ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA FROM 1963 TO 1996

THESIS SUBMITTED FOR THE DEGREE OF MASTER OF MEDICAL SCIENCE,

UNIVERSITY OF ADELAIDE

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CONTENTS

ABSTRACT

This thesis provides an overview of accidental childhood death and reports original work analysing specific types of injuries, mechanisms of death and recommendations for the prevention of such fatalities. It is based on a detailed review of 369 cases of childhood accidents taken from the records of the Women's & Children's Hospital Department of Histopathology over a 34 year period from 1963 to 1996. Given the range of the subject matter only certain areas have been selected for detailed examination; these include unsafe sleeping environments, unsafe eating practices, dangerous situations on farms and drowning. These were chosen following identification of significant child safety problems in these areas as a part of the 'Keeping Your Infant and Child Safe' programme run by the author through the Department of Histopathology, Women's & Children's Hospital, Adelaide. Previously unrecognised dangers to children detected during this study include mesh-sided cots, 'V'shaped pillows and certain types of stroller-prams. In addition, particular dangers associated with home swimming pools, cosleeping with adults, breast feeding and infant holding practices are described. Analyses of series of fatal cases are provided with clinicopathological features of individual cases where appropriate. A case report and review format has been used as this lends itself to the examination of particular problems that may occur with individual infants or children.

CERTIFICATION

This study resulted from an expansion of the author's investigations into deaths attributed to sudden infant death syndrome (SIDS). While a thesis submitted to the University of Adelaide for the award of an MD degree in 1994 on 'Sudden Natural Death in Infancy and Early Childhood' briefly mentioned problems with eating and sleeping environments, this was in the context of differentiating such deaths from SIDS (specifically pp 87-97 on sleeping environments, 97-102 on rocking cradles and 238 on airway obstruction due to food). In contrast, the current thesis focuses on unnatural death up to the age of 16 years and provides analyses of different cases.

I, therefore, certify that to the best of my knowledge and belief this thesis contains no material which has been accepted for the award of any degree or diploma in any University and no material previously published or written by another person, except where an acknowledgement has been made or where due reference is made in the text.

I consent to the thesis being made available for photocopying and loan if applicable if accepted for the award of the degree.

Assoc. Prof. Roger W. Byard

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ACCIDENTAL CHILDHOOD DEATH - INTRODUCTION

Children are at increased risk of injury and death from accidents for a variety of reasons. They have a natural curiosity which leads them to explore their environment and investigate situations where they often do not recognise potential hazards. Lack of physical strength, small size, inexperience and immature coordination may also result in infants and children being unable to extricate themselves from potentially perilous positions that may not present a danger to an adult.

AIMS

The aims of this study were:

1) To provide an overview of cases of accidental death in children aged from 0 to 16 years presenting to the Adelaide Children's Hospital and Women's and Children's Hospital over the past 34 years from 1963 to 1996, with an outline of autopsy approaches, review of fatal mechanisms, and an analysis of:
   - i) Dangerous sleeping environments that may result in childhood fatalities.
   - ii) Dangerous eating practices that may result in childhood fatalities.
   - iii) Dangerous farm environments that may result in childhood fatalities.
   - iv) Deaths due to drowning.
   - v) Previously underappreciated, or unrecognised, situations that may result in childhood fatalities.

In addition, the study:

2) Assessed the effectiveness of the process of identifying and diagnosing subtle accidental childhood deaths through the 'Keeping Your Infant and Child Safe' programme.
3) Analysed potential problems that arose in the pathological determination of the cause of death in cases of unexpected infant fatalities.
4) Provided recommendations for changes to child care practices by parents and child carers in the form of pamphlets and information for public distribution.

MATERIALS and METHODS

It is a legal requirement that all 'violent or unusual' deaths in South Australia are reported to the State Coroner. Childhood deaths from accidents fall within that description. While the majority of these cases are autopsied at the Women's and Children's Hospital, occasional cases are autopsied by pathologists at State Forensic Science, the State's centre for forensic pathology.

In this study an analysis was undertaken of all cases of accidental death in children aged between 0 and 16 years recorded at the Women's and Children's Hospital (formerly the Adelaide Children's Hospital) Department of Histopathology over the past 34 years from 1963 to 1996. The hospital is the major referral and treatment centre in South Australia for cases of paediatric trauma and treats the majority of such cases. Cases were identified through the department's computerised database and also by manually reviewing autopsy records. Categories of death included motor vehicle accidents (passengers, pedestrians, cyclists, motorcyclists and 'not otherwise specified'), drowning (freshwater, saltwater, other liquid, and 'not otherwise specified'), accidental asphyxia, burns, poisonings, electrocutions and miscellaneous trauma. In addition, manual review was undertaken of all paediatric autopsy records at State Forensic Science, Adelaide, South Australia. Further State and National data have also been incorporated, where appropriate, from the National Injury Surveillance Unit Database to help place the study data into context.

The cases were examined for the types of injury, the age and sex distribution, and the year of occurrence. Analysis of specific subgroups of infants and children was performed from this pool of cases. It should be noted that documentation of the circumstances of death was often not optimal in the earlier years of the study, and varied depending on the type of accident Thus, time periods for studies of the different accident groupings are not uniform, as they have been chosen to include only those cases where specific detailed information was available and verifiable, or where the author had conducted a specific prospective study. As well, description of occasional

earlier cases in a previous thesis, as mentioned on page 4, has resulted in their exclusion from certain sections of the current work. Deaths due to iatrogenic and non-accidental injuries have not been included.

COMMENT

The computerised autopsy database at the Women's and Children's Hospital Department of Histopathology contains details of all autopsies performed at the hospital since 1963. Cases are classified according to the SNOMED system and are readily accessible if standard diagnoses are searched for. In addition the author has set up the South Australian SIDS Database in the Department which not only contains information on SIDS infants, but has information on approximately 100 control infants who died suddenly and unexpectedly. The control database includes accidental deaths and provides further background information on these cases.

Although the study is based primarily on cases from the Women's and Children's Hospital, a small number of cases (< 5% of the total) present in the database were provided by the State Coroner' Office. Unfortunately reports of paediatric cases autopsied elsewhere have not always been sent to the Women's and Children's Hospital, and so the data do not include all of the state's paediatric coronial cases over the period of the study. Over the 34 year time frame of the study, there has also been variable referral of cases for autopsy to different centres in South Australia. For example, gunshot wounds and aviation accidents have always been autopsied at State Forensic Science rather than the Women's and Children's Hospital. Not all of the children involved in fatal accidents died instantaneously and therefore a certain number may have been treated, and later died, at peripheral hospitals. Additionally, not all of these children would have undergone autopsies.

Coding of cases in the past has also not been optimal, and failure of certain injuries to appear within the group is most likely due to a failure of accessioning, rather than to an absence of such cases over the study period.

Manual search of records at State Forensic Science, Adelaide, was undertaken by the author from the commencement of documentation in 1976, to 1996. As a formal state forensic service was not established until 1976, records before this time are unavailable. Unfortunately, case classification, particularly in the earlier years, is again rudimentary with minimal details, and so the State Forensic Science data are not of particular use in analysing factors contributing to the various types of injuries. For this reason the data have not been combined with the Women's and Children's Hospital data, unless autopsy reports had already been forwarded and incorporated into the Women's and Children's Hospital database in previous years.

Because of these factors the study cannot be used to provide completely accurate epidemiological information on accidental childhood deaths in South Australia over the past 34 years. For example, only one case of drowning under the age of 9 years is present in the 1994 Women's and Children's Hospital data compared to two drownings under 9 years registered for the state for that year (National Injury Surveillance Unit, 1996). In certain categories, however, the Women's and Children's Hospital and State data concur, for example both register three fire/flame/scald deaths in 1994 in children under 9 years of age. In some instances the Women's and Children's Hospital data are more accurate than State data. For example, although no drowning deaths were registered in 1991 in State data for children under 14 years (National Injury Surveillance Unit, 1996), the death of a 2-year-old boy who drowned in a home pool in November, 1991 is recorded in the Women's and Children's Hospital database. Presumably, partial resuscitation followed by brain death two days after the immersion resulted in different coding, hence the exclusion of the boy from the drowning group in State data.

Further information on fatal accident rates from 1979 to 1995 is available through the Australian Institute of Health and Welfare National Injury Surveillance Unit which provides ICD9 External Causes classification of national and state deaths, based on data supplied by the Australian Bureau of Statistics, State Registers of Death and

Coroners. Data from 1990 to 1994 is also available from this Unit on the Internet (webmaster@www.nisu.flinders.edu.au).

Given that detailed information on the rates of state and national accidental childhood deaths is already available, this thesis focuses instead on:

i) formulating one institution's experience of accidental childhood death over more than three decades;

ii) providing a clear picture of certain types of childhood activities that may be associated with lethal outcomes, with an approach to such cases at autopsy;

iii) providing further assessment of mechanisms of death and characteristics of environments which may be dangerous to children;

iv) delineating the advantages of formal death scene investigation in cases of accidental childhood death;

v) developing recommendations and strategies for accident prevention.

SUMMARY

Brief summaries of each section of the thesis follow.

1) ACCIDENTAL DEATH IN INFANCY AND EARLY CHILDHOOD - AN OVERVIEW AND REVIEW

Objective: To review causes of accidental death in children aged between 0 and 16 years in cases which have been studied by autopsy at the Women's and Children's Hospital, and to compare these data to reports in the literature.

Methodology: An analysis was undertaken of all cases of accidental childhood death that have been recorded at the Adelaide Children's Hospital and Women's and Children's Hospital Department of Histopathology during the past 34 years from 1963 to 1996. The cases were examined for the types and circumstances of injury, age and sex distribution.

Results: A total of 369 cases of accidental childhood death were accessioned including 189 motor vehicle accidents (69 pedestrians, 66 not specified, 27 passengers, 24 cyclists, 3 motorcyclists); 63 drownings; 40 asphyxiationss associated with unsafe sleeping conditions; 24 burns/scalds; 15 farm-related (5 of these cases are also listed in other categories); 14 foreign body aspirations/asphyxias; 10 poisonings, 6 electrocutions, and 13 miscellaneous (age range = 1m - 15y 11m; mean = 5y 2m; M:F = 1.7:1).

Conclusions: Variable referral patterns to the hospital during the past 34 years preclude accurate epidemiological comments on rates of traumatic childhood deaths in South Australia. However, motor vehicle accidents (51%) and drownings (17%) have accounted for the largest number of deaths. The data also demonstrate significant problems with sleeping and eating practices of young children, and with fire and rural safety.

STUDY 2) AN ANALYSIS OF HAZARDOUS CHILDHOOD SLEEPING ENVIRONMENTS

Objective: To review recent local experience of accidental death in early childhood associated with unsafe sleeping environments.

Methodology: The records of the Department of Histopathology at the Women's and Children's Hospital were searched over a four year period from January 1993 to December 1996, for cases of sudden infant and early childhood death caused by unsafe sleeping environments.

Results: Fourteen cases were found where unsafe sleeping environments had caused accidental asphyxial death in infants and young children in recent years. Lethal situations involved wedging/positional asphyxia (N = 5), hanging (N = 4), suffocation from pillows (N = 2) and overlaying (N = 3). If a 4y-old severely mentally retarded boy is not included in the series because of his underlying medical condition, the age range is 1m to 18m, (mean age = 5.6m), with a male to female ratio of 2:1.

Conclusions: Despite previous studies and reports on unsafe infant sleeping environments, accidental deaths in this situation are still occurring. This fact prompted the production of two safety information pamphlets aimed specifically at parents and child carers. In addition, a safety checklist for parents and hospital policy guidelines on safe sleeping have been prepared and distributed (see Appendix I).

STUDY 3-1) AN ANALYSIS OF HAZARDOUS CHILDHOOD EATING PRACTICES

Objective: To review local and overseas experience of food asphyxia in children and to examine aspects of safe childhood eating practices.

Methodology: Inpatient Separation Information Data for childhood hospital admissions in South Australia were searched for episodes of food-induced airway obstruction, and case records of the Department of Histopathology at the Women's and Children's Hospital were searched for cases of fatal food asphxia from 1989 to 1994.

Results: While other forms of injury to young children appear to be declining in numbers, episodes caused by choking on food have remained relatively constant. The increase in average length of hospital stay (from 2.8d in 1989-1990 to 5.2d in 1993-1994) also suggests that the episodes have been more severe. Two fatal cases were also found within the same period.

Conclusions: Choking due to food inhalation is a problem with potentially fatal consequences. Young children are particularly at risk as they have immature dentition, often poor control of swallowing, and lack experience of food. Although young children should avoid potentially dangerous foods, such as raw carrot sticks and raw apples, certain currently available information packages for parents recommend these foods.

STUDY 3-2) AN ANALYSIS OF MECHANISMS OF UNEXPECTED DEATH FOLLOWING FOREIGN BODY INGESTION

Objective: To determine the range of possible mechanisms of death following foreign body ingestion in childhood by case and literature review.

Methodology: Cases of accidental asphyxia due to foreign body ingestion were accessioned from the Department of Histopathology (Women's and Children's Hospital) and from referral cases. Mechanisms whereby the foreign material led to death were ascertained in each instance.

Results: Fourteen cases were identified in the Women's and Children's Hospital database. Death most often resulted from asphyxia due to compromise of major airways. Other less common mechanisms involved haemorrhage, acute cardiac tamponade, arrhythmia, centrally-mediated respiratory arrest and sepsis.

Conclusions: Unexpected and often sudden death following foreign body ingestion/inhalation most commonly results from acute airway obstruction. There are, however, a variety of other lethal mechanisms which are not often considered, but which should be looked for at autopsy. Sudden death may follow a protracted asymptomatic period and may also be due to foreign bodies impacting in the oesophagus.

STUDY 3-3) AN ASSESSMENT OF EATING PRACTICES IN CHILDCARE CENTRES

Objective: To assess the safety of toddler and early childhood eating practices in childcare centres.

Methodology: A search of cases of accidental deaths on file at the Women's and Children's Hospital Department of Histopathology from 1963 to 1996 was conducted to identify cases of accidental food asphyxia that had occurred in childcare centres.

Results: Of the 369 cases of accidental death identified over this 34 year period only two cases of food asphyxia in childcare centres (0.6%) were found. These involved an 18m-old boy who choked on a carrot stick and a 19m-old boy who choked on a piece of sausage.

Conclusions: As deaths due to food asphyxia in childcare centres are rare, there have been no guidelines set in place to advise on safe food preparation in such places. This is of particular concern given that children attending these centres may be exposed to food that is inappropriate for their level of dental and swallowing maturation. A pamphlet for parents and childcarers detailing safe feeding practices for toddlers has been prepared and distributed (see Appendix I).

STUDY 4) A STUDY OF HAZARDOUS CHILDHOOD FARM ENVIRONMENTS IN SOUTH AUSTRALIA FROM 1981 TO 1996

Objectives: To analyse the circumstances surrounding farm and tractor-related childhood fatalities in South Australia, to identify potentially dangerous situations and to delineate prevention strategies.

Methodology: A retrospective search of files at the Women's and Children's Hospital Department of Histopathology from 1981 to 1996, and the State Coroners Office from 1988 to 1996, was conducted for cases of accidental deaths of children on farms.

Results: Fifteen fatalities were identified comprising 11 boys and 4 girls (age range = 2y 11m to 13y; mean = 6y). Activities prior to death included riding on a tractor/trailer (N = 8); playing near a field grain storage bin (N = 2); playing near machinery (N = 2); sleeping in a car near a burn off (N = 1); walking around a dam (N = 1) and riding as a passenger on a motorcycle (N = 1). Causes of death included multiple skull fractures and cerebral damage (N = 11), asphyxia (N = 2), drowning (N = 1) and incineration (N = 1). (Please note: 5 of these cases have also been listed under 'Drowning, 'Fire deaths' 'Foreign body asphyxias' and 'Motorcycle deaths).

Conclusions: The most dangerous activity involved children riding unrestrained on tractors; falls resulted in extensive injuries from tractor rear wheels or towed machinery. Allowing children on farms to ride on tractors or machinery only if there is a safe seat with a restraining harness would substantially reduce the number of fatal farm-related childhood injuries in South Australia. An information package for farm families detailing safety issues is currently being prepared (Appendix I).

STUDY 5) AN ANALYSIS OF DROWNING DEATHS IN CHILDHOOD IN SOUTH AUSTRALIA FROM 1963 TO 1996

Objective: To study deaths during immersion in water in childhood in South Australia from 1963 to 1996, to determine the circumstances of death and to ascertain the number of cases where underlying conditions either caused death or prediposed to drowning.

Methodology: Autopsy records at the Women's and Children's Hospital over a 34 year period from 1963-1996 were searched for cases coded under drowning.

Results: Sixty-seven cases were found where the coding was 'drowning'. Two children died six months and one year after 'near-drowning' episodes from pneumonia secondary to severe brain damage, leaving 65 cases. In 9 cases, however, examination of the history and post mortem findings suggested a more complex terminal sequence of events. Specifically, death was attributed to haemorrhage from an intracranial arteriovenous malformation in one case, and a hypoplastic right coronary artery in another, leaving only 63 cases where death could be attributed to drowning. Seven children, aged between 3m and 12y 7m (mean = 8y 7m), had conditions predisposing to accidental drowning such as epilepsy (N = 6) and/or mental impairment (N = 2). The majority (25) of the remaining 56 children (age = 3m to 12y; mean = 2y 11m; M:F = 2.4:1) drowned in home pools. Fourteen children drowned in the bath, 6 in freshwater lakes or creeks, 3 in fishponds, and the remainder in a variety of different locations.

Conclusions: In South Australia children are most at risk of drowning in home swimming pools. This gives support for moves to legislate for compulsory fencing around pools. Children who drowned in baths tended to be younger (mean age = 2y 3m) than those who drowned in pools (mean age = 3y 11m). Seven of the children (11% of the group) had additional underlying medical problems that could either have initiated the drowning episode or caused death due to alternate mechanisms. These results indicate that a high index of suspicion must be maintained in all cases of paediatric drowning, not only for unnatural causes of death, but also for additional natural disease processes which may have significantly contributed to the fatal episode. This may have particular relevance in jurisdictions where full post mortem examination in such cases is not always required by law.

STUDY 6-1) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS AND YOUNG CHILDREN ASSOCIATED WITH MESH-SIDED COTS

Objective: To investigate the possibility that mesh-sided cots may be associated with accidental asphyxial deaths in infants.

Methodology: Review was undertaken of all cases of sudden infant death presenting to the Women's and Children's Hospital from 1963 to 1996 involving infants who had been found in mesh-sided cots.

Results: Two cases of accidental asphyxia were found involving an 11.5m-old boy and a 3.5m-old boy who each died after being trapped between the elastic mesh side of their cots and the cot mattress. In both cases the original cot mattress had either been replaced or augmented by a less well fitting, thicker mattress.

Conclusions: Particular problems that exist with mesh sided cots are the potential for considerable stretching of the side of the cot admitting the relatively larger, poorly supported infant head, with elastic recoil of the mesh holding the head in potentially dangerous positions. Such cots should only, therefore, be used with the mattress that is provided by the manufacturer. In both of the described cases the initial assessment at the death scene failed to implicate the cot, and the deaths were thought most likely to be due to SIDS. Subsequent investigation and death scene reconstructions determined that accidental asphyxia had occurred.

STUDY 6-2) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS AND YOUNG CHILDREN ASSOCIATED WITH STROLLERS AND STROLLER-PRAMS

Objective: To investigate the possibility that certain kinds of strollers may be associated with accidental asphyxial deaths in infants.

Methodology: Review was undertaken of all cases of sudden infant death presenting to the Women's and Children's Hospital from 1963 to 1996 involving infants who had been found in strollers or stroller-prams.

Results: Two cases of sudden and unexpected death occurring in a 3m-old boy and an 8m-old boy, respectively, were found in which accidental positional asphyxia and hanging occurred in stroller-prams. Both infants, after being placed to sleep, had moved down towards the fronts of the stroller-prams and had slipped out. The younger infant who had fallen out of the stroller-pram when the foot plate collapsed, was found hanging from a metal bar on the side. The older infant had partly slipped through the front and was suspended with his head and arms within the stroller-pram. His face was pushed firmly into the mattress by a horizontal metal bar that his body had passed under.

Conclusions: Although infants are sometimes left unattended in strollers/stroller prams, these may not be safe environments for sleeping. Problems which may occur include collapse of the footplate, collapse of the stroller and slipping of the infant through the front of the stroller. Death may result from hanging, airway obstruction and/or positional asphyxia.

STUDY 6-3) AN INVESTIGATION OF POSSIBLE DANGERS TO SLEEPING INFANTS ASSOCIATED WITH ADULT SIZED 'V' PILLOWS

Objective: To investigate the possibility that adult size 'V' pillows may be associated with accidental asphyxial deaths in infants.

Methodology: Review was undertaken of all cases of sudden infant death presenting to the Women's and Children's Hospital from 1963 to 1996 involving infants who had been placed to sleep on adult size 'V' pillows.

Results: Four cases of sudden infant death were identified in which infants were found sleeping on 'V' pillows. All of the deaths had occurred in 1995. In two of the cases the pillows were considered to be implicated in the cause of death as the potential for obstruction of the infants' airways could be demonstrated on death scene reconstruction.

Conclusions: Adult size 'V' pillows may be unsafe for use with small infants who may accidentally suffocate if trapped between the two arms of, or under, the pillow. The use of such pillows to maintain the body position of sleeping infants should be discouraged.

STUDY 6-4A) AN INVESTIGATION OF A POSSIBLE CAUSAL RELATIONSHIP BETWEEN APPARENT LIFE THREATENING EVENTS (ALTE), INFANT HOLDING PRACTICES AND BREAST FEEDING

Objective: To investigate the possibility that infant holding practices and/or breast feeding may induce ALTEs, or cause death, in susceptible infants.

Methodology: Clinical study of two infants who sufferred ALTEs when held incorrectly was undertaken. Prospective examination of autopsy cases was then undertaken from 1992 to determine whether any cases of sudden infant death could be attributed to incorrect holding practices or to breast feeding.

Results: ALTEs could be induced in two 1m-old infants with incorrect holding techniques. Once these were modified the ALTEs ceased to occur. A 2m-old girl was also identified subsequently who died while being breast fed.

Conclusions: These cases demonstrate that certain infants may not respond normally to airway occlusion while being held or nursed, and show that careful study of the events surrounding ALTEs may reveal contributing environmental factors. If ALTEs occur around feeding time, observation of how caretakers place the infant during and after feeding may be informative. The occurrence of an infant death during breast feeding by an awake, although distracted, mother would be supportive of airway occlusion being potentially responsible for death in these circumstances.

STUDY 6-4B) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS ASSOCIATED WITH ADULT COSLEEPING

Objective: To investigate the possibility that cosleeping with adults may be associated with accidental asphyxial deaths in infants.

Methodology: Review was undertaken of all cases of sudden infant death presenting to the Women's and Children's Hospital from 1983 to 1996 involving infants who had died while sleeping with an adult.

Results: Between 1983 and 1996 the proportion of cosleeping of adults and infants in cases of sudden infant death in South Australia has risen from 7.5% to approximately 30%. This compares to 1.5% of control infants aged 3m in 1988 who were bed sharing. Although the circumstances of death failed to reveal anything specific in most cases, three infants were identified whose histories suggested the possibility of accidental asphyxia: a 1m-old boy who died in bed with an intoxicated mother, and two additional infants who died after their breast feeding mothers fell asleep with them in bed.

Conclusions: If cosleeping with adults is a potentially lethal practice for certain infants then it is vital to be able to identify infants who may be at risk in this situation. If this is not possible, then alternative sleeping arrangements must be adopted until the situation is further clarified. Given the potential that exists for accidental asphyxia when infants are being breast fed by their mothers in bed, breast feeding in this situation may only be appropriate in the presence of a second awake adult who can ensure the safety of the infant if the mother falls asleep. Alternatively breast feeding may be more safely conducted away from the bed.

STUDY 7) TO EXAMINE STEPS IN THE POSTMORTEM ASSESSMENT OF CASES OF SUDDEN INFANT DEATH TO DETERMINE THE EFFECTIVENESS OF THE PROCESS OF IDENTIFYING CASES OF SUBTLE ACCIDENTAL DEATH

Objective: To determine the range of diagnoses and to evaluate the contribution made by various steps in the postmortem assessment of cases of accidental infant death.

Methodology: Review of cases of sudden infant death presenting to the Women's and Children's Hospital from 1 Jan 1995 to 31 Dec 1995 was undertaken.

Results: A total of 24 cases were found, each of which had a death scene examination, clinical history review, postmortem examination and subsequent formal case review. Obvious trauma was involved in three cases: a drowning, a motor vehicle accident and a hanging. Death in a further case was attributed to accidental asphyxia involving a mesh sided crib, based on death scene reconstruction (for further details see Study 6-1). A lingual thyroglossal duct cyst was found in another infant. This left 19 cases in which SIDS was considered the final diagnosis before formal case review. At review, however, additional information that was not forthcoming at the time of the initial investigation resulted in three further deaths being attributed to accidental asphyxia. These involved suffocation under large 'V' shaped pillows in two cases and overlaying in the third (for further details see Studies 6-3 & 6-4B).

Conclusions: Of 24 cases of sudden infant death: 3 were due to obvious trauma, 4 to accidental asphyxia and 1 to a lingual thyroglossal duct cyst, leaving only 16 which were eventually attributed to SIDS. Twenty-four percent of cases of sudden infant death were, therefore, due to causes other than SIDS; 5% were identified at death scene examination/reconstruction, 5% were found at autopsy and a further 14% were only found during subsequent case review.

STUDY 8) AN ANALYSIS OF CERTAIN PROBLEMS THAT MAY OCCUR IN ESTABLISHING THE CAUSE OF SUDDEN INFANT AND CHILDHOOD DEATH

Study 8-1) This descriptive study deals with alteration of the presenting history or death scene in cases of accidental infant death.

Study 8-2) Significant coincidental findings at autopsy in accidental childhood death

Objective: To ascertain the number of cases of accidental childhood deaths where there is significant unsuspected and potentially lethal underlying pathology.

Methodology: An examination of the 369 cases of accidental childhood death in this study was undertaken to identify cases where significant occult disease was found at autopsy.

Results: Three cases of unexpected childhood death were found where significant potentially-life threatening lesions were identified that were considered coincidental to the cause of death. Two children died of accidental asphyxia and one died of potassium fluoride poisoning. Abnormalities identified at autopsy included established myocarditis (N = 2) and cardiac rhabdomyomas (N = 1). In one case a previously unsuspected abnormal familial karyotype was found.

Conclusions: These cases emphasize the value of complete autopsy investigations in all types of sudden childhood death, even when the cause of death appears obvious; they also demonstrate that potentially-lethal organic disease may be asymptomatic in childhood and unrelated to death.

* * * *

CHAPTER 1

ACCIDENTAL DEATH IN INFANCY AND CHILDHOOD - AN OVERVIEW AND REVIEW

OVERVIEW

A) INTRODUCTION

Accidents account for the majority of sudden deaths in childhood and adolescence. In the United States, where childhood injury rates are high, accidents are responsible for nearly 50% of deaths in the age range 1 to 24 years and 71% of deaths between the ages of 15 and 19 years (Accident Facts, 1990). Between 1984 and 1986 the mortality rate from accidents for individuals aged between 1 and 19 years was 30.5/100,000 in the United States, 26.1/100,000 in Canada, 22.3/100,000 in Norway, 21.5/100,000 in France, 15.6/100,000 in England and Wales and 13.1/100,000 in the Netherlands (Williams & Kotch, 1990). This compares to Australia where the mortality rate in 1994 from accidents was 10.8/100,000 in 0 to 4 year olds, 5.3/100,000 in 5 to 9 year olds, 6.4/100,000 in 10 to 14 year olds and 29.2 in 15 to 24 year olds. The corresponding rates for South Australia were 7.1, 6.9, 4.0 and 30.1/100,000 (National Injury Surveillance Unit Data, 1996).

Although the percentages for the different categories vary among studies, the most common causes of accidental death in children are usually motor vehicle accidents, drowning and fires/burns (Colombani et al, 1985; Peclet et al, 1990; Runyan et al 1985). This trend was observed in the present study, with motor vehicle accidents accounting for 51% of deaths overall, followed by deaths due to drowning which accounted for 17%. These data are summarised in Figure 1-1 and Table 1-1. In other studies motor vehicle accidents have been responsible for 23% of deaths in infants and 80% of deaths in adolescents aged between 15 and 19 years (Byard & Cohle, 1994). A significant number of cases of vehicle related deaths and drowning in adolescents involve the consumption of alcohol (Rosenberg, Rodriguez & Chorba, 1990). Details of alcohol intake or blood alcohol levels were not available in the reported adolescent road fatalities in the present series.

Under one year of age accidental mechanical asphyxia is also a significant contributor to preventable deaths (Corey et al, 1992) and in the study data, accidental

FIGURE 1-1: TYPES OF FATALITIES FOUND IN A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH (0-16y) FROM 1963 TO 1996 IN SOUTH AUSTRALIA

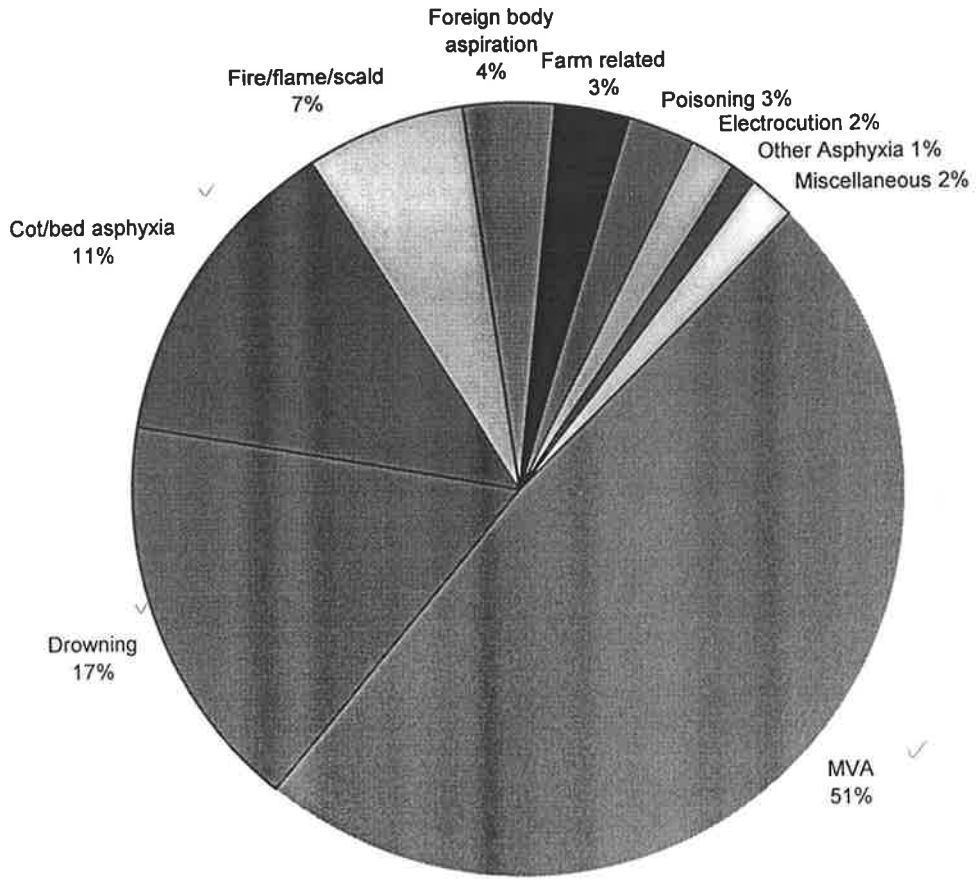


TABLE 1-1: DETAILS OF 369 CASES OF ACCIDENTAL DEATH IN CHILDREN AGED BETWEEN 0 AND 16 YEARS IN SOUTH AUSTRALIA FROM 1963-1996

CATEGORY		No.	M	F	AGE RANGE	MEAN
1)	**MOTOR VEHICLE ACCIDENT**					
	Unspecified	66	35	31	1y-14y	6y5m
	Passenger	27	13	14	1m-13y	4y9m
	Pedestrian	69	47	22	6m-15y11m	7y5m
	Cyclist	24	17	7	6y-13y6m	10y2m
	Motorcyclist	3	2	1	6y5m-13y	9y10m
2)	**DROWNING**	63	46	17	4m-12y7m	3y6m
3)	**COT/BED ASPHYXIA**	40	25	15	1m-4y	10m
4)	**FIRE/FLAME/SCALD**	24	11	13	1y1m-14y2m	5y1m
5)	**FOREIGN BODY ASPIRATION**	14	12	2	4m-8y	2y2m
6)	**POISONING**	10	5	5	1y1m-3y3m	2y6m
7)	**FARM RELATED***	10	6	4	2y6m-11y6m	4y10m
8)	**ELECTROCUTION**	6	4	2	2y-12y	5y8m
9)	**OTHER ASPHYXIA**	5	5	0	3y-9y	5y5m

TABLE 1-1: ACCIDENTAL DEATH (cont.)						
10)	**SPORTING**	4	4	0	3y9m-14y9m	9y3m
11)	**FALLS**	2	1	1	3y2m-14y	8y7m
12)	**INDUSTRIAL**	1	0	1	9y6m	9y6m
13)	**TRAIN**	1	1	0	12y3m	12y3m
TOTAL		369	234	135	1M-15Y11M	5Y2M

* In addition there are 5 other deaths on farms: 2 asphyxias, 1 drowning, 1 incineration, 1 motorcycle accident.

asphyxia due to unsafe sleeping circumstances was the next major category (11% of deaths), followed by fire/scald deaths (7%). Less common causes of accidental death include poisoning and falls (Accident Facts, 1990). These were only responsible for 3% of the 369 deaths in the data. Accidents are, however, much less common in infants under one year of age, reflecting their relative immobility, and have been responsible for only 3% of fatalities in this age group (Byard & Cohle, 1994). Other categories of death that were found in the current study included foreign body aspirations, farm-related fatalities, electrocutions, traumatic asphyxia, sporting accidents, industrial accidents and train-related fatalities.

Causes of death also vary depending on the country studied; for example, firearms are the third most common cause of accidental death in 15 to 19 year-olds in the United States (Byard & Cohle, 1994). Although firearm deaths were not included in the Women's and Children's Hospital data, review of files at State Forensic Science reveals that gunshot deaths are relatively rare in South Australia under the age of 16 years. While both accidents and suicides are included in the State Forensic Science data, a significant number of childhood gunshot deaths in South Australia appear to have been associated with family murder/suicides perpetrated by one of the parents.

Motor Vehicle Accidents

Fatalities due to motor vehicle accidents in this age group are readily identifiable by history and autopsy examination. The extent of injury depends on the circumstances and speed of impact; the autopsy findings are often similar to adults, involving craniocerebral, chest and abdominal trauma (Cooper & Taylor, 1989). Aortic laceration occurs predominantly in pedestrians (Eddy et al, 1990).

Since 1990 there has been a steady decline in the rate of death from on-road motor vehicle accidents (ICD9 E810-819) in children in Australia. Under the age of 14 years the yearly numbers of fatalities have been 200 (in 1990), 141 (in 1991), 150 (in 1992), 132 (in 1993) and 127 (in 1994) (the 1995 data are not yet available). These data compare to the numbers of late adolescent and young adult motor vehicle accident

deaths (15 to 24 years) of 785 in 1990 and 559 in 1994 (National Injury Surveillance Unit Data, 1996). (Local data may be found later in this chapter).

Drowning

Situations where children are at greatest risk of drowning vary with age (Pearn, 1992). For example, toddlers left unattended in baths may drown, whereas adolescents are more at risk in outdoor situations, particularly if alcohol has been consumed (Hyma, 1990; Jumbelic & Chambliss, 1990; Pearn et al, 1979). It should be recognised that the diagnosis of drowning may not be easy and it is important to look for pre-existing conditions that may have caused death or precipitated drowning (Modell, 1971; Smith, Byard & Bourne, 1991).

In the United States more than 2000 children per year have drowned, and in addition, significant neurological disabilities afflict a percentage of survivors (Pearn, 1992). Peak ages of drowning are under 4 years and between 15 and 19 years (Rodriguez & Brown, 1990). National Australian data confirm the preponderance of drowning deaths in children under the age of 4 years with 42 drownings in swimming pools in 1992, compared to only 2 in that year between the ages of 5 and 14. Similarly, there were 26 and 25 childhood pool drownings under 4 years in 1993 and 1994 repectively, compared to only 4 each year in the 5 to 14 years age group (National Injury Surveillance Unit Data, 1996).

Drowning in baths showed a similar trend nationally, with 10 deaths of children under 4 years in 1992, 3 in 1993 and 7 in 1994, compared to 1, 3 and 1 in the same years in the age range 5 to 14 years. Ten, 11 and 2 'unspecified' drownings in children under 4 years occurred in 1992, 1993 and 1994, compared to 0, 2 and 1 over the same time for children in the older age group (National Injury Surveillance Unit Data, 1996).

Drowning in the 15 to 24 year old group, as would be expected, also tended to occur at sites other than baths and pools with a total of 80 fatalities in this age range occurring nationally between 1991 and 1994, inclusive, compared to only 33 cases in

children under 15 years (National Injury Surveillance Unit Data, 1996). (Local data may be found in Chapter 5).

Asphyxial Deaths

Accidental asphyxia may be a problem in younger children and infants who may move into positions in their beds where their airways become occluded, or their bodies become wedged so that they are unable to breathe, or where they become suspended from their clothing or restraining harnesses and hang. A recent Australian review found the mean age of such infants to be 10 months with an upper limit in this study of 3 years (Byard, Beal & Bourne, 1994).

Hanging deaths are readily identifiable at autopsy by ligature marks around the neck and by the presence of facial petechiae. Deaths from wedging of the body (Sturner, 1980) between a mattress and cot side, or wall or furniture, are less easily diagnosed, however, as the autopsy findings may be identical to SIDS (Moore & Byard, 1993), a feature which may result in misdiagnosis.

Scene examination in cases of accidental asphyxia may implicate plastic pillow and mattress coverings, beanbags, defective or badly constructed cots, seat or bouncinette harnesses, waterbeds, rocking cradles or adult beds, as contributing or causal factors in the fatal episode (Gilbert-Barness & Emery, 1996; Kemp & Thach, 1991). Not only are such findings of importance in determining the cause of death, but they may have direct implications for public health policy and legislation.

Suicidal hanging occurs in adolescence and the rare possibility of misadventure during an autoerotic episode should always be considered as the death scene findings may be subtle or altered. The youngest age for a victim of autoerotic asphyxia in the medical literature is 9 years (Byard, Hucker & Hazelwood, 1990).

Australian national data confirm that children under 4 years of age are most at risk from 'mechanical suffocation' with 11 deaths registered under 4 years, compared to 3 deaths in children aged between 5 and 14 years in 1992; 14 compared to 6 in

1993; and 11 compared to 3 in 1994 (National Injury Surveillance Unit Data, 1996). Reasons for this age-related vulnerability are discussed in Chapter 2.

Overlaying

Accidental suffocation of an infant due to overlaying by a parent in a shared sleeping environment was for centuries considered the cause of SIDS. However, in recent times there has been considerable debate as to the likelihood of overlaying being a cause of sudden infant death. As the autopsy findings in overlaying are identical to SIDS resolution of this issue must rely on death scene reconstruction and on epidemiological study. (Local data may be found in Chapter 6).

Foreign Body Inhalation

Compromise of childrens' airways may also occur due to inhalation of foreign bodies, most often parts of toys or food (Freidman, 1988; Harris et al, 1984). Although the presenting history is usually of sudden collapse while eating, foreign bodies may not cause immediate symptoms (Humphries Wagener & Morgan, 1988), and cases have occurred of infants being found dead in their cots after appearing quite well when being put down to sleep (Byard, Moore & Bourne, 1990). An impacted foreign body as the cause of death may only be revealed at autopsy when material is found in the airways, in the oesophagus with external compression of the airways, or causing perforation of the heart or great vessels (Grey, 1988).

Toddlers are particularly at risk of aspirating foreign material, although older children may also be vulnerable if there are factors which interfere with the assessment, mastication and swallowing of food or other materials. Situations where there is a higher risk of fatal aspiration include conditions such as mental retardation, or neurological conditions which may interfere with swallowing, such as cerebral palsy.

Australian data confirm the increased risk of fatal aspiration for children under the age of 4 years with 37 cases of fatal aspiration in the five year period 1990 to 1994, compared to only 12 such deaths over the same time in children aged between 5

and 14 years (National Injury Surveillance Unit Data, 1996). Twenty-one of the deaths in the under-four year group were due to food aspiration, with the remaining 16 cases being classified merely as due to 'non-food'. (Local data may be found in Chapter 3).

Fire Deaths and Burns

Childhood deaths due to fires often involve domestic house fires. Death may be due to extensive burns or to carbon monoxide inhalation. The presence of soot in the airways and elevated levels of carbon monoxide indicate that the child was alive at the time of the fire. As well as testing for carbon monoxide at autopsy, measurement of cyanide levels may be informative as this is liberated when plastics burn. Failure to find soot in the airways or elevated carbon monoxide or cyanide levels indicates that the child was dead before the fire started and suggests homicide (Byard, In press).

The pattern of burns from accidental scalding should be explainable by the caretaker's history, should correspond with the position of the infant or child in the bath and should include satellite burns from splashing. In all of these situations a thorough history and careful death scene examination are essential components of the postmortem examination.

Deaths due to house fires in Australia showed a striking predominance in the under 4 year group compared to children aged between 5 and 14 years, presumably due to infants' and toddlers' relative immobility and lack of understanding of dangers. Nationally, there were 15 housefire deaths in children under 4 years of age, compared to 6 in the 5 to 14 year group in 1991; 12 compared to 6 in 1992; 14 compared to 7 in 1993 and 12 compared to 10 in 1994. In South Australia over the same time there were 11 deaths of children under 9 years and only one death of a child aged between 10 and 14 years from 'fires/flame/scalds' (National Injury Surveillance Unit Data, 1996). (Local data may be found later in this chapter).

Electrocution

Children are at risk of electrocution when they play outside near low hanging wires or when they are investigating live appliances. Cases also occur when children have dropped electrical appliances into their baths. Deaths are relatively rare, however, with only 4 deaths under 15 years from electrocution in Australia in 1990, 2 in 1991, 1 in 1992 and 4 in both 1993 and 1994 (National Injury Surveillance Unit Data, 1996). The autopsy findings are similar to those found in adults (Knight, 1991). (Local data may be found later in this chapter).

Fatal Falls

Fatal accidental falls in children are rare and usually involve falls from considerable heights. The National Injury Surveillance Unit (1996) registered no deaths from falls of children under the age of 14 years in South Australia in 1990 or 1991, with only four fatal falls over the following 3 years. Only two cases of fatal falls were found in the present data.

Accidental fractures of the skull following falls tend to be linear, and nondisplaced and do not usually cross suture lines. Depressed skull fractures caused by accidents usually involve falls of more than 4 feet, or down stairs, or result from impact with a moving or sharp object, features which should be reflected in the history (Byard & Cohle, 1994). In contrast, skull fractures from nonaccidental injury tend to be larger, involving several bones, with a complex pattern and separation of the bone edges by more than 3mm. This reflects the greater force involved in assault compared to a simple fall.

