of the spinal axis (2, 6, 9). Since the surgical removal of bony and cartilaginous spurs does not directly threaten function in the contiguous hemicord, any attenuation in the spinal SEP of the waveform should be due to a remedial cause. There is however the possibility of an anterior cord insult occurring with no change in the spinal SEPs since these are relayed up the dorsal columns. Because of this possibility Levy and associates (7) have recently described monitoring motor potentials following percutaneous stimulation of the motor cortex.

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