Although the presenting history in cases of fatal nonaccidental injury with skull and other bone fractures is often of a fall at home, children show considerable resilience in falling. For example, a study of 165 witnessed accidental falls from beds and cots in hospital in children under the age of 16 years revealed only two relatively minor skull fractures and two extremity fractures (Levene & Bonfield, 1991). In another study of 61 children who fell from buildings, no deaths occurred in falls from

three stories or less (Barlow et al, 1983). Thus, it is difficult to perceive how a minor fall at home could result in death or even serious injury unless a plausible witnessed event can be described.

Intracranial haemorrhage may result from skull fracture or may occur without bony damage. Subdural haemorhage may be caused by tearing of dural bridging veins and in cases of abusive shaking may be associated with retinal haemorrhages. Subarachnoid haemorrhage may also be found (Adams, 1984; Leestma, 1988).

Injury to the substance of the brain may consist of direct tissue damage associated with skull fracture or may be due to rotational and shearing forces associated with acceleration/deceleration from direct blows or from violent movement of the head. Injuries consist of contusional tears of the subcortical white matter and haemorrhage, with or without the presence of diffuse axonal injury. Diffuse axonal injury is found in the white matter and consists of so-called 'retraction balls', which are swollen damaged nerve tracts reflecting previous damage.

Poisoning

Fatal accidental poisoning may occur in toddlers who discover and ingest prescription adult medicines during the course of investigating their environments. Other toxic substances which may be swallowed include household or garden products, alcohol and, less commonly, plants. A thorough inventory of household medications and substances accessible to a child victim is required. The National Injury Surveillance Unit (1996) registered only one death from pharmaceutical poisoning in children under 14 years of age in South Australia between 1990 and 1994, and only two deaths from other kinds of poisoning. This compares to a total of 48 cases of fatal poisoning between the ages of 15 and 24 years over the same time. The reason for this disparity is that fatal adolescent poisoning may result from suicidal activity or from recreational drug abuse, including inhalation of volatile substances (Garriott & Petty, 1980; King, Smialek & Troutman, 1985; Steadman et al, 1984; Yamamoto, Wiebe & Matthews, 1991). (Local data may be found later in this chapter).

Iatrogenic Deaths

Infants undergoing surgery are at increased risk of anaesthetic-related deaths due to respiratory or cardiovascular complications. These include inadequate ventilation, airway obstruction, cardiac arrhythmia and fluid and electrolyte imbalance (Graff et al, 1964; Mancer, 1989). Air embolism may result from misconnected gas lines (Murphy, 1986), and anaesthesia may worsen airway obstruction by causing pharyngeal relaxation (Berry, 1981).

Accumulation of fluid within the pericardial sac surrounding the heart may follow intracardiac procedures or the insertion of central intravascular catheters, and may result in death from cardiac tamponade (Byard et al, 1992). As death may not occur immediately there may not be a history of such a procedure available at the time of autopsy. The finding of significant amounts of fluid within the pericardial sac therefore necessitates biochemical analysis to assist in identifying its origins; for example, it may represent intravenous fluid derived from a misplaced catheter.

Postmortem investigation of childhood anaesthetic deaths should include review of the clinical and operative records, discussion with the involved surgeon and anaesthetist, and possibly consultation with a surgeon and anaesthetist who were not involved with the case (Reay et al, 1985). No iatrogenic deaths were included in the current data.

Commotio Cordis

Rarely a blow to the precordium may result in sudden death with no apparent damage to the heart or coronary arteries (Froede, Lindsey & Steinbronn, 1979). While it is likely that the terminal event is an arrhythmia, the aetiology is uncertain (Frazer & Mirchandani, 1984). Occasionally an occlusive thrombus will be found within a major coronary artery (Bor, 1969). The only possible case encountered was in the State Forensic Science records of a 12y-old boy who collapsed and died after being hit in the

chest during sporting activity, however, hypertrophic cardiomyopathy was found at autopsy.

Temperature-related Deaths

Infants and young children are at risk of hyperthermia due to their high metabolic rates, incompletely developed thermoregulatory control and inability to extricate themselves from hot environments (Oshima et al, 1992). While signs of dehydration may be found at autopsy, the findings in fatal cases of hyperthermia may be minimal. Again, investigation of the death scene is vital, with assessment required of the scene temperature, humidity and ventilation. Measurement of the infant's temperature is also needed. Particularly dangerous situations include children left inside closed automobiles on hot days and infants overwrapped in front of heaters on colder days. Occasionally occult medical conditions such as congenital adrenal hyperplasia or cystic fibrosis may be found at autopsy which may predispose to unexpected death on hot days.

Similarly, deaths due to hypothermia may have no characteristic findings at autopsy (Benz, 1980; Coe, 1984). Important information to be obtained as part of the postmortem investigation includes a history with specific details of time of exposure to cold, adequacy of bedding and clothing, environmental and body temperatures, humidity and windchill factor.

Nationally there have been only four cases of deaths under 14 years of age due to excessive heat registered by the National Injury Surveillance Unit (1996) between 1990 and 1994, and only one fatality attributed to excessive cold in this age group over the same time. There were no cases in the current data.

Nonaccidental Deaths

The features of nonaccidental deaths in children are a reflection of their inability to defend themselves from more powerful adult assailants. The autopsy findings range from the quite obvious in cases of starvation/neglect and repetitive assault, to those cases where the findings are quite subtle. Unique causes of death in infants/young

children include suffocation in cases of Munchausen syndrome by proxy and brain damage from whiplash shaking injury (Byard & Beal, 1993; Wissow, 1995). Rib fractures are very rarely, if ever, caused by cardiopulmonary resuscitation (Spevak et al, 1994).

Diagnostic Difficulties in Infancy

While SIDS has been the most common cause of unexpected death in infants and young children in Western countries, including Australia, there are many other fatal occult conditions that occur in this age group (Byard & Cohle, 1994; Byard, 1994a). Death may result from accidental or deliberate trauma, or from unsuspected organic disease. Unfortunately infants may be quite seriously ill and yet have minimal external manifestations. Reliance on caretakers for the clinical history is also a problem in cases of fatal abuse as details of the child's condition prior to death may not be accurate. In addition the autopsy findings in cases of accidental or nonaccidental asphyxia are often nonspecific (Byard, 1995; Valdes-Dapena et al, 1993). Thus, the possibility of falsification of aspects of the presentation, the lack of a suggestive clinical history or clinical findings, and the subtlety of pathological features may make determination of the precise cause of death difficult. The readiness to use 'SIDS' as a diagnostic label in such cases of sudden infant death has led to the suggestion that it has been used as a 'convenient diagnostic dustbin' (Emery, 1989). Thus the advantage of having postmortem examinations conducted by a paediatric pathologist, or a forensic pathologist with an interest in paediatrics, is obvious. Close liaison between such individuals and other physicians with specialist training in paediatrics, paediatric radiology and child abuse will only improve the investigation of such unexpected deaths.

Of note, the numbers of reported cases of sudden death due to other conditions occurring in infants and young children who have presented in a manner similar to SIDS have varied greatly. Although a North American report has suggested that nearly 50% of cases of infants being found unexpectedly dead in their cots have diagnoses other

than SIDS made at autopsy, Australian and English studies have found this number to be less than 10% (Byard, Carmichael & Beal, 1994). This variation may be due in part to the rigor with which the infant's medical history is reviewed for evidence of underlying organic illness and to the thoroughness of the death scene examination and parental interviews looking for evidence of accidental and nonaccidental injury. The percentages may, however, be changing with the recent marked fall in the SIDS rates in many communities (for further details on diagnostic difficulties in accidental death see Chapters 7 & 8).

B) REVIEW OF SPECIFIC CATEGORIES

As certain types of fatal accidents in childhood have received quite intensive study, this thesis will not deal in detail with characteristics and safety recommendations that are readily available elsewhere. Thus, the remainder of the chapter will be limited merely to a summary of local data involving motor vehicle accidents, fire and flame deaths, electrocutions, poisonings and a miscellaneous grouping. Detailed studies of other categories may be found in subsequent chapters.

Motor Vehicle Accidents

Review of the 369 cases of accidental childhood deaths since 1963 revealed 189 motor vehicle accidents of which 69 involved pedestrians, 27 passengers, 24 cyclists and 3 motorcyclists (66 cases were unspecified). The mean age of pedestrian fatalities was 7y 5m (M:F = 2:1), compared to passengers who were younger (mean age 4y 9m; M:F = 1:1). Children dying in cycle and motorcycle accidents tended to be older than children in these two groups, reflecting increasing independent mobility with age. The mean age of cylists was 10y 2m (M:F = 2.4:1) and of motorcyclists (including both riders and pillion passengers) was 9y 10m (M:F = 2:1). Case details are summarised in Table 1-2 and Figures 1-2 to 1-5.

South Australian State data for children under 14 years from the National Injury Surveillance Unit (1996) show a decline in childhood deaths from on-road motor vehicle

TABLE 1-2: DETAILS OF 189 CASES OF MOTOR VEHICLE/CYCLE RELATED DEATHS IN CHILDREN AGED FROM 0-16 YEARS IN SOUTH AUSTRALIA FROM 1963-1996

i) **MVA UNSPECIFIED**

No.	Sex	Age	Year	No.	Sex	Age	Year
1.	F	1y	1964	21.	F	2y2m	1972
2.	F	1y7m	1964	22.	F	2y11m	1973
3.	M	3y2m	1964	23.	F	10y2m	1973
4.	F	3y5m	1964	24.	M	2y4m	1973
5.	M	11y	1965	25.	M	7y5m	1974
6.	M	11y6m	1966	26.	F	8y	1974
7.	F	7y6m	1966	27.	F	9y5m	1974
8.	F	2y6m	1966	28.	F	12y8m	1975
9.	M	10y	1966	29.	F	2y10m	1975
10.	M	10y10m	1966	30.	F	7y8m	1975
11.	M	9y4m	1967	31.	M	6y6m	1976
12.	M	5y8m	1968	32.	M	2y3m	1976
13.	F	2y3m	1968	33.	M	2y10m	1976
14.	M	4y1m	1969	34.	M	7y2m	1976
15.	M	10y4m	1969	35.	M	9y4m	1977
16.	M	10y1m	1970	36.	M	4y5m	1978
17.	M	5y4m	1970	37.	M	11y4m	1978
18.	F	3y7m	1970	38.	M	9y11m	1978
19.	F	9y9m	1971	39.	M	1y4m	1978
20.	M	1y11m	1972	40.	F	13y6m	1979

i) **MVA UNSPECIFIED (cont.)**

No.	Sex	Age	Year	No.	Sex	Age	Year
41.	M	6y	1979	54.	M	1y2m	1982
42.	F	8y2m	1979	55.	M	3y11m	1982
43.	M	7y	1979	56.	M	5y5m	1982
44.	F	1y2m	1980	57.	F	1y1m	1982
45.	F	4y3m	1980	58.	M	2y11m	1983
46.	M	1y7m	1980	59.	F	9y6m	1985
47.	F	9y5m	1980	60.	F	9y4m	1986
48.	M	12y11m	1980	61.	F	8y2m	1988
49.	F	4y	1980	62.	F	5y6m	1989
50.	F	14y	1980	63.	M	5y4m	1989
51.	F	5y11m	1982	64.	M	13y6m	1990
52.	M	7y4m	1982	65.	F	9y6m	1991
53.	F	1y1m	1982	66.	M	6y5m	1991

ii) **MVA PASSENGER**

1.	M	6y6m	1964	11.	F	2y11m	1981
2.	F	1m	1967	12.	M	9y7m	1982
3.	M	1m	1971	13.	F	1y3m	1983
4.	M	9y3m	1974	14.	M	2y9m	1983
5.	M	4y9m	1975	15.	F	4m	1983
6.	M	4y10m	1976	16.	M	1m	1984
7.	F	3m	1976	17.	F	3y4m	1984
8.	F	4y10m	1978	18.	F	7y10m	1985
9.	M	10y6m	1980	19.	F	7y10m	1986
10.	M	12y8m	1980	20.	F	3y4m	1987

ii) **MVA PASSENGER (cont.)**

21.	F	13y	1988	25.	F	10y9m	1995
22.	F	2y5m	1989	26.	M	5m	1995
23.	M	5m	1991	27.	F	2y9m	1995
24.	M	6y	1993				

iii) **MVA PEDESTRIAN**

1.	M	6y6m	1964	18.	M	5y7m	1972
2.	M	5y2m	1967	19.	F	7y11m	1972
3.	M	6y8m	1967	20.	M	10y5m	1974
4.	M	7y	1969	21.	M	3y5m	1975
5.	M	8y	1969	22.	F	10y	1975
6.	M	9y6m	1969	23.	M	9y1m	1975
7.	F	11y5m	1970	24.	M	2y7m	1976
8.	F	3y5m	1971	25.	M	9y4m	1976
9.	F	9y4m	1971	26.	M	6y11m	1978
10.	M	3y6m	1971	27.	M	3y4m	1978
11.	M	3y6m	1972	28.	M	3y4m	1978
12.	M	8y	1972	29.	F	8y2m	1979
13	M	4y8m	1972	30.	M	6y	1979
14.	M	3y3m	1972	31.	F	9y5m	1980
15.	M	1y9m	1972	32.	F	4y	1980
16.	M	7y	1972	33.	F	6y11m	1981
17.	F	11y10m	1972	34.	M	8y11m	1981

iii) MVA PEDESTRIAN (cont.)

35.	M	4y1m	1981	53.	M	2y8m	1986
36.	M	11y2m	1981	54.	M	14y6m	1986
37.	M	7y4m	1982	55.	M	6y	1986
38.	M	5y5m	1982	56.	F	3y1m	1986
39.	M	8y6m	1982	57.	M	8y9m	1987
40.	M	3y4m	1983	58.	F	2y7m	1987
41.	F	11y10m	1983	59.	M	8y11m	1987
42.	M	1y6m	1983	60.	M	6y	1989
43.	F	2y10m	1983	61.	F	7y11m	1992
44.	F	2y11m	1983	62.	M	6y5m	1992
45.	M	12y2m	1984	63.	M	5y6m	1994
46.	M	4y4m	1984	64.	M	5y2m	1994
47.	F	7y6m	1984	65.	M	4y3m	1995
48.	F	7y10m	1984	66.	M	3y6m	1995
49.	F	6m	1985	67.	M	4y6m	1995
50.	M	12y9m	1985	68.	M	8y	1996
51.	F	15y11m	1985	69	M	3y	1996
52.	F	7y5m	1986				

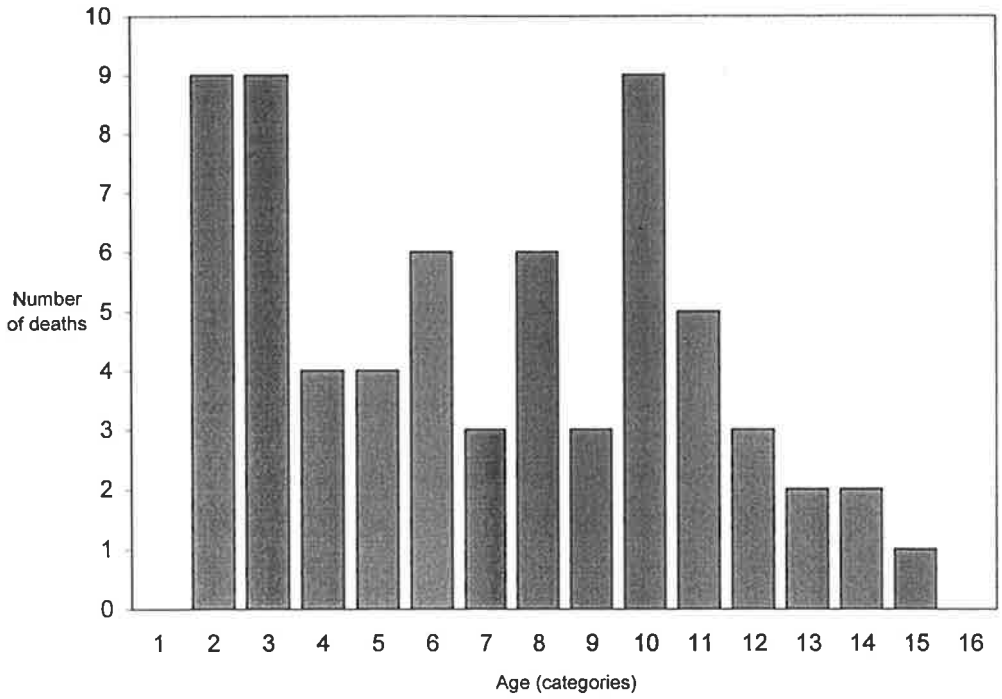
iv) CYCLISTS

No.	Sex	Age	Year	No.	Sex	Age	Year
1.	M	15y8m	1967	4.	M	10y5m	1971
2.	M	8y6m	1969	5.	M	10y3m	1971
3.	M	13y7m	1969	6.	M	11y9m	1971

iv)	CYCLISTS (cont.)						
7.	M	13y6m	1972	16.	F	6y	1981
8.	F	13y	1972	17.	M	5y7m	1981
9.	F	7y6m	1972	18.	F	8y1m	1982
10.	M	6y10m	1975	19.	M	8y5m	1982
11.	F	10y7m	1975	20.	M	7y7m	1982
12.	F	8y6m	1977	21.	F	6y7m	1983
13.	M	13y4m	1977	22.	M	12y5m	1984
14.	M	11y9m	1979	23.	M	11y11m	1984
15.	M	10y3m	1981	24.	M	13y	1987

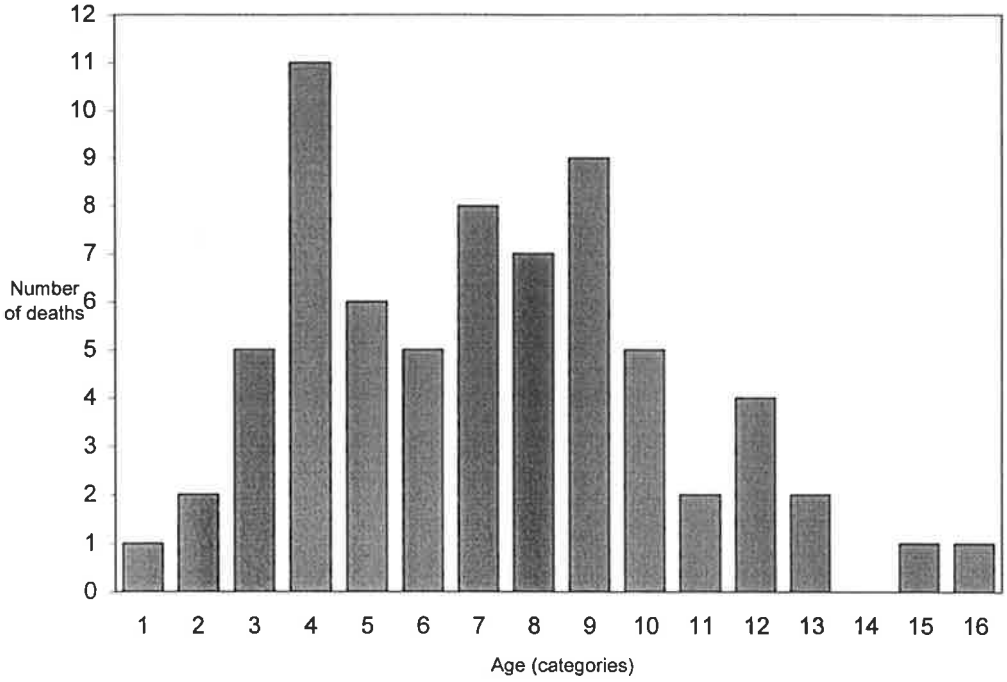
v)	MOTORCYCLISTS						
1.	M	6y6m	1966	3.	F	13y	1996
2.	M	13y	1987				

FIGURE 1-2: MOTOR VEHICLE FATALITIES (DETAILS UNSPECIFIED) - NUMBER AND AGE OF VICTIMS (0-16y) TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH FROM 1963-1996 IN SOUTH AUSTRALIA



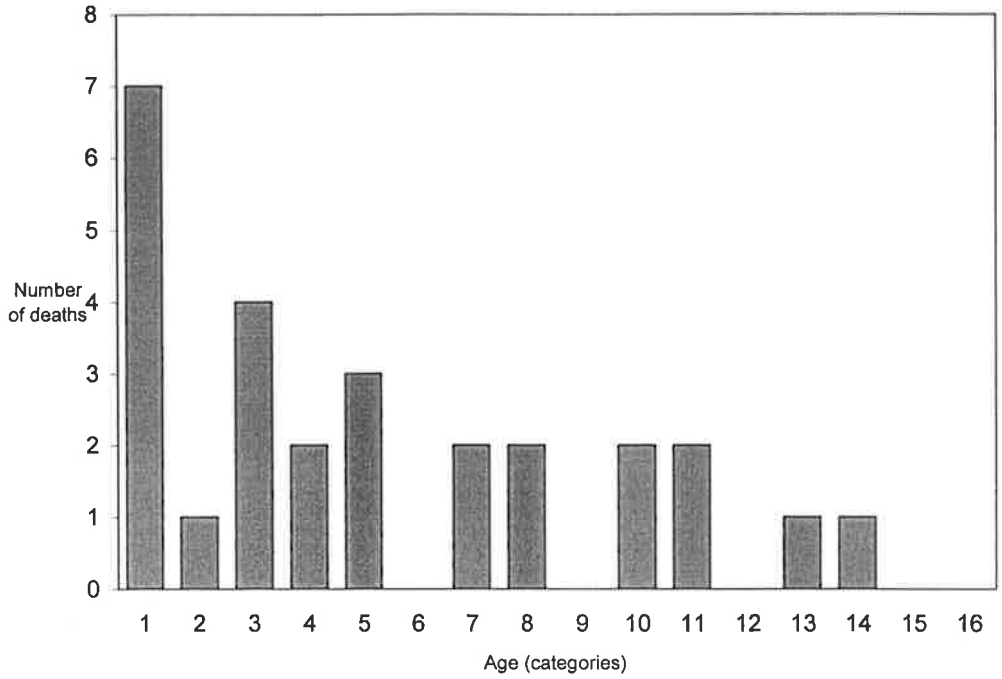
AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

FIGURE 1-3: MOTOR VEHICLE PEDESTRIAN FATALITIES - NUMBER AND AGE OF VICTIMS (0-16y) TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH FROM 1963-1996 IN SOUTH AUSTRALIA



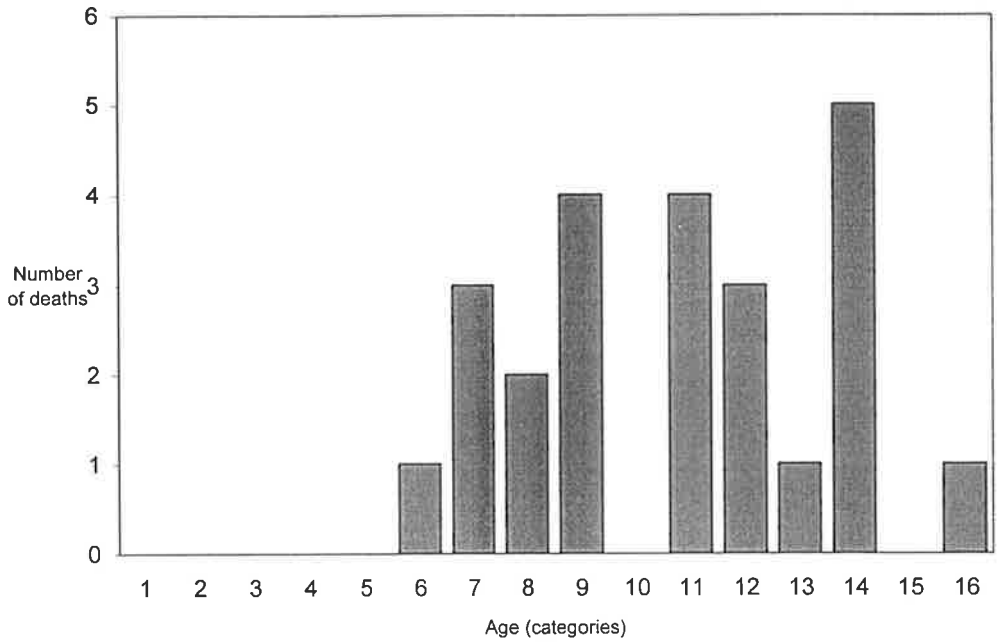
AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

FIGURE 1-4: MOTOR VEHICLE PASSENGER FATALITIES - NUMBER AND AGE OF VICTIMS (0-16y) TAKEN FROM 369 CASES OF ACCIDENTAL CHILDHOOD DEATH FROM 1963-1996 IN SOUTH AUSTRALIA



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

FIGURE 1-5: NUMBER AND AGE OF CASES OF CYCLIST FATALITIES TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16Y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

accidents with 19 'Transportation' deaths registered for 1990, 9 for 1991, 15 for 1992, 12 for 1993 and 7 for 1994. Again there is a dramatic difference in the number of these deaths in the 15 to 24 year age range, in which there were 70 deaths in 1990 and 51 deaths in 1994 (National Injury Surveillance Unit Data, 1996).

In children under 14 years of age in South Australia there were four motor vehicle passenger deaths in 1991, 8 in 1992, 8 in 1993, and 2 in 1994. Over this time there were two deaths of children aged between 10 and 14 years who were driving on public roads and four deaths of cyclists aged between 5 and 14 years (National Injury Surveillance Unit Data, 1996).

Fire Deaths and Burns

In the local data there were 4 house fire deaths and 6 deaths due to scalding, out of a total of 24 fire/scald deaths (6.5% of the 369 cases). The age range was from 1y 1m to 14y 2m with a mean age of 5y 1m (M:F = 1:1). Case details are provided in Table 1-3. Figure 1-6 demonstrates a preponderance of younger children under the age of 7 years. Four of the fatalities involved either the victim or a sibling playing with matches or a cigarette lighter. There were 5 deaths in car fires and death in 3 cases resulted from clothing catching fire.

The safety messages are clear; matches and hot water should be kept away from young children; flammable clothing should not be used and young children in cars should be supervised and not allowed near cigarette lighters.

Poisoning

In the present study, 10 cases of fatal poisoning were found, representing 3% of the total number of accidents. The ages ranged from 1y 1m to 3y 3m with a mean age of 2y 6m, and a M:F ratio of 1:1. Six fatalities resulted from accidental overdoses of pharmaceutical substances, 2 involved insecticides, and 2 involved other toxic substances. Case details are summarised in Table 1-4 and in Figure 1-7.

Electrocution

A total of 6 cases of electrocution were identified ranging in age from 2 to 12y (mean = 5y 8m; M:F = 2:1). Causes of electrocution included a faulty airconditioner, a faulty washing machine, a fall into a transformer and an electric hair curling wand falling into a bath. Details of the circumstances of death were not available in two cases. Case details are summarised in Table 1-4 and autopsy findings are illustrated in Figures 1-8 and 1-9.

Miscellaneous

Details of fatalities involving other forms of asphyxia, sporting injuries, industrial accidents, falls and train accidents are summarised in Table 1-4.

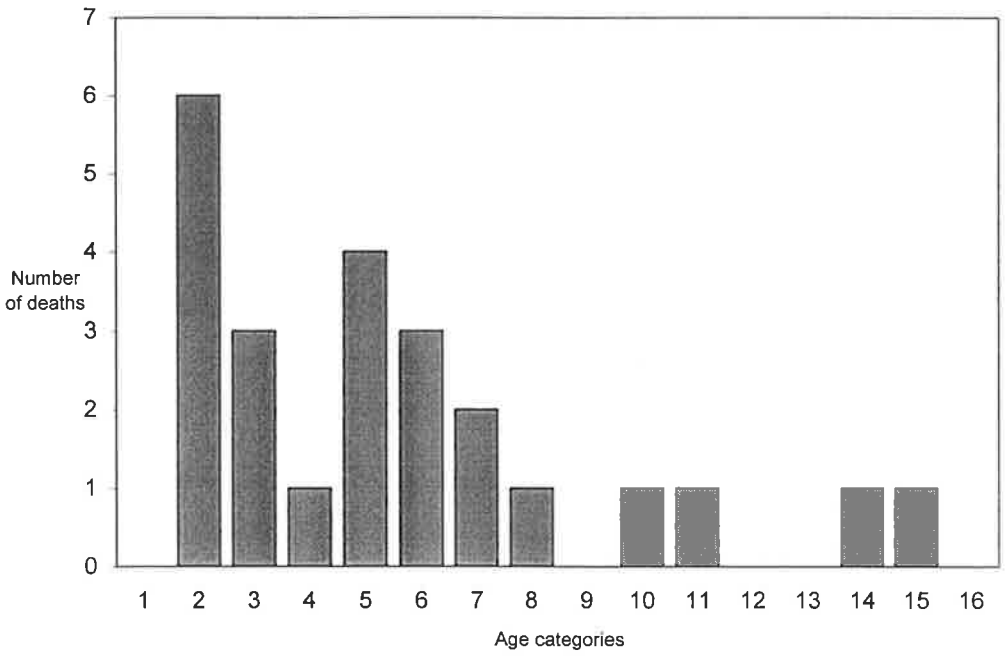
* * * *

TABLE 1-3: DETAILS OF 24 CASES OF FIRE/FLAME/SCALD DEATHS IN CHILDREN AGED BETWEEN 0 AND 16 YEARS IN SOUTH AUSTRALIA FROM 1963 TO 1996

No.	SEX	AGE	YEAR	CIRCUMSTANCES
1.	F	4y9m	1963	House fire
2.	F	2y4m	1963	Scald in bath
3.	F	3y	1963	Kerosene fire
4.	M	10y6m	1964	Unspecified fire
5.	F	5y	1966	Petrol fire
6.	M	4y2m	1966	Clothes on fire
7.	F	1y10m	1966	Scald (bucket of hot water)
8.	F	6y11m	1966	Clothes on fire (cigarette lighter)
9.	F	1y6m	1966	Scald in bath
10.	F	4y5m	1966	Clothes on fire (matches)
11.	M	13y	1967	Unspecified burns
12.	M	1y9m	1969	Scald
13.	F	5y10m	1969	Petrol fire
14.	F	7y11m	1969	Petrol fire
15.	M	9y11m	1978	Car fire
16.	M	1y1m	1982	Scald
17.	M	1y6m	1984	House fire (sibling - cigarette lighter)

TABLE I-3: BURNS (cont.)				
18.	F	14y2m	1988	Scald
19.	M	6y	1993	Car fire
20.	M	5y9m	1994	House fire
21.	F	2y8m	1994	House fire
22.	M	1y9m	1994	Car fire (sibling - matches)
23.	F	2y8m	1996	Car fire
24.	M	4y	1996	Car fire

FIGURE 1-6: NUMBER AND AGES OF CASES OF BURN/FLAME/SCALD FATALITIES TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

TABLE 1-4: DETAILS OF MISCELLANEOUS CATEGORIES OF ACCIDENTAL DEATHS IN CHILDREN AGED BETWEEN 0 AND 16 YEARS IN SOUTH AUSTRALIA FROM 1963 TO1996

No.	SEX	AGE	YEAR	CIRCUMSTANCES
i)	**POISONING (N = 10)**			
1.	M	1y3m	1964	Amyl acetate ingestion
2.	M	3y	1964	Selenium ingestion
3.	F	2y7m	1966	Aspirin ingestion
4.	M	1y1m	1966	Soldering flux (KF) ingestion
5.	M	1y8m	1969	Pheniramine ingestion
6.	F	1y10m	1971	Dibenzepin ingestion
7.	F	2y5m	1972	Amitriptyline ingestion
8.	F	1y6m	1973	Insecticide ingestion
9.	F	6y	1973	Promethazine ingestion
10.	M	3y3m	1990	Insecticide (thiodan) ingestion
ii)	**ELECTROCUTION (N = 6)**			
1.	F	2y	1970	Faulty washing machine
2.	M	12y	1973	Fall onto a transformer
3.	M	2y	1979	Electric curling wand in bath
4.	M	6y	1979	Not specified
5.	M	7y	1981	Faulty airconditioner
6.	F	5y	1987	Not specified

iii) OTHER ASPHYXIA (N = 5)

1.	M	9y	1978	Hanging
2.	M	3y2m	1981	Traumatic asphyxia in factory
3.	M	7y	1985	Crush asphyxia
4.	M	5y	1991	Crush asphyxia
5.	M	3y	1996	Crush asphyxia

iv) SPORTING INJURIES (N = 4)

1.	M	11y7m	1967	Head injury
2.	M	7y	1976	Head injury
3.	M	3y9m	1978	Head injury
4.	M	14y10m	1991	Javelin injury

v) FALLS (N = 2)

1.	M	3y2m	1978	At home
2.	F	14y	1996	From horse

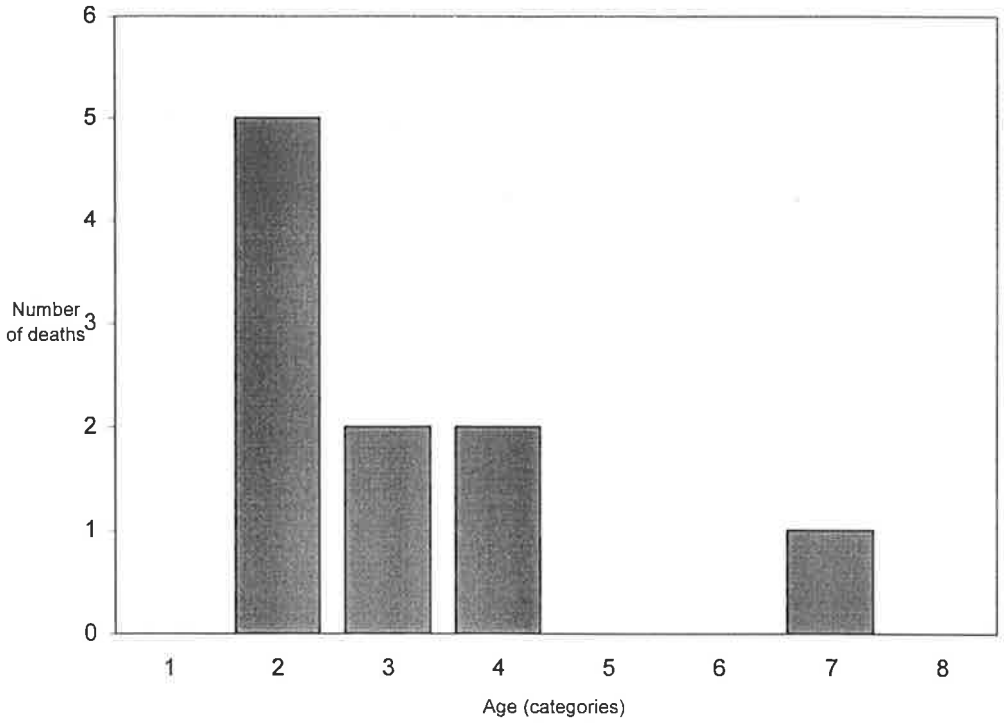
vi) INDUSTRIAL (N = 1)

1.	F	9y6m	1991	Forklift truck injury

vii) TRAIN (N = 1)

1.	M	12y4m	1979

FIGURE 1-7: NUMBER AND AGE OF FATAL POISONINGS TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

Figure 1-8: Burns of the inner aspects of the fingers in a child who was electrocuted after grasping a live wire.

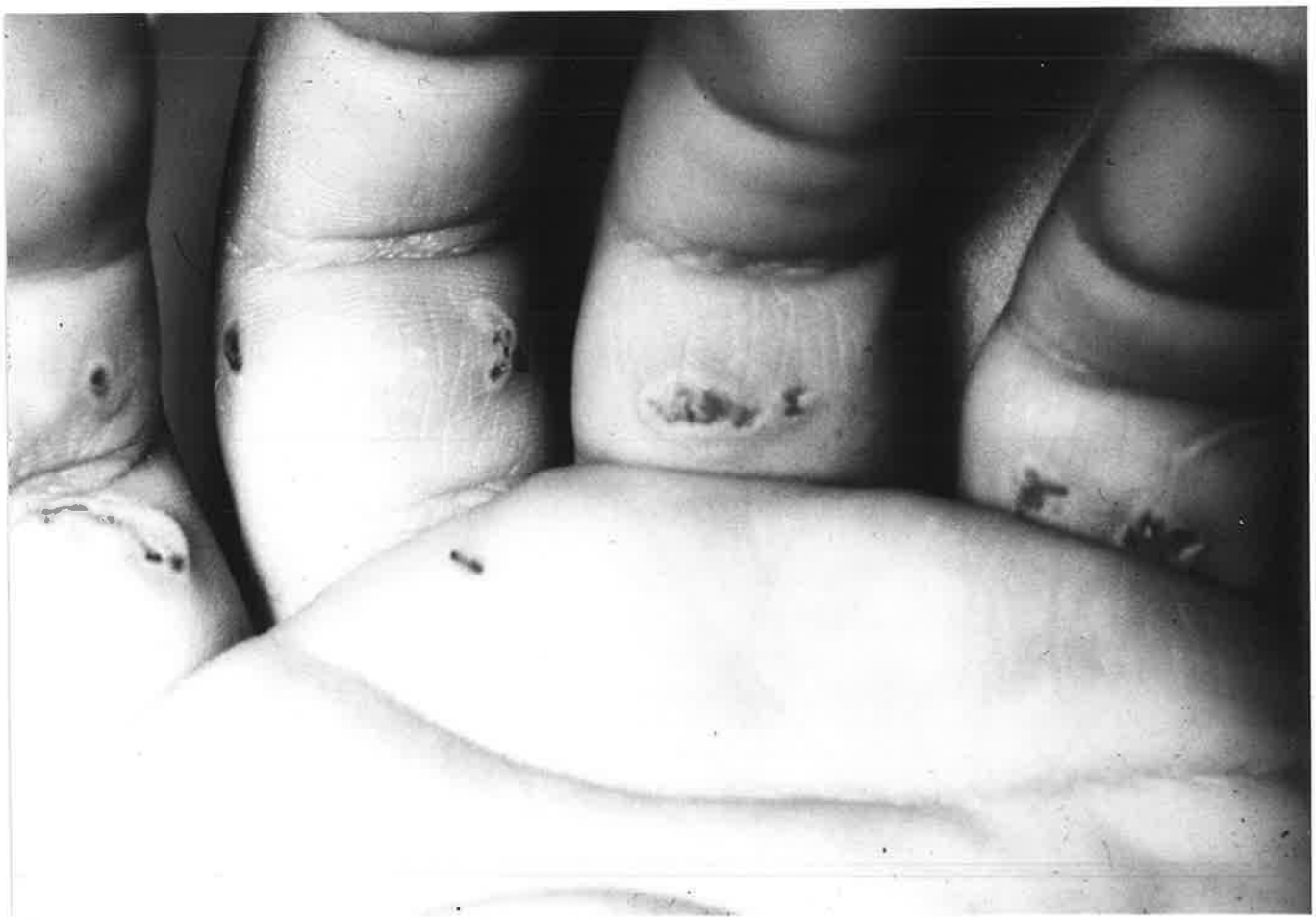
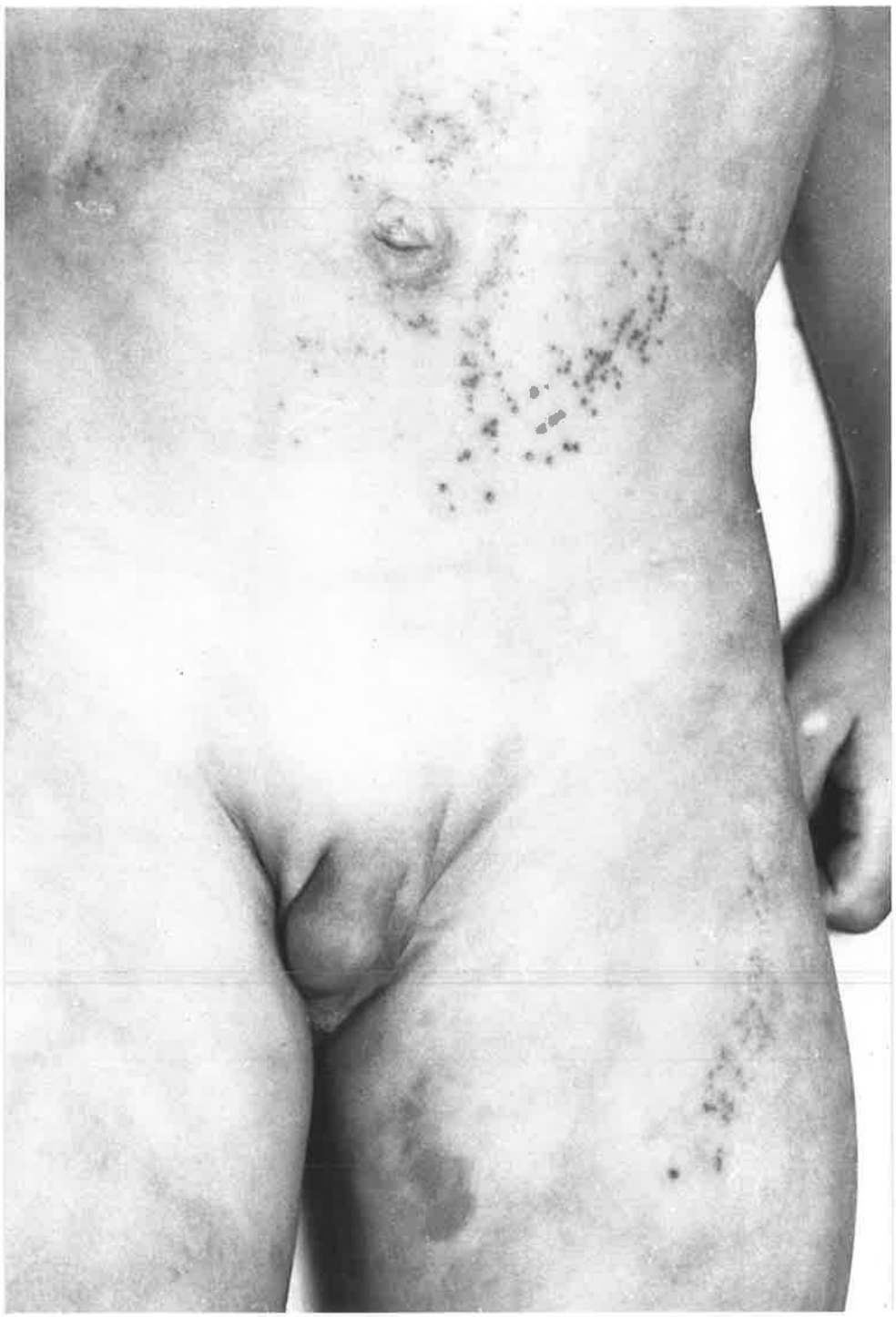


Figure 1-9: Exit burns on the abdomen and upper leg of a 4y-old boy who touched live power lines while playing on the roof of a shed.



CHAPTER 2

HAZARDOUS SLEEPING ENVIRONMENTS

INTRODUCTION

The environment where infants sleep is an example of a potentially hazardous situation which may result in accidental death from asphyxia (Bass, 1977; Byard, Beal & Bourne, 1994; Smialek, Smialek & Spitz, 1977). As the autopsy findings in cases of asphxyia in this age group are often entirely nonspecific, determination of the manner of death may therefore rely more on an assessment of the death scene/sleeping environment than on postmortem dissection (Beal & Byard, 1995).

MATERIALS AND METHODS

As the pathological features and circumstances of death of infants and young children who accidentally asphyxiated were dealt with briefly in a previous thesis (Byard, 1994b; pp 87-102, 238), the main body of the following study deals only with recent cases which were not included in that study.

Specifically, this study deals with cases of sudden infant and early childhood death caused by unsafe sleeping environments, taken from the records of the Department of Histopathology at the Women's and Children's Hospital for the 4 year period from January 1993 to December 1996. Circumstances of death and autopsy findings were reviewed in detail. Death scene visits and/or reconstructions were undertaken by the author in 8 of the 14 cases.

RESULTS

A total of 40 cases of accidental asphyxia resulting from unsafe sleeping circumstances were identified over the 34 years of the study. These included 20 wedging/positional asphyxias, 13 hangings, 4 suffocations and 3 overlayings. The average age of death of 10m (age range 1m to 4y; M:F = 1.7:1) demonstrates an age-related vulnerability.

In the specific subgroup of 14 cases of accidental asphyxial deaths from 1993 to 1996 being analysed, the age range was 1m to 4y. If the oldest child in this group is excluded because of additional complicating features of profound mental retardation and physical immobility, the age range is 1m to 18m, with an average age of 5.6m.

Deaths in this group involved hanging (N = 4), wedging between a mattress and a mesh cot/bed side (N = 2), suffocation under adult 'V' pillows (N = 2), wedging between adult beds and the wall (N = 2), entrapment between a defective sliding cot side and a mattress (N = 1), and overlaying (N = 3). The case details are summarised in Table 2-1 and illustrated in Figures 2-1 to 2-7

Details of the remaining 26 cases of accidental asphyxia related to unsafe sleeping circumstances over the years 1963 to 1992 may be found in Table 2-2. Figure 2-8 provides a breakdown of the number of deaths for each year of life.

DISCUSSION

The most recent group of accidental asphyxial infant and toddler deaths is of particular concern as these fatalities have occurred in spite of publicising of such preventable occurrences in the literature, with local media attention. Failure to eradicate such preventable fatalities is not, however, restricted to South Australia. For example, in 1977 two series of accidental paediatric asphyxial deaths in the United States were published which involved 13 and 15 children, respectively (Bass, 1977; Smialek, Smialek & Spitz, 1977). As with the more recent report (Byard, Beal & Bourne, 1994), attention was drawn to dangerous sleeping environments for infants, and recommendations were made for improving bed and cot safety. One author had also produced an illustrated pamphlet for parents detailing potentially lethal situations (Bass, 1977). In spite of this information, preventable infant asphyxial deaths still occur in the United States (Bass, Kravath & Glass, 1986; Byard & Cohle, 1994), as they do in South Australia. The continued occurrence of similar deaths suggests that parents and child carers are either not receiving information on dangerous sleeping environments, or are not acting appropriately on this advice when it is made available.

Wedging deaths involve the victims slipping between an adult's or older child's mattress and a wall, between a thick cot mattress and cot side, or between sliding side railings of a cot and the mattress/cot base (Figure 2-3). Both gravity and movement of the infant assist in the downward displacement of the body into these narrow spaces.

TABLE 2-1: DETAILS OF 14 CASES OF ACCIDENTAL ASPHYXIA IN INFANCY AND EARLY CHILDHOOD ASSOCIATED WITH UNSAFE SLEEPING ENVIRONMENTS IN SOUTH AUSTRALIA FROM 1993 TO 1996

No.	Sex	Age	Year	Circumstances
1.	F	3m	1993	Hanging from baby bouncer
2.	F	10m	1993	Wedged between adult bed & wall
3.	F	6m	1993	Head wedged between adult bed & wall
4.	M	16m	1994	Hanging from child car seat belt
5.	M	3m	1995	Hanging from stroller pram
6.	M	3m	1995	Suffocated under 'V' shaped pillow
7.	M	1m	1995	Overlayed in parent's bed
8.	M	3m	1995	Wedged down side of mesh sided cot
9.	M	18m	1995	Hanging from cord attached to door knob
10.	M	2m	1995	Suffocated under 'V' shaped pillow
11.	F	8.5m	1996	Wedged down side of mesh sided cot
12.	M	4y	1996	Wedged between mesh bed side and mattress (mentally retarded)
13.	M	1m	1996	Overlayed while being breastfed (mother asleep)
14.	M	1m	1996	Overlayed while being breastfed (mother asleep)

Figure 2-1: Reconstruction of the position in which a 3m-old girl was found after having been left unattended for 2 hours in a baby bouncer. Death was due to accidental hanging.

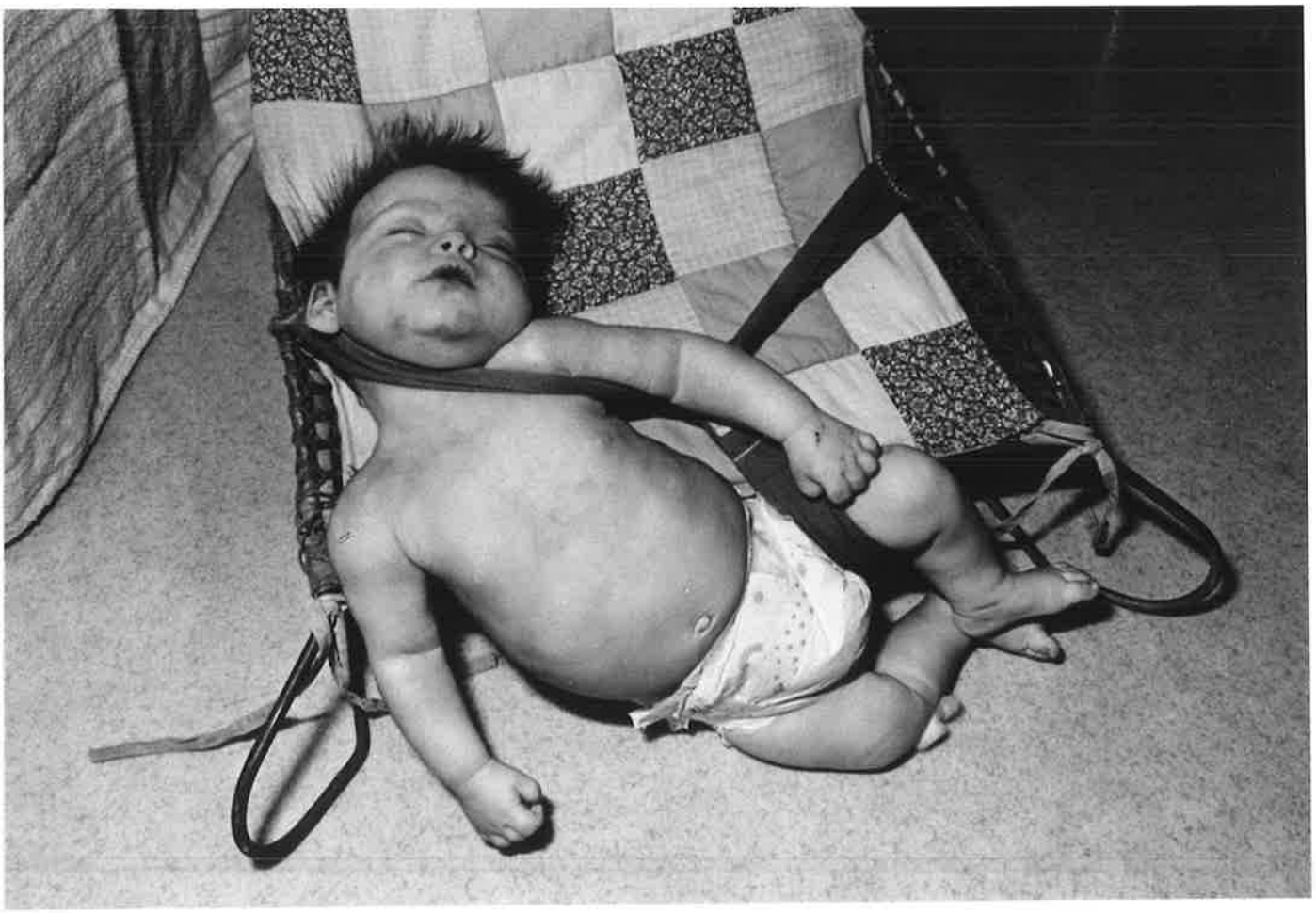


Figure 2-2: A 16m-old boy who was left asleep for 20 minutes in a child restraint harness in a car. The cause of death was neck compression due to hanging. Facial petechiae are visible and the arrow indicates the position of a product safety label which specifically warns against leaving a child in the harness unattended. Although folded under his chin in this photograph, the label was normally quite clearly displayed. Further safety information is present in a label above the boy's left ear.

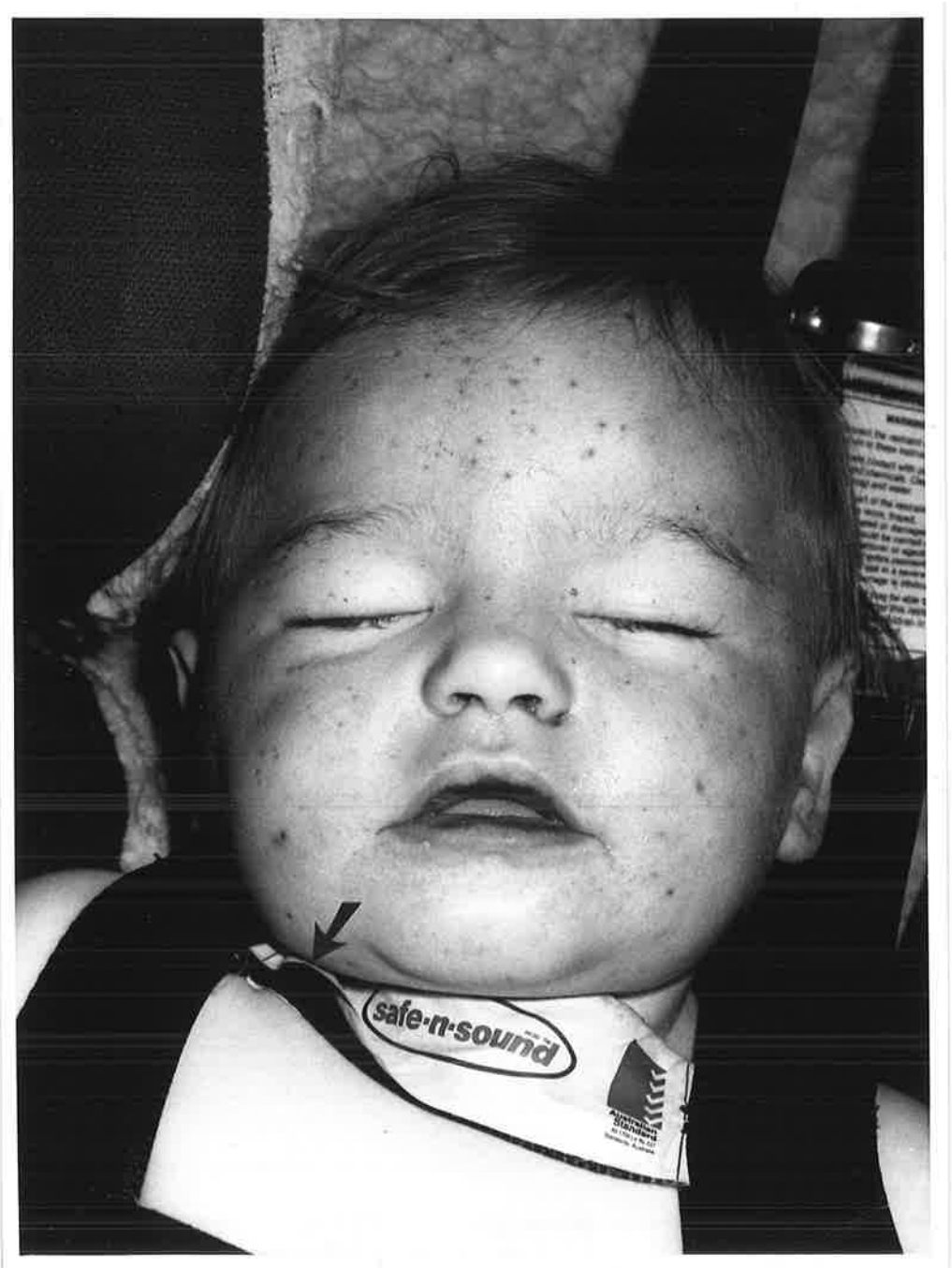


Figure 2-3: A cot in which an 8.5m-old girl was found wedged between the sliding side and the mattress and frame. The large arrow indicates the unrestrained bottom of the sliding side which has also pulled free of the metal guiding rod (A). The position where the body was found with the face pointing inwards, is indicated by the toy bear (*) (B). A similar case was recently referred for opinion from interstate, and an almost identical situation is shown in Figure 3 of the paper by Smialek et al in 1977.


B
*

Figure 2-4: A typical ligature mark with facial petechiae in a 16m-old boy who was hanged. No conjunctival petechiae were present.

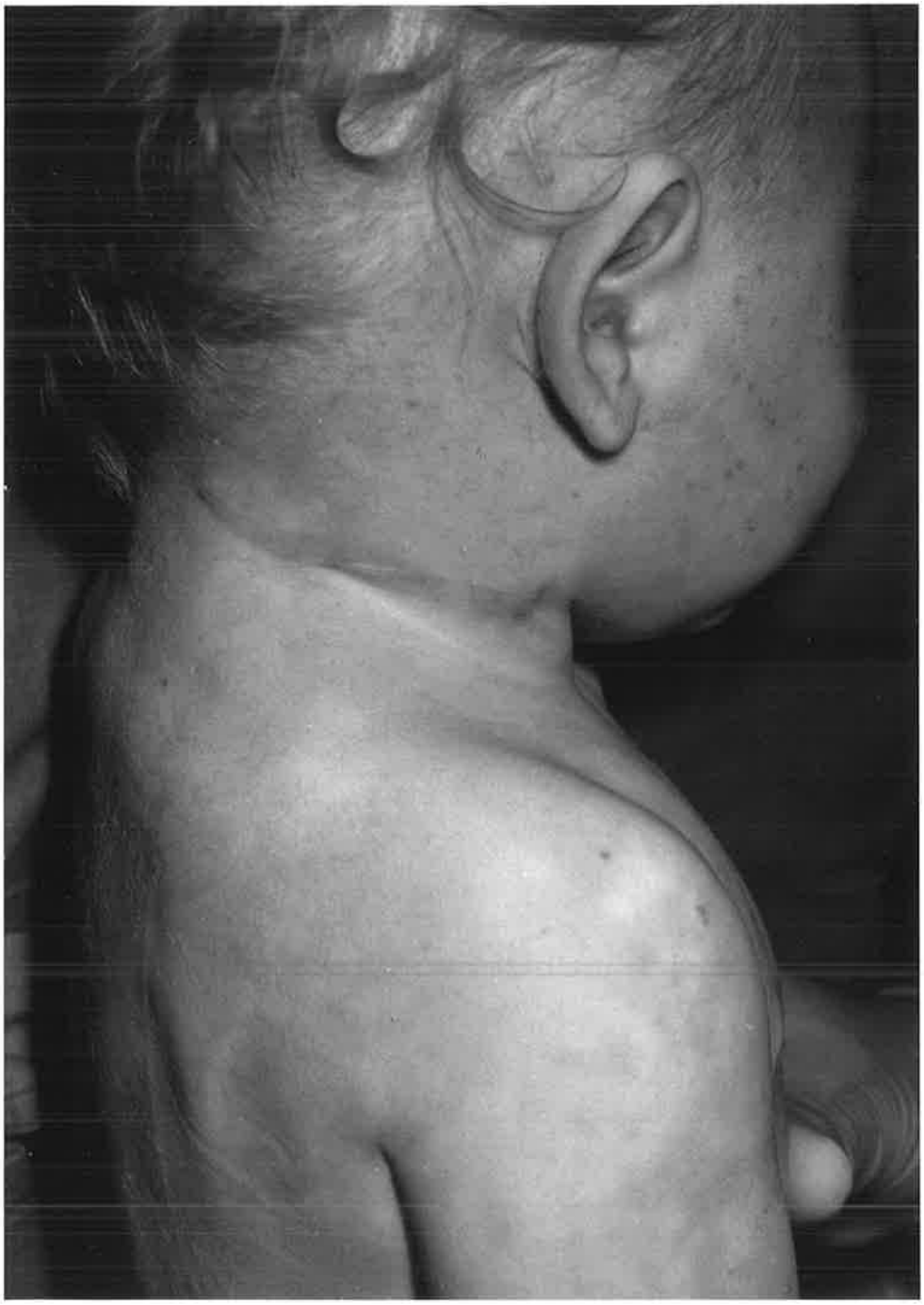


Figure 2-5: Linear marks caused by the edge of a mattress on the face of a 10m-old girl who was found wedged between her parent's mattress and the bedroom wall.

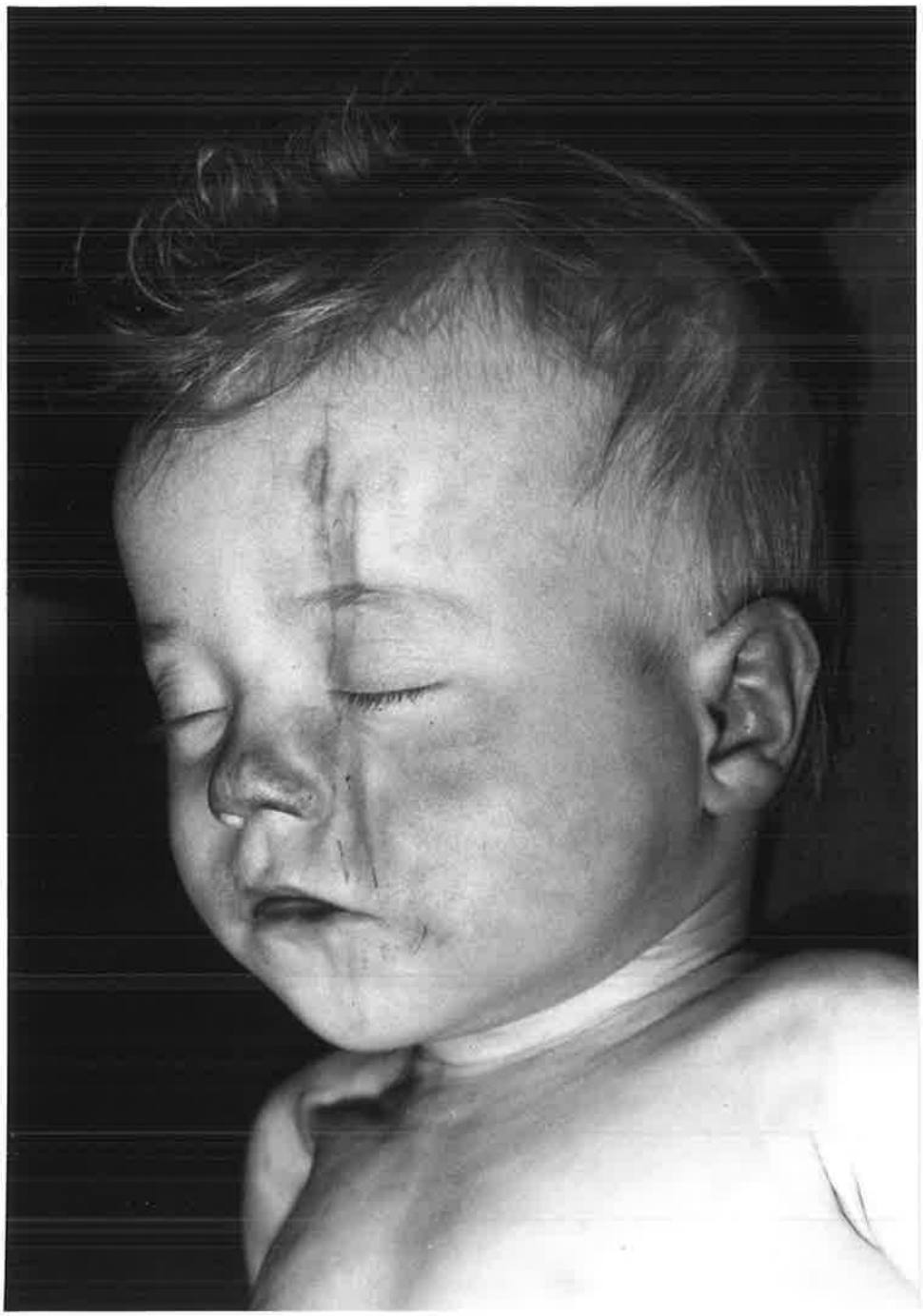


Figure 2-6: Two very faint linear bruises on the back of an infant's head (seen more clearly in the insets at higher magnification) correspond to wooden bars which were holding the infant's head into the side of the mattress. The 6 cm distance between the marks corresponds exactly with the distance between the wooden bars on the side of the cot.

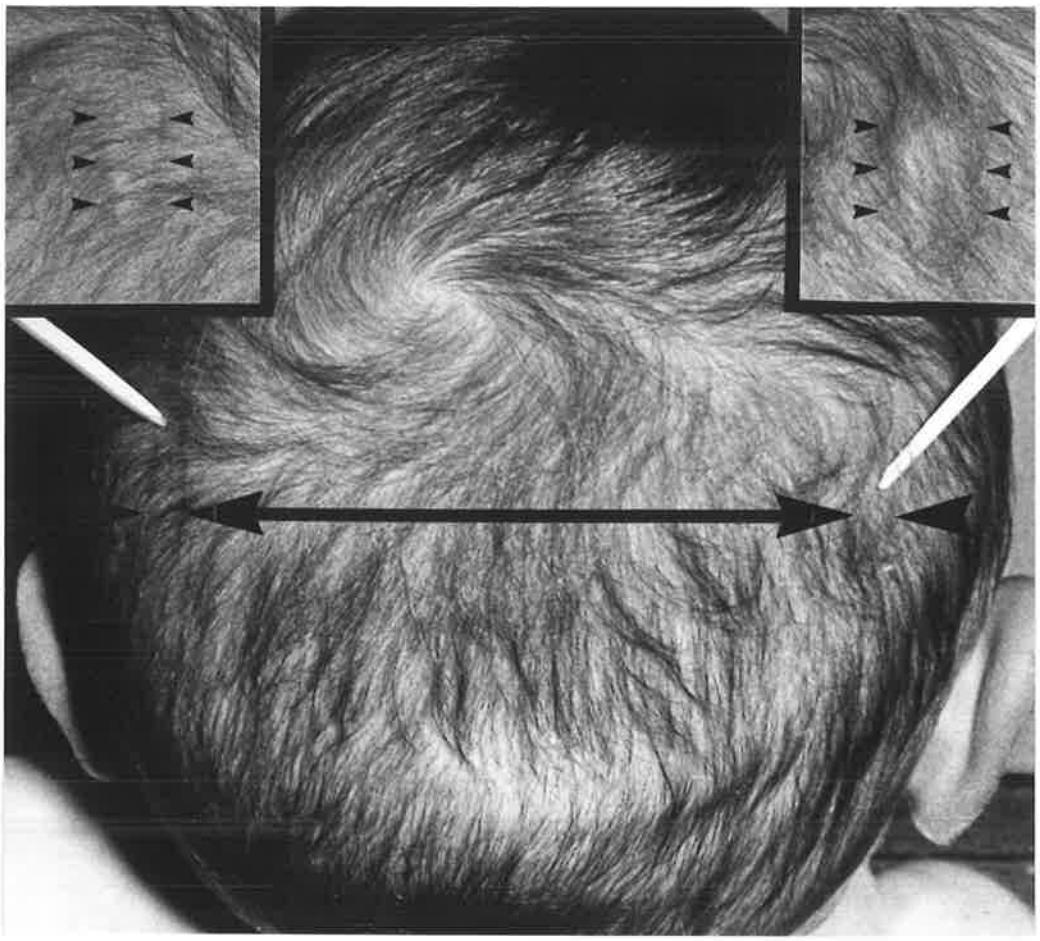
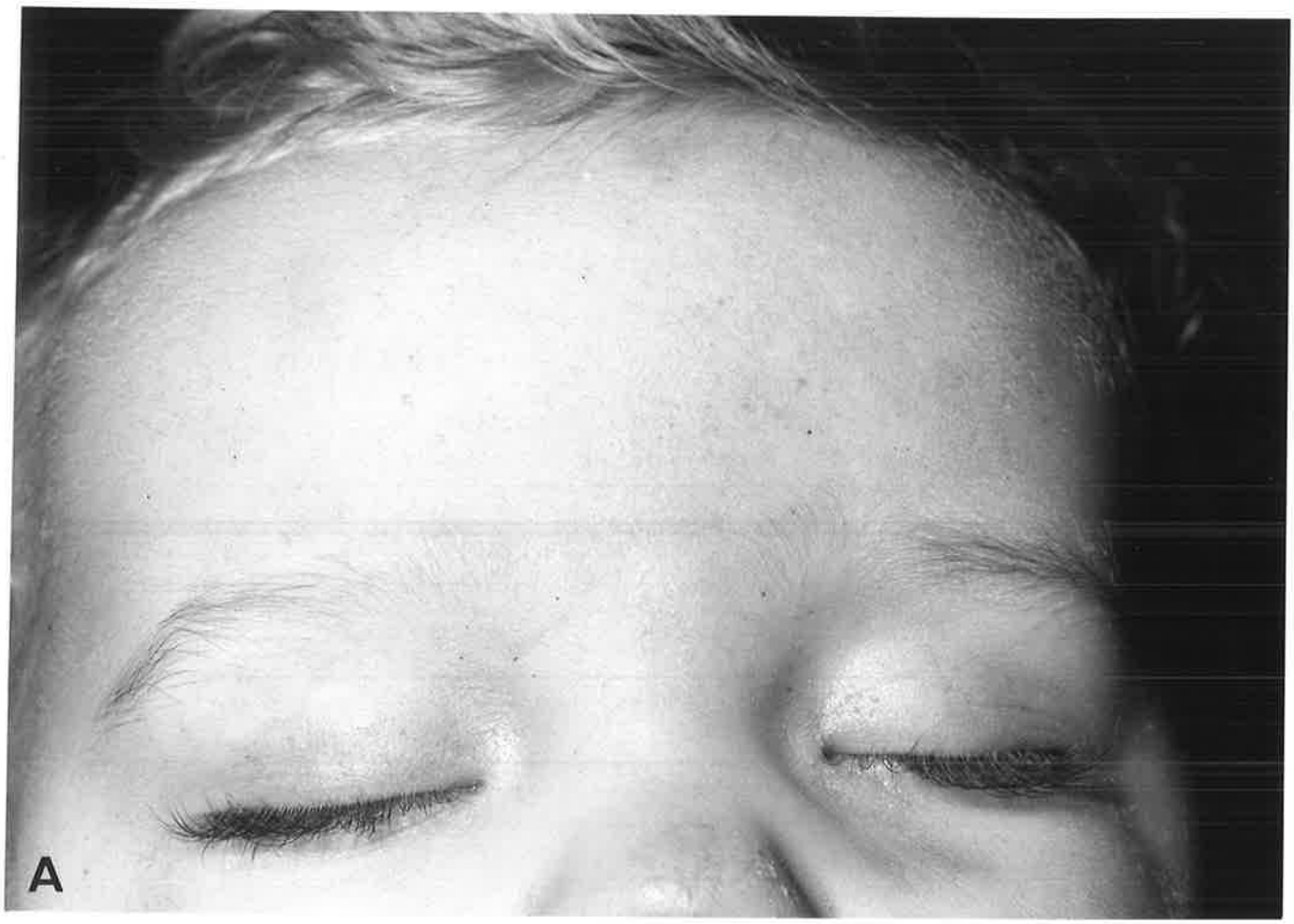


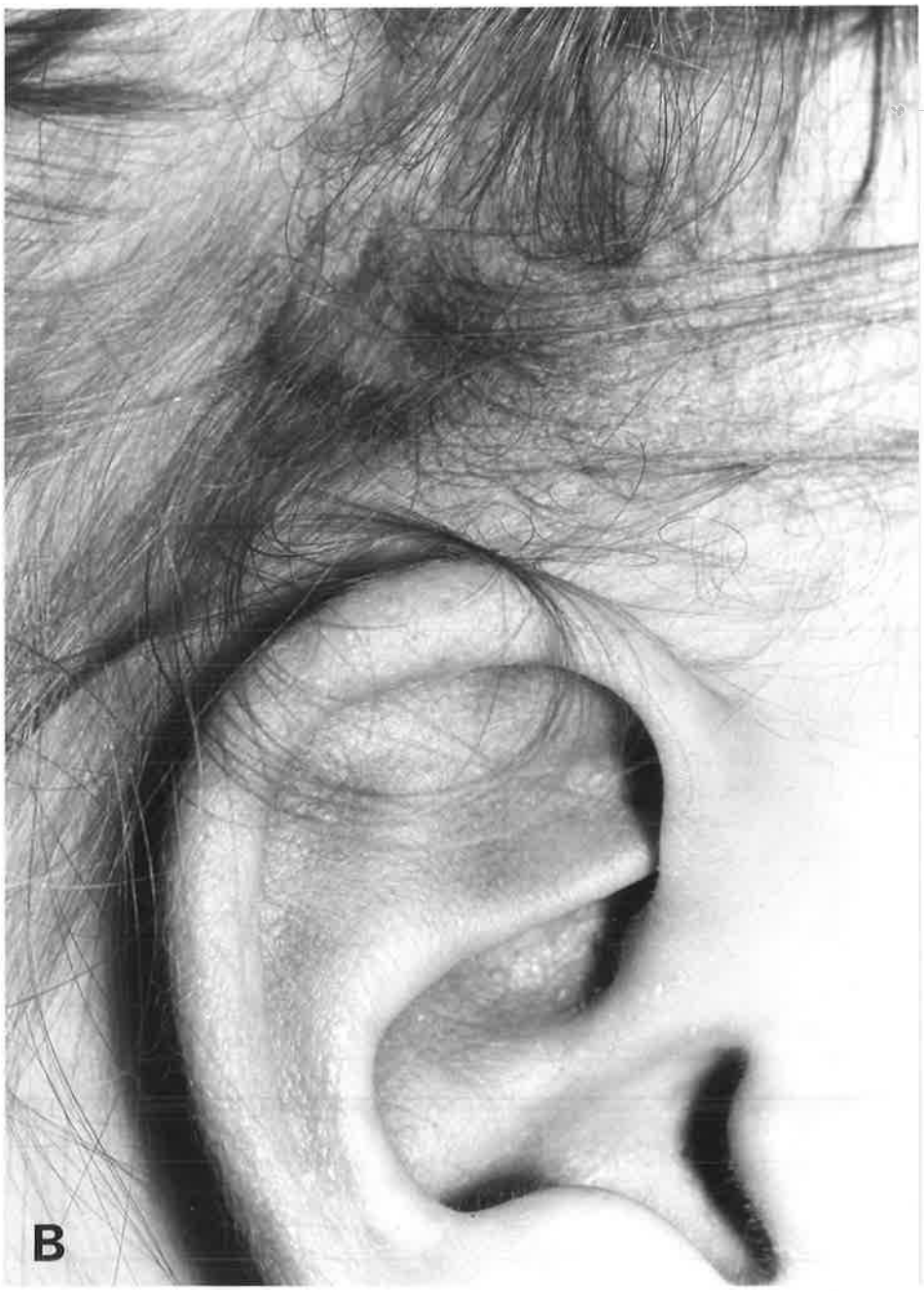
Figure 2-7: Autopsy findings in cases of accidental asphyxia in infants and young children may be very subtle. For example pathological features in the following three infants were only demonstrable after a careful examination had been undertaken:

(A) Faint petechiae over the forehead and upper eyelids in an 8 1/2m-old girl who died from hanging;

(B) Bruising over the right ear caused by head entrapment in a 9m-old boy who was found wedged between a mattress and cot side;

(C) Bruising over the base of the occiput in a 10m-old boy also caused by head entrapment in similar circumstances to the above case.




B

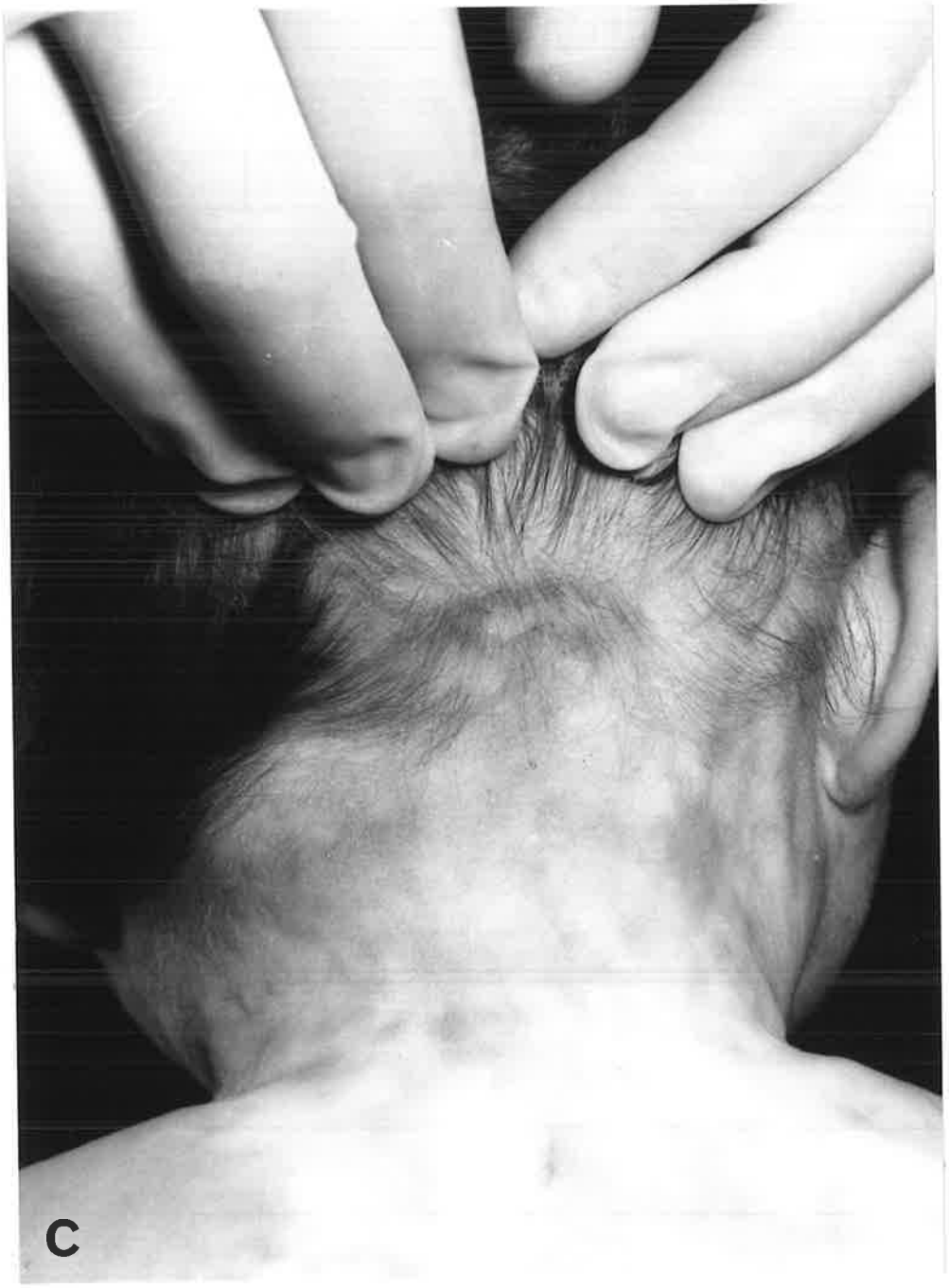

C

TABLE 2-2: DETAILS OF 26 CASES OF ACCIDENTAL ASPHYXIA ASSOCIATED WITH UNSAFE SLEEPING ENVIRONMENTS IN CHILDHOOD IN SOUTH AUSTRALIA FROM 1963 TO 1992 (SEE POSTSCRIPT*)

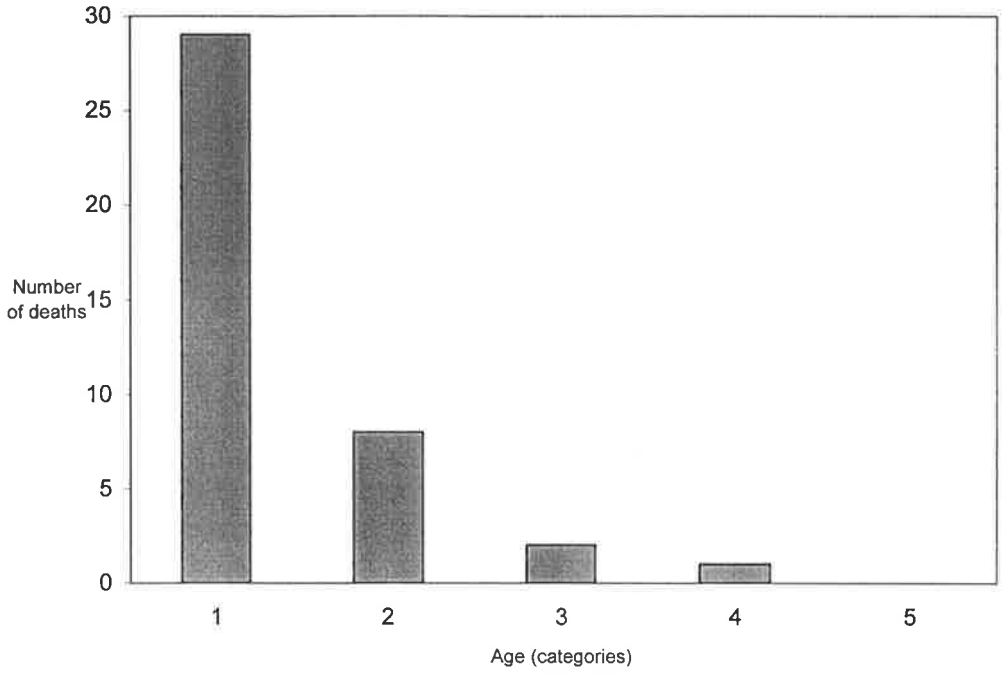
No.	SEX	AGE	YEAR	MECHANISM
1.	M	3y	1966	Hanging
2.	M	1y6m	1970	Hanging
3.	F	8m	1970	Wedging/positional asphyxia
4.	F	8m	1974	Hanging
5.	M	1y6m	1976	Hanging
6.	M	6m	1976	Wedging/positional asphyxia
7.	F	1m	1978	Suffocation (plastic sheet)
8.	F	8m	1978	Hanging
9.	M	7m	1981	Hanging
10.	M	8m	1981	Wedging/positional asphyxia
11.	M	8m	1981	Wedging/positional asphyxia
12.	F	3y	1982	Hanging
13.	M	1y2m	1983	Wedging/positional asphyxia
14.	F	1y3m	1984	Hanging
15.	F	11m	1984	Hanging
16.	M	10m	1985	Wedging/positional asphyxia
17.	M	8m	1986	Wedging/positional asphyxia
18.	F	1m	1987	Suffocation (plastic sheet)
19.	F	5m	1988	Wedging/positional asphyxia
20.	M	3m	1989	Wedging/positional asphyxia
21.	M	3m	1989	Wedging/positional asphyxia
22.	M	1m	1989	Wedging/positional asphyxia

TABLE 2-2: ACCIDENTAL ASPHYXIA (cont.)

23.	M	1y	1991	Wedging/positional asphyxia
24.	F	1y2m	1991	Wedging/positional asphyxia
25.	F	3m	1992	Wedging/positional asphyxia
26.	M	3m	1992	Wedging/positional asphyxia

*POSTSCRIPT: Cases of accidental asphyxia from 1964 to 1992 have been included for completeness, but have not been described in detail as their clinicopathological features were discussed in the thesis 'Sudden Natural Death in Infancy and Childhood - an Analysis of Aetiological Mechanisms & Pathological Features' (Byard, 1994c, pp. 87-102, 238). A more detailed description of cases taken only from 1993 to 1996 is given in Table 2-1. The two groups have been pooled when analysis of age/sex trends has been made, for example in Figure 2-8.

FIGURE 2-8: NUMBER AND AGE OF 40 CASES OF ACCIDENTAL ASPHYXIA RELATED TO DANGEROUS SLEEPING ENVIRONMENTS TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

Immature motor coordination and poor muscle development prevent particularly young infants from extricating themselves. Factors resulting in death include not only wedging with splinting and compression of the chest, but also obstruction of the nose and mouth, and hanging (Byard, 1996a). A combination of airway obstruction and exhaustion may play a role in deaths where infants are suspended by their heads between the side railings of the cot and the mattress when there is hyperextension of the neck and no apparent obstruction to the nose and mouth. Cots with defective construction, for example, with excessive gaps between railings or between the sides and mattress, may also result in such deaths (Variend & Usher, 1984).

Hanging usually involves infants and toddlers who slip after being left to sleep in restraining harnesses, and toddlers who are able to stand and reach curtain cords or catch their clothing on projections within the cot. Additional dangers are cords attached to toys or pacifiers which may wrap around infants' necks. The first group includes victims who have been left asleep in bouncinettes (baby bouncers) or in car seat harnesses (Figures 2-1 & 2-2). A feature common to all is that the victims have been left unsupervised for variable periods of time.

Obstruction of the mouth and nose may interfere with breathing until death occurs from suffocation. This may happen, for example, when thin plastic sheeting has been used to cover pillows and mattresses, or where infants are lying face down in the trough formed on the surface of incompletely filled water beds (Gilbert-Barness et al, 1991).

Although there are suggestions that infants who sleep under a thick layer of bedding are at increased risk of sudden death, it is difficult to determine the contribution to the lethal episode of this type of sleeping environment. Certainly, overheating, carbon dioxide rebreathing and prone sleeping position, all of which may occur in this situation, have been implicated as risk factors in sudden infant death (Byard, 1995; Kemp & Thach, 1991; Ponsonby et al, 1993). However, given the considerable uncertainties that exist in determining fatal mechanisms in these cases, it has been recommended that deaths in infants who are found sleeping prone with no

indications of asphyxia should still be classified as SIDS (provided that all of the standard criteria are met) (Byard et al, 1996).

Cases of repetitive parentally-induced asphyxia have been well documented under the rubric Munchausen syndrome by proxy (Samuels et al, 1992). In these cases apnoeic episodes are produced in an infant which result in considerable medical attention and investigation (Byard & Beal, 1993). A common presentation of these infants is as an unexpected death indistinguishable from SIDS, except for previous infant deaths within the family and a history of resuscitation for ALTEs, sometimes with recent hospital investigation. Statements from grandparents may sometimes be extremely helpful in cases where nonaccidental asphyxia is suspected. The author is not aware of any such cases being disguised as accidental deaths such as hanging or wedging.

Although the ideal situation in investigating cases of accidental infant death would be for the pathologist to attend the death scene, this is not always possible. In addition to usual death scene protocols, therefore, adequate investigation requires a careful description of the position of the body when found, as well as the position that the infant had been in when last checked. Diagrams of the sleeping arrangements and colour photographs of the bed, bedding and room should be taken. It is extremely valuable to have photographs of a manikin or doll placed in the position of the body and to have the cot brought to the mortuary for assessment with the body.

In Australia standards exist which recommend that new cots meet minimum safety requirements, for example, with respect to the distance between side bars and the presence of potentially hazardous projections within cots. However, such standards are not mandatory, nor can they be applied effectively to second hand, home made or broken cots. An example of a defective home constructed cot which resulted in the wedging death of an 8.5m-old girl is shown in Figure 2-3. The side of the cot was designed to slide on metal rods, but unfortunately the bottom ends of the rods were not secured, resulting in the lower edge of the side swinging out, thus creating a sizable gap between it and the base of the cot. In addition, a further design fault

resulted in the bottom bar of the cot side slipping off the side rods thus exacerbating the problem. Although the girl's mother was aware of the problem and had placed a chest of drawers against the side to prevent it from moving, this was not effective and her daughter was found dead, wedged in the gap between the side and base of the cot.

Another reason why accidental asphyxial deaths continue to occur is that to date not all potentially dangerous situations have been recognised. As these situations may be only rarely responsible for infant deaths, their detection has been understandably delayed. Several potentially lethal situations identified by the author and his research colleagues include, rocking cradles, stroller-prams, 'V' shaped pillows and mesh-sided cots. Further details are given in Chapter 6.

The cases described demonstrate a number of important points. Even though there are no suspicious circumstances in most of these deaths, the histories may be altered by parents who are attempting to disguise the true sequence of events (see Chapter 8). While this may represent an attempt to avoid prosecution for criminal negligence (Bass, 1977) it may simply be a reaction to an overwhelming sense of guilt, as the children have usually been left unsupervised for variable lengths of time (Byard, Beal & Bourne, 1994). Whatever the reasons, the attending police officers and pathologist should not assume that all nonsuspicious deaths in cots are 'cot deaths'.

It is also important to have all sudden infant deaths reviewed at a later date by an independent committee of experts who have access to all of the pertinent information on the case. It is always possible that new information may become known, that was not available to the pathologist at the time of autopsy (see Chapter 7). On occasion this information will change dramatically the original pathological opinion.

Finally, how can such deaths be prevented? It is recognised that there have been many suggestions over the years which have improved the safety of infant sleeping environments, both through the establishment of better safety standards and through public education. Although this has resulted in a decline in accidental infant asphyxial deaths (Davidson, 1945; Moore et al, 1995), local experience suggests that

we may have reached a plateau stage with a constant number of cases occurring every year - dangerous cots still exist; standards do not cover safety aspects of all new products used for infant sleeping and are not mandatory; parents and child carers modify cots; and product safety warnings are lost or ignored (Figure 2-2).

Perhaps the most constructive step that a pathologist can take in these cases is not to dismiss them as merely another example of one of the inevitable risks of early life. The pathologist is in an excellent position to identify these hazards and has a responsibility to recommend that cots in which infants and toddlers have died are formally assessed by product safety experts. This step alone may lead to the withdrawal from sale of defective cots and the introduction or modification of design standards (Moore et al, 1995). Liaison between the pathologist and hospital clinicians and health promotion departments may also provide another avenue for highlighting the dangers of certain situations. The author was involved in the setting up of a 'safe sleeping' committee which has recently produced safety guidelines and information for parents. The production of such child safety pamphlets (see Appendix I) and the presentation of this information at hospital grand rounds, to medical students, to residents and to parent groups, are all steps that can be taken to disseminate this information. These activities will demonstrate that the pathologist can have a very important input into community and child health safety issues, with significant impact on the lives of young families.

* * * *

CHAPTER 3

HAZARDOUS EATING PRACTICES

STUDY 3-1) AN ANALYSIS OF HAZARDOUS CHILDHOOD EATING PRACTICES

INTRODUCTION

Fatal and near-fatal upper airway obstruction caused by inhaled foreign material may occur at all ages, however it is a particular problem for infants and young children due to a variety of predisposing factors (Al-Hilou, 1991; Baker & Fisher, 1980; Black, Johnson & Matlak, 1994; Brown & Clark, 1983; Esclamado & Richardson, 1987; Harris et al, 1984; Lima, 1989; Mantel & Butenandt, 1986; Mittleman, 1984; Puhakka et al, 1989). The potential for prevention is great, and this is one of the most tragic aspects of fatal cases. A difficulty which occurs in toddlers relates to the development of incisor teeth which erupt 10 months to 2 years before the second molars (at 20 to 30 months) (Warwick & Williams, 1973). Thus for a significant period children are able to bite off portions of food without being able to successfully grind the food prior to swallowing. In addition, neural coordination of swallowing is not as precise as in older children. Problems with food therefore arise when it is ingested in amounts that are too large to be successfully masticated, or when the food has a firm or nonfriable consistency. Unfortunately young children do not yet possess the ability to assess the potential problems associated with inappropriate food ingestion and are thus absolutely dependent upon their caretakers for the provision of safe food.

MATERIALS AND METHODS

Inpatient Separation Information Data for childhood hospital admissions in South Australia due to food-induced airway obstruction were obtained from the South Australian Health Commission from 1989 to 1994. In addition, case records of the Department of Histopathology at the Women's and Children's Hospital were searched for cases of fatal food asphxia over the period 1989 to 1994.

RESULTS

Inpatient Separation Information Data from the South Australian Health Commission showed that from June 1989 to June 1990 29 children were admitted to hospitals in South Australia for airway obstruction due to food, with an average length of stay of 2.8 days. This compared to 1990-1991 with 13 admissions, average stay 1.9 days; 1991-1992 with 21 admissions, average stay 3.9 days; 1992-1993 with 27 admissions, average stay 5.5 days; and 1993-1994 with 23 admissions, average stay 5.2 days. Two cases of fatal food asphyxia in the records of the Women's and Children's Hospital were found between 1989 and 1994.

The results of a number of Australian and overseas studies of childhood foreign body inhalation are summarised in Table 3-1. Specific cases are illustrated in Figures 3-1 & 3-2.

DISCUSSION

Although a number of mechanisms may be responsible for fatalities in infants and young children following foreign body ingestion, death most often results from acute upper airway obstruction (Byard, In press). In this age group choking may be caused by a variety of foreign materials, most often food or parts of toys (Al-Hilou, 1991; Altmann & Nolan, 1995; Baker & Fisher, 1980; Black, Johnson & Matlak, 1994; Brown & Clark, 1983; Byard, 1994c; Esclamado & Richardson, 1987; Harris et al, 1984; Lima, 1989; Mantel & Butenandt, 1986; Mittleman, 1984; Puhakka et al, 1989). The most vulnerable age for airway blockage lies between 1 and 3 years with 90% of deaths occurring before 5 years (Banerjee et al, 1988; Harris et al, 1984; Gay et al, 1986). During this time toddlers are learning to explore their environment, a process which often involves placing newly discovered objects in their mouths. It has been estimated that foreign body aspiration causes more than 300 childhood fatalities each year in the United States (Black, Johnson & Matlak, 1994). In Australia there were 7 cases of fatal food aspiration in children under 4 years in 1989, 4 in 1990, 2 in 1991, 7 in 1992 and 6 in 1993 (National Injury Surveillance Unit, 1995).

TABLE 3-1 : DETAILS OF STUDIES DEALING WITH FOREIGN BODY/FOOD INHALATION IN CHILDHOOD

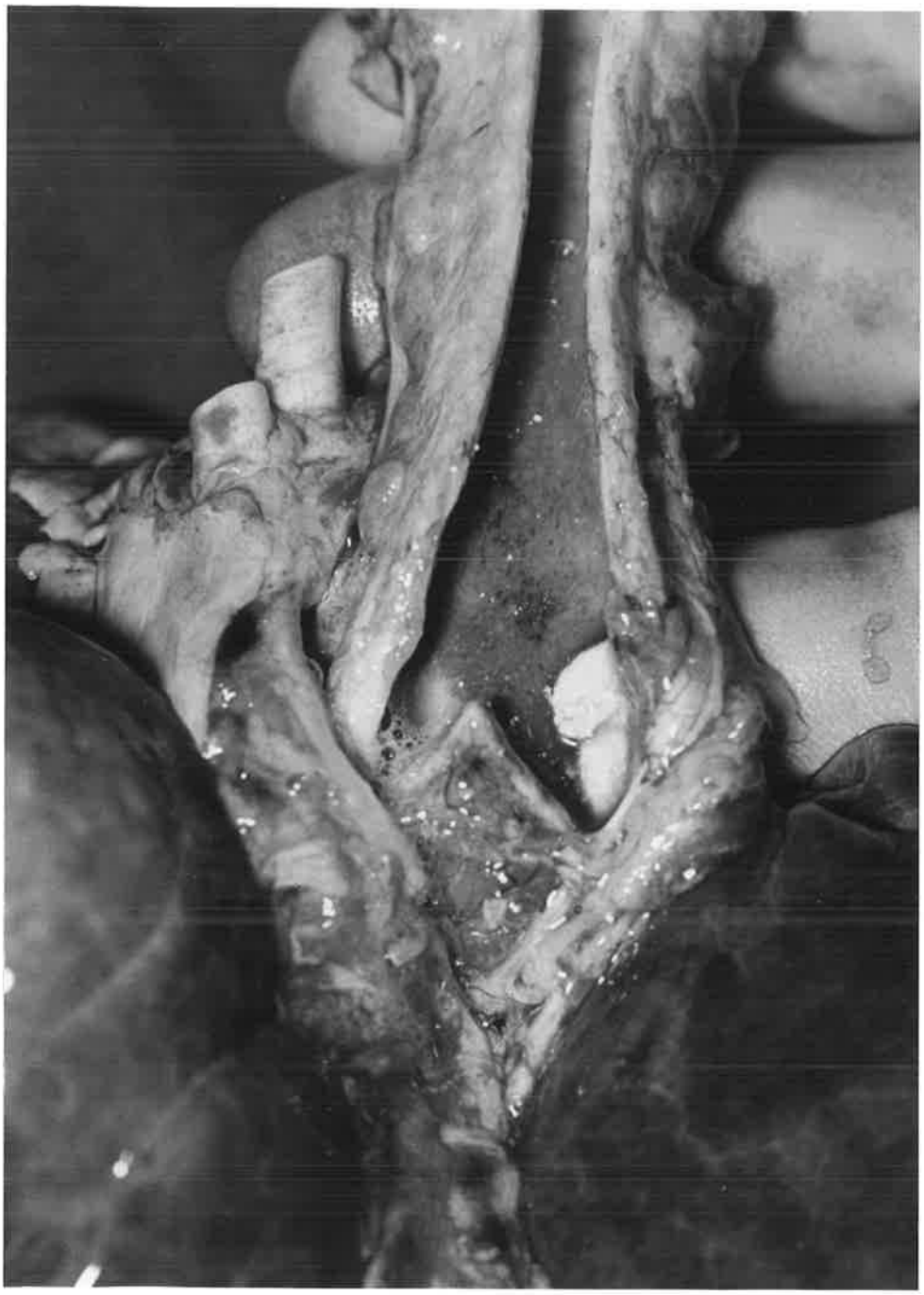
Report	Year	Location	Total No. of Cases	Patient Status	Age	Sites of Obstruction	Foods Specifially Documented
1. Baker and Fisher	1980	Maryland, USA	42	Dead	7m-5yr	Oropharynx Pharynx Larynx	Hotdogs - 6 Sweets -2 Sandwich - 2 Nuts - 1 Potato hamburger - 1
2. Brown and Clark	1983	Melbourne, Australia	115	Alive	Paediatric	Trachea Bronchi	Peanuts - 53 Other nuts - 7 Seeds and vegetables - 7 Carrot - 6 Apple - 4 Chicken bone - 2
3. Harris *et al.*	1984	USA	103	Dead	0-9yr	Not stated	Hotdogs - 16 Sweets - 13 Peanuts/nuts - 9 Grapes - 8 Meat - 7 Biscuits - 7 Carrot - 6 Apple - 5 Popcorn - 5 Peanut butter - 5 Bean - 4 Bread - 4 Macaroni/noodle - 3 Chicken bone - 2 Miscellaneous - 9

4. Mittleman	1984	Dade County, USA	16	Dead	9m-9yr	Oropharynx to Bronchi	Hotdog - 4 Meat - 2 Bean - 2 Sweets - 2 Limes - 2 Sandwich - 1 Popcorn - 1 Peanut - 1 Prune - 1
5. Mantel and Butenandt	1986	Munich, Germany	224	Alive	7m-14yr	Tracheobronchial	Peanut - 83 Other nuts - 66 Carrot - 5 Seeds - 5 Apple - 4 Corn kernel - 2 Miscellaneous - 6
6. Esclamado and Richardson	1987	Seattle, USA	20	Alive	6m-17yr	Laryngotracheal	Peanut - 5 Bone - 3 Peanut shell -2 Meat - 1 Sunflower seed - 1
7. Lima	1989	St. Louis, USA	11	5 Dead	8m-4yr	Larynx	Peanut - 1 Bologna - 1 Bone - 1 Sweets - 1 Hotdog - 1 Popcorn - 1 Chicken - 1
8. Puhakka *et al.*	1989	Turku, Finland	83	Alive	<16yr	Tracheobronchial	Peanut - 25 Apple - 10 Carrot - 7
9. Al-Hilou	1991	Dubai, U.A.E.	49	Alive	3m-23yr	Not stated	Watermelon seed - 11 Peanut - 8 Other nuts - 7 Miscellaneous - 11

10. Black *et al.*	1994	Salt Lake City USA	440	Alive	4m-18yr	Bronchus	Peanut - 165 Other nuts - 49 Popcorn - 27 Seeds - 21 Chicken bone - 3

Figure 3-1: Mince meat found obstructing both main bronchi in a 9m-old boy who collapsed while being fed.

Figure 3-2: Tablets obstructing the upper airway in a 2y-old boy who collapsed soon after ingesting them.



While the presentation of inhaled foreign material is classically of sudden coughing, gagging or choking, followed by stridor, wheezing and reduced air entry, cases not infrequently occur where there is no history suggestive of inhalation resulting in the diagnosis being missed for weeks or months (Cotton & Yasuda, 1984; Friedman, 1988; Kenna & Bluestone, 1988; Linegar et al, 1992; Wiseman, 1984). It has been suggested that any child with persistent wheezing or a respiratory condition with an unusual history or failure of response to usual therapy should undergo chest X-ray to check for differences in aeration between the two lungs (Brown & Clark, 1983). Unfortunately a normal X-ray may occur and so diagnostic bronchoscopy is indicated in cases where there is any doubt (Al-Hilou, 1991; Brown & Clark, 1983; Gay et al, 1986). Fluoroscopy may be useful in demonstrating mediastinal shift (Laks & Barzilay, 1988).

Particular foods which have resulted in death are often rounded in shape, with firm to hard consistencies and diameters less than 32mm (Baker & Fisher, 1980). For example, the two paediatric choking deaths reported from Adelaide in the 6 year period 1989-1994 involved a carrot stick (Byard, 1994c) and a piece of raw apple. A North American study found that 40% of food deaths in children were caused by hot dogs, sweets, nuts and grapes (Harris et al, 1984). Sausages and hot dogs present a particular problem if the covering skin is not removed and the sausage or hot dog is not cut up into small pieces. Sausages/hot dogs accounted for 17% of food deaths overall and 71% of deaths from meat products in the study of Harris et al (1984) (see Table 3-1).

Risk factors for fatal choking include foodstuffs such as these, inadequate supervision, distractions while eating, lack of parental awareness, and medical conditions with feeding difficulties such as Treacher-Collins syndrome and mental retardation (Altmann & Nolan, 1995; Mittleman, 1984; Nixon et al, 1995). Variation in diet among different communities may explain differences that are observed in the predominant type of food associated with choking (Al-Hilou, 1991; Baker & Fisher, 1980; Black, Johnson & Matlak, 1994; Brown & Clark, 1983; Esclamado &

Richardson, 1987; Harris et al, 1984; Lima, 1989; Mantel & Butenandt, 1986; Mittleman, 1984; Puhakka et al, 1989). For example, watermelon seeds caused the largest number of cases in a study from Dubai, compared to most American studies in which hotdogs head the list of dangerous foods (Al-Hilou, 1991; Baker & Fisher, 1980; Harris et al, 1984; Mittleman, 1984). Fish bones are cited as the most common foreign bodies in Japan (Goldsher, Eliachar & Joachims, 1978) and pumpkin seeds were the most frequent inhaled foreign body (25% of all cases) in a study from Greece (Daniilidis et al, 1977).

A disturbing trend emerges in reviewing South Australian data on admission to hospitals for airway obstruction due to food inhalation in children under 5 years of age. While other forms of injury to young children appear to be declining in numbers, episodes caused by choking on food have remained relatively constant. The increase in average length of hospital stay (from 2.8 days in 1989-1990 to 5.2 days in 1993-1994) also suggests that the episodes have been more severe. Although data from England and Wales demonstrate a steady decline in childhood deaths from choking on food from 1972 to 1989 (Nixon et al, 1995; Roper & David, 1987) the numbers of fatal cases in Australia have remained relatively constant over the period from 1989 to 1993 (National Injury Surveillance Unit, 1995). The numbers of fatal cases in South Australia over this time are too small to draw any conclusions.

Another disturbing feature in the area of childhood food safety is the dissemination of inaccurate recommendations in books, newspapers and pamphlets aimed at educating parents, childcare providers and other preschool workers in food presentation and nutrition. Publications commonly recommend feeding toddlers hard foods such as carrot sticks and apples before their dentition is capable of handling such foods. While being readily available from a variety of sources, ranging from newsagents to government agencies, there appears to be no control or monitoring of the publication of such potentially dangerous information. Specific recommendations for improving the safety of childhood eating practices are made in Study 3-3 and in Appendix I.

STUDY 3-2) AN ANALYSIS OF MECHANISMS OF UNEXPECTED DEATH FOLLOWING FOREIGN BODY INGESTION

INTRODUCTION

The aim of this study was to determine the range of possible mechanisms that may cause death in childhood following foreign body ingestion.

MATERIALS AND METHODS

Cases of accidental death due to foreign body ingestion were obtained from the Women's and Children's Hospital Department of Histopathology over the period 1963 to 1996, and from referral cases. Each case was reviewed in detail and mechanisms whereby the foreign material led to death were ascertained.

RESULTS

Local data show a total of 14 cases (4%) of food or foreign body aspiration out of the 369 cases. The age range of the victims was 4m to 8y with a mean age of 2y 2m (M:F = 6:1). There were 3 cases of impaction of pieces of vegetable matter in the upper airway, 2 cases of meat/sausage impaction (the latter case within the oesophagus), and 2 each involving toy pieces and tablets. Further details of these and the remaining cases are listed in Table 3-2 and illustrated in Figures 3-3 & 3-4.

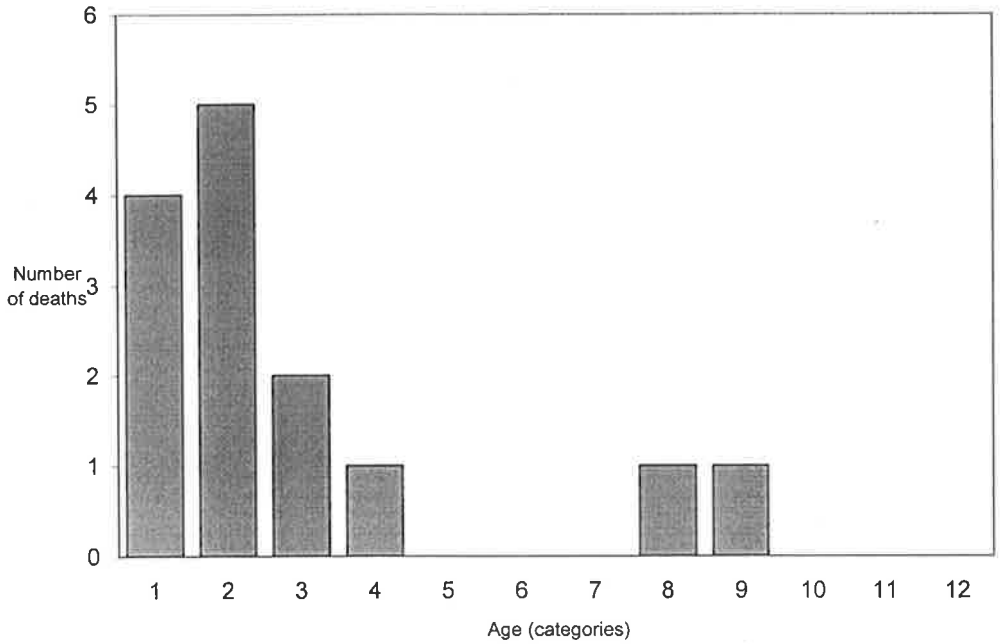
Death most often resulted from asphyxia due to compromise of major airways. Other less common mechanisms involved haemorrhage, acute cardiac tamponade, arrhythmia, centrally-mediated respiratory arrest and sepsis. A summary of possible pathways that may lead to death in children who have ingested foreign material is included in Table 3-3.

TABLE 3-2: DETAILS OF 14 CASES IN CHILDHOOD OF FOREIGN-BODY ASPIRATION/ASPHYXIA IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)

No.	SEX	AGE	YEAR	ASPIRATED MATERIAL
1.	M	11m	1964	Sand in upper airways
2.	M	9m	1970	Mincemeat in bronchi
3.	M	1y3m	1971	Woodscrew in larynx
4.	M	1y7m	1974	Sausage in oesophagus
5.	M	3m	1974	Tablet in bronchi
6.	M	2y2m	1976	Chess piece (pawn) in larynx
7.	M	1y7m	1976	Peanut in trachea
8.	M	4m	1978	Coin in oesophagus
9.	M	7y	1984	Wheat in upper airways
10.	F	1y3m	1985	Plastic ball in larynx
11.	M	2y2m	1986	Tablet in bronchi
12.	M	1y6m	1990	Carrot stick in trachea
13.	M	8y	1991	Wheat in upper airways
14.	F	3y1m	1994	Apple in trachea

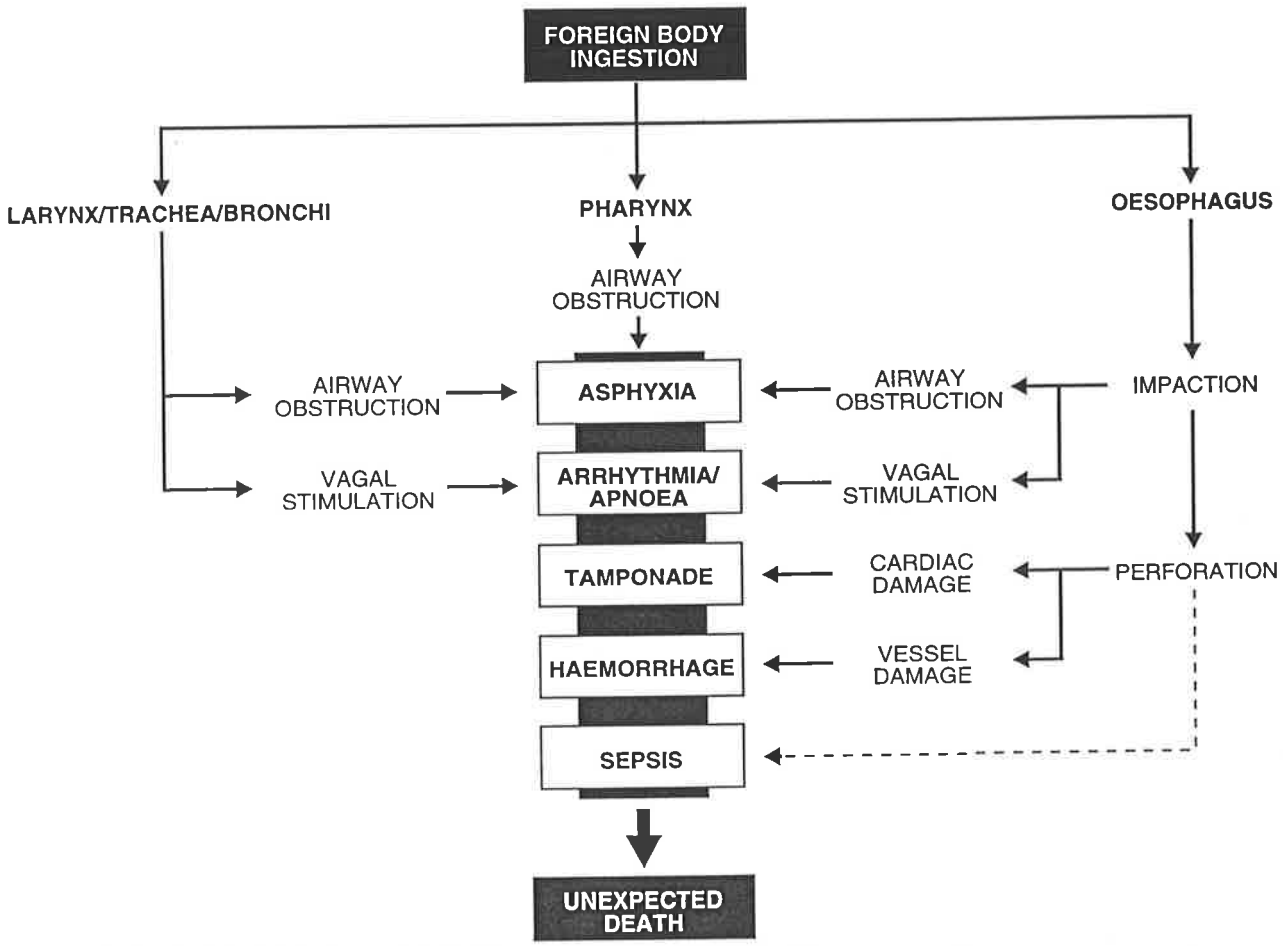
Figure 3-3: A wood screw found firmly wedged in the larynx and upper trachea of a 1y-old boy.

FIGURE 3-4: NUMBER AND AGE OF 14 CASES OF FOREIGN BODY ASPHYXIA TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

TABLE 3-3: A SUMMARY OF POSSIBLE CAUSES AND MECHANISMS OF SUDDEN/UNEXPECTED DEATH IN CHILDHOOD FOLLOWING FOREIGN BODY INGESTION



DISCUSSION

It is well recognised that acute asphyxia may occur if a foreign body occludes any part of the upper airway from the oropharynx to the major bronchi (Byard, 1994c; Byard & Cohle, 1994). Less commonly asphyxia may also result from foreign bodies that lodge in the oesophagus due to compression of the adjacent trachea (Byard, Moore & Bourne, 1990). Fatal cases demonstrating both of these mechanisms were identified in the current data (Byard, 1996b). Other cases demonstrated alternative mechanisms of death including cardiac tamponade and sepsis. The possibility of a fatal vagally-mediated cause of death was also considered.

The role of reflex vagal stimulation in contributing to sudden death following ingestion of foreign material is, however, difficult to assess. The rapidity with which death occurs in adults who suffer a 'cafe coronary' (collapse while eating due to food impaction in the airways) suggests that cardiac arrhthymia, rather than airway occlusion, is the more likely terminal event (Knight, 1991; Mittleman & Wetli, 1982). In infants it has been proposed that oesophageal dysmotility may also cause cardiorespiratory arrest secondary to a vasovagal reflex mechanism (Schey et al, 1981), and experimental observations have shown that instilling dilute acid into the oesophagus of infants causes bradycardia and apnoea (Herbst, Minton & Book, 1979). Animal studies have also demonstrated reflex apnoeas following intralaryngeal and intraoesophageal fluid instillation (Downing & Lee, 1975; Gaultier, 1990; Kenigsberg et al, 1983; Kovar et al, 1979), giving support for reflex neurological mechanisms as a possible cause of death in cases of foreign body impactions.

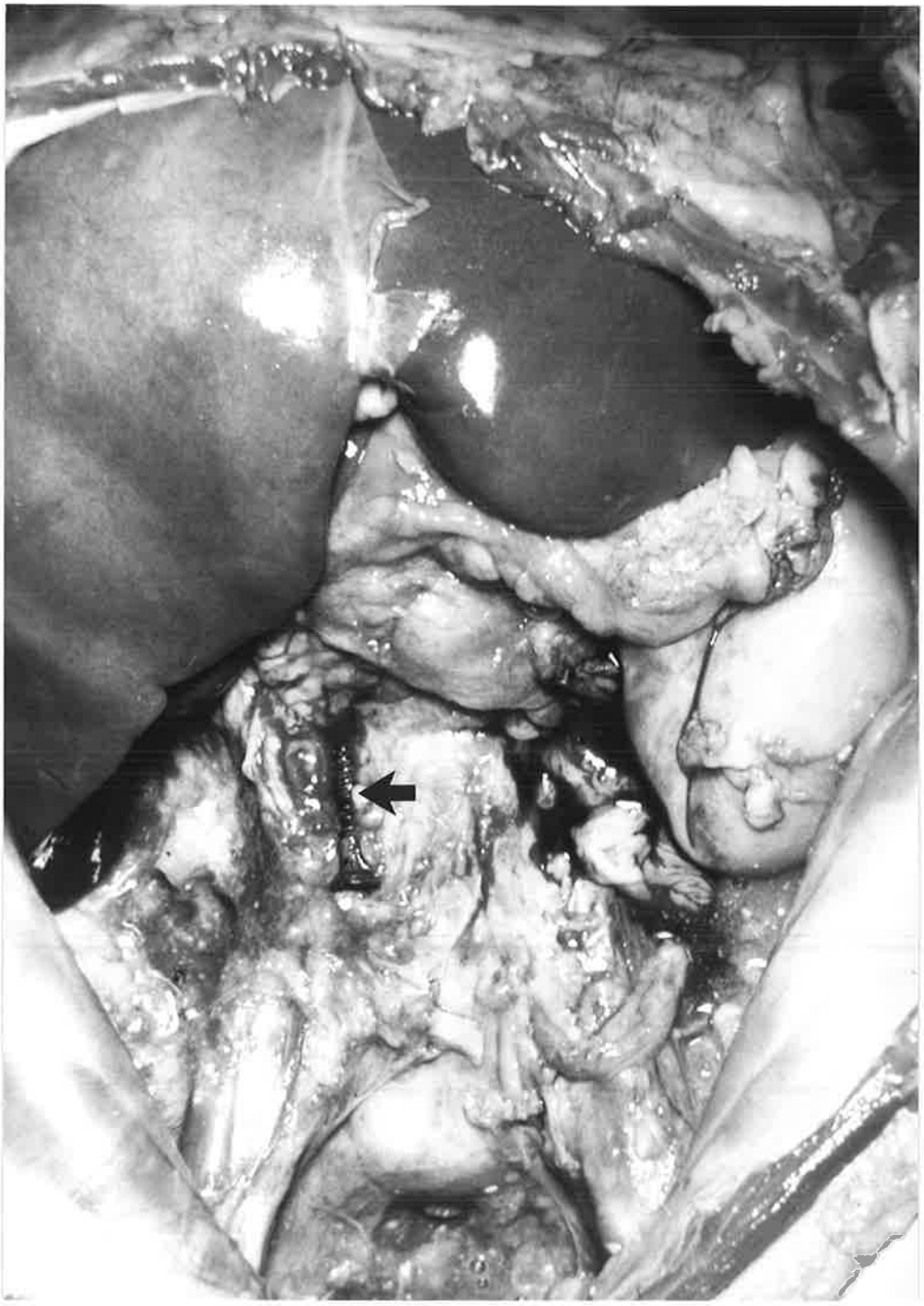
Thus, while the cause of death in cases of intraoesophageal food impaction may be asphyxia due to compression of the membranous portion of the trachea by a large bolus of intraesophageal food, reflex vagal mechanisms may also contribute to the fatal episode. Support for this possibility is gained from the apparent absence of coughing or choking prior to death in certain cases (Byard, 1996b). However, it should be noted that other studies have failed to demonstrate a temporal relationship between gastroesophageal reflux and apnoea in infants (Ariagno et al, 1982), or an association

between reflux and clinical outcome (Rosen, Frost & Harrison, 1983; Walsh et al, 1981). In addition, it may not necessarily be possible to extrapolate from infant and animal studies to older children.

Less commonly, death results from foreign body migration. In children, migration of foreign material has occurred from the pharynx and all levels of the oesophagus, with death resulting from aortic and innominate artery erosion, carotid artery thrombosis, stroke and parapharyngeal sepsis (Jiraki, 1996; Remson et al, 1983). Death from acute cardiac tamponade has been described following ingestion of safety pins (Norman & Cass, 1971; Peeler & Riley, 1957), and was reported recently in a case where an opened safety pin had pierced the anterior wall of the oesophagus, the pericardium and the left ventricle causing fatal tamponade (Byard, 1996b). Death from aorto-oesophageal fistula formation may also occur due to massive uncontrolled haemorrhage.

Although fatal sepsis may also result from migrating foreign material, this does not usually present as unexpected death unless there are confounding factors such as mental retardation or neglect (Byard & Cohle, 1994). For example, although fulminant sepsis occurred in a boy following migration of a woodscrew into the retroperitoneum (Figure 3-4) (Byard, 1996b), it is possible that significant mental retardation may have masked preceding symptoms.

Figure 3-5: Death resulted from bacterial sepsis in a mentally retarded boy who ingested a woodscrew which had migrated into the retroperitoneum.



STUDY 3-3) AN ASSESSMENT OF EATING PRACTICES IN CHILDCARE CENTRES

INTRODUCTION

As noted previously, sudden collapse due to acute upper aerodigestive tract obstruction in infants and young children is most often caused by ingested food or toy parts (Friedman, 1988; Mittleman, 1984). Although attention has been previously drawn to the different pattern of food asphyxiation in children compared to adults (Mittleman, 1984), the following two cases demonstrate particular risk factors associated with young children eating in childcare centers. Both cases have been briefly mentioned in a previous thesis but without specific details (Byard, 1994b; pg 238).

CASE REPORTS

Case 1: A 19m-old healthy boy was happily eating a sausage in a childcare centre when last observed by a caretaker who was then absent for several minutes. Upon return he was found dead. There was no significant previous or family history.

At autopsy a large, approximately spherical, portion of partly chewed sausage measuring 30mm in maximum diameter was found within the lower oesophagus. Several similar portions of sausage were also present within the stomach. The only other findings of note were multiple petechial hemorrhages on the surface of the thymus. Death was initially attributed to undetermined causes, however, upon review of both the presenting history and autopsy findings it was considered that accidental death due to upper airway obstruction was the most likely cause.

Case 2: An 18m-old boy with a previous history of stable mild pulmonary stenosis collapsed at a childcare centre while eating raw carrot sticks, after having attempted to cry/call out with a mouth full of food. He had otherwise been quite well. Asystole was documented by ambulance crew, resuscitation was attempted and a piece of carrot was removed from the trachea via bronchoscopy after transfer to hospital. His neurological status progressively deteriorated and brain death was confirmed. Death occurred 5 days following the episode of food asphyxia.

At autopsy the major findings were of marked cerebral oedema with uncal and cerebellar tonsillar herniation and diffuse hypoxic-ischaemic encephalopathy. Also present were ischaemic necrosis of the colon and minimal, insignificant narrowing of the pulmonary valve. Death was attributed to diffuse hypoxic ischaemic encephalopathy following aspiration of food material with subsequent cardiac arrest and resuscitation.

DISCUSSION

As already described young children are at increased risk of fatal food asphyxiation for a number of reasons, with the most vulnerable age range being between 1 and 3 years (Banerjee et al, 1988; Gay et al, 1986). A lack of understanding of the appropriate size of a food bolus, combined with inadequate control of mastication and swallowing puts them at particular risk for airway obstruction (Banerjee et al, 1988). As noted in Study 3-1 a further problem involves incisor teeth erupting before molars.

The reported cases in this study demonstrate particular features which may predispose to food asphyxia at childcare centers. In such centres children may be confronted with food to which they are not accustomed, and which may not be appropriate for their level of dental, masticatory or swallowing maturation. Although individual meals may be checked, food swapping may still result in potentially dangerous food items being obtained by young children. Increased levels of activity in large groups of children may also make supervision during meal times difficult. Children

may be moving from the table, or calling out or crying while eating, situations which predispose to food inhalation. For example, the infant given portions of carrot was not used to this as a food, and although he had incisor teeth, secondary molars were lacking. He also aspirated when attempting to cry out. In the other case, the infant who asphyxiated on a portion of sausage had been left unsupervised for a short period. It is uncertain from the history whether he had been exposed to this form of food previously, and what the level of interaction with surrounding children was in either case immediately prior to death. It is reasonable to assume that both children were interacting with more children than they were accustomed to at home.

To ensure the safety of children while eating in childcare centers there are a number of requirements which should be met. Close supervision must be undertaken at all times, with greater numbers of staff being present for younger children, or when there is a mix of younger and older children. For example, in South Australia a staff/child ratio of 1:5 is required for children under 2 years of age with a minimum of one trained person being present (Regulations under the Children's Services Act, SA Government Gazette, 1985). Children should not be left unsupervised, and at least one staff member currently certified in cardiopulmonary resuscitation should be readily available to initiate emergency first aid measures if required. Posters demonstrating first aid for choking in situations both where children can breathe and where they cannot breathe should be on display for staff (Thomson, 1991).

Unfortunately in examining the situation in childcare centres more closely, uniform policies regarding safe eating practices for young children appear to be lacking, as does consistent training in this area for childcare workers. Attention appears to be paid to providing food with a high nutritive value without corresponding attention to ensuring its safety. Ironically the recent trend of improving the nutritive value of meals and snacks by serving raw vegetables and fruit may result in inappropriately prepared food being presented to toddlers. It is important that there is a clear understanding of child development and why certain foods are dangerous at certain ages. Guidelines

should be formulated to ensure that vegetables are steamed to the point of softness, or diced and mashed, so that no firm/hard food is presented to children under the age of 4 years or until a child is able to chew well. It is also important to test that food and drinks are not too hot before they are served (Thomson, 1991).

Interviews with parents are helpful in determining the type of foodstuffs that individual children are used to and in ensuring that inappropriate food is not sent with the child to the center. Lunch boxes from home should be inspected to check for potentially dangerous food. Careful control of the meal area is also important so that children are not running around when they eat, but are seated and thus able to be easily watched and supervised, away from distracting situations. Small toys and other potentially dangerous ingestable objects should be removed from both the meal and general play areas.

In summary, safe food practices should include feeding young children under 4 years of age only food that they will be capable of chewing and swallowing with ease. Foods to be avoided include hard foods such as nuts, raw carrot sticks, pieces of raw apple, celery sticks and corn chips, and round foods such as grapes, popcorn, and sweets. Caution must also be taken with nonfriable food or food with skin such as chicken, sausages and hotdogs. Certain of these foods such as carrots and apples may be appropriate for children under 4 years only if cooked, mashed or grated. Sausages, hotdogs, chicken and other meats are appropriate if cut into small pieces for children who are able to chew them. Tough sausage skins, gristle and bone should be removed prior to cutting meat into small pieces.

Children should not be fed forcibly, and should be sitting quietly under observation while eating. Eating while running or crying should not be permitted. Potentially dangerous foods should be identified and introduced to a young child's diet only under close supervision, tailored to the child's level of dental development and ability to handle food safely.

A variety of strategies have been suggested to reduce the number of children choking on food (Harris et al, 1984). Dissemination of information to parents, childcare and child health professionals on potentially dangerous foods and the need for safe eating practices has been recommended, along with approaches to the food industry. In Sweden packaged foods contain labels specifying safe ages for consumption, with warnings about possible dangers of children choking (Harris et al, 1984). The author was involved in the setting up of a 'safe eating' committee which has produced an information pamphlet (see Appendix I) which has been distributed to childcare workers, health professionals and parents. In addition the committee has formulated policy guidelines on safe childhood feeding which has been distributed to childcare workers, hospitals and other organisations responsible for the care of young children (also see Appendix I).

* * * *

CHAPTER 4

HAZARDOUS FARM ENVIRONMENTS

INTRODUCTION

Trauma at all ages on farms in Australia is an ongoing problem with estimates of fatal accidents ranging as high as one death every 3 days nationally, with one death every 10 days due to tractors (Fragar, 1996). Unfortunately due to the unique combination of workplace and home environment that most farms incorporate, a number of these accidents involve children; in fact, proportionately more children than adults have died in farm accidents (Wolfenden & Sanson-Fisher, 1993).

In the United States agriculture has been ranked second only to mining in terms of occupational danger, with as many as 300 fatal and 23,500 nonfatal childhood farm accidents occuring each year (Rivara, 1985). Both mining and rural industries also have the highest occupation fatality rates in Australia (Harrison et al, 1989). In South Australia between 1986 and 1992, 82% of the 201 childhood hospital admissions from workplace injuries were caused by farm accidents, and 73% of the 37 children admitted with machine injuries had sustained the accident on a farm. Nationally over the same period, 83% of the 847 children admitted to selected hospitals following workplace injuries had sustained the injuries on farms (South Australian Occupational Health & Safety Committee, 1992). Accurate mortality figures are more difficult to obtain due to variable reporting practices, however, 34 childhood agricultural deaths occurred nationally in Australia during the 3 year period 1982-1984 (Erlich et al, 1993). Given this significant childhood morbidity and mortality, the following review was undertaken to examine the circumstances of tractor and farm-related deaths in a group of children in South Australia.

MATERIALS AND METHODS

To specifically investigate fatal farm and tractor accidents in children under the age of 16 years, the Department of Histopathology computerised autopsy database at the Women's and Children's Hospital was searched for the 15 year period from October 1981 to September 1996 for such cases. In addition, a listing of rural industry fatalities from 1988 to July 1996 was obtained from the Department of Industrial

Affairs. Additional cases on this list were then accessioned from the files of the State Coroner's Department. Coding of such cases prior to 1980 was so variable that the study did not include the earliest years.

The circumstances of death and autopsy findings in individual cases were reviewed with specific attention being paid to the age and sex of the child, the year of death, the activity being engaged in prior to death, the type of accident, the type of injuries and the cause of death.

RESULTS

Fifteen fatalities were identified comprising 11 boys and 4 girls (age range = 2y 11m to 13y; mean = 6y). Activities prior to death included riding on a tractor/trailer (N = 8; age range = 2.5y to 11.5y; mean = 5y 3m; M:F = 1:1); playing on a field grain storage bin (N = 2); playing near machinery (N = 2); sleeping in a car near a burn off (N = 1); walking around a dam (N = 1) and riding on a motorbike (N = 1). Causes of death included multiple skull fractures and cerebral damage (N = 11), asphyxia (N = 2), drowning (N = 1) and incineration (N = 1). (The results are summarised in Table 4-1).

DISCUSSION

Farm environments are particularly dangerous places for young children as they combine elements of both home and industry (Dunn & Runyon, 1993; Wolfenden & Sanson-Fisher, 1993). For example, a report from the United States noted that in one year the number of deaths caused by farm machinery in children aged between 3 and 4 years almost equalled the number of deaths due to home falls and poisonings, despite the fact that only 2% of children lived on farms (Davis, 1988). Children's curiosity leads them to investigate situations where they often do not recognise potential hazards. Falls, tractor/wagon accidents and large animal trauma constituted the majority of childhood farm injuries documented in a 12-year American study (Cogbill et al, 1991).

TABLE 4-1: SUMMARY OF FEATURES OF 15 TRACTOR AND FARM-RELATED DEATHS IN CHILDREN IN SOUTH AUSTRALIA FROM 1981 TO 1996

No.	Year	Age	Sex	Activity	Accident	Injury/Cause of Death
1	1982	4y	M	Riding on tractor tool box mounted on rear mudguard; tractor driven by father - towing truck.	Fall - run over by tractor rear tyre (DOA).	Multiple fractures of skull, contusions and lacerations of brain, lacerations of liver.
2	1983	4y	F	Riding on tractor pulling rotary hoe; driven by mother.	Fall - run over by rotary hoe (DOA).	Multiple skull and other fractures, contusions and lacerations of brain, transection of upper thoracic spine and cord, avulsion of liver, laceration of thoracic and peritoneal organs, extensive soft tissue damage.
3	1984	7y	M	Playing on field grain storage bin being emptied by auger into truck; operated by father.	Fall - drawn into depths of bin (DOA).	Asphyxia - filling of bronchi with wheat.
4	1986	2y11m	M	Playing in farmyard; tractor driven by father.	Run over by rear wheel of tractor (brain death after six days).	Multiple fractures of skull, laceration and infarction of brain.
5	1987	13y	M	Pillion passenger on motorbike on farm; wearing a helmet.	Fall (brain death after 7 days).	Cerebral contusion and intraparenchymal haemorrhage.
6	1988	5y	M	Riding on tractor towing mower; driven by uncle.	Fall - run over by mower (DOA).	Decapitation injury with compound fracture of skull and avulsion of brain; amputation of right upper limb; multiple soft tissue injuries and fractures.
7	1988	3y	F	Riding in tractor cabin towing hay cart; driven by mother.	Fall - having opened door of cabin - run over by rear tractor wheel and wheels of trailer (DOA).	Massive fractures of skull, laceration of brain, laceration of heart, rupture of diaphragm and crush injury of liver.
8	1989	6y	M	Riding in trailer attached to reversing tractor driven by father.	Fall - trailer wheel ran over head (DOA).	Massive fractures of skull and fragmentation of brain.

9	1991	8y2m	M	Sitting on top of field grain bin being emptied by auger into truck; operated by father.	Fall - drawn into depths of bin (DOA).	Asphyxiation - filling of bronchi and trachea with wheat.
10	1993	6y	M	Sleeping in car while father was burning off grass.	Incineration (DOA).	Soot in trachea, extensive charring of body, CO - 11%.
11	1995	2½y	F	Riding on tractor driven by father.	Fall - rear wheel ran over head (DOA).	Massive fracture of skull with laceration and contusion of brain.
12	1996	11½y	M	Riding on tractor towing mower driven by grandfather.	Fall - under mower (DOA).	Extensive compound comminuted fracture of skull, laceration and contusion of brain, rupture of spleen, laceration of viscera (kidneys and liver), fractures of ribs.
13	1996	3y	M	Playing near power take-off of auger operated by father and grandfather.	Caught in machinery (DOA).	Massive compound comminuted fracture of skull, fracture dislocation of cervical spine and cord, avulsion of thoracic aorta, avulsion of liver, spleen and left kidney, extensive visceral, soft tissue and skeletal injuries.
14	1996	6y	F	Riding on fork of tractor driven by father.	Fall (brain death one day after accident).	Compound fracture of skull, laceration and contusion of brain.
15	1996	8y	F	Walking around dam	Slipped into water.	Drowning

DOA = dead on arrival

Swanson et al (1987), in analysing reasons for the high rates of injury and death on farms, implicated long working hours at peak seasons, work occurring in all types of weather, a great diversity of types of work with a variety of potentially hazardous machinery, involvement of family members of all ages in activities, and failure to upgrade older less-safe machinery due to the substantial costs involved.

Although current Occupational Health, Safety & Welfare regulations in South Australia specify that 'no person other than the operator is permitted to ride on an industrial lift truck or tractor unless the person is seated in a seat specifically designed for carrying a passenger, and the seat is fitted with appropriate seat restraints and is located within the zone of protection afforded by the required operator protective devices' (OHS&W Regulations, Government of SA, 1995), the current study has shown that the majority of deaths (53%) occurred in children who were riding on tractors or trailers without provision of safe seating with suitable restraining harnesses. The drivers of the tractors were all close relatives (father - 4; mother - 2; uncle - 1; grandfather - 1), in keeping with the finding that 60% of farms in Australia are operated under family partnerships (Coleman et al, 1996). The preponderance of boys over girls in our series (11:4) has also been noted by other investigators (Salmi et al, 1989).

While it has been claimed that tractor rollover accidents have been responsible for up to 35% of rural child deaths (Erlich et al, 1996), no examples occurred in our series. It is interesting that the NSW WorkCover Authority found a similar relatively low number of childhood fatalities due to rollovers. It has recently been noted that on subsequent review of the Erlich paper by one of the coauthors, an error in interpretation of the original data had occurred, with the majority of childhood tractor deaths in their series being due to *run*overs, rather than rollovers as originally stated (Coleman et al, 1996). This revised opinion would certainly be more in keeping with South Australian and New South Wales data. The three major forms of tractor-associated injury found in a Canadian study were runover, rollover and entanglement injuries (Pickett & Brison, 1995). Overall, 50% of farm deaths in the United States, and 40% in Australia, have been caused by tractors (Wolfenden & Sanson-Fisher, 1993).

Changing socioeconomic conditions on farms have accentuated the issue of child safety. In recent times there has been an increase in the number of women working full or part-time on farm activities outside the home, resulting in an increase in the number of young children being taken into the workplace. This is reflected in two of the tractor drivers in the fatal cases being the mothers of the dead children. Obviously it is often not possible to babysit and work simultaneously, and yet alternative strategies for child care may not be available due to financial and geographic constraints. Recent economic difficulties in the rural sector also mean that issues of safety may be neglected for financial reasons with money being unavailable for upgrading unsafe equipment. A disturbing trend was noticed in the mid-1980's in the United States, with fatalities caused by machinery not associated with farms falling by 79% in the 50 years before 1980, compared to machinery deaths occurring on farms which had increased by 44% over the same time (Swanson et al, 1987). In the United Kingdom childhood farm deaths other than those due to tractor rollovers have 'not fallen appreciably in recent years' (Cameron, Bishop & Sibert, 1992).

While the major problem in this study involved children falling from moving equipment, other dangerous activities that were identified included playing around moving machinery, including tractor power take-offs where clothing or limbs could get caught, dams, grain silos and mobile field storage bins (Figure 4-1). (A power take-off is an extension to the drive shaft of a tractor which provides power for other equipment) (Kalenak et al, 1978). Different farms also have different safety issues; for example wheat farms have more agricultural equipment related injuries compared to dairy and beef farms where animal-related or other injuries are more common (Edmonson, 1987; Wolfenden & Sanson-Fisher, 1993). Problems may occur if insecticides, drenches and sprays are left accessible to children, if firearms and electrical outlets are not protected from tampering with, if ladders are left upright and if children are allowed unsupervised access to animals that they may fall from, or be kicked by. Examples of the latter include horse riding and playing in animal yards. Motorcycles, all terrain vehicles and farm bikes may be ridden by young children who

do not have the ability to control such equipment, and safety helmets may not be worn. High stacks of hay may fall, or children may fall from them. City visitors may also be a problem as they may have even less appreciation of potential dangers (Doyle & Conroy, 1989). Injuries which occur in children on farms when machinery is involved tend to be severe, and include fractures, lacerations and amputations (Figures 4-2 & 4-3) (Swanson et al, 1987; Vanneuville et al, 1992).

The types of injury vary with age, with toddlers more likely to be injured in falls or from animal kicks while exploring, compared to older children who fall from horses or are injured in tractor accidents while working without adequate supervision (Cameron, Bishop & Sibert, 1992; Cogbill, Busch & Syeirs, 1985). Cogbill et al (1985) also found in their study of 105 injured children that the age of presentation at injury had a bimodal distribution, with peaks at 4 and 14 years. Australian data from 1982 to 1984 show a similar pattern with 14 deaths of children under 4 years of age, 7 deaths from 5 to 9 years and 13 deaths from 10 to 14 years (Clarke & Coleman, 1995). In our study there is a clustering around the age of 5 years with only two cases in older children (Figure 4-4). This may, however, reflect the small size of the series, or problems with accessioning cases. We also found that fatal tractor related accidents occurred at all ages from 2.5 to 11.5 years.

Unfortunately, this study does not include every case of fatal childhood farm accident in South Australia over the designated time, as not all cases have been entered into computerised databases, coding of childhood fatalities does not always indicate whether or not a death occurred on a farm, and not all cases have had autopsies. For this reason the study again cannot be used to provide accurate epidemiological information on childhood farm deaths in South Australia. It does, however, give a clear picture of certain types of activities that may be associated with lethal outcomes for children on farms.

A number of strategies can be adopted to make farms safer for children. Riding on tractors/equipment should not be allowed unless a secure seat with a safety harness is provided and the tractor has a rollbar fitted. Unfortunately having a child within the

Figure 4-1: Filling of the main bronchi with wheat pellets in a 7y-old boy who fell into a field grain storage bin (Case 3).

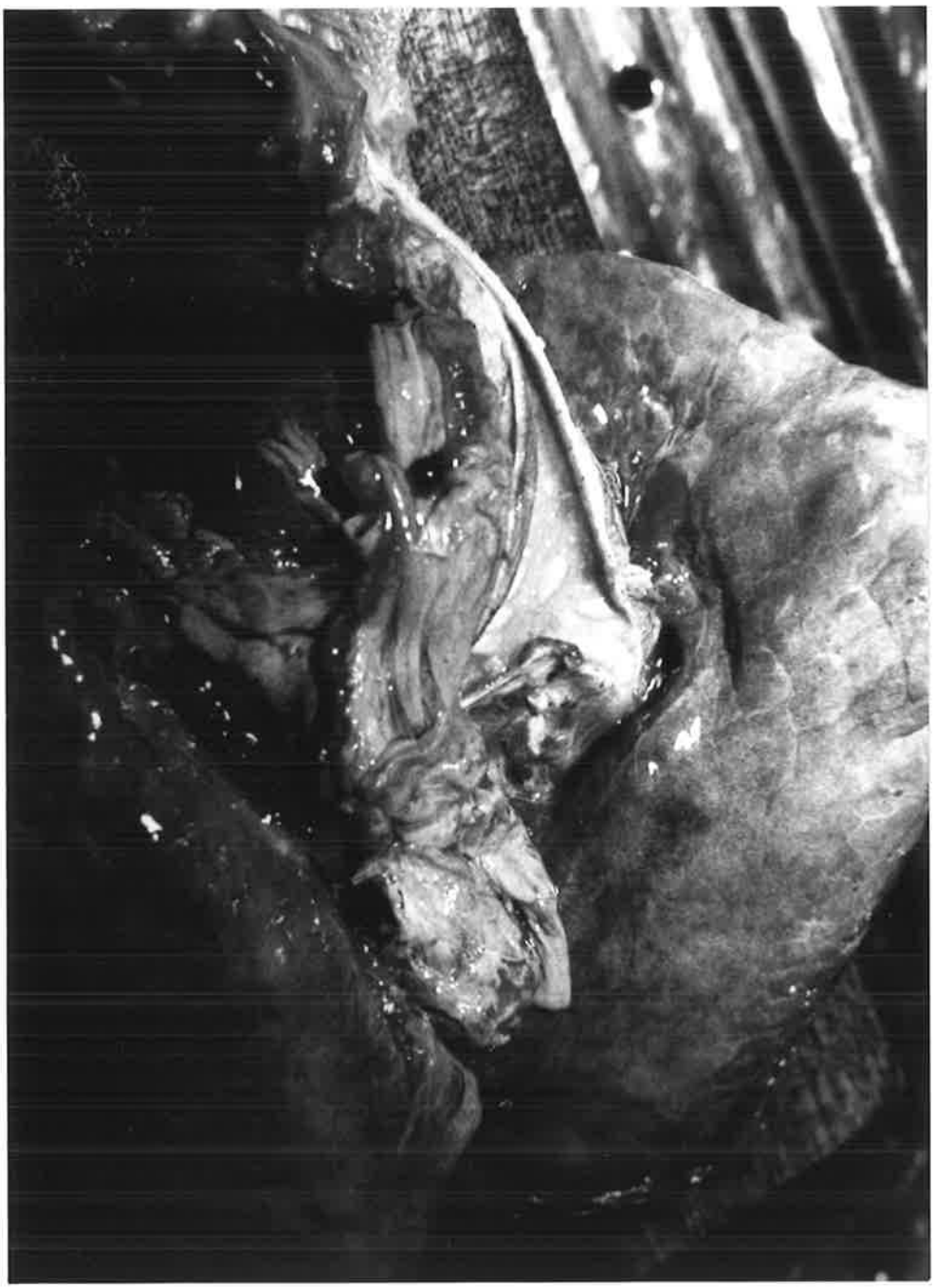


Figure 4-2: Extensive soft tissue, bone and organ damage in a 4y-old girl who was run over by a rotary hoe following a fall from a tractor (case 2).

Figure 4-3: Massive limb and organ damage occurred in this 3y-old boy who was caught in a tractor power take-off (Case 13).

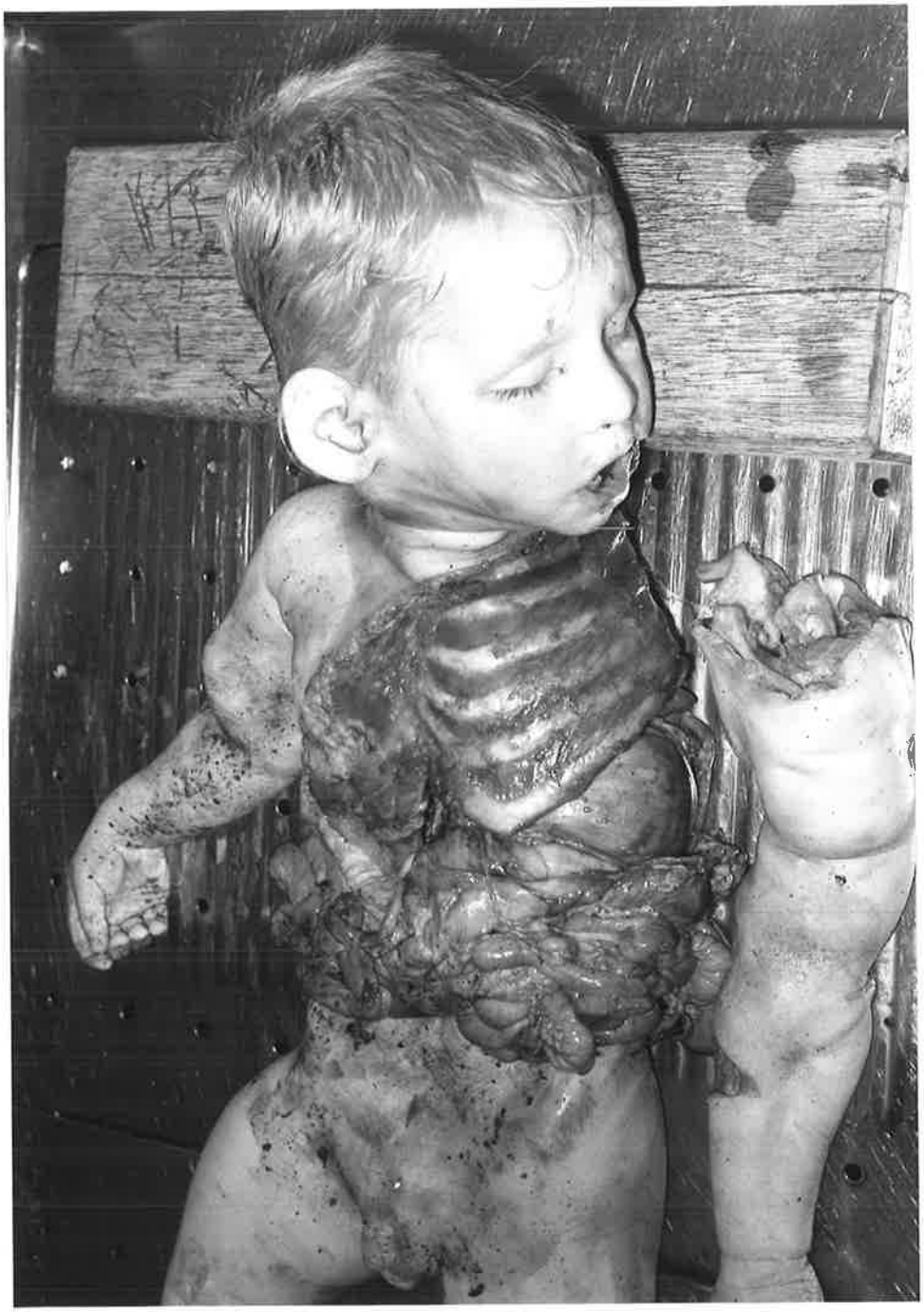
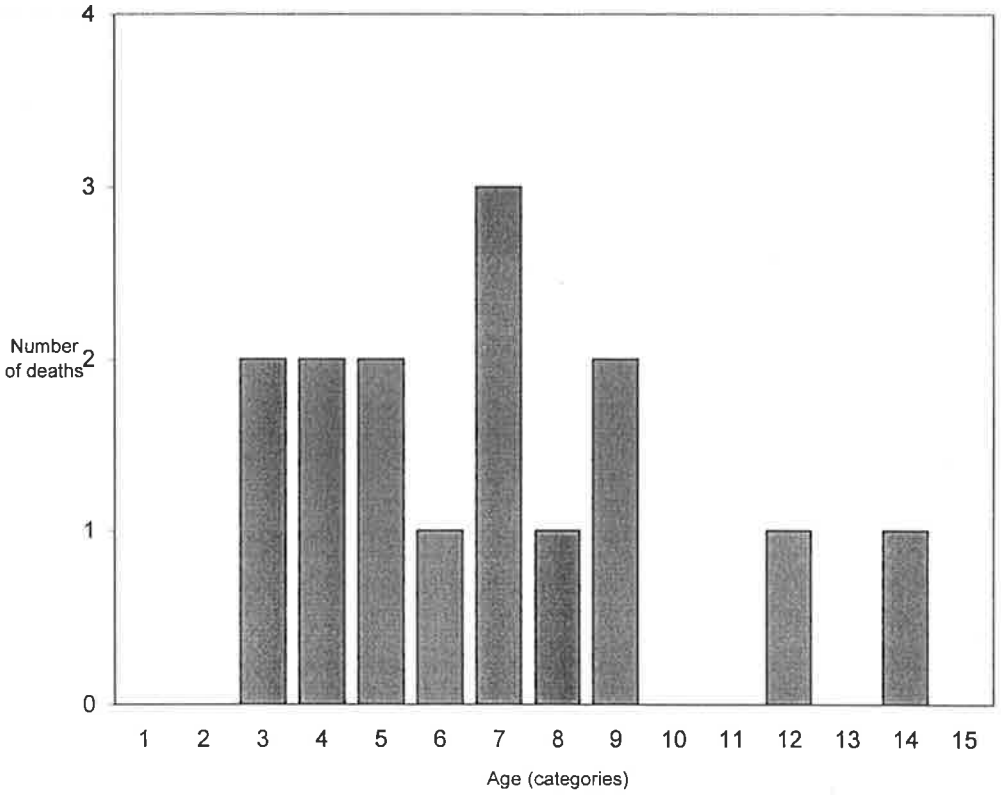


FIGURE 4-4: NUMBER AND AGE OF CASES OF FARM-RELATED FATALITIES TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

tractor cabin is not sufficient to prevent injury as demonstrated in one of the cases (Case 7), where the child opened the cabin door and fell under the tractor wheels. Starter keys should also not be left in tractors and equipment should have automatic shutoff devices. Young children should be carefully watched at all times and kept away from machinery that is in operation, particularly machinery with universal joints, belts, shafts, pulleys, and power take-offs. These moving parts should have nonremovable safety guards. Such safety guards have in the past been found to be 'inadequate, damaged or discarded' (Kalenak et al, 1978). Children should be taught to avoid dangerous areas such as watercourses and livestock enclosures unless accompanied.

Providing a safe and secure area to play in is one way of ensuring that children do not stray into workshop areas or animal enclosures. Although many farms are geographically isolated, shared child care arrangements are sometimes a possibility when several farms are in proximity. Fencing around dams, irrigation ditches, creeks and waterholes should be undertaken if possible, and sheep dips, septic and water tanks should be kept secure. Ladders should be either out of reach of children or taken down, and poisons should be in childproof containers or locked up. Access to explosives and firearms should not be possible. Safety helmets must be worn when children are riding bicycles, motorcycles or horses.

The success of any farm safety campaign will depend on its acceptance by rural communities. For this reason community education programs through women's groups, schools and farmer's groups may be more likely to be successful than legislation imposed without consultation. For example, a survey of rural areas in South Australia found strong resistance to government regulation of child safety on farms (Mandryk & Harrison, 1995). However, in the past the failure of voluntary safety standards has been well documented and formal legislation to protect children in the work place has been advocated (Karlson & Noren, 1979; Polack, Landrigan & Mallino, 1990). General practitioners and paediatricians in rural areas are in an excellent position to engage in community education activities and to promote child safety issues (James, Christie &

English, 1994), although funding may be an issue. Recommendations need to be practical, as inexpensive as possible, and sustained so that initial enthusiasm does not wane (McKnight & Hetzel, 1985). Initiatives currently being developed in Australia include *Health and Safety Guidance Notes* by the Australian Agricultural Health Unit which will cover all aspects of farm safety, and *Child Safety on Farms* by the WorkCover Corporation, South Australia which will focus specifically on the issues of rural childhood injury and death. The author is a member of the 'Child Safety on Farms' committee responsible for producing the latter document. These publications and educational packages which will include recommendations and suggestions for improving farm safety will be released in mid-1997 (see Appendix I).

* * * *

CHAPTER 5

CHILDHOOD DROWNING

INTRODUCTION

Drowning is responsible for a significant number of paediatric deaths, accounting for 24% of deaths in some series (Cass, Ross & Grattan-Smith, 1991; Lawson & Oliver, 1978). Rates of childhood drowning range from 7.6/100,000 in Mexico to 2.5/100,00 in Minnesota, and 1.5/100,000 in New South Wales (Cass, Ross & Lam, 1996; Hedberg et al, 1990). Australian national data show a rate of 7.2/100,000 fatal drownings in children under the age of 4 years in 1990, compared to a rate of 3.7/100,000 in 1994 (National Injury Surveillance Unit, 1996). The post-mortem diagnosis of drowning is, however, occasionally less than straightforward. In some cases an underlying medical condition may have initiated the drowning episode and in others careful distinction between death during, rather than death due to, immersion in water must be made (Smith, Byard & Bourne, 1991b). In all cases contributory medical conditions should be sought. The following cases, where uncomplicated drowning was often initially considered likely, demonstrate the need for modification of the diagnosis in a significant number of cases of childhood drowning once the patient's history is evaluated and additional post-mortem findings become apparent.

MATERIALS AND METHODS

The autopsy database of the Women's and Children's Hospital for the 34 year period from January 1963 to December 1996 was searched for cases coded as 'Drowning Freshwater', 'Drowning Saltwater', 'Drowning Other Liquid', and 'Drowning Not Otherwise Specified'. Case histories and autopsy findings were reviewed.

RESULTS

Sixty-seven cases were were found where the circumstances of death were strongly suggestive of drowning. Two cases where death had occurred 6 months and 1 year, respectively, from the sequelae of near-drowning episodes were excluded i.e. the children had suffered severe hypoxic brain damage at the time of accidental immersion

and subsequently died of pneumonia. Of the remaining 65 cases there were two children (Cases 1 & 2 below) who died of natural disease while in water. Excluding these cases left 63 children, representing 17% of the total number (369) of cases of accidental death. Seven children had documented medical conditions which may have predisposed to drowning. This latter group consisted of 5 children with epilepsy, 1 with epilepsy and mental retardation and 1 with hydrocephalus. The age range was 3m to 12y 9m with an average age of 8y 7m and a male to female ratio of 6:1. Two of these children drowned at the beach, 2 in public pools, and 3 in baths.

Fifty-six children drowned with no predisposing disease (age range 3m to 12y; mean 2y 11m; M:F = 2.4:1). Of these, 23 children drowned in home swimming pools compared to only two children in public pools (age range of the 25 pool deaths = 1y 1m to 11y 2m; mean = 3y 11m; M:F = 3.8:1). The next major category consisted of 11 children who drowned in baths (age range 6m to 3y 4m; mean = 1y 9m; M:F = 1:1). All of these children had been left unsupervised for variable lengths of time. Although multiple bruises raised the possibility of nonaccidental injury in a 2y 6m and a 3y-old girl, both of whom drowned unattended in baths, no further evidence could be obtained to support this possibility. While two of the children who drowned in baths (ages 2y 6m & 3y 4m, respectively) had histories of febrile convulsions there was no suggestion of convulsions being involved in the lethal episode. One 7m-old boy drowned in the bath despite the use of a 'flotation' device.

In two cases there were specific problems with the public swimming pools related to the young age of the victims. In one case, a 3y 6m-old girl was sucked underwater into a pool filter, and in the second, a 6y-old girl, who was a poor swimmer, was pushed into the deep end of the pool by the forceful water return.

The remaining children drowned in a variety of different locations: freshwater lake/creek (N = 6); fishpond (N = 3); bucket/sink (N = 2); ocean, sewage pit,

irrigation ditch, and farm dam, (N = 1 each). Histories in the remaining 5 cases are lacking. The clinicopathologic details of selected cases are presented below.

SELECTED CASE REPORTS

Case 1: An 8y-old boy failed to surface at a public swimming pool and was rapidly removed from the water. Spontaneous respirations had ceased but were re-established after resuscitation, although these became intermittent during transfer to hospital. There was no significant previous medical history. On admission a systolic blood pressure of 150mm Hg was noted in association with papilloedema. Intravenous mannitol and dexamethasone were given and a right frontal burr-hole and ventricular tap performed which revealed slightly blood-stained cerebrospinal fluid. Rapid deterioration occurred, culminating in respiratory arrest. At autopsy the lungs were oedematous with normal airways. Histology showed small intra-alveolar haemorrhages. The brain was mildly oedematous with basal subarachnoid haemorrhage. Histological examination showed a disrupted arteriovenous malformation in a cerebral sulcus. The cause of death was subarachnoid haemorrhage due to a disrupted arteriovenous malformation, and not drowning.

Case 2: An 11y-old boy was found unconscious in a public swimming pool. Poolside and casualty room resuscitative efforts failed to elicit spontaneous respiratory efforts and death ensued. Eight months previously the child had collapsed and lost consciousness while sprinting at school. Resuscitation on this occasion was successful and he woke quickly and was fully alert. Investigations (EEG, ECG, X-rays of chest and skull) showed no abnormality. At post mortem the lung surfaces were spongy, without excess fluid and the airways contained a small amount of gastric contents. The brain was normal. Although the heart was normal, the calibre of the right coronary artery

was markedly reduced compared to the left coronary artery. Death was therefore attributed to cardiac arrest due to marked hypoplasia of the right coronary artery, and not to drowning (Byard, Smith & Bourne, 1991a). This case has also been briefly described in a previous thesis (Byard, 1994b pg 161).

Case 3: A boy aged 6y 10m was found at the bottom of a relative's swimming pool. He was known to be epileptic and had suffered up to 6 seizures per day despite medical treatment. No seizures had been witnessed in the 2 weeks prior to death. At post mortem the lungs were dry and well aerated. Histology showed mild oedema and scanty foreign material in occasional bronchioles. The brain was unremarkable. Death was ascribed to drowning in a patient with epilepsy. (Serum anticonvulsant levels were not measured.)

Case 4: A boy aged 6y 11m was found face down in the bath. The taps were running and water was overflowing. He had suffered a single grand mal seizure 10 months prior to death. At that time an electroencephalogram (EEG) was abnormal and medication had been prescribed. At post mortem the lungs were bulky and exuded frothy blood-stained fluid into the airways. The brain was oedematous. Death was ascribed to drowning in a patient with epilepsy. (Serum anticonvulsant levels were not measured.)

Case 5: A boy aged 11y was found unconscious in his school swimming pool. Resuscitatory efforts were unsuccessful. He had suffered mental retardation and epilepsy subsequent to an episode of acute cerebral damage due to ethanol poisoning at 4 years of age. Seizures were increasing in frequency during the last year of life. At post-mortem the lungs were bulky, airless and exuded frothy blood-stained fluid. The

brain was externally and histologically normal. Blood levels of anticonvulsants were in the low therapeutic range. Histology showed lung oedema and vegetable matter in airways. Death was ascribed to drowning in a patient with epilepsy and mental retardation.

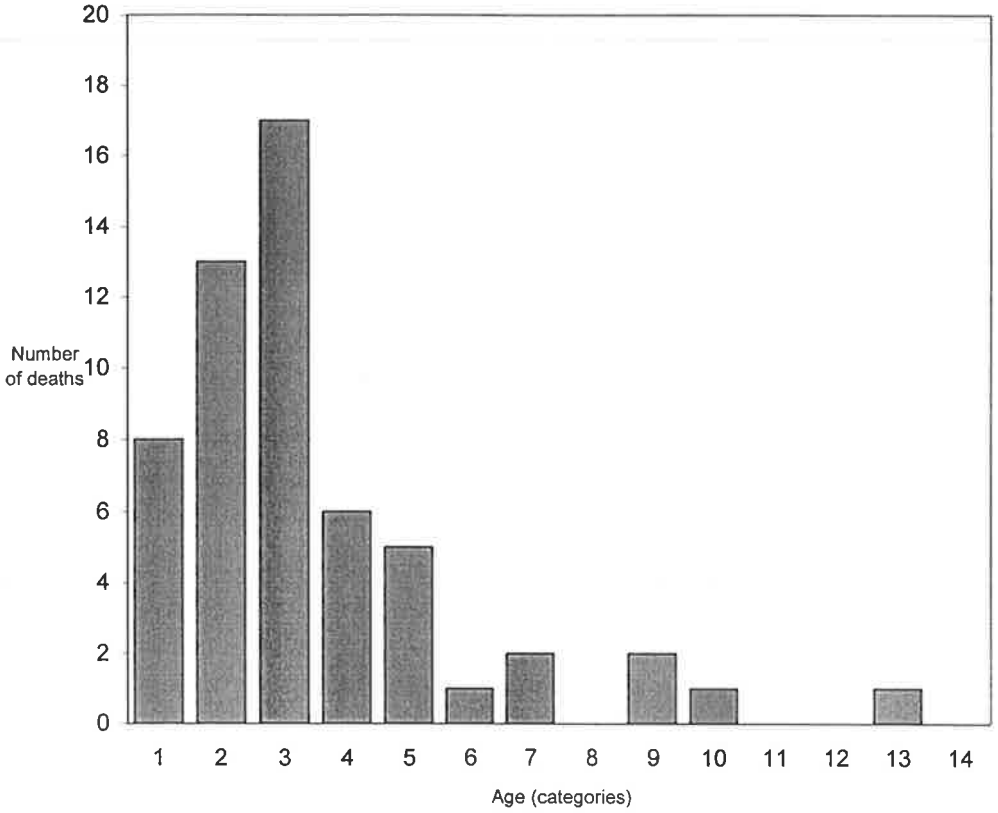
Case 6: A girl aged 10y 10m was under instruction at a public swimming pool when she suddenly became unconscious. A pool attendant removed her from the water and placed her in the coma position and her mother, who was present in the pool, observed what she thought were the usual signs of recovery from a seizure. The child was known to be epileptic and was on anticonvulsant therapy. Resuscitation was unsuccessful. At autopsy the lungs showed oedema and intra-alveolar haemorrhage. The brain was unremarkable. Analysis of blood showed subtherapeutic trace levels of anticonvulsant. Death was ascribed to drowning in a patient with epilepsy.

Numbers of drowning deaths classified according to age are summarised in Figure 5-1 and details of individual drowning cases are listed in Table 5-1. Table 5-2 and Figure 5-2 provide a breakdown of numbers of drowning deaths in 5-year periods from 1965 to 1994 inclusive, comparing bath and pool death numbers.

DISCUSSION

Drowning most often occurs in infants who have been left in bathtubs, in toddlers who have overbalanced into heavy buckets, and in children of all ages who have fallen into swimming pools (Hyma, 1990; Jumbelic & Chambliss, 1990; Mann, Weller & Rauchschwalbe, 1992; Pearn et al, 1979). The high centre of gravity of infants and younger children combined with muscular weakness and lack of coordination prevent them from extricating themselves from these situations. Peak ages of drowning are

FIGURE 5-1: NUMBER AND AGE OF CASES OF DROWNING TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (AGE 0-16y; 1963-1996)



AGE CATEGORIES			
1	0-11m	9	8y-8y11m
2	1y-1y11m	10	9y-9y11m
3	2y-2y11m	11	10y-10y11m
4	3y-3y11m	12	11y-11y11m
5	4y-4y11m	13	12y-12y11m
6	5y-5y11m	14	13y-13y11m
7	6y-6y11m	15	14y-14y11m
8	7y-7y11m	16	15y-15y11m

TABLE 5-1: DETAILS OF 63 CASES OF DROWNING IN CHILDREN AGED FROM 0-16y IN SOUTH AUSTRALIA FROM 1963-1996

No.	SEX	AGE	YEAR	CIRCUMSTANCES/SITE OF DROWNING
1.	M	1y6m	1964	Home swimming pool
2.	F	3m	1964	Unknown
3.	M	1y3m	1965	Sink in yard
4.	M	11y3m	1965	Convulsion while swimming in sea (epilepsy)
5.	M	2y6m	1966	Bath (multiple bruises)
6.	M	9y10m	1969	Home swimming pool
7.	M	6y9m	1970	Bath (epilepsy)
8.	M	6y11m	1970	Bath (epilepsy)
9.	M	12y	1971	Unknown
10.	M	2y10m	1972	Home swimming pool
11.	M	1y8m	1972	Home swimming pool
12.	M	1y1m	1972	Home swimming pool
13.	M	4y5m	1972	Open effluent pit
14.	M	2y7m	1972	Home swimming pool
15.	M	3y	1973	Home swimming pool
16.	F	1y1m	1973	Large bucket
17.	M	2y	1973	Fishpond
18.	M	3m	1974	Bath (multiple malformations, hydrocephalus)
19.	M	2y	1975	Estuary (fell from boat while mother was sleeping)
20.	F	1y10m	1976	Bath
21.	M	11y2m	1976	School swimming pool (epilepsy and mental retardation)
22.	F	3y4m	1976	Bath (multiple bruises, previous febrile convulsions)
23.	M	1y	1978	Fishpond
24.	F	4y2m	1978	Home swimming pool
25.	M	8y1m	1979	Home swimming pool

TABLE 5-1: DROWNING (cont.)

No.	SEX	AGE	YEAR	CIRCUMSTANCES/SITE OF DROWNING
26.	F	10y10m	1979	Public swimming pool (epilepsy)
27.	F	9m	1979	Unknown
28.	M	8m	1979	Creek (swept from car in flood)
29.	M	2y8m	1980	Bath
30.	M	3y2m	1981	Home swimming pool
31.	M	1y7m	1981	Fishpond
32.	F	1y6m	1981	Irrigation ditch
33.	M	11m	1981	Unknown
34.	M	1y6m	1982	Home swimming pool
35.	M	6y6m	1982	Freshwater lake
36.	F	2y	1982	Home swimming pool
37.	M	1y7m	1982	Home swimming pool
38.	M	2y	1982	River
39.	F	3y6m	1983	Public swimming pool (sucked into filter)
40.	F	2y4m	1983	Bath
41.	M	4y	1983	Home swimming pool
42.	M	1y5m	1983	Bath (died 2 weeks later)
43.	M	2y2m	1983	Home swimming pool
44.	F	9m	1984	Bath
45.	F	8m	1985	Bath (died 3 days later)
46.	M	4y6m	1986	River (fell off jetty, died 1 week later)
47.	F	2y8m	1986	Home swimming pool
48.	M	5y9m	1987	Home swimming pool
49.	F	2y1m	1987	Unknown
50.	M	2y8m	1988	River
51.	M	3y7m	1988	Home swimming pool (died 2 days later)

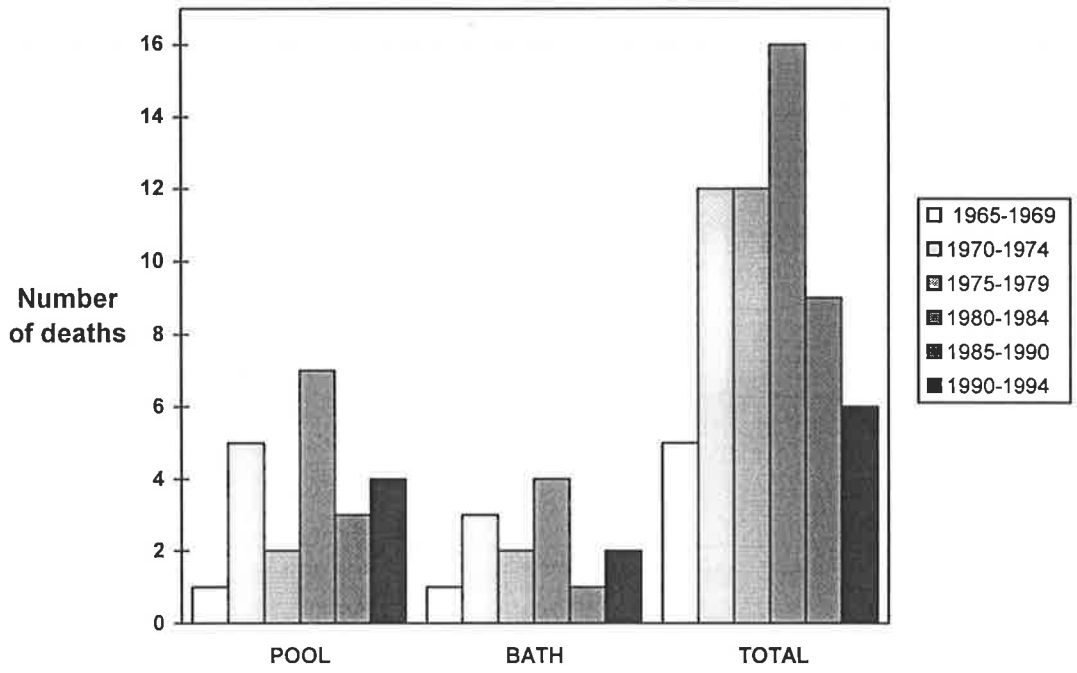
TABLE 5-1: DROWNING (cont.)

No.	SEX	AGE	YEAR	CIRCUMSTANCES/SITE OF DROWNING
52.	M	12y8m	1989	Sea (fell off jetty; epilepsy; died 4 days later)
53.	M	1y7m	1989	Home swimming pool
54.	M	9m	1990	Bath
55.	M	3y6m	1990	Home swimming pool
56.	M	2y	1991	Home swimming pool (died 2 days later)
57.	F	6y2m	1992	Public swimming pool (caught in water return)
58.	M	2y5m	1993	Bath (previous febrile convulsions)
59.	M	4y6m	1994	Home swimming pool
60.	M	7m	1995	Bath (flotation device used)
61.	M	2y2m	1996	Home swimming pool
62.	F	8y	1996	Farm dam
63.	M	2y	1996	Home pool

TABLE 5-2: NUMBER OF CASES OF CHILDHOOD DROWNING PER 5-YEAR PERIOD FROM 1965 TO 1994 TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA

YEAR	POOL	BATH	OTHER	TOTAL
1965-1969	1	1	3	5
1970-1974	5	3	4	12
1975-1979	2	2	8	12
1980-1984	7	4	5	16
1985-1990	3	1	5	9
1990-1994	4	2	0	6
TOTAL	22	13	25	60

FIGURE 5-2: NUMBER AND CASES OF CHILDHOOD (0-16y) DROWNING PER 5-YEAR PERIOD FROM 1965 TO 1994 TAKEN FROM A SERIES OF 369 CASES OF ACCIDENTAL CHILDHOOD DEATH IN SOUTH AUSTRALIA (POOL VS BATH)



under 4 years and between 15 and 19 years (Rodriguez & Brown, 1990), with infants and young children being at greatest risk (Calder & Clay, 1990; Ellis & Trent 1995; Jenson et al, 1992; Kemp & Sibert, 1992; Pitt & Balanda, 1991; Wintemute, 1990). As in the present data, most childhood drowning series demonstrate a higher proportion of boys than girls (Kibel et al, 1990; Nixon et al, 1986; Warneke & Cooper, 1994).

As in this study, other data show that drowning in Australia is second only to deaths from motor vehicle accidents as the most common form of fatal childhood accident (Nixon et al, 1995). There has, however, been a 56% fall in paediatric drowning cases nationally since 1979 following community education and public safety initiatives, with a fall in New South Wales between 1990 and 1995 from 2.0/100,000 to 1.5/100,000 population (Cass, Ross & Lam, 1996; National Injury Surveillance Unit Data, 1996; Pearn, 1988; Pearn & Nixon, 1977a & b). A recent study from New South Wales also demonstrated that although numbers of children drowning in home pools and waterways had declined, there had been no fall in numbers of children drowning in baths or dams (Cass, Ross & Lam, 1996). Table 5-2 demonstrates that there has been a decline in drowning cases autopsied at the Women's and Children's Hospital from a peak of 16 cases in 1980-1984 to 6 cases in 1990-1994. The numbers are too small to ascertain trends in either pool or bathtub deaths.

In South Australia there were 8 drownings in children under 4 years in 1990, compared to 2 cases between the ages of 5 and 14; 6 under 4 years, and 1 between 5 and 14 years in 1992; 7 under 4 years, and 2 between 5 and 14 years in 1993; and only 1 under 4 years compared to 2 between 5 and 14 years in 1994. There were no cases registered for 1991 (National Injury Surveillance Unit Data, 1996).

In young children 90% of drownings occur in home swimming pools and in 40 to 50% of adolescent drownings there has been alcohol consumption. In this study no history of alcohol intake was, however, available and no blood alcohol levels were performed. Although in most cases of toddler drowning, pools lack fencing (Pearn, 1991), the author is aware of at least one drowning occurring in a pool with a

surrounding fence. In this case the child was able to force a way through a poorly-constructed trellice fence to enter the pool area. The usefulness of enforcing legislative requirements for secure pool fencing is self evident. Two children fell from jetties and drowned, one of whom was epileptic, and a 2y-old boy fell from his mother's boat while she was sleeping.

Drowning in the bath tends to occur in infants (mean age 9 months) who are able to sit and pull themselves up to stand (Byard & Cohle, 1994). However, bathtub drowning also raises the possibility of nonaccidental injury and homicide (Feldman, Monasterky & Feldman 1993; Griest & Zumwalt, 1989; Nixon & Pearn, 1977) and this was suspected in two of our cases, given a number of variably-aged bruises in both children and unusual histories. The older ages of several of the bath drowning victims is also a point of concern. No proof of inflicted injury could, however, be established although the extent of the investigation conducted at the time is uncertain. In all of the cases the children had been left unattended for variable periods.

An accurate account of events leading up to death is often necessary to enable the performance of an appropriately detailed autopsy. This is particularly important in cases where a body is recovered from water as post mortem findings may be characteristic but not specific; e.g. the large, bulky, oedematous lungs and foam in airway and trachea seen in typical cases of drowning can also be seen in cases of drug overdose (DiMaio & DiMaio, 1989). Intracranial events may also produce similar changes. Studies of cases of near drowning have shown that electrolyte abnormalities and red cell lysis, as reflected by haemoglobinemia, have minimal clinical significance (Modell & Davis, 1969; Pearn, 1985). Tests such as the Gettler chloride test (Gettler, 1921) which compares the chloride level in blood from the left and right sides of the heart are no longer considered reliable, and diatom analysis is considered by some only to be of use if positive (Knight, 1982), as a negative result does not exclude drowning. The diagnosis is often, therefore, one of exclusion, and can only be made if other causes of death are discounted. The mode of death in drowning is thought to be

hypoxaemic in nature due either to laryngeal spasm or to inhalation of water occluding the airway (Pearn, 1985).

In epileptic patients the diagnosis of a fatal epileptic episode is also by exclusion of other causes, as the mode of death, cardiac arrhythmia triggered by autonomic discharge (Leestma, 1988), will provide no definitive morphological abnormalities for the prosector to describe. The finding of sclerosis of Ammon's Horn is confirmatory evidence that there have been previous epileptiform episodes, but has little relevance to the final event in most cases, and many epileptics will not have this feature (DiMaio & DiMaio, 1989).

There is, therefore, a problem in cases where both epilepsy and drowning may be implicated. When comprehensive descriptions of the terminal event from witnesses, full autopsy examination, and biochemical estimation of anticonvulsants are obtained it may, however, be possible to give an opinion as to the likely terminal sequence of events.

Subarachnoid hemorrhage is, after epilepsy, the second most common cause of sudden unexpected death due to disease of the brain (DiMaio & DiMaio, 1989). Most cases are due to ruptured berry aneurysms in the adult population with the most frequent cause in the paediatric age group being rupture of an arteriovenous malformation (Byard, Bourne & Hanieh, 1991-92; Smith, 1989). These lesions can be very small or inconspicuously sited and may be detected only after careful fixation and histological examination of the brain. This may be a source of difficulty when rapid definitive results are being demanded. In the case described, the existence of an arteriovenous malformation was only proven after appropriate fixation and histological sampling of the brain was performed.

The possibility of a very rare abnormality is always present. Coronary artery hypoplasia as an isolated finding is very much less frequently found than other abnormalities of coronary circulation such as aberrant origin of a coronary artery or

stenosis secondary to arteritis (Lipsett et al, 1994). In this case it was felt that the existence of coronary artery hypoplasia was very likely to be contributory to death as documented episodes of exercise-induced collapse had occurred previously (Byard, Smith & Bourne, 1991a).

This study has shown that 7/63 (11%) of paediatric cases where drowning was initially thought responsible for death, were much less straightforward on detailed examination. This group was significantly older than the remaining children who did not have medical conditions (mean age = 8y 7m, compared to the remaining 56 children whose average age was 2y 11m). It is, therefore, emphasized that the fullest possible history must be obtained from family, medical attendants and from witnesses to the terminal event, and made available to the prosector before autopsy. The case notes should be studied if any are in existence. Where a history of epilepsy is apparent, anticonvulsant levels may be useful, although they are not of themselves conclusive. Uncommon but significant lesions may occur in these cases and meticulous and thorough dissection must be performed even in cases where the cause of death appears obvious. This is of particular importance in jurisdictions not requiring full autopsy in every case of apparent drowning (Kringsholm, Filskov & Kock, 1990). It is possible that death may be misattributed in a number of cases in these regions.

Measures to prevent childhood drowning include publicising common dangers such as unfenced home pools. Potential problems with heavy buckets, sinks and fishponds should also be made known to parents and child carers. Pools should be secured by properly constructed and maintained fences with child-proof gates. Parents should receive information about fences and legislation should be introduced to ensure compliance. Children should be taught to swim, however, young children and particularly toddlers need to be supervised in water at all times. Infants and toddlers should not be left alone in baths, and flotation devices should not be used (Cass, Ross & Lam, 1996). Children in boats and on jetties also need to be constantly monitored

and should be wearing safety vests if they are near the water. Epileptic children should also be supervised at all times in baths, pools and at the beach.

* * * *

CHAPTER 6

SPECIFIC RECENTLY IDENTIFIED DANGERS

STUDY 6-1) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS AND YOUNG CHILDREN ASSOCIATED WITH MESH-SIDED COTS

INTRODUCTION

Identification of a number of risk factors for SIDS has resulted in closer attention being paid to infant sleeping environments as detailed in Chapter 2. The following cases demonstrate another potentially dangerous specific sleeping situation for infants, with possible pitfalls that may occur during death scene examination of such cases (Byard, Bourne & Beal, In press).

CASE REPORTS

Case 1: An 11.5m-old boy was found dead in his cot at around 0630 hrs by his mother. He was lying face down on a 5cm thick foam insert. Although his mother stated that he had being lying in the gap between the wall of his cot and the side of the foam mattress, the investigating police officer reported that the space was only 2 to 5cm in width. The only other history was of delivery by caesarian section at 35 weeks for cephalopelvic disporportion and mild croup at 4 to 5 months of age. He had been otherwise quite well.

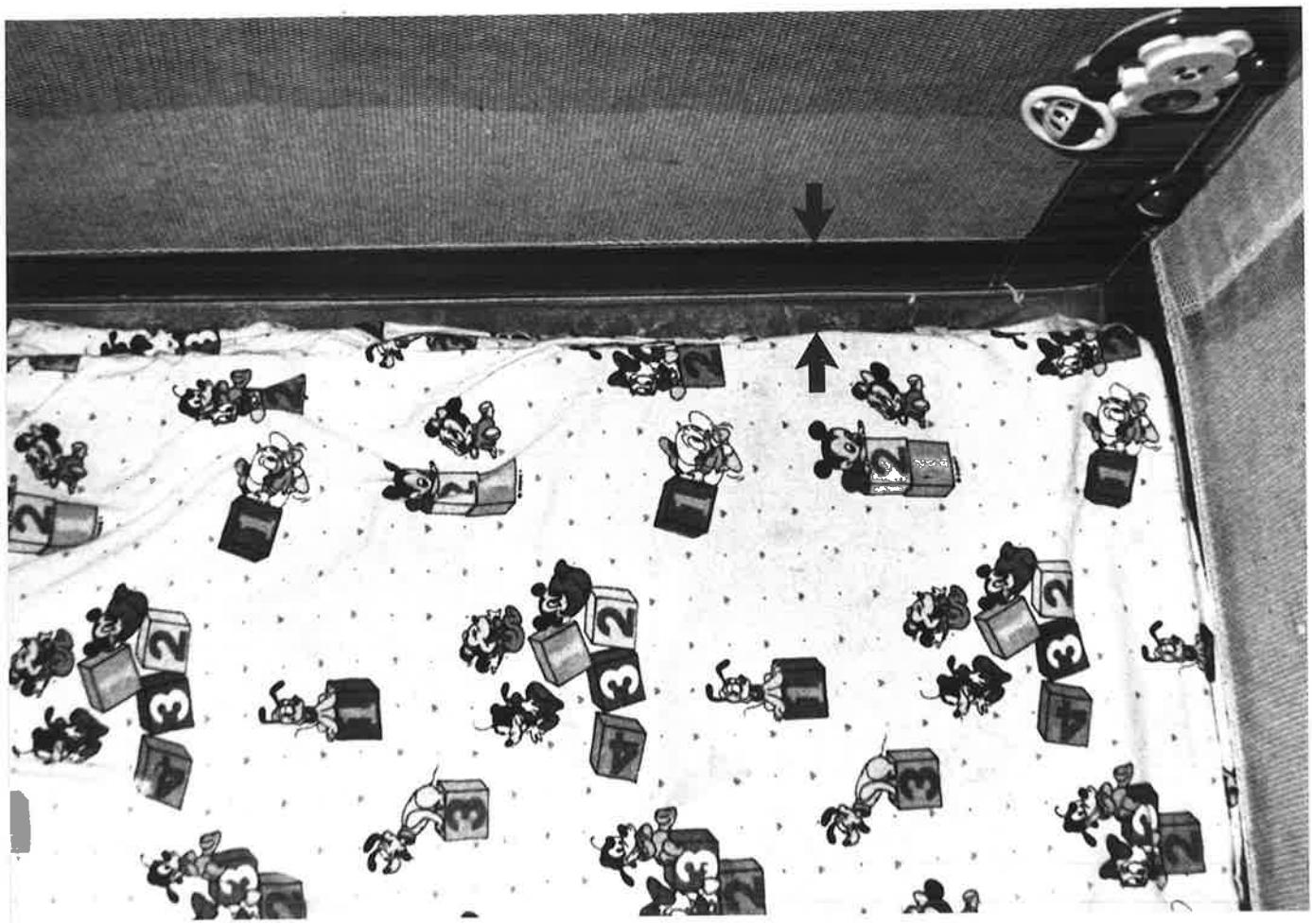
Autopsy examination revealed a normally formed male infant with no evidence of injury. Marked thymic petechiae, congestion and oedema of the lungs and cerebral oedema were nonspecific autopsy findings. Although the features were quite compatible with SIDS the cause of death was considered undetermined given the discrepancy in history between the mother and the investigating officer.

Subsequent investigation, however, revealed that the cot had distensible mesh sides which had allowed the infant's head to slip down between the mesh and foam mattress. The head had been found firmly held by the mesh with the face pushed into

Figure 6-1: A mesh-sided cot in which a 3.5m-old boy was found lying face down between the side of the mattress and the stretched mesh wall (Case 2) (A). The tear in the fabric represents another potentially dangerous problem with this particular cot. Postmortem reconstruction of the position of the body showing a firmly entrapped head in the face down position. The impressions left by clothing on the back occurred after death when the body was left supine (B).

B

Figure 6-2: Size of the gap that existed between the mesh wall and the new mattress in Case 2 (arrows).



the side of the mattress. Based on this new information death was considered due to accidental asphyxia.

Case 2: A 3.5m-old boy was found dead in his mesh-sided cot (Figure 6-1). The original mattress had been augmented with a 9 cm thick mattress covered with synthetic material. Although the body and head had been found along the side of the mattress against the mesh wall, the investigating SIDS worker felt that 'wedging' was unlikely given the relative narrowness of the gap between the mattress and cot side (8 cm). There was no other relevant medical history.

Reconstruction of the death scene with the body placed into the position where it had been found, however, convincingly demonstrated that death had been caused by head entrapment with accidental asphyxia. The head was held firmly in a face down position by its own weight and the elastic recoil of the mesh, against the synthetic material that formed the base of the cot. A 7.5 cm strip of similar synthetic webbing, forming the lower part of the cot wall, was pressed against the left side of the head, and the mattress abutted the right side of the head. Although the gap measured only 8 cm in the unstretched state (Figure 6-2), it readily accomodated the infant's head which had a bifrontal diameter of 9.5 cm and a bitemporal diameter of 11 cm.

Autopsy examination revealed a normally formed male infant with blanching and flattening of the tip of the nose. There was also a small area of superficial excoriation on the left cheek, but no other evidence of injury. Scattered thymic petechiae and congestion and oedema of the lungs were the only other autopsy findings.

Given the facial features at autopsy and the death scene reconstruction, death was considered due to accidental asphyxia following head entrapment.

DISCUSSION

The cases discussed highlight yet another potentially dangerous sleeping environment which may present particular problems at the time of death scene examination. For example, the cause of death in Case 1 was initially considered not determinable given

the apparent conflicting information from the child's mother and the investigating officer. It was only after subsequent investigation was undertaken that the elastic nature of the cot side became known. Although the gap between the mattress and the cot wall was only 2 to 5 centimetres, the mesh wall readily stretched to allow the infant's head to become wedged between the mesh and the soft mattress. In this case the elastic recoil of the mesh pushed the infant's face firmly into the mattress resulting in accidental asphyxia.

In Case 2 the investigating SIDS worker did not consider that accidental asphyxia could have occurred given that the gap between the mattress side and cot wall was less than the width of the infant's head. It was only after the cot was examined at the Women's and Children's Hospital mortuary, with reconstruction of the position of the body, that it became obvious that the infant's head easily slipped into the enlarged gap between the mesh and the mattress. Again the elastic recoil of the mesh played an additional role in the fatal episode by holding the infant's head quite firmly with the nose and mouth compressed against the synthetic material that formed the floor of the cot, resulting in accidental asphyxia due to head entrapment.

In both of the reported cases the original cot mattresses had been either replaced or augmented by thicker, softer, mattresses which left gaps at the side between the mattress and the mesh wall of the cot. However, even at points where the mattress closely abutted the cot wall it was quite easy to place an object the size of an infant's head between the mattress and mesh wall. It also appeared that less force was required to distend the mesh side at points away from the edges and the base. Thus a thick mattress which lifts an infant's head up off the floor of the cot places the head next to an area of more distensible mesh. Unfortunately the thin mattresses which are provided with these cots may not appear comfortable enough to parents who may then replace the mattress with thicker less well-fitting material with its resultant dangers.

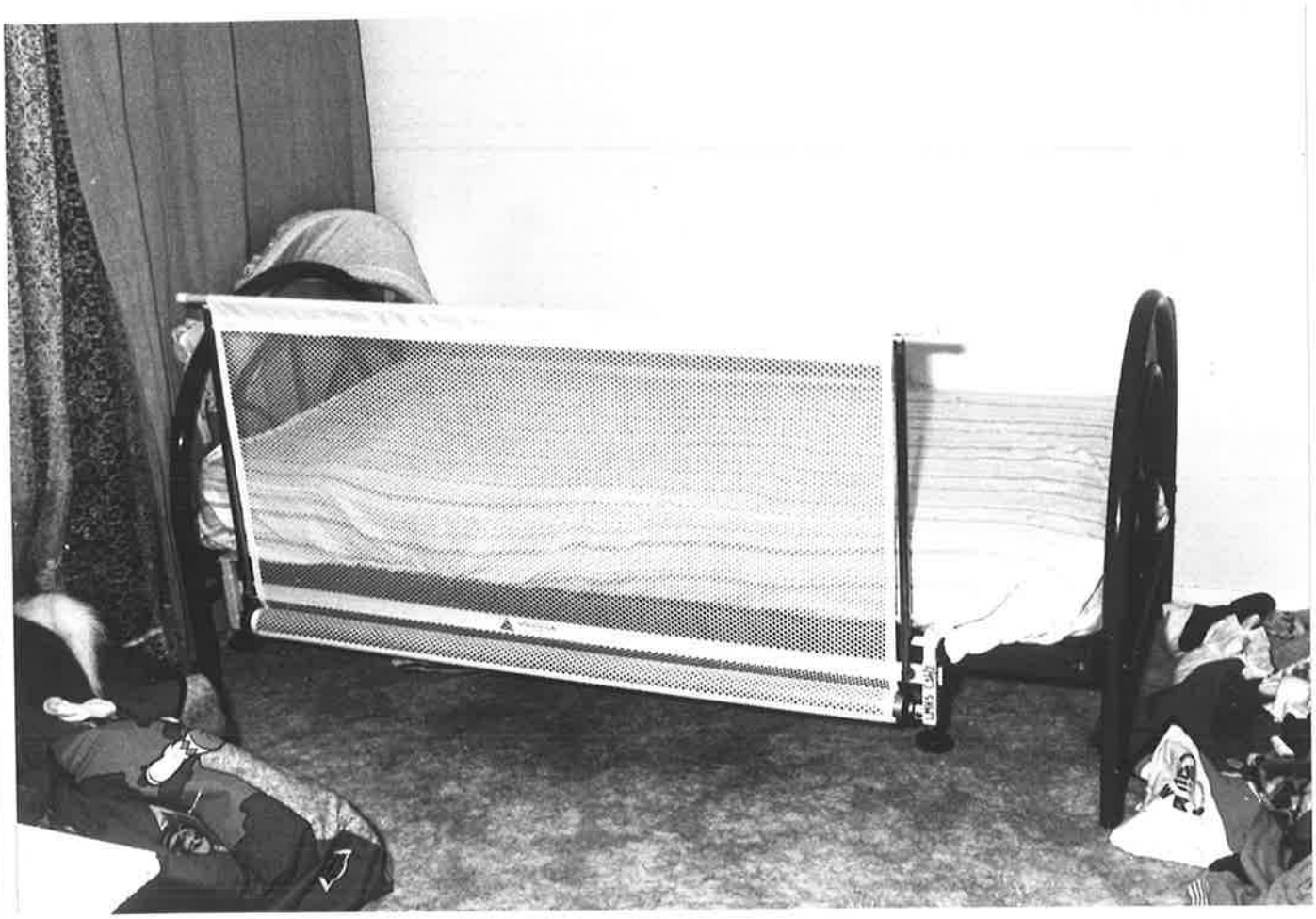
Particular problems which therefore occur with mesh sided cots with added thick mattresses are the potential for entrapment of the head in the trough between the

mattress and wall, with the unique feature of the elastic recoil of the stretched mesh maintaining the infant's head and face in an unsafe position. Although it may appear that the mesh side should allow breathing to occur, the trough where the infant's face is held may be lined by solid synthetic webbing on two sides and mattress material on the third. While warnings not to replace the mattress are present on some of these cots, neither cot in this series had such a warning in place.

Other features which predispose to problems in this situation involve the shape and characteristics of the infant head. The infant head is relatively larger and heavier at this age than at any other, and has weaker supporting neck muscles. It may also be wedge shaped with the bifrontal diameters being smaller than the bitemporal diameters. All of these aspects predispose to the head being able to work its way down into the potential or actual space between the mesh wall and mattress without being able to be easily extricated.

An additional more recent case not involving a cot, but with similar fatal mechanisms, involved a 4y-old mentally retarded boy who was found wrapped in a heavy quilt, wedged between the side of his bed and a retractable mesh netting (Figure 6-3). The mesh was composed of a heavy plastic material and was held in a retractible roller on the side of the bed. It was designed to be pulled up at night and pinioned to provide a safety barrier to prevent the child falling out of bed, while being able to be retracted and hidden during the day. Once the boy had pushed against the mesh and created a large enough space to fall into, the mesh recoil had held him firmly against the side of the bed. At autopsy there were no facial or conjunctival petechiae, and the padding of the body with the quilt had resulted in no unusual patterns of lividity or cutaneous impressions being present to suggest wedging. Thus, the diagnosis of accidental asphyxia was only possible following death scene examination. Although this boy is older than the usual age of children found accidentally wedged in bed, complicating factors included severe mental retardation with poorly developed motor skills.

Figure 6-3: A profoundly retarded 4y-old boy was found wedged between the retractable mesh safety barrier and the side of his bed.



STUDY 6-2) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS AND YOUNG CHILDREN ASSOCIATED WITH STROLLERS AND STROLLER-PRAMS

INTRODUCTION

While it is generally well recognised that broken cots may be a problem, there remain a variety of less well appreciated situations that may be hazardous to sleeping infants resulting from specific design flaws of cots. The following two cases demonstrate this type of hazardous sleeping situation for infants involving stroller-prams (Byard et al, 1996).

CASE REPORTS

Case 1: A 3m-old previously well twin boy was placed to sleep at 0230 one morning prone in a stroller-pram with the foot plate in the upright position. He was found dead at 0600 hanging by his neck from a metal supporting bar attached to the side (Figure 6-4). It appeared that his body had moved down in the stroller-pram causing the foot plate to depress. This had resulted in his body falling out of the now-open front, with his head passing over the metal bar and becoming suspended. At autopsy the major finding was a linear mark running horizontally across the anterior aspect of his neck corresponding to the shape of the metal bar that he had been suspended from. No other patterned skin lesions or bruises were found. There were no facial petechiae. Thymic and visceral pleural petechial haemorrhages were present, as well as pulmonary congestion and oedema.

Examination of the stroller-pram revealed that the foot plate was held in position only by pressure from a grooved plastic strip on a rivet head. The foot plate maintained its position while a weight equivalent to that of the deceased infant (5.3 kgs) was moved slowly down the bedding. Once the weight had moved a short distance onto the upright footplate, it suddenly collapsed resulting in the weight being thrown out of the front of the stroller-pram. Thus, based on the death scene and autopsy findings,

and event reconstruction, death was attributed to neck compression from accidental hanging.

Case 2: An 8m-old previously-well boy was placed to sleep in a stroller-pram with the foot plate in the down position. He was later found dead, his feet and body having passed through the front of the stroller-pram under a horizontal metal bar. (Further details of this case are available in the thesis 'Sudden Natural Death in Infancy and Early Childhood - An Analysis of Aetiological Mechanisms and Pathological Features' (Byard, 1994b; Case 14, p 93).

DISCUSSION

These cases demonstrate that strollers which convert into prams may not be the most appropriate places to put infants to sleep unless certain precautions are taken. In both of these cases the infants had moved down from their original positions until they had slipped through the fronts of the stroller-prams and become suspended by the neck and head, respectively. Although the foot plate in Case 1 had been elevated when the infant was placed in the pram, the weight of the infant's body had been enough to cause it to collapse through the night. This resulted in the infant hanging from the side of the pram. The angle of tilt of the base of the pram also favored the infant moving down towards the foot plate.

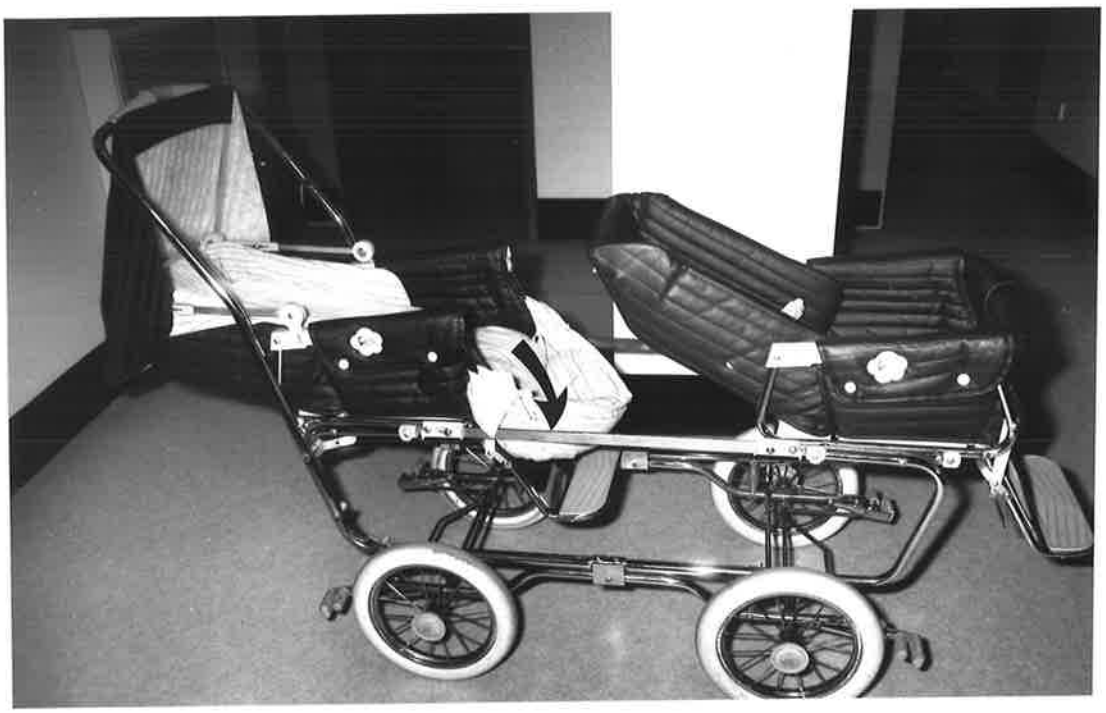
While it is well recognised that strollers may contribute to infant injury, the possibility of lethal accidents has not been emphasized. For example, in their publication 'Prams and strollers- safety requirements', Standards Australia/Standards New Zealand concentrate on important issues of durability, hazardous projections, efficiency of restraints and stability, rather than on potential problems related to sleeping infants (AS/NZS 2088, 1993). This reflects the rarity of fatal cases, however, even standards for the prevention of nonlethal injuries are not mandatory. It is also difficult to determine how often these fatalities occur on a national scale as there has not been a nation-wide review of such cases. In a study of injuries related to strollers

and prams the National Injury Surveillance Unit found 526 infants and toddlers presenting to selected emergency departments between 1991 and 1993. Injuries involved falls, overbalancing, entrapments (mainly fingers), failure of harnesses, collapse and brake failure. No information was available on near-fatalities; for example although 19 cases involved collapse of prams/strollers, the significance was regarded as uncertain given the absence of detailed information on each case (Moller & Dolinis, unpublished). Of interest, in our department records we also have details of a 14-month-old girl who was found dead in a partially collapsed pram. The case occurred nearly 20 years ago and the lack of an adequate death scene investigation at the time, with inconclusive autopsy results, unfortunately precluded its inclusion in the present study.

In a review by Couper et al, (1994) the most common injuries consisted of trauma to the head, face and teeth with concussion. This occurred in 60% of the 149 attendances for stroller-related injuries. Digital entrapment was another injury that was identified, but again there were no fatalities. The cause of the accidents was most often incorrect use of the stroller with failure to use shoulder harnesses and alteration of the centre of gravity by shopping bags which increased stroller instability. The authors noted that no previous systematic study of stroller-related injuries had been undertaken.

Thus, the two cases described extend the type of possible injuries to infants sleeping in stroller-prams to include fatal positional asphyxia and hanging. Unless direct supervision of an infant sleeping in a stroller-pram is possible, or complete blocking of access to the foot plate is achieved, it would be advisable to select a safer sleeping environment where there is less potential for the occurrence of lethal accidents.

Figure 6-4: The stroller-pram from Case 1 in which a 3m-old boy was found dead hanging from a metal bar (arrow) on the side of the pram.



STUDY 6-3) AN INVESTIGATION OF POSSIBLE DANGERS TO SLEEPING INFANTS ASSOCIATED WITH ADULT SIZED 'V' PILLOWS

INTRODUCTION

The following cases involving infants who died while sleeping on adult size 'V' pillows were reviewed following evidence suggesting that the cause of death in one case was suffocation.

MATERIALS AND METHODS

Following the reclassification of a case of sudden infant death from 'SIDS' to 'accidental asphyxia' when suffocation of the infant in a 'V' pillow was suggested, re-assessement of all of the cases of infants who had been sleeping on adult size 'V' pillows at the time of their deaths was undertaken.

Autopsy files at the Women's and Children's Hospital were examined for all cases of sudden infant death associated with the use of adult 'V' shaped pillows. Each case had an examination of the death scene, review of the clinical history and full autopsy examination performed or supervised by a paediatric pathologist. In addition a formal case review was conducted several months after the death, with input where pertinent from clinicians, nurses and social workers.

RESULTS

A total of 4 infants were found who had died while sleeping on adult 'V' pillows; all had died in 1995. The age range was 2m to 6m (mean = 3.9m); male to female ratio = 3:1. Death was thought to be unrelated to the use of the pillow in 2 cases. In the remaining 2 cases, however, there was evidence that the pillows had caused upper airway occlusion/suffocation.

In Case 1, reconstruction of the death scene with a doll demonstrated that, although the infant had been initially placed in a position where the face and nose were

unobstructed (Figure 6-5A), this position had changed. Specifically, the infant had slipped down into the crevice formed by the two sides of the pillow that had been wedged into the bassinette (Figure 6-5B). This had resulted in the infant being unable to extricate herself from a vulnerable position where the upper airway was occluded.

A similar problem occurred in Case 2 where an infant had slipped off the pillow where he had been placed (Figure 6-6A) resulting in the head passing under the overhanging edge of the pillow (Figure 6-6B). Elevation of the pillow by another pillow had exacerbated the situation by increasing the space beneath the 'V' pillow. On review, death in both of these cases was attributed to accidental asphyxia rather than to SIDS.

DISCUSSION

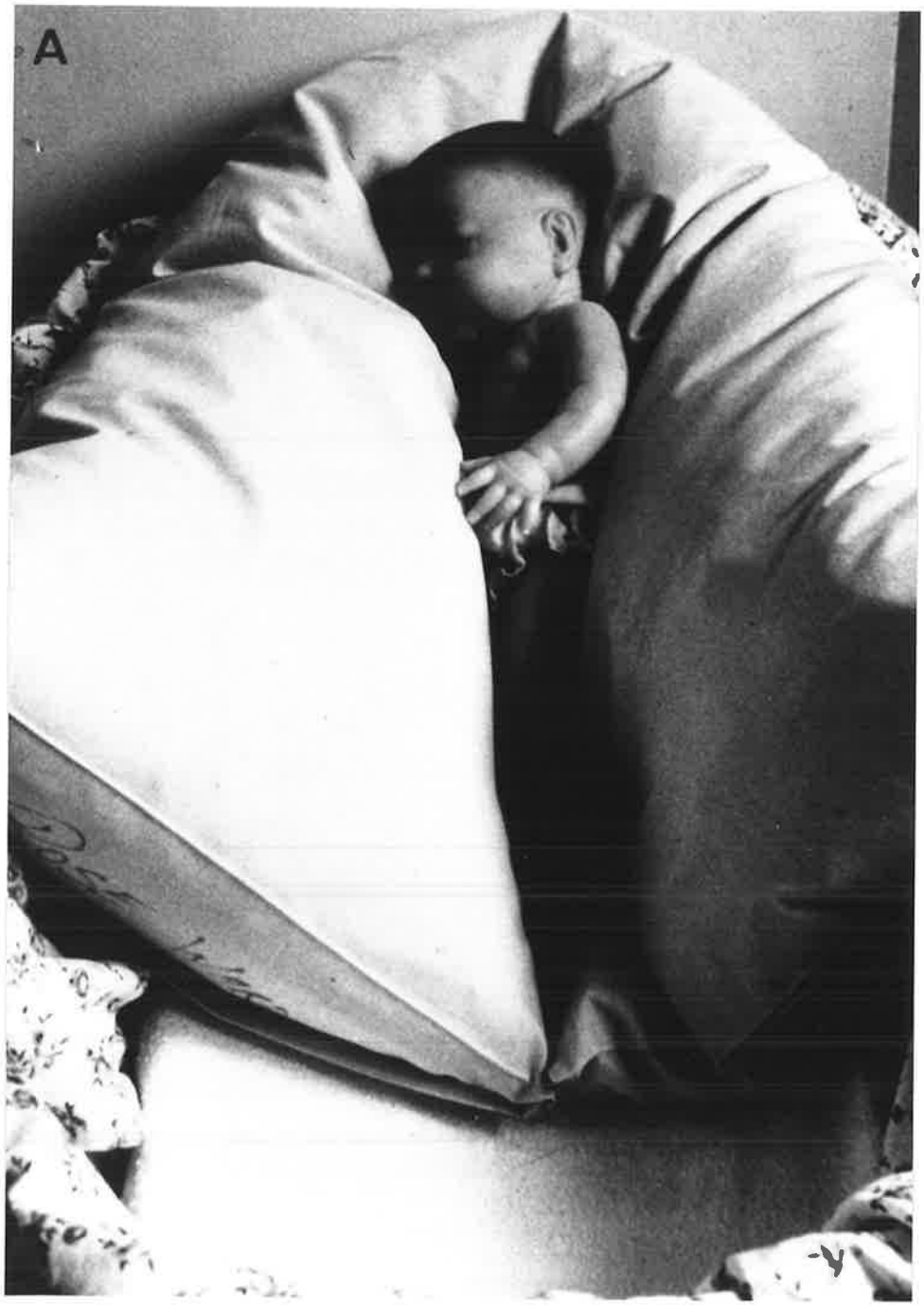
As has been shown, infants are very vulnerable to airway compromise both from dangerous sleeping positions and from certain physiological characteristics. For example, infants have relatively small nasal passages and mandibular rami with close proximity of the epiglottis to the soft palate and relatively large tongues (Byard, 1991; Golding, Limerick & Macfarlane, 1985; Tonkin & Beach, 1988). Once the pharynx closes, considerable positive pressure is required to reopen the lumen (Tonkin, 1983). In addition certain infants fail to respond normally to complete or partial airway occlusion (Byard & Burnell, 1995; Newman et al, 1986). All of these factors may contribute to fatal asphyxia following minor airway obstruction at a young age.

The two cases draw attention to yet another potentially dangerous sleeping environment which may be hazardous to young infants involving adult size 'V' pillows. These pillows have become popular recently in part due to their usefulness in maintaining infant sleeping position. As can be seen in the reconstruction of Case 1, movement from side to face down was not possible due to the pressure of the pillow on the infant's body. Unfortunately this pressure was also applied to the infant's face and nose once the infant had slipped down between the folds of the pillow. The

second case demonstrates another hazardous situation where an infant had slipped under the 'overhang' of the pillow which was raised on a second pillow.

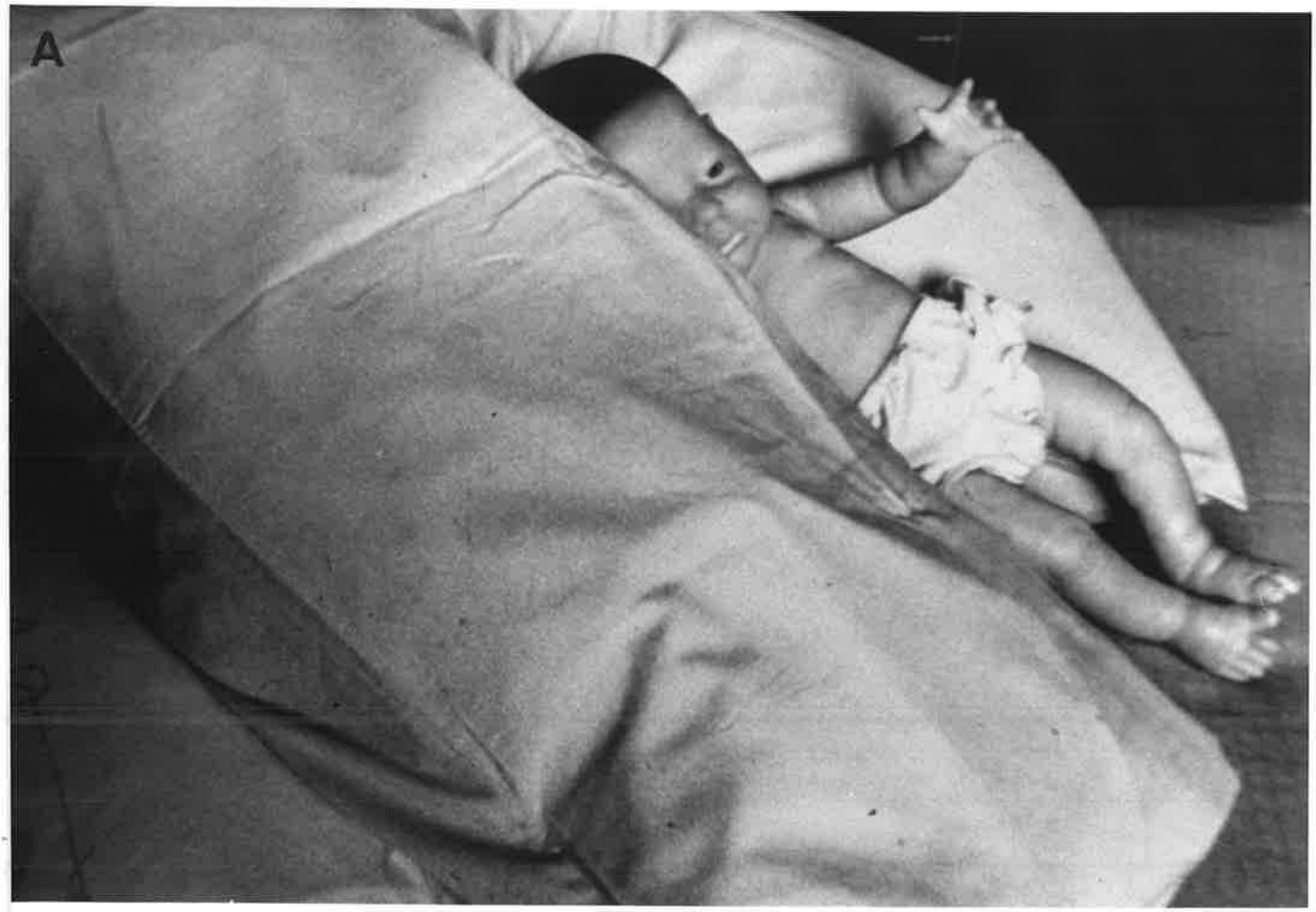
This report, therefore, demonstrates that adult size 'V' pillows may be unsafe for use in infants who may accidentally suffocate if left unsupervised. Given the possibility of accidental asphyxia the use of such pillows to maintain the body position of sleeping infants should be discouraged.

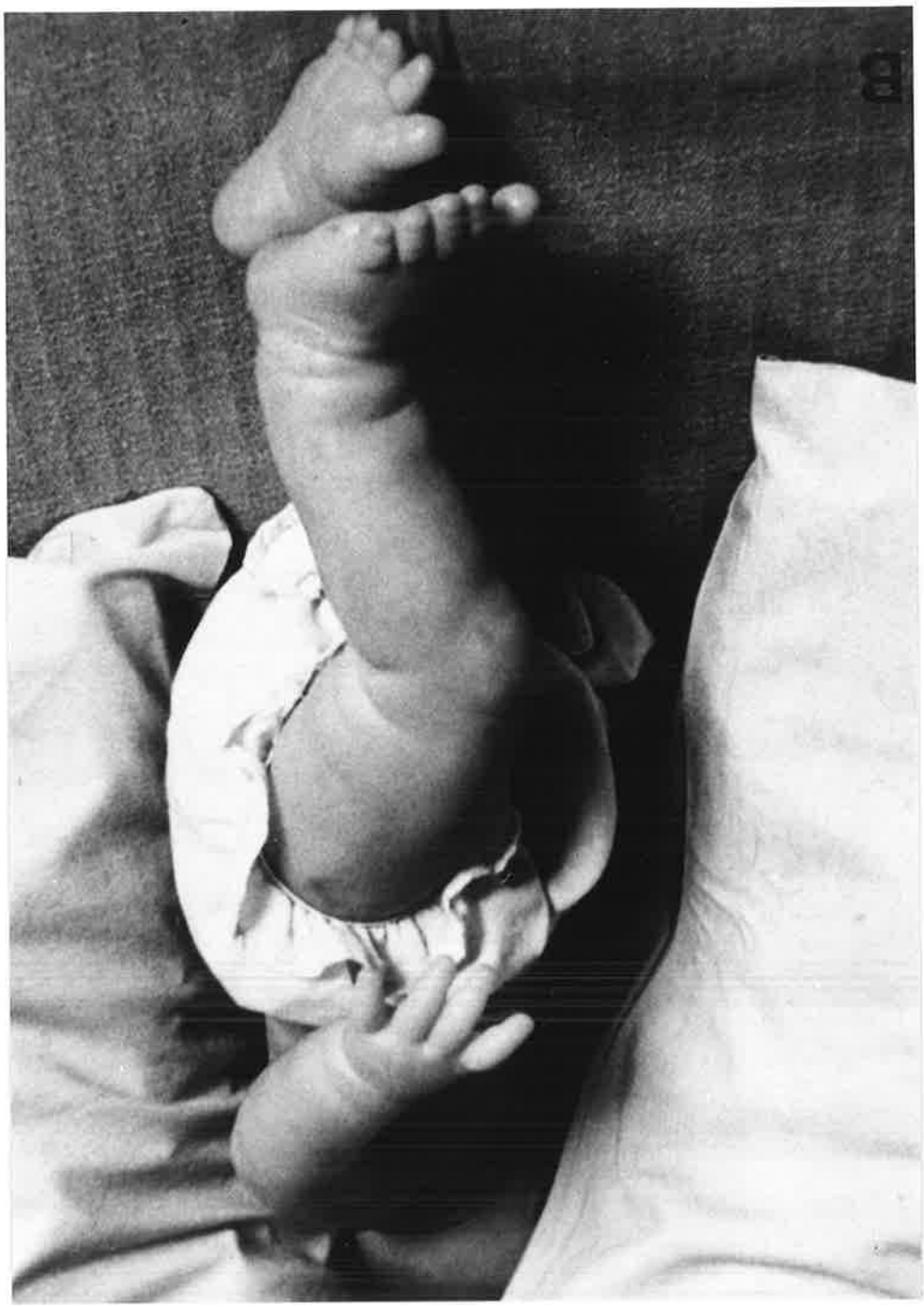
Figure 6-5: Reconstruction of the initial position that the infant in Case 1 was placed in using a doll. Initially the doll is lying on its side with no airway obstruction (A). The second reconstruction shows the position that the infant was found in, having slipped into the depths of the pillow. Complete covering of the face has now occurred (B).




B

Figure 6-6: Reconstruction of the initial position that the infant in Case 2 was placed in using a doll. The doll is lying on its side with no airway obstruction (A). The second reconstruction shows the position that the infant was found in, having slipped beneath the pillow. Complete covering of the face has now occurred (B).





STUDY 6-4A) AN INVESTIGATION OF A POSSIBLE CAUSAL RELATIONSHIP BETWEEN APPARENT LIFE THREATENING EVENTS (ALTE), INFANT HOLDING PRACTICES AND BREAST FEEDING

INTRODUCTION

Airway obstruction is a recognised cause of apparent life threatening events (ALTEs) and sudden unexpected death in infancy. The following cases provide evidence that some infants may fail to respond normally to accidental occlusion of the nose and mouth when being either incorrectly held in the arms of an adult, or breast fed (Byard & Burnell, 1995). Careful investigation of the circumstances of ALTEs may be crucial in determining possible contributing environmental factors and underlying mechanisms.

CASE REPORTS

Case 1: A 1m-old boy, who had been born 3 weeks prematurely, was investigated at a country hospital following two episodes of apnoea with cyanosis. Each episode had occurred in the early hours of the morning approximately 20 minutes after breast feeding and had required mouth to mouth resuscitation by the maternal grandmother. The infant had been otherwise well and thriving although significant gastro-oesophageal reflux was documented by barium swallow and a small ventricular septal defect was confirmed on echocardiography. He remained well for the week-long hospital admission. The day after discharge a similar event occurred at 0200 hrs while being held in his grandmother's arms, resulting in referral to the Adelaide Children's Hospital for monitoring and further investigation. He remained well for the 5 day admission with a normal overnight pneumogram. The day after discharge, however, a similar event requiring resuscitation again occurred at 0200 hrs with his grandmother.

In reconstructing events around early morning feeding it transpired that the grandmother would also rise with her daughter and would hold the infant in her arms

after feeding had finished. On the first two occasions the mother had returned from making tea to find the infant's feet blue. The remainder of the infant was not visible as he was being clasped under his grandmother's ample breast in a fold of a large quilted polyester dressing gown.

Reconstruction of events in the involved clinician's office clearly demonstrated obstruction of the infant's nose and mouth with apnoea and mild cyanosis resulting. This occurred when the infant was held in the usual position by his grandmother wearing her quilted dressing gown. The infant had been fed and was asleep at the time of the supervised ALTE and made no attempts to breathe or struggle at any time. Following identification of the cause of the ALTEs, the grandmother altered her pattern of infant holding and no further episodes have occurred. The child is now 6 years old and has no medical problems.

Case 2: A 1m-old boy, who had been born 2 weeks prematurely, was admitted to hospital following a prolonged period of apnoea that had been labelled as a 'near-miss SIDS' episode. Extensive investigations including chest X ray, electrocardiogram and an overnight pneumogram were undertaken. These failed to reveal any abnormalities. No apnoeic episodes occurred in hospital.

Following interview by the involved clinician it was discovered that just as in the previous case, the ALTE had occurred while the infant was being held firmly in his grandmother's arms. His grandmother also had large breasts and wore a quilted dressing gown.

A similar supervised event reconstruction was undertaken with the grandmother and infant which showed that the deeply-sleeping infant made no effort to breathe or struggle whilst being sufficiently obstructed to become mildly cyanotic. The reconstruction was terminated at this point. Again, following identification of the cause of the ALTEs, the grandmother altered her pattern of infant-holding and no further episodes have occurred. The child is now 5 years old with no medical problems.

Case 3: A 2m-old girl was noted by her mother to be limp and unresponsive while being breast fed. The infant had been held firmly with her face to the mother's left breast with the nipple in her mouth while her mother sat on the floor and leaned forward to watch another child through a window. Resuscitation was attempted with only partial response and brain death was diagnosed in hospital 2 days later. The only significant clinical history was of three episodes in an obstetric hospital, soon after delivery, of cyanotic episodes associated with apnoea while breast feeding. One episode was witnessed by nursing staff. Respiration recommenced after each episode once the infant was removed from the breast. There had been no further episodes since discharge from hospital.

At autopsy the infant was small (height and weight below the third percentile) but normally formed. There was no evidence of congenital cardiovascular, pulmonary or central nervous system disease except for a small (2mm diameter) patent ductus arteriosus not associated with cardiomegaly. All organs were normal except for mild microvesicular steatosis of the liver and diffuse hypoxic/ischaemic encephalopathy of the brain. Although death was attributed to hypoxic/ischaemic encephalopathy secondary to cardiorespiratory arrest of unknown cause, circumstantial evidence implicated failure to respond to airway occlusion.

DISCUSSION

The possibility of inappropriate holding of infants resulting in airway obstruction gains support from the reported ALTEs occurring in two apparently healthy infants while being cradled in their grandmothers' arms. Both infants had cyanotic episodes successfully reproduced under careful medical supervision while being held by their grandmothers who were wearing quilted dressing gowns. In each case the infant's face was, at the time of the ALTEs, being pushed firmly, albeit inadvertently, against large breasts covered by thick clothing. Neither infant struggled or made any attempt to breathe while being unintentionally suffocated. The concerned reactions of the

grandmothers when confronted with the cause of the ALTEs and the absence of similar episodes in other family members left in the care of these women in the past and subsequently, argued against a diagnosis of Munchausen syndrome by proxy (Byard & Beal, 1993).

Although reconstruction of the events surrounding the ALTEs in these cases provided evidence implicating mechanical airway occlusion, rather than an occult medical condition, the infants' lack of response to airway occlusion was unusual. Whether this reflected an underlying abnormality in the two infants which interfered with a normal response to airway blockage is uncertain. Reduction in arousal response to partial airway occlusion has been demonstrated in sleeping infants (Newman et al, 1986) and thus may also have played a role in the ALTEs of these infants. Carbon dioxide accumulation with rebreathing (Kemp & Thach, 1991) is another theoretical mechanism which may have contributed to the ALTEs in Cases 1 & 2, given that both had their faces pushed into bulky clothing. Neither infant had any further episodes once the problems with holding practices had been rectified.

The cardiorespiratory arrest of a 2m-old infant while breast feeding in her mother's arms is also unusual. Subsequent investigation revealed that she had been held to her mother's chest while her mother had been sitting on the floor, stretching forward to look out of a window at an older child playing outside. Although death was attributed to hypoxic-ischaemic encephalopathy secondary to cardiorespiratory arrest of unknown cause there is circumstantial evidence implicating failure to respond to possible airway obstruction from breast feeding. The previous history of cyanotic episodes while being breast fed on at least three occasions is also supportive of ALTEs triggered by airway obstruction occurring in this position. Unfortunately the autopsy findings in cases of accidental or non-accidental airway obstruction in infancy are often identical to those found in sudden infant death syndrome. Thus although a 'SIDS-like' event cannot be excluded in Case 3, the circumstances surrounding the terminal episode are not typical.

As previously noted, an investigation of the circumstances surrounding death is necessary in all cases of unexpected infant death. The current cases show that a similar investigation or reconstruction of events may also provide additional information which might not otherwise be available in cases of ALTEs. This could take the form of a carefully supervised re-enactment of the usual situation in which ALTEs have occurred, with continued close observation by an attending paediatrician with resuscitation equipment at hand. The possibility of inappropriate positioning, or of failure of normal response to airway blockage, should be considered in these cases. If ALTEs occur around feeding time, observation of how caretakers place the infant during and after feeding may be informative.

STUDY 6-4B) AN INVESTIGATION OF POSSIBLE DANGERS TO INFANTS ASSOCIATED WITH ADULT COSLEEPING

INTRODUCTION

It has been hypothesized that a parent sleeping in the same bed as an infant, i.e. cosleeping, is a desirable activity which, as well as promoting infant development and health, may reduce the risk of SIDS (McKenna, 1986; McKenna et al, 1993). However, Scragg et al (1993) have recently produced data which demonstrate that cosleeping is associated with an apparent greater risk of sudden infant death. Despite these data, there remains considerable resistance to accepting that the risk of death may be increased when infants cosleep with adults.

CASE REPORTS

Case 1: A 1m-old boy was found dead in bed with his mother, father and 3y-old brother. He was positioned between his parents with his head pointing towards the head of the bed. No other relevant history was forthcoming and the autopsy findings of thymic, pleural and epicardial petechiae were relatively nonspecific and consistent with a diagnosis of SIDS. Although no markings were found on the body and there were no cutaneous or conjunctival petechiae, it was noted that 'overlaying' could not be excluded.

Subsequently in a second statement from the mother to police about a related matter, further information became available that both adults had consumed a considerable amount of alcohol on the night of the infant's death resulting in the mother losing consciousness for some time prior to going to bed. In the second statement the mother also commented that when she regained consciousness/woke up she had found the dead infant underneath her; she had not realised that she had been 'sleeping on

him'. These features strongly suggest a diagnosis of asphyxia due to overlaying, rather than natural disease.

Case 2: A 1m-old boy was placed by his grandmother against his mother's breast to feed in the early hours of the morning. His mother had been sleeping in her bed and had only partially roused when her son was placed in bed with her. She had then fallen back asleep. Several hours later when the grandmother went to check on the mother and son, she found them in the exact position that she had left them, with the mother on her right side and the infant on his left side. The infant was dead with his face against his mother. Although the autopsy findings were nonspecific, and SIDS could not be excluded, the circumstantial evidence strongly implicates accidental asphyxia as the cause of death while the infant was breast feeding.

Case 3: Another mother took her 1m-old son to bed to breast feed at around 0100 hrs. She did not awake until 0850 hrs when she went to check his bassinette, where she thought that she must have placed him after feeding. Upon rechecking her own bed, she found him dead, on his side covered in bed clothes, facing where she had been sleeping. The autopsy findings were nonspecific. Again, while those mechanisms that cause SIDS cannot definitely be excluded in this case, the circumstantial evidence points strongly to accidental asphyxia associated with breast feeding and maternal fatigue.

COSLEEPING STUDY

Examination of cosleeping rates from the South Australian SIDS Database has shown that between 1983 and 1995 there has been an absolute, as well as a relative, increase in the numbers of infants who were asleep in bed with an adult at the time of their death. The percentage of SIDS infants found dead in bed with an adult between 1983 and 1990 was 7.5% compared with 30 to 33% in later years. The percentage of control infants aged 3 months who were bed sharing in South Australia in 1988 was

1.5% (Bourne, Beal & Byard, 1994). More recent control data are not available for South Australia.

DISCUSSION

The concept that sharing a bed with an infant is dangerous goes back thousands of years and was first mentioned in the Bible in the Judgement of Solomon (Byard & Cohle, 1994). One of the women claiming an infant as her own had lost a child due to 'overlaying'. The theory of overlaying was quite straightforward, in that a mother was thought to have accidentally/deliberately suffocated her infant by laying over him or her during the night. In eighteenth century Europe, and later, various types of wooden and metal frames were constructed under which infants were placed while in an adult bed to safeguard against this perceived danger. However, during the nineteenth century a realisation grew that infants might die suddenly and unexpectedly without having been exposed to the risk of overlaying (Fearn, 1834). From this observation developed an understanding of the the complex system of characteristics, risk factors and mechanisms now associated with SIDS.

Having thus fallen out of favour as a possible explanation for unexpected infant death, overlaying remained in the historical literature, along with entities such as *status thymicolymphaticus*, as an interesting but hardly plausible lethal mechanism (Byard & Cohle, 1994).

In recent times, however, there has been a resurgence of interest in problems associated with cosleeping. Bass and Kravath (1986) found 6 cases of suspected overlaying in a series of 26 consecutive cases of presumptive SIDS. Although Templeman (1892) observed in 1889 that 46% of infant deaths in Dundee, Scotland occurred on Saturday nights when alcohol consumption was high, little attention was paid to the role of sedation until recent authors suggested that parental sedation (including alcohol consumption), as well as obesity and fatigue may predispose to accidental infantile asphyxia (Bass & Kravath, 1986; Gilbert-Barness et al, 1991; Rintahaka & Hirvonen, 1986).

Conversely, Scragg et al (1993) suggested that the risk of infant death in a cosleeping situation is not due to overlaying, but instead is associated with passive smoking. In this series, infants exposed to higher levels of smoking had an increased risk of sudden death, even if cosleeping had not occurred on the night of death. A possible explanation of this association was of passive smoking by the infant with resultant hypoxia. Overlaying was further discounted as there was no demonstrable increase in rates of sudden death in cosleeping situations where there were two adults in bed, as might be expected due to the smaller amount of space available to the infant. Although alcohol was not considered a risk factor in this group, alcohol consumption on the night of death was not specifically documented in the study.

The demonstration of dangerous sleeping environments such as partly filled water beds and polystyrene-filled cushions has drawn attention to other potentially significant factors in cosleeping situations, such as rebreathing of carbon dioxide (Gilbert-Barness & Barness, 1993; Gilbert-Barness et al, 1991; Kemp & Thach, 1991) For example, it has been shown that carbon dioxide may accumulate around an infant's face under bed covers, and animal studies have shown the accumulation of potentially lethal levels in rabbits breathing into polystyrene-filled cushions(Kemp & Thach, 1991). Overheating of infants due to poor heat radiation in certain situations such as the prone position has also been associated with an increased risk of SIDS (Fleming et al, 1990; Ponsonby et al 1992; Ponsonby et al 1993), although Scragg et al (1993) did not demonstrate an increased risk of infant death with hyperthermia in their series.

Thus, although there are contradictions in the literature, potential dangers arising when an infant is sleeping under the covers alongside a parent may include not only positional asphyxia from compression by the adult's body, but also the possibility of overheating and carbon dioxide rebreathing. Unfortunately it is not possible at present to predict which infants are going to be vulnerable to these particular stressors.

Another problem which arises in trying to determine the effect of cosleeping is in defining precisely what is meant by the term. For example, the position of infants in parental beds varies from the head of the bed to the centre, or distant from a single

cosleeping adult to wedged between two or more adults and/or siblings. The time spent cosleeping also varies greatly from a short interval occasionally after the morning feed, to regularly every night. An important point to ascertain in taking histories from parents with an infant death who regularly coslept with their child is whether or not cosleeping occurred on the night of death.

Recently it has been proposed that cosleeping is a more appropriate form of sleeping for an infant as it more closely resembles tribal and animal behaviour. It is suggested that exposure of an infant to sensory stimuli in the parental bed improves sleeping patterns and enhances neurosensory development (McKenna, 1986; McKenna et al, 1993). This concept has been taken up by the popular press which has emphasized the more 'natural' aspect of shared sleeping. However, it appears that the pathophysiology may be more complex than mere alteration of sleep patterns, given a report from New Zealand which demonstrated a reduced risk of SIDS for infants who slept in the same room as their parents, but not in infants who slept in rooms with siblings (Scragg et al, 1996). The increase in cosleeping rates in South Australia among parents whose infants subsequently die suddenly and unexpectedly is also of concern (Bourne, Beal & Byard, 1994), and may lend support to suggestions that cosleeping can be potentially dangerous.

It seems reasonable, therefore, to accept that a small infant asleep or being breast fed in a soft bed could be in danger of accidental suffocation if the mother falls asleep. Certainly if the adult is exhausted and/or sedated, an infant's struggles may not cause the parent to wake (Anon, 1992). Alternatively, certain infants may not show normal arousal responses when their airways are occluded (Byard & Burnell, 1995). In retrospect, the occurrence of nearly half of the infant deaths in Templeman's series on Saturday nights, a time traditionally associated with intoxication, does lend credence to the possibility of alcohol playing a role in the fatal episodes (Templeman, 1892). More than one adult in the bed, such as in the case described, may be an additive risk factor.

It is recognised that the closeness of cosleeping may have positive effects such as improved parental-child bonding, synchronised parental and infant sleep patterns and

longer breast feeding. Given the evidence for an increased risk of death in a cosleeping situation, and the lack of alternative data to demonstrate its safety, however, health care workers and others recommending cosleeping may find it difficult to defend their position in the event of an infant death.

Although the New Zealand Cot Death Study found no data to support overlaying or infant holding practices as causes of death (Scragg et al, 1993), an early study by Abramson attributed death due to overlaying in 19 out of 139 infants (Abramson, 1944). It was claimed by Abramson that death had resulted from occlusion of the nose and mouth of these infants who had been pressed against the breast or arm of mothers who had fallen asleep. Unfortunately lack of detailed death scene descriptions prevents reassessment of these cases.

The possibility of maternal sedation as a contributing factor to accidental infantile asphyxiation during breast feeding was demonstrated by the case of a 23-year-old mother who fell asleep in a local South Australian hospital. Her 2 day-old boy was later found by nursing staff to be unresponsive beneath her right breast. A coronial inquest attributed death to asphyxia due to overlaying, with a contributing factor being maternal drowsiness due to sedative ingestion (Anon., 1992). Sedation or intoxication were not implicated in the infant deaths in Cases 2 & 3.

The occurrence of two infant deaths in association with breast feeding, and the documentation of a previous infant death while breast feeding during the day (Study 6-4A), suggests that certain infants have to be carefully monitored for evidence of airway occlusion in this circumstance. These cases also indicate that breast feeding of infants in the parent's bed while the mother is fatigued may not be a safe child care practice.

As we are at present unable to predict which infants are at risk from cosleeping, are there acceptable alternatives which combine the possible advantages of cosleeping while reducing the risk of death? One solution for parents who want close contact with their infant may be to place the cot next to the bed within arms reach, paying attention to other risk factors such as avoidance of the prone position and exposure to cigarette smoke (Byard, 1994d). This would have the infant in a more carefully

controlled environment while still permitting parental interaction and stimulation. Pinning the infant's clothing to a pillow at the head of the bed or immobilising with swaddling may also prevent movement down into the bed. If breast feeding is to be undertaken by the infant's mother in her bed the presence of a second awake person to remove the infant if the mother falls asleep may be a wise precaution. Until further information is available on the role of cosleeping in infant death, alternatives such as these may be the safest options.

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CHAPTER 7

IDENTIFICATION OF SUBTLE ACCIDENTAL INFANT DEATHS

INTRODUCTION

The following study was undertaken to determine the percentage of cases that have presented as probable 'SIDS' deaths in which an accidental cause of death was found. In addition, this study evaluates the contribution of death scene examination, autopsy examination and particularly retrospective formal case review to the establishment of the final diagnosis.

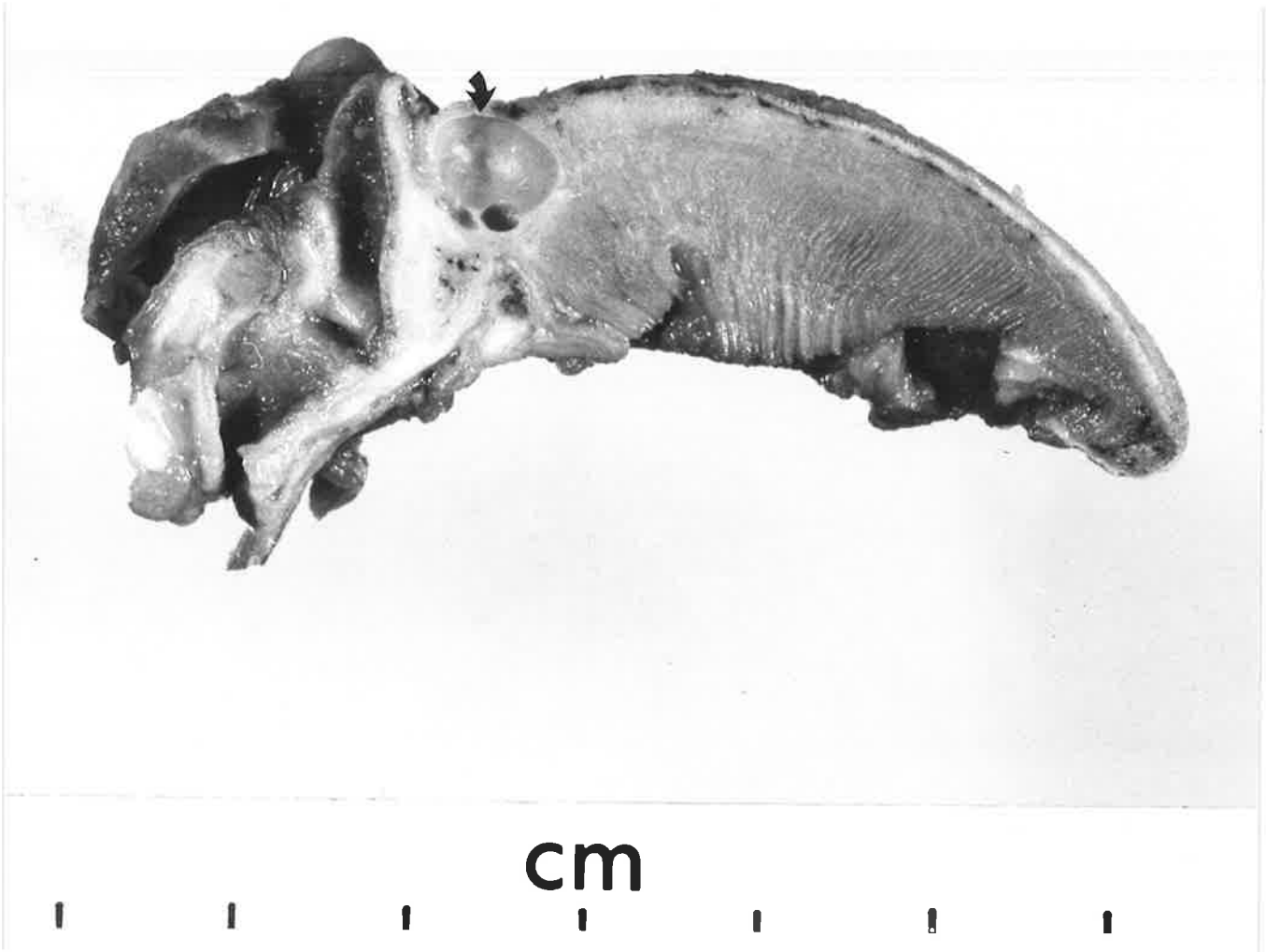
MATERIALS AND METHODS

All cases of sudden and unexpected infant death presenting to the Department of Histopathology at the Women's and Children's Hospital between January 1st and December 31st 1995 were evaluated to determine the percentage of cases where death could be attributed to accidents. In addition, determination of the contribution of death scene examination, autopsy dissection and retrospective formal case review to the establishment of the final diagnosis was undertaken. The cases included all infants who had died suddenly and unexpectedly in South Australia aged between 1 week and 1 year of age. Formal death scene examinations had been conducted by police officers, a trained worker from the SIDS Association of South Australia and/or a paediatric pathologist. Clinical histories were reviewed, and autopsies were performed or supervised by a paediatric pathologist. Formal review of each case was retrospectively conducted prior to the cases being entered into the South Australian SIDS Database. Information was sought from involved paediatricians, nurses and social workers where appropriate.

RESULTS

A total of 24 sudden and unexpected infant deaths occurred. Three deaths were immediately attributable to accidents due to clear histories and death scene and autopsy findings that were quite consistent with the histories provided. These involved a drowning, a motor vehicle accident and a hanging.

Figure 7-1: Sagittal section of the tongue in an infant who died suddenly and unexpectedly in whom a large thyroglossal duct cyst was found at the base of the tongue.



Death in one of the remaining 21 cases was attributed to accidental asphyxia based on the death scene reconstruction and autopsy findings. This infant had been found lying against the mesh side of his cot (further details are available in Chapter 6).

All cases had bacteriological and virological examinations, the results of which were not significant, and none of the infants exhibited evidence of nonaccidental injuries.

Only one case was identified as being due to natural disease based purely on pathological grounds. This infant was found at autopsy to have a sizable lingual thyroglossal duct cyst at the base of the tongue (Figure 7-1), a condition recognised as a cause of sudden infant death due to upper airway obstruction (Byard, Bourne & Silver, 1990; Hanzlick, 1984).

In the remaining 19 cases, SIDS was considered the final diagnosis before formal case review. At review, however, additional information that was not forthcoming at the time of the initial investigation resulted in 3 further deaths being attributed to accidental asphyxia; 2 involved large 'V' shaped pillows and the other, heavy parental alcohol intake and the finding of the infant under the mother when she awoke/regained consciousness (More information on these cases may again be found in Chapter 6).

Thus, of 24 cases of sudden death, 3 were due to obvious accidents, 4 to accidental asphyxia and 1 to a lingual thyroglossal duct cyst. Deaths in the remaining 16 cases are attributed to SIDS. Twenty-four percent of cases of sudden infant death were, therefore, due to causes other than SIDS; 5% were identified at death scene examination/reconstruction, 5% were found at autopsy and a further 14% were only found during subsequent case review.

DISCUSSION

In recent years one of the major developments in paediatric autopsy pathology has been in obtaining international consensus on the manner in which infant deaths are investigated (Krous, 1995). This has not been without debate, and there has been disagreement on such basic matters as the diagnosis and on the significance of

common pathological findings (Byard & Krous, 1995; Mitchell et al, 1994). Progress has, however, been made and there is growing support for a standardised international autopsy protocol and death scene examination.

The current study was undertaken to determine what percentage of cases of sudden infant death were due to accidents, and at what stage in the investigative process SIDS was excluded. In addition, the usefulness of formal case review in assigning a final cause of death was assessed. The results demonstrate that formal case review provides a forum whereby significant information that may not be available at the time of autopsy can be brought forward and discussed. This information may have considerable impact on other cases as well, For example, information regarding the possible involvement of a large 'V' shaped pillow in the death of one infant enabled further investigation of a similar case with the result that death in both cases was attributed to accidental asphyxia (Byard & Beal, In press). There may also be considerable public health consequences to this finding.

One of the advantages that a retrospective review has over information gathering at the time of autopsy is that parents and others involved in the infant's death will have had time to reconsider aspects of the case that they may not have thought important at the time of death. Additional pertinent information may also become available coincidentally; for example further details of the overlaying case only became known after the mother had given police a second statement about a related matter which involved details of the night of the infant's death. As well, later review of information pertaining to a particular case provides a way of ensuring that both the original death scene examination and data collection were performed adequately. If information appears to be lacking, a review of that part of the process can then be undertaken. Thus formal case review may act as a quality control mechanism to ensure that an investigation has been performed optimally.

Considerable disagreement exists in the literature as to the number of cases of sudden and unexpected infant death that present in a manner typical of SIDS in which a diagnosis other than SIDS is made following autopsy examination. For example,

Gilbert-Barness & Barness (1992) have claimed that nearly 50% of cases presenting as SIDS are due to other diagnosable conditions. This contrasts with Byard, Carmichael & Beal (1994) who found that less than 9% had an alternative diagnosis and Fleming et al (1991) who found only 8% were due to definable causes. In the literature, however, it has been proposed that of cases presenting as SIDS, 10% may be due to homicide, 10% to accidental asphyxia, 39% to occult infection, 5-20% to metabolic disorders and 15% to miscellaneous conditions (Byard & Krous, 1995; Gilbert-Barness & Barness, 1992; Emery, 1989; Rambaud et al, 1992; Emery et al, 1988).

It is interesting to note that the current study has demonstrated that 24% of cases presenting as 'SIDS' had an alternative diagnosis made once further investigations had been performed. This contrasts with the figure of less than 9% published by the same group for the period 1983 to 1992 (Byard, Carmichael & Beal, 1994), and raises the possibilities that diagnostic shift, a change in autopsy protocols, or an increase in accidental infant deaths are occurring in this community. However, as the overall number of cases of sudden infant death attributed to causes other than SIDS has remained steadily under 10 per year over the past decade (Byard & Beal, 1995), the seven cases in 1995 show that there has been no absolute increase in the number of accidental infant deaths. Although with more rigorous investigation of recent cases it could be expected that a greater number of accidental deaths with only subtle findings would be diagnosed, this does not appear to have happened. The data show, therefore, that the increase in percentage of non-SIDS cases in 1995 has not been due to an increase in other causes of death, but is due instead to the dramatic fall in numbers of SIDS cases, from 52 in 1987 to 16 in the study year.

In conclusion, this study has demonstrated the value of retrospective formal case review in cases of subtle accidental infant death. More accurate determination of the cause of death was possible in a significant percentage of cases and a possible dangerous sleeping environment was identified that was not obvious at the time of autopsy in 2 infants. In addition, the increase in percentage of cases where death was

attributed to an accident has been shown to be a relative, not an absolute, increase due to the marked decline in SIDS deaths.

* * * *

CHAPTER 8

PROBLEMS IN DIAGNOSIS

STUDY 8-1) ALTERATION OF THE PRESENTING HISTORY OR DEATH SCENE

INTRODUCTION

Even when there are no suspicious circumstances in cases of accidental infant or childhood death, parents may not provide a factual account of events, or may subsequently alter their statements. Two recent examples illustrate certain problems which may occur.

Case 1: Once the possibility of accidental asphyxia became known to the parents of one of the infants who was found dead in a mesh-sided cot (Case 2 in Study 6-1), the history of the initial position of the body was changed. Specifically, the parents denied that the body had been wedged down the side of the cot as they had originally described. It was only after considerable counselling that the parents finally agreed that their original statements had been correct. It appears that they would have prefered a diagnosis of SIDS to one of accidental asphyxia which might imply fault on their part.

Case 2: Another example of a false statement from a parent occurred in the hanging death of an 18m-old boy. Although his mother initially claimed to have found him dead on the floor of his room, the presence of an obvious ligature mark around his neck with facial petechiae indicated an asphyxial death. It was only after a cord was found looped over a wardrobe handle during careful inspection of the child's room by the attending pathologists (Figure 8-1) that the mother admitted that she had found him hanging from the door. It is presumed that her guilt at leaving the cord dangling from the handle within her son's reach had caused her to try to disguise the circumstances of his death (Byard, 1995).

Figure 8-1 A cord looped between wardrobe doors in a room where an 18m-old boy was found dead by his mother. The cord position had been altered before investigating officers and pathologists arrived at the scene to a position which would have been out of the boy's reach (A). The boy had, however, been found hanging from the cord which had been left looped around only one handle. (B).

B

DISCUSSION

These two examples show the difficult situation of parents and investigators when faced with a potentially preventable accidental infant death. Even in cases of childhood death where there is no parental culpability, there will still be strong feelings of guilt which will require sensitive handling and skilled counselling. This is even greater in situations where possible parental error has resulted in death, however, it is vital to clearly identify the sequence of events so that other children within the family will not be put at risk. Careful assessment is essential in these cases to ensure that there has been no alteration of the death scene with disguising of the true sequence of events. The broader issue of community safety is ill served if potentially dangerous childhood environments such as the ones illustrated are not identified.

STUDY 8-2) SIGNIFICANT COINCIDENTAL FINDINGS AT AUTOPSY IN ACCIDENTAL CHILDHOOD DEATH

INTRODUCTION

Establishing the cause of death at autopsy may be difficult in cases where there are minimal findings, or where a plausible mechanism linking abnormal findings with death cannot be found (Leadbeatter & Knight, 1987). Even when lesions known to cause death are present, determining their precise role in the fatal episode may be difficult (Cordner, 1993). The following cases emphasize aspects of this problem by demonstrating significant, potentially lethal disorders at autopsy which were considered entirely coincidental to death.

MATERIALS AND METHODS

The 369 cases of accidental death taken from the files of the Department of Histopathology at the Women's and Children's Hospital over a 34 year period from 1963 to 1996 were searched for circumstances where significant, potentially-lethal, but coincidental disease was found at autopsy. Clinical histories and autopsy findings were reviewed.

RESULTS

Three cases of unexpected accidental death were found in which major clinically occult and potentially lethal, disease was identified that was considered to be coincidental to death (0.2% of coronial autopsies). The clinicopathological details are summarized below.

CASE REPORTS

Case 1: An apparently previously well 19m-old boy ingested peanuts while sitting at the kitchen table with his parents. This was followed by a coughing fit, cyanosis and respiratory arrest. He was dead on arrival at hospital. (Case 7; Table 3-2).

At autopsy the boy showed no dysmorphic features and the major finding was of a peanut wedged in the trachea immediately below the larynx. The lungs were congested and oedematous but otherwise unremarkable, as was the heart macroscopically.

Microscopically, however, florid myocarditis was found with infiltration of necrotic myocardium by mononuclear cells (Figure 8-2). The serological titre for Coxsackie B4 was positive at 1/256. The only other finding of note was of an abnormal karyotype with a balanced translocation - 46 XY,t(12;16)(q24;p11). (The karyotype had been routinely performed as part of an ongoing study unrelated to the case.) The boy's father was subsequently found to have an identical karyotype.

Death was attributed to accidental asphyxia following inhalation of a peanut with coincidental findings of established viral myocarditis and an abnormal familial karyotype.

Case 2: A 3m-old boy had slipped off pillows where he had been placed to sleep, into the hollow at the back of a couch. He was found wedged between the pillows and couch with his face turned into the couch. (Case 20; Table 2-2). His past history included a presumed upper respiratory infection treated with paracetamol in the week leading up to death.

At autopsy the major finding was of a moderate focal myocardial lymphocytic infiltrate associated with myocyte necrosis (Figure 8-3). Given the position of the body, death was attributed to accidental asphyxia, with an incidental finding of myocarditis. Viral studies were uninformative.

Case 3: A 13m-old boy collapsed and died 12 hours after ingesting soldering flux containing potassium fluoride. (Case 4; Table 1-4-i) His previous health had been unremarkable with no history of developmental delay or epilepsy.

At autopsy markedly elevated fluoride levels were demonstrated. Also present were numerous cardiac rhabdomyomas (Figure 8-4), facial angiofibromas and cortical tubers, diagnostic of tuberous sclerosis (Byard, Smith & Bourne, 1991c.) Death was attributed to accidental potassium fluoride poisoning with an incidental finding of tuberous sclerosis.

DISCUSSION

The reported cases demonstrate the importance of thorough autopsy examinations in cases of sudden childhood death. Although the causes of death were obvious, careful examination revealed further occult organic disease in all three children, two of whom had heritable conditions. Although coincidental to death, potentially-lethal conditions such as these may be an important discovery in the paediatric autopsy as they may have considerable impact on surviving family members. The reported cases also show that significant disease may be present in infants and young children with minimal histories and clinical signs, which remains unsuspected until autopsy examination has been performed. It is, of course, recognized that death may be due to a combination of factors; for example in Case 2 it is possible that myocarditis played a role in the death, although scene examination implicated positional asphyxia/suffocation.

Two important functions of a pathologist are to identify disease accurately at autopsy, and to render an opinion as to the likelihood of such disease contributing to or causing death. Making the latter decision is not always straightforward. For example, determining the role played by minor congenital malformations or inflammatory infiltrates in sudden infant and childhood death is often difficult, given the variability that exists in the clinical manifestations of such lesions (Byard & Krous, 1995). Conversely, when an abnormality or disease process is found which has caused extensive disruption of vital organs, with interruption of blood supply, disturbance of

cardiac conduction pathways, widespread tissue necrosis, or profound hemorrhage, it is usual to accept this as the cause of death given appropriate circumstances.

The present cases demonstrate, however, that care should be exercised in proposing a cause of death even when obvious significant disease is present. For example, each of the reported children had coincidental lesions which could have been held responsible for sudden death under different circumstances. Similar results were also found in a study by Cohle and Lie of cardiac conduction tracts from 30 individuals aged between 15 and 30 years who died of trauma or from noncardiac causes. Coincidental lesions included conduction tract abnormalities, nodal arteriopathy and myocarditis, conditions which may also have been considered lethal in the absence of other findings (Cohle & Lie, 1990). Knight has emphasized that the 'overlay of degenerative cardiovascular disease' present in the elderly may also result in incorrect assumptions about possible causes of death (Knight 1991), i.e. death may be attributed to a particular lesion or disease because of a lack of another explanation, rather than from specific evidence of organ dysfunction.

While death is often confidently ascribed to lesions found during postmortem assessment, the nature of autopsy material makes it difficult to precisely correlate histological lesions with functional impairment. For example, it is often impossible to determine why apparently identical lesions result in death in one individual and yet cause minimal disturbance in another. This reflects the complex and idiosyncratic nature of pathophysiological processes and the limitations of autopsy pathology. It also underlines the importance of obtaining good clinical and death scene information so that the final decision on the cause of death can be guided by clinicopathological correlation rather than based on pathological findings in isolation.

* * * *

Figure 8-2: Marked lymphocytic infiltrate and myocyte necrosis in occult myocarditis in Case 1 (Haematoxylin & eosin, magnification x 80).

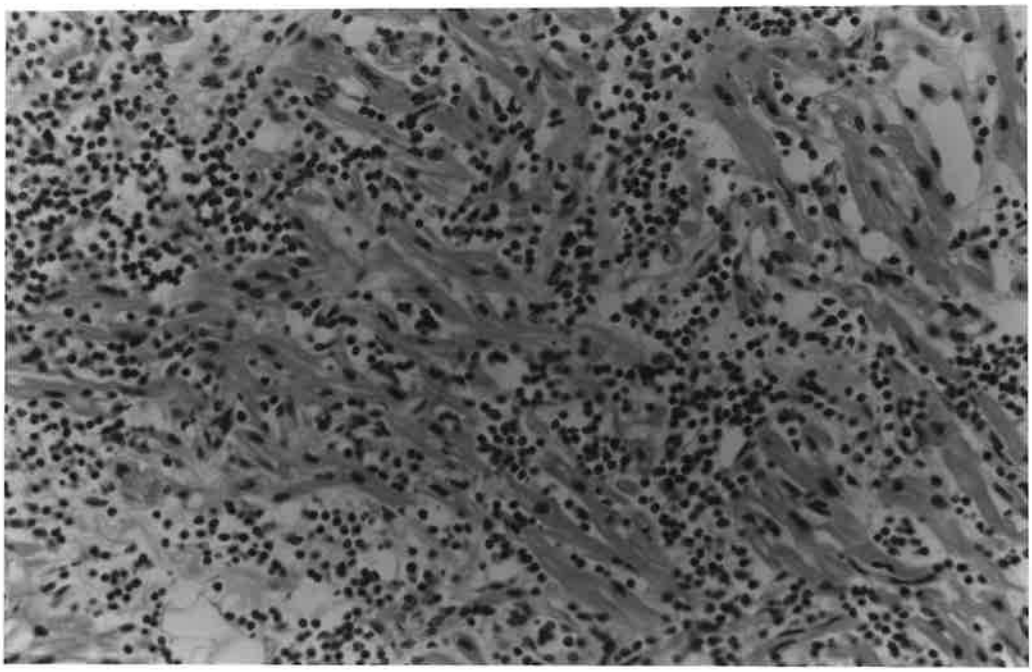
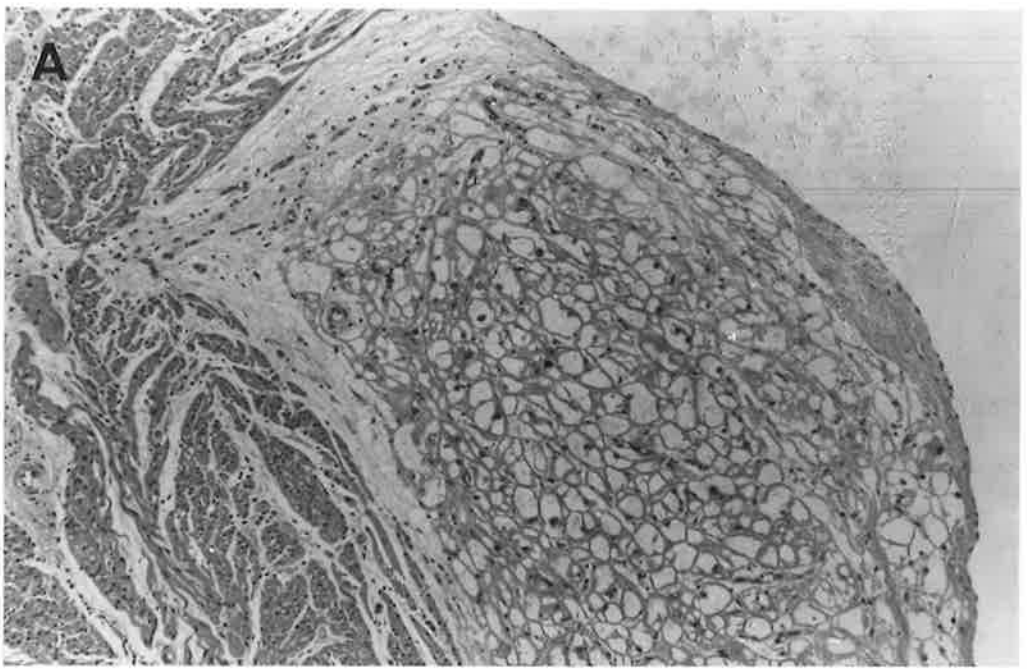
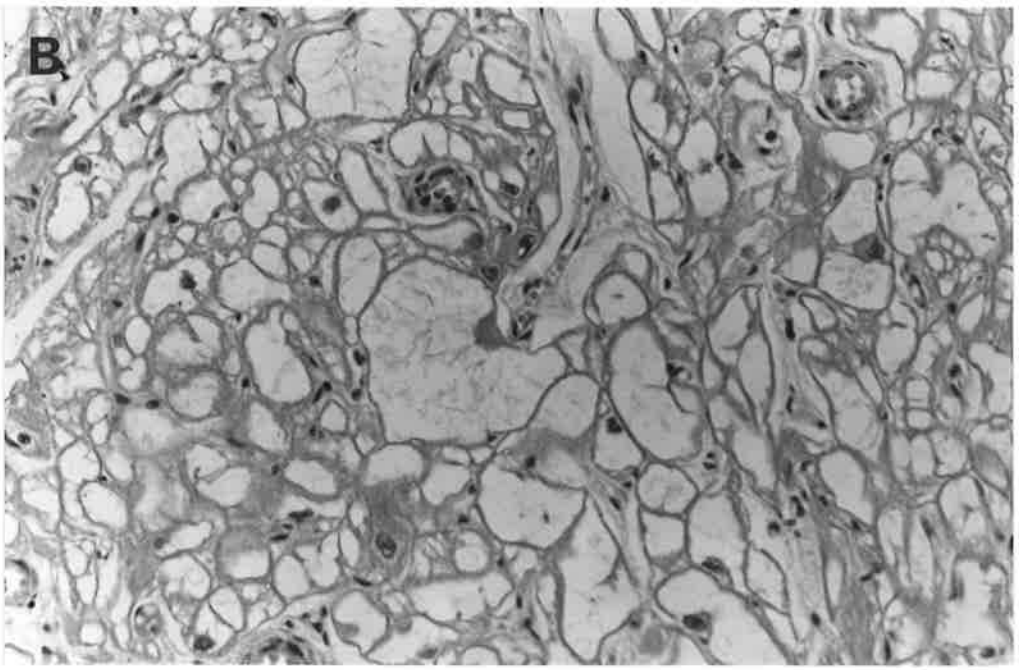


Figure 8-4: Circumscribed subendocardial rhabdomyoma within the heart with adjacent normal myocardium in Case 3 (A). Higher power demonstrating typical 'spider' cells (B) (Haematoxylin & eosin, magnifications x 50; x 100).





CHAPTER 9

CONCLUSIONS

This thesis has provided one institution's experience of accidental childhood deaths with assessment of individual cases and series of cases. Not every case of accidental childhood death in South Australia over the 34 years of the study has been included in the thesis for reasons that have been outlined previously. However, by carefully defining specific groups and time periods within the overall study, particular problems have been identified which have led to recommendations and actions which will have direct public health implications and effects.

Review of the data demonstrates that the rates of certain subgroups of accidental childhood death have been following national trends. For example, the number of children killed in motor vehicle accidents has fallen both locally and nationally in recent years. This is most likely the result of a number of public health initiatives which include the compulsory use of seat belts, utilisation of baby capsules, and anti-speed and anti-drink driving campaigns. Similarly, the number of childhood deaths involving bicycles have declined, most likely influenced by compulsory helmet wearing legislation. Reduction in deaths from poisoning in young children has occurred since the introduction of childproof containers and the launch of campaigns aimed at increasing adult awareness of potential dangers in the house and workshop. Similar falls have occurred in the rates of fatal childhood immersion.

The data also unfortunately demonstrate that childhood deaths from accidents still occur. Problem areas include drownings in unfenced pools and in baths, and fire deaths of children who have been playing with matches, car lighters or flammable liquids. In addition the study has highlighted a need for action in the areas of farm and childcare centre safety, as well as in the specific areas of unsafe feeding and sleeping practices. Because of these problems, the author has been an initiator and/or an active participant in recent campaigns which have resulted in the preparation and/or launch of four safety packages for parents and childcare workers. These are detailed further in Appendix I.

It is too early to assess the impact of these initiatives, however, the safe eating project pre- and post- campaign surveys suggest that local parents and child carers are

now better informed about safe feeding practices for toddlers. Additionally, very preliminary data appear to show that the number of admissions to hospitals in South Australia from choking on food have fallen in the time since the campaign launch. No fatalities have been recorded in children in South Australia from food aspiration over the past two years since the campaign. To date only a precampaign survey has been completed for the safe sleeping project, and the farm safety material is at present being distributed for public comment.

Although not traditionally recognised as being involved in the field of child safety, the paediatric pathologist is in an excellent position to achieve significant results. For example, by careful documentation of cases of accidental childhood death the author has set up the 'Keeping Your Baby and Child Safe Programme' (see Appendix 1), and the South Australian SIDS Database which includes a database of both SIDS and control infants. The programme and database enable careful monitoring of trends in accidental childhood death, and the rapid identification of new dangers. As a number of the cases studied have presented 'dead on arrival' or directly to the mortuary, they have bypassed the hospital system and therefore are not included in clinical assessements of unsafe childhood environments. It is left to the pathologist to be the key figure in collating information on such deaths.

Examples of successes that have arisen from work in the Department of Histopathology include the identification of potential dangers to young infants of overhead suspended rocking cradles. i.e. Following the deaths of two infants which were attributed to these cradles, the cradles were withdrawn from public sale, investigated by the Office of Consumer Affairs, and voluntarily modified by manufacturers. New Australian standards were established and there have been no further deaths in infants in these cradles in South Australia in the four years since the study. Other potentially dangerous sleeping environments which have been identified through the programmme include mesh-sided cots, stroller-prams and 'V' shaped pillows. As well as publishing details in the medical literature the author has used media interviews and public lectures to increase awareness of these dangers. The

author is also a member of the International Society for Child and Adolescent Injury Prevention and will be co-director and plenary speaker at two conferences in the United States this year which will specifically deal with aspects of child safety.

Although cot safety standards are not at present compulsory, both the Federal and NSW Ministers for Consumer Affairs are investigating the possibility of legislation. Both offices have been supplied with research papers and recommendations from the author with regard to safe sleeping environments. The success and cost effectiveness of legislation in other areas of child safety, such as in the prevention of drowning, has been convincingly argued (Pearn, 1991).

If pathologists are to have an effect in fatal injury prevention in children, certain basic requirements must be achieved to ensure that cases are investigated to the fullest extent. For example, autopsies must be performed to a consistent and acceptable standard, and death scene assessments must be made either by a pathologist or by trained personel with whom close liaison is maintained. The author has been involved with the section of the International SIDS Global Strategy Pathology Group, headed by Dr P McFeeley (Albuquerque, USA) and Prof H Krous (San Diego, USA), that is establishing a standardised international protocol for autopsy examination and death scene investigation of cases of sudden infant death. It is also essential that information and standardised diagnoses are stored in an appropriate computerised database to enable ready access. Although formal case review is not usually carried out in cases of sudden infant and childhood death, Chapter 7 demonstrates the importance of such an exercise, with 14% of cases having alternative diagnoses to SIDS made after this type of review.

The underlying philosophy of this thesis is that aspects of paediatric pathology can be made into a preventative science. Parents cannot be expected to be aware of all of the risks to their children, and therefore require assistance in the form of instructions, guidance and legislation where appropriate. As has been demonstrated in the thesis, once risks to children have been identified by the pathologist, recommendations for change can be made and steps can be taken to implement them.

The author firmly believes that undertaking such activities must result in a decline in the number of fatal and nonfatal injuries that may occur in children. The validity of this belief will be determined by the numbers of deaths and hospital admissions from childhood accidents that will occur over the ensuing years in South Australian hospitals.

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APPENDICES I & II

APPENDIX I: DETAILS OF THE 'KEEPING YOUR BABY AND CHILD SAFE' PROGRAMME

During the course of autopsy work and research into aspects of sudden childhood death the author became concerned that certain types of childhood fatalities were not decreasing in incidence. As a result of this observation a programme entitled 'Keeping Your Baby and Child Safe' was initiated in the Department of Histopathology, WCH, aimed at increasing public awareness of childhood dangers and their avoidance. The process involves review by the author of every case of sudden death under the age of 16 years that presents either to the WCH or State Forensic Science (where the author is a visiting consultant pathologist). The cases are also reviewed by at least one paediatrician (for example, Dr. Susan Beal, WCH). The circumstances of death, lethal mechanisms and causes of death are analysed with formulation of possible preventative measures. This thesis represents a compilation of much of that work. As a result of this programme the author has worked with hospital colleagues and a number of groups from outside the hospital, including Kidsafe, Farmsafe, Child, Adolescent and Family Health Service, SA SIDS Association, SIDA (NSW), SIDS Association (ACT), National SIDS Association, The Menzies Centre for Population Health Research & the University Department of Paediatrics (Tasmania), the SA Farmers Federation, the SA Police Association and WorkCover, to produce educational packages for parents and child carers. Examples of four of the projects with which the author has been involved include:

a) **"Safe Eating for Young Children - Prevent Choking on Food"** 1995. (Group coordinator - Ms Anne Johnson, Health Promotions Officer, WCH). A copy of the pamphlet is attached.

b) **"Create a Safe Sleeping Environment for Your Baby"** 1995 (Group coordinator - Dr Anne-Louise Ponsonby, Menzies Centre for Population Health Research). A copy of the pamphlet is attached.

c) **"Safe Sleeping for the Under 2's"** 1996-97. (Group coordinator - Ms Anne Johnson, Health Promotions Officer, WCH). A copy of the pamphlet, safety checklist and hospital policy guidelines are attached.

d) **"Child Safety on Farms - A Guide for Child Safety in the Farming Industry"** 1996-97. (Group coordinator - Mr Phillip Clark, WorkCover, SA). A copy of the draft discussion document is attached.

In addition in the last year the author has contributed to the South Australian Police Association Child Safety Handbook and the National SIDS Association pamphlet on "Reducing the Risk of SIDS", as well as giving media interviews and public lectures on aspects of child safety.

* * * *

APPENDIX II: RELEVANT PUBLICATIONS

Much of the information presented in this thesis has been previously published in peer-reviewed journals, is 'in press', or has been submitted for publication. Papers with data that have been incorporated into the text are listed below in chronological order to acknowledge the coauthors contributions. The author of this thesis has played a major role in the production of the textbook (11 1/2 out of 13 chapters), and in the chapters, papers, proceedings and letters cited.

Textbook

1. **Byard RW**, Cohle S. *Sudden Death in Infancy, Childhood and Adolescence.* Cambridge University Press, Cambridge, England, 1994.

Book chapters

1. **Byard RW**. Sudden and unexpected death in infancy and early childhood - Diagnostic possibilities. In : *Expert Evidence.* Eds. Freckleton I, Selby H. The Law Book Company, North Ryde, (In press).

Papers/proceedings

1. Smith NM, **Byard RW**, Bourne AJ. Death during immersion in water in childhood. Am J Forensic Med Pathol 1991; 12: 219-221.

2. **Byard RW.** Is cosleeping in infancy a desirable or dangerous practice? J Paediatr Child Health 1994; 30: 198-199.

3. **Byard RW.** Unexpected death due to acute airway obstruction in child care centers. Pediatrics 1994; 94: 113-114.

4. **Byard RW,** Beal SM, Bourne AJ. Potentially dangerous infant sleeping environments. Arch Dis Child 1994; 71: 497-500.

5. Beal SM, **Byard RW.** Accidental death and sudden infant death syndrome. J Paediatr Child Health 1995; 31: 269-271.

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7. **Byard RW.** Mechanisms of unexpected death in infants and young children following foreign body ingestion. J Forensic Sci 1996; 41: 438-441.

8. **Byard RW.** Hazardous infant and early childhood sleeping environments and death scene examination. J Clin Forensic Med 1996; 3: 115-122.

9. **Byard RW,** Beal SM, Simpson A, Carter RF, Khong TY. Accidental infant deaths and stroller prams. Med J Aust 1996; 165: 140-141.

10. **Byard RW,** Gallard V, Johnson A, Barbour J, Bonython-Wright B, Bonython-Wright D. Safe feeding practices for infants and young children. J Paediatr Child Health 1996; 32: 327-329.

11. **Byard RW**, Bourne AJ, Beal SM. Mesh-sided cots: yet another potentially dangerous infant sleeping environment. Forensic Sci Int 1996; 83: 105-109.

12. **Byard RW**. Significant coincidental findings at autopsy in accidental childhood death. Med Sci Law (In press).

13. **Byard RW**, Beal SM. 'V' shaped pillows and unsafe infant sleeping. J Paediatr Child Health (In press).

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15. **Byard RW**. Does sudden collapse in a child care centre warrant the designation 'creche coronary' syndrome? Proceedings of the 14th Meeting of the International Association of Forensic Sciences, Tokyo, Japan, 1996 (In press).

16. **Byard RW**. The role of death scene examination in separating accidental asphyxia from SIDS. Proceedings of the 14th Meeting of the International Association of Forensic Sciences, Tokyo, Japan, 1996 (In press).

17. **Byard RW**, Hilton JMN. Infant suffocation and bed sharing. Proceedings of the 13th Australian and New Zealand International Symposia on the Forensic Sciences, Sydney, Australia, 1996 (In press).

18. **Byard RW**. "Crib or Coffin?', the problem continues. Proceedings of the 13th Australian and New Zealand International Symposia on the Forensic Sciences, Sydney, Australia, 1996 (In press).

19. **Byard RW**, Gilbert J, Lipsett J, James R. Farm and tractor-related fatalities in children in South Australia. Med J Aust (Submitted).

20. **Byard RW**, MacKenzie J, Beal SM. Formal retrospective case review and sudden infant death. Acta Paediatr Scand (Submitted).

21. **Byard RW**, Hilton JMN. Overlaying, accidental suffocation and sudden infant death. J SIDS Infant Mort (Submitted).

Letters

1. Bourne AJ, Beal S, **Byard RW**. Cosleeping and sudden infant death. Brit Med J 1994; 308: 537-538.

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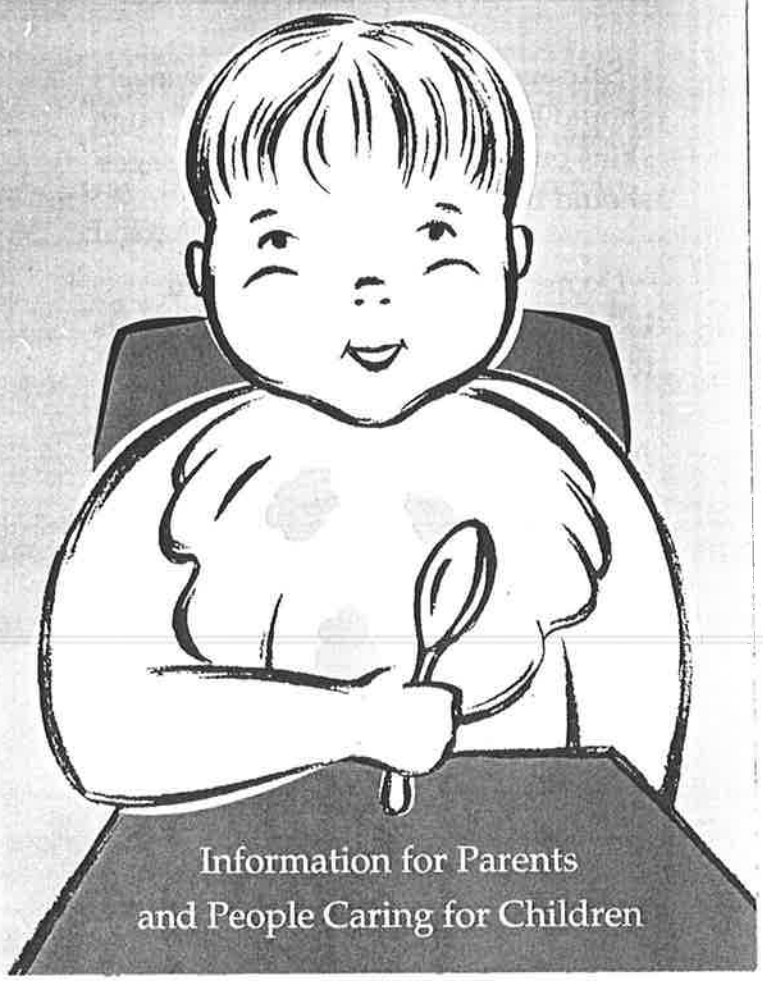
Safe eating for young children

PREVENT CHOKING ON FOOD



Information for Parents
and People Caring for Children

How to make eating safer for young children

Food

- Do not give foods that can break off into hard pieces.
- Avoid raw carrot, celery sticks and apple pieces, for example. These foods should be grated, cooked or mashed.
- Sausages, frankfurts and other meats should be cut into small pieces. Tough skins on frankfurts and other sausages should be removed.
- Do not give popcorn, nuts, hard lollies, cornchips or other similar foods to young children.

At eating times:

- Always stay with young children and supervise them while eating.
- Make sure that young children sit quietly while eating.
- Never force young children to eat, as this may cause them to choke.

What to do if a young child chokes on food

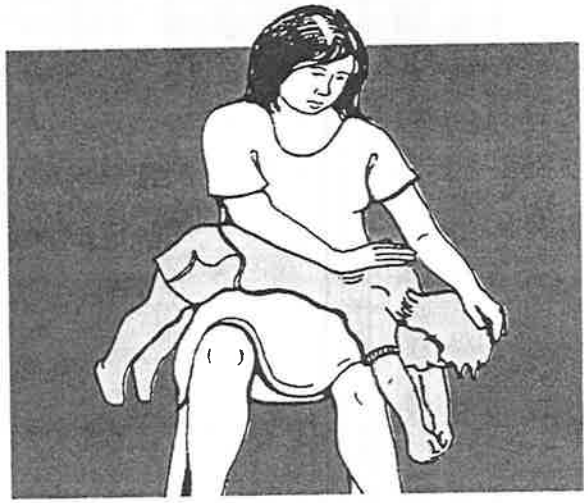
Check first if the child is still able to breathe, cough or cry.

If the child IS breathing, coughing or crying, the child may be able to dislodge the food by coughing:

- Do not try to dislodge the food by hitting the child on the back because this may move the food into a more dangerous position and make the child stop breathing.
- Stay with the child and watch to see if their breathing improves.
- If the child is not breathing easily within a few minutes, phone 000 for an ambulance.

If the child is NOT breathing

- Try to dislodge the piece of food by placing the child face down over your lap so that their head is lower than their chest.
- Give the child 4 sharp blows on the back just between the shoulder blades. This should provide enough force to dislodge the food.
- Check again for signs of breathing.
- **If the child is still not breathing, urgently call 000 and ask for an ambulance.** The ambulance service operator will be able to tell you what to do next.



Why young children are at risk of choking on food

- Young children do not have the back teeth needed to chew and grind lumps of food properly; these may not be fully developed until around 4 years of age. Young children are still learning to eat solid food.

- Food swallowed in large pieces is more likely to get stuck and block off the airways. If it goes "down the wrong way" this can cause young children to choke.

- If young children run, play, laugh or cry while eating they are more likely to choke on their food.

We strongly suggest that you do a First Aid course either with Australian Red Cross or St John Ambulance Australia (SA Inc) so you feel confident to deal with any situation where first aid care is needed.

Australian Red Cross
Phone: (08) 267 7624
Toll free: 1800 188071

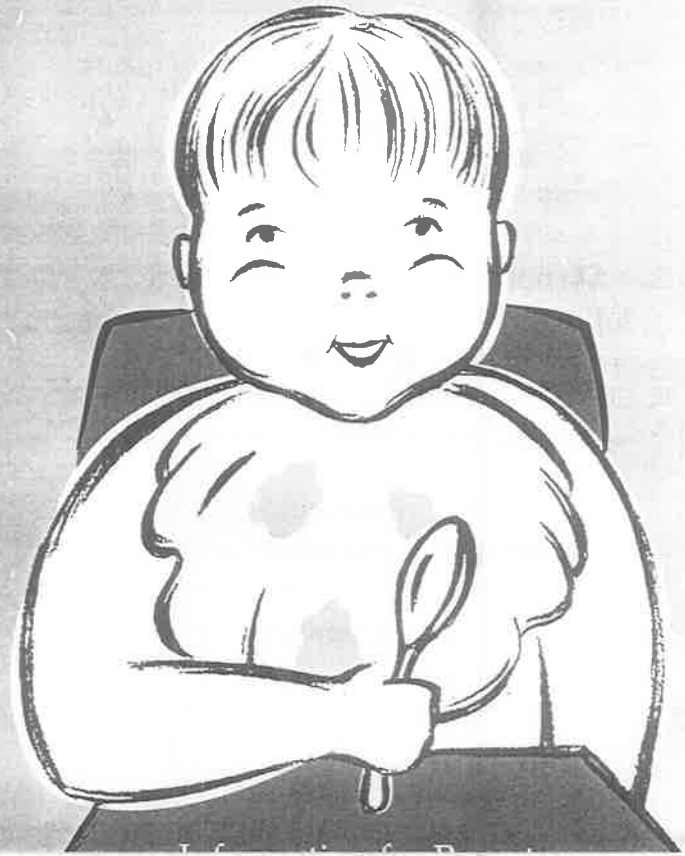
St. John Ambulance Australia (SA Inc)
Phone: (08) 274 0331

For more information contact your:
General Practitioner
Community Child Health Nurse
Nutritionist

Women's & Children's Hospital ADELAIDE
Child and Youth Health
Kidsafe
Foundation SA

Safe eating for young children

PREVENT CHOKING ON FOOD



Policy Guidelines - Safe Eating Practices for Young Children

These policy guidelines for Safe Eating Practices for Young Children have been developed for childcare centres, family day care, creches, kindergartens, child parent centres and hospitals.

Background

Statistics show that in recent years a consistent number of young children (0-5 years of age) have been admitted to hospital as a result of choking on food. While many other forms of injury to young children are declining, injury caused by choking on food has remained constant. The average length of hospital stay has increased, which may indicate that the children now presenting after choking on food are more severely affected.

During 1993/94 41 children in the 0 - 5 year age group were admitted to South Australian hospitals with "airway obstruction due to a foreign body in the larynx". In another 23 children, airway obstruction was identified as having resulted from inhalation of food.

These figures represent only a small percentage of young children who actually choke on food as many young children have choking incidents which do not require medical attention or admission to hospital.

Information from Injury Surveillance data for the Women's and Children's Hospital over the past 8 years indicates that the main types of food that children choke on are:

- raw carrot sticks, celery sticks and other raw vegetables
- raw apple pieces
- peanuts, walnuts, almonds
- chicken
- fish bones

A review of the literature revealed a number of other foods commonly associated with choking episodes including frankfurts, sausages, sweets, popcorn, grapes, seeds and corn chips.

While some children who choked on food were seated and supervised, other children were involved in various unsupervised activities at the time of choking, such as jumping on a trampoline, watching TV, and running.
Young children under 4 years of age are particularly at risk of choking on food because the back teeth which are used to chew and grind down lumps of food

into small pieces are not present, or are incompletely developed. As the incisor teeth develop first, young children can bite food, but they can't chew and grind the food properly. The food they swallow is in larger pieces and therefore more likely to obstruct their airway causing them to choke. This problem is compounded by their very small airways. It is therefore important that food such as carrot and apple is either cooked or grated. It is also important that young children sit quietly and are supervised when eating, and are never fed forcefully.

Many community nutrition programmes which have been implemented in recent years advocate the eating of foods such as raw vegetables, fresh fruit and nuts to improve the nutritional status and health of the general public. While there is no dispute that these foods are very healthy for people of all ages, young children require special food preparation and attention to their eating environment. These messages are not being conveyed to the community within current nutrition programmes. Also of concern are several widely distributed books and pamphlets which actively promote the feeding of carrot sticks and apples to toddlers.

These policy guidelines for Safe Eating Practices for young children have been developed for childcare centres, family day care, creches, child parent centres, kindergartens and hospitals as these are venues where a growing number of young children spend many hours a day. There is an added danger of choking in these situations because of the number of children needing supervision at meal times and the various ages of children requiring different types of meals.

Target Groups

These policy guidelines are designed specifically for organisations whose staff have young children in their care, or work with parents of young children in an advisory capacity.

These organisations include:

- Department of Education and Children's Services (DECS)
- Child and Youth Health
- Hospitals
- Community Health Centres
- Universities and TAFE
  - health professional training
  - childcare training programs
- Nanny Training Schools
- Gowrie Skill Centre

Policy Objectives

The objectives of the policy guidelines are as follows:

- **Reduction in the number of** :

  - children who choke on food
  - hospital admissions for choking during childhood
  - deaths from choking during childhood

- **Increased confidence in parents and childcare providers that they can**:

  - reduce the risk of choking on food
  - effectively manage a choking episode should it occur

Policy Guidelines

i) Make eating safer for young children

Food :

- Do not give foods that can break off into hard pieces.

- Avoid raw carrot and celery sticks and apple pieces, for example. These food should be grated or cooked.

- Sausages, frankfurts and other meats should be cut into small pieces. The tough skins on frankfurts and sausages should be removed.

- Do not give nuts, popcorn, hard lollies, cornchips or other similar foods to young children.

At eating times:

- Always stay with young children and supervise them while eating.
- Make sure that young children sit quietly while eating.
- Never force young children to eat, as this may cause them to choke.

ii) Know what to do if a young child chokes on foods.

Check first if the child is still able to breathe

<u>If the child IS breathing, coughing, or crying the child may be able to dislodge the food by coughing.</u>

- DO NOT try to dislodge the food by hitting the child on the back because this may move the food into a more dangerous position and make the child stop breathing.
- Stay with the child and watch to see if their breathing improves.
- If the child is not breathing easily within a few minutes, phone 000 for an ambulance.

<u>If the child is NOT breathing:</u>

- Try to dislodge the piece of food by placing the child face down over your lap so that their head is lower than their chest.
- Give the child 4 sharp blows on the back just between the shoulder blades. This should provide enough force to dislodge the food.
- Check for signs of breathing.
- **If the child is still not breathing, urgently call 000 and ask for an ambulance.** The ambulance service operator will be able to tell you what to do next.

* *Reference* : Policy Statement - The Management of Choking due to Suspected Impaction of Foreign Material in or just above the Windpipe. Australian Resuscitation Council. 1995

Recommendations

- This policy should be adopted by all organisations whose staff have young children in their care.
- The 'Safe Eating Practices for Young Children' guidelines should be readily accessible to all staff.
- All staff should be trained in 'Safe Eating Practices for Young Children' and in the management of choking.
- All staff working with young children should do a First Aid Course.

For Further information

Kidsafe
Community Child Health Nurse
Dietitian - Nutritionist

Policy Developed by :

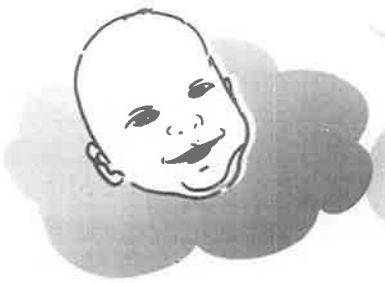
Women's and Children's Hospital
Child and Youth Health

October, 1995

Create a safe sleeping environment for your baby

Protect your baby with a safe sleeping environment

There are nine important steps you can take to ensure your baby is properly put into bed.



1 **A side or back sleeping position should be used for healthy babies.**

2 **There should be a good circulation of fresh air around your baby's face.**

3 **Make sure your baby cannot bury his or her face into any surface, including soft surfaces.**

4 **Do not use a pillow.**
You can raise the head of your baby's bed simply by placing some books under the legs of the cot.

Rather than use a pillow, raise the head of your baby's bed

5 **Use only light bedding to cover your baby and make sure it can't move over your baby's face.**

6 **Check the mattress to make sure it is firm and is well fitted within the cot.**

7 **When making up the cot, your baby's feet should be at the bottom of the cot.**
This might mean that the bedding will cover only the lower part of the cot, but it will ensure that your baby's head is free of bedclothes.

8 **Select an infant cot that meets the Australian Safety Standards.**

9 **Avoid allowing your baby to become too hot or cold.**
Generally, a room temperature of 16–20 degrees Celsius is comfortable for babies, and light bedding covers only should be used (see reference 1 on back page).
If your baby has a fever, use fewer bed coverings, or even none at all.

When making up the cot, your baby's feet should be at the bottom of the cot to ensure that his or her head remains free of bedclothes.

Developed by the:

- Menzies Centre for Population Health Research, University of Tasmania

in conjunction with:

- Department of Paediatrics and Child Health, University of Tasmania
- Family and Child Health Services, the Tasmanian Department of Community and Health Services
- Department of Paediatrics, Women's and Children's Hospital, University of Adelaide

Kmart

TASMANIAN INJURY SURVEILLANCE AND PREVENTION PROGRAM

For further information contact:
THE MENZIES CENTRE FOR POPULATION HEALTH RESEARCH
17 Liverpool Street, Hobart TAS 7000
Phone: (002) 357 700

Avoiding dangerous situations

As well as doing the right things to protect your baby when he or she is asleep, there also are a number of dangerous situations to look out for. These include:

- **Projections in the cot such as knobs and nails.** These can catch onto and cause your baby to be hanged and suffocate (see reference 2 on back page).

- **Other objects in the cot.**
These include anything which could choke or scald your baby, or assist your baby to climb the cot.

- **Ill-fitting mattresses and defective cots.**

- **The incorrect placement of the cot in a room.** The cot should be placed away from windows, cords, heaters, power points and lights. This will prevent falls, choking, burns and electrocutions.

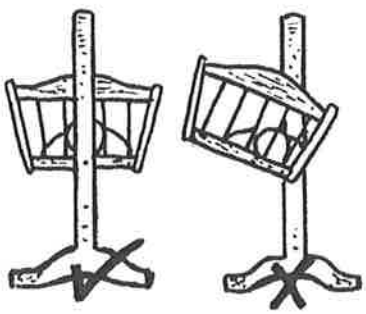
- **Rocking cradles with a tilt of more than 5 degrees.**
A tilt of more than 5 degrees can create the potential for your baby to become trapped in the angle between the wall and floor of the cot. Make sure the cradle cannot tilt while your baby sleeps.

- **In certain circumstances, bed-sharing with a parent or with a brother or sister (co-sleeping).**
This may be unsafe if your baby:
  - slips down under the bedding or into pillows;
  - becomes trapped between the head of the bed and the wall;
  - falls out of bed;
  - gets too hot with too much bedding; or
  - is overlaid by a person who has impaired consciousness (eg a person who has taken drugs, alcohol or has epilepsy).

  Bed-sharing has been found to be a risk factor for SIDS among infants of mothers who smoke. (see reference 3 on back page).

  Until further information is available on the role of co-sleeping in infant deaths, alternatives such as placing the cot next to the bed may be the safest option (see reference 4 on back page).

- **Restraining harnesses on car seats, strollers or bouncinettes.**
If your baby is left unsupervised and slips, these harnesses can cause him or her to be hanged (see reference 2 on back page).



Make sure the rocking cradle cannot tilt while your baby sleeps.

- **A "face down" sleeping position must be avoided for your baby.**

- **Bedding which covers baby's face.**
  Your baby's face should not be covered or blocked by bedding.

- **Adult waterbeds.**
  If a baby is placed in an adult waterbed, he or she may:
  - change position easily;
  - sink their head into the mattress; or
  - get too hot on heated waterbeds.

  The American Academy of Pediatrics has endorsed a warning that waterbeds are unsafe for infants (see reference 5 on back page).

- **An unsupervised infant in an adult bed.**
  In this situation, your baby may:
  - roll off;
  - become wedged between the bed and a wall;
  - be submerged under adult bedding; or
  - get too hot.

- **Plastic coverings near your baby's face – these may cause suffocation.**

- **Situations which encourage the build-up of stale, exhaled air around your baby's face.**
  Gas-trapping environments should be avoided (see reference 6 on back page). Examples of these include bumpers, pillows or large soft toys which can trap exhaled air. Doonas or thick quilts should not be used for infants under one year (see reference 1 on back page).

REFERENCES

1) Report of the Chief Medical Officer's Expert Group on the Sleeping Position of Infants and Cot Death. Department of Health. Her Majesty's Stationery Office London 1993.

2) Byard RW, Beal S, Bourne AJ. Potentially dangerous sleeping environments and accidental asphyxia in infancy and early childhood. Archives of Disease in Childhood 1994; 71:497-500.

3) Scragg R, Mitchell EA, Taylor BJ et al. Bed sharing, smoking and alcohol in the sudden infant death syndrome. British Medical Journal 1993; 307:1312-8.

4) Byard RW. Is co-sleeping in infancy a desirable or dangerous practice? Journal of Paediatrics and Child Health 1994; 30:198-199.

5) Gilbert-Barness E, Barness LA. Contemporary Pediatrics 1992; 9:13-28.

6) Infant Sleep Position and Sudden Infant Death Syndrome (SIDS) in the United States: Joint Commentary from the American Academy of Pediatrics and Selected Agencies of the Federal Government. Pediatrics 1994; 93:820.

Information for Parents and Caregivers

SAFE SLEEPING FOR THE UNDER 2's

Why are babies at risk?

While sleeping, babies may get into dangerous situations. They may be:

- suffocated under bedding.
- choked on toys or other objects.
- caught between the cot side and mattress.
- strangled on cords and ribbons.

The reasons for this are that they:

- are not able to control their own sleeping situations.
- cannot understand danger.
- may not be able to move out of a dangerous situation.
- may place things in their mouths or around their necks and choke.

There are positive steps that parents and caregivers can take to make sleeping situations safer for babies.

STEPS TO TAKE TO REDUCE THE RISKS

What sleeping position is safest?

- Place your baby on his/her back to sleep.
- When putting your baby to sleep, place his/her feet at the end of the cot. Make up the cot so that your baby's head is not able to slide under the bed clothes, nor can his/her head get trapped against the head of the cot.

What safety factors should I consider?

Cot

- The cot should be strong and safe.
- Look for the Australian Standards label.

- Ensure bars in cots meet safety standards ie. they are between 5 cm and 8.5 cm apart and are strong and do not bend.
- Ensure the cot does not have corner posts, screws or knobs which your baby's clothes can get caught on and cause hanging.
- Make sure that there are no gaps where your baby could fall through, or where his/her head could get caught.
- Ensure catches on the cot-side can't be undone by little fingers.
- Ensure your baby's cot does not have any sharp edges or corners, or holes he/she can poke his/her fingers into.
- Do not use a cot which is broken.
- Make sure that all bolts and screws are tight.
- Ensure cots have high sides ie. from base of mattress to top of cot side needs to be at least 60 cm. This is important because as babies grow and can pull themselves to a standing position in the cot, they are at risk of falling over the side.
- If your baby's cot has been painted, ensure the paint is lead free. Leaded paint is toxic. When babies are teething they may chew the cot bars and swallow the leaded paint. If you can't be sure if the paint is leaded or not, strip and repaint the cot with a lead free paint. For further information check with your paint shop.

Mattress

- Make sure the mattress is firm and well fitting and there is no more than a 2.5cm gap between the mattress and the cot sides and ends.
- A thick mattress placed into a portable mesh-sided cot is not safe. Only use the mattress provided with the cot. The baby can stretch the side of the mesh cot and get caught in the gap between the mattress and cot side.
- Remove loose plastic mattress coverings. Mattress protectors need to be strong and fit the mattress firmly.

Strollers and bouncinettes

- If your baby is sleeping in a stroller, make sure your baby can't slide out of it and get caught and strangle.
- Make sure the stroller has a stable footrest which can't collapse.
- If harnesses are used in strollers or bouncinettes make sure that they are fitted firmly and that your baby can't get caught and strangle.

Rocking cradle

- Make sure the rocking cradle cannot tilt while your baby sleeps.
- Ensure the locking pin/bolt is securely in place when you leave your baby.

Beds

- Do not let your baby sleep on a water bed or bean bag.
- Children over the age of 2 years are generally old enough to sleep on a mattress on the floor, or in a regular bed with guard rails.
- Bunk beds are not safe for children under the age of 9 years.

What should I take into account when placing the cot in a room?

- Place the cot away from curtain cords and other cords/ropes in which your baby could become entangled.
- Have mobiles well out of reach to avoid your baby becoming entangled.
- Your baby's cot should be well away from heaters, power points, electrical appliances and lights, as babies like to play with anything within their reach.

What bedding should I use for my baby?

- Keep your baby's head uncovered. Make up the cot so that your baby's head is not able to slide under the bed clothes.

- Cot bumpers are not needed to protect your baby. Babies have been found with their faces pressed against bumpers. This can cause breathing problems.
- Use only light bedding. Doonas, thick quilts or heavy blankets may cause your baby to over heat.
- If your baby needs to be wrapped for settling, use a sheet over light clothing to avoid overheating.
- It is best not to use pillows in a cot. Babies do not need pillows to sleep comfortably.
- It is not safe to use a hot water bottle or electric blanket, due to the risk of overheating and burns.
- It is best to remove toys from the cot when your baby is sleeping.

What should I consider when dressing my baby for sleep?

- Always keep your baby's head uncovered when sleeping. Do not cover your baby's head with hats, hoods or rugs when sleeping.
- To avoid your baby being strangled, ensure that clothing does not have any long drawstrings, ribbons or cords.
- If you use cord or ribbon to attach a dummy to your baby's clothing, make sure it is no longer than 10 cm.

Is it safe for my baby to sleep with cuddly toys and pets?

- No. Large toys and pets can smother

esigned by Foundation Studios. January 1997

Is it safe for my baby to sleep with others?

Babies may suffocate if they sleep in the same bed with an adult or child. If you want your baby to sleep near you, place him/her in a separate bassinette or cot near to your bed, not in your bed.

Do not smoke in the same room as your baby, as the smoke can be harmful.

Where can I get more information?

Health Information Centre, Women's and Children's Hospital (08) 8204 6875

Kidsafe Centre (08) 8204 6318

SIDS Association (08) 8363 1963 or 1800 656 566

Parent Help Line 1300 364 100

Your local Child Health Centre (Child and Youth Health)

We strongly suggest that you do a First Aid course either with Australian Red Cross or St John Ambulance Australia so you feel confident to deal with any situation where first aid care is needed.

Sponsored by the Women's and Children's Hospital Shops Auxiliary

Women's and Children's Hospital ADELAIDE

Child and Youth Health

SIDS

Kidsafe

A Buyer's Guide To Cot Safety

A Checklist for Parents

When buying a cot for your baby it is important to consider safety above all other factors. The following is a checklist of cot safety factors you should consider to reduce the risk of your baby being injured or dying.

☐ **1. Does the cot meet the Australian Standard (AS 2172)?**
If you are buying a new cot, look for the Australian Standard logo, or ask the retailer if the cot meets the Australian Standard.

☐ **2. Is the cot in good repair?**
Broken cots can be dangerous. Make sure there are no bent or broken parts which would allow the drop side to move away from the cot, or the base to collapse.

☐ **3. Are the spaces between the vertical bars between 5cm and 8.5cm wide, and are the bars unbendable?**
This is to prevent your baby's head or neck becoming stuck between bars (see over-top of page).

☐ **4. Is the distance from the top of the mattress to the top of the cot frame at least 60cm?**
This is to reduce the risk of your baby falling over the top of the cot (use this ruler to measure).

☐ **5. Are the edges of the cot smooth and rounded?**
This is to prevent injury from splinters of wood/metal or from your baby hitting a sharp corner.

☐ **6. Is the cot free from knobs or things sticking out eg. screws, wingnuts or bolts?**
This is to prevent clothes becoming caught and your baby being hung, or hitting a protrusion and being injured.

☐ **7. Is the frame sturdy?** (It should not collapse when being moved and the bars must not bend)
This is to prevent your baby becoming trapped between bars or in a collapsed cot.

☐ **8. Is the base strong?** (It shouldn't sag or collapse)
This is to prevent your baby becoming trapped.

☐ **9. Is the inside free from small openings or holes?**
This is to prevent your baby's fingers becoming trapped.

☐ **10. Is the inside of the cot free from footholds eg. cross bars, horizontal bars or decorative features?**
This is to prevent your baby being able to climb up and fall out of the cot.

PTO

SAFE too wide

Use this scale to measure the gaps between vertical bars

☐ **11. Does the cot have brakes on the wheels or castors?**
To prevent the cot from moving, if four legs are fitted with wheels or castors, at least two must have brakes. If not remove two of the wheels, or castors.

☐ **12. Are the catches on the dropside child-resistant?**
This is to prevent your baby opening the catches and being injured.

☐ **13. Is the cot painted with lead free paint?**
Lead paint can be toxic. When babies are teething they may chew the cot bars and swallow the lead paint. If you have a second hand cot and can't be sure if the paint contains lead, strip the cot and repaint it with a lead free paint. For further information check with your paint shop.

☐ **14. Is the mattress the right size for the cot?**
To prevent your baby becoming trapped between the mattress and the cot frame, make sure the mattress fits snugly. There should be no more than a 2.5cm space between the mattress and the cot frame.

☐ **15. Is the mattress firm and covered with a strong cover?**
It is important to remove any loose-fitting plastic coverings to prevent suffocation. Mattress protectors need to be strong and fit the mattress firmly.

☐ **16. Are the manufacturer's instructions included?**
Make sure that you strictly follow the instructions when putting the cot together.

Be wary of second hand and older style cots as they might not meet current safety standards. They may be fashionable, but they may be a death trap for your baby.

For further information contact:

Kidsafe (SA) (08) 8204 6318

Health Information Centre,
Women's and Children's Hospital, Adelaide (08) 8204 6875

Child and Youth Health Parent Help Line 1300 364 100

Child and Youth Health Centre,
for location of your nearest centre phone (08) 8303 1500

Project Safety Section,
Office of Consumer and Business Affairs (08) 8204 9751

Women's and Children's Hospital ADELAIDE

SIDS

Child and Youth Health

Kidsafe

Sponsored by: Office of Consumer and Business Affairs

Women's and Children's Hospital

Policy

Safe Sleeping Environments for Children under 2 years of age who sleep in cots in the Hospital

1. Aim

To ensure that staff are effective role models in providing a safe sleeping environment for children under 2 years of age who sleep in cots in the hospital.

2. Background

Children under 2 years of age are particularly susceptible to environmental hazards, partly because of their lack of understanding of potentially dangerous situations, and also because of their inability to extricate themselves from harmful locations due to poorly developed motor skills and limited muscle strength. The sleeping environment is an example of a potentially hazardous situation which may result in accidental death from asphyxia or injury in children under 2 years of age.

While sleeping, babies may get into dangerous situations. They may:

- suffocate under bedding.
- choke on toys or other objects.
- get caught between the cot side and mattress.
- strangle on cords and ribbons.

A significant number of injuries and deaths are due to:

- wedging/entrapment between the mattress and cot frame;
- heads becoming caught between cot rails;
- strangulation/hanging from cords, ribbons, necklaces and clothing;
- and suffocation under bedclothes (1-3) .

Babies are at risk of injury from falls and from faulty cots. Falls from cots become more frequent as babies gain mobility, starting at 7 to 8 months. The frequency peaks between the ages of 12 and 18 months and then reduces significantly as children move out of cots into beds (1). The most common faults which cause serious injuries and deaths relate to instability and collapse of the side or bottom of cots due to poor design. There also have been faults with rocking cradle locking pins causing babies to die from positional asphyxia. Projections on the inside of cots may ensnare clothing causing hanging, as may restraining harnesses. There has been alarming evidence from testing a variety of cots against the Australian and New Zealand standard for regular cots (AS/NZ 2172:1995) that many manufacturers do not design their cots to meets these standards (4). There are now moves to make these standards mandatory.

Finally, external pressure by adults in shared sleeping situations or while breast feeding may result in fatal chest compression or airway occlusion (5,6). There also are concerns that shared sleeping may result in overheating of the baby.

Health professionals play an important role in promoting safe sleeping practices to parents and caregivers, not only in the provision of accurate and consistent information, but in modelling safe sleeping practices whilst caring for children under 2 years in a hospital setting.

The Hospital needs to provide safe sleeping environments for young children. Cots purchased should meet the Australian Standard 2130 - 1981 and mattresses used should fit securely with no more than a 2.5 cm gap between the mattress and cot frame.

3. Outcomes

3.1 Staff will be aware of and provide a safe sleeping environment for all children under 2 years of age using a cot, whilst in hospital.

3.2 Staff will promote and encourage the application of safe sleeping principles to parents and caregivers of children under 2 years of age who use a cot.

4. Policy Application

4.1 Cots

All cots used in the hospital must meet Australian Standards (AS 2130-1981).

Ensure all cots are in a good state of repair.

No mesh sided cots are to be used.

4.2 Mattresses

Mattresses must be well fitting with no more than 2.5cm gap between the mattress and the cot frame.

Mattresses need to be firm, and on a firm cot base, as mattresses which dip and sink can interfere with a baby's breathing.

Plastic mattress covers must be intact and fit the mattress securely.

Mattress protectors need to be strong and fit the mattress firmly.

Ensure mattresses are in a good state of repair.

4.3 Bedding

Pillows should not be used in cots to support a baby's head unless under medical advice. The head of the cot may be elevated by the use of blocks/cot lifter or a pillow under the mattress.

Cot bumpers are not to be used as babies have been found with their faces pressed up against bumpers. This can cause breathing problems.

Electric blankets, hot water bottles or wheat bags are not to be used to heat babies because of the risk of burns and overheating. Electric blankets may be used under close clinical monitoring (Theatres and Intensive Care Unit) when thermoregulation is a problem.

Use only light bedding to avoid overheating. Doonas, thick quilts or heavy blankets should not be used.

If a baby requires wrapping for settling, use a light sheet/cloth over minimal clothing and make up the cot with light bedding. Ensure the head is kept uncovered to avoid overheating (unless thermoregulation is a problem).

4.4 Clothing

Ensure that the cord/ribbon/chain attaching a dummy to clothing is no longer than 10 cm.

Do not use clothing with cords/ribbons/drawstrings around the neck which may become caught and cause strangulation.

4.5 Positioning

Place babies to sleep on their back unless clinically contraindicated.

Place babies to sleep in cots with their feet at the foot of the cot (unless clinically contraindicated eg. babies with reflux) and make up the cot to facilitate this position. This way babies cannot slide under the bedclothes, nor can their heads get trapped against the head of the cot.

4.6 Co-Sleeping

Co-sleeping should be discouraged because of the risk of suffocation, overheating and falls from beds.

5. Implementation

It is recognised that to ensure this policy is known to all clinical staff it will require general awareness raising through the following means:

- distribution of policy to all wards and departments.
- distribution of parent/caregivers information "Safe Sleeping in the under 2's" to all staff.
- discussion at Divisional meetings with department/unit heads.
- discussion at ward/unit meetings and display of information in the ward/unit communication book and new notices board.
- inclusion in the WCH Policy and Procedures Manual.
- specific inservice training on the principles of safe sleeping in the under 2 years olds for clinical staff.

6. References

1. Moller J. (1995) Injuries Associated with Cots among Children from Birth to 35 months. National Injury Surveillance Unit. (unpublished)
2. Byard R. (1996) Hazardous infant and early childhood sleeping environments and death scene examination. Journal Clinical Forensic Medicine. 3:115-122.
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6. Byard R, Burnell R. (1995) Apparent life threatening events and infant holding practices. Archives of Disease in Childhood. 73: 502-504.

January, 1997

draft

Discussion Document

Child Safety on Farms

A guide for child safety in the farming industry

WorkCover Corporation
South Australia

December 1996

CONTENTS

1. DANGERS ON A FARM

Farms are a unique environment in which to raise children. They are a workplace, a place to live and a place to indulge in leisure activities. Children can lead a healthy lifestyle while developing skills for later life, however, dangers for children on farms are numerous.

This guide is designed as a practical help for busy farmers and other agricultural workers who need to reduce the everyday dangers that their own children, and their children's friends face about the farm.

Childrens' mental and physical development plays a large part in child injury on the farm. Children who may appear physically mature enough to carry out a task often lack the necessary co-ordination, judgment and understanding of the dangers to complete the task or activity safely. An understanding of child development is essential in identifying the dangers for children on farms and reducing the risks of those dangers. Physical size, the way children behave as well as exposure to a danger can combine to make the farm a very dangerous place.

Some of the child development issues that need to be considered include:

- Size of the child in relation to the environment (can squeeze through small openings);
- Small body mass/weight affects the ability to control animals, motor bikes, machinery (limited strength and reach)
- Poorly developed sense of danger which involves risk taking behaviour (jumping off sheds, climbing ladders etc.);
- Increased independence, mobility and curiosity (want to see what is on the other side of the fence, door or what's in the shed);
- Peer pressure from the older children (will take on a dare very quickly);

Risks of every type exist for children on farms, however, the methods of injury are generally physical, (slips - trips - falls) mechanical (tractors and other machinery) and chemical (poisons - fertilizers - veterinary products).

2. MAJOR SOURCES OF INJURY

2.1 Machinery

- If children are allowed to play in the vicinity of machinery, they are at increased risk of being run over or caught in moving parts.
- If children are carried on machinery, they are at a very great risk of falling and being run over. This is particularly so if the terrain is rough or undulating.

- Inadequate guarding of machinery increases the risk of child death or injury from entanglement.

2.2 Motorcycles

- The relationship between the height and weight of the child and that of the agbike or ATV is important, particularly when children cannot comfortably reach the controls and the ground; injury is more likely due to loss of control.

- The weight of the machine can make it difficult to manoeuvre if it is not matched by the correct weight of the child.

- Four-wheeled motorcycles or All Terrain Vehicles (ATV) require more co-ordination than most children have.

- Inadequate training in riding techniques and an inadequate awareness of the hazards associated with motorcycle riding increases the level of risk of injury or death.

- Riding of motorcycles without a helmet dramatically increases the risk of head injury.

2.3 Animals

- Toddlers are at risk of being kicked or trampled by cattle, horses, pigs and even smaller animals such as sheep or goats.

- Older children are at increased risk of falling from horses.

- All children are at greater risk of injury if they have access to animals with young or enclosed animals.

2.4 Chemicals and Poisons

- Where chemicals are not stored safely, children are at greater risk of poisoning because of increased access.

- Toddlers are particularly at risk because they are curious and readily put things in their mouths.

- Because of their low body weight, poison and chemicals will have more effect on children than adults.

2.5 Firearms

- The presence of firearms on the farm increases the risk of death or injury for children. If firearms are not stored safely children of all ages are at great risk due to their curiosity and lack of awareness of the potential danger.

- Where children are allowed to carry firearms or participate in shooting animals, the risk of death or injury is very high.

An area that can be easily overlooked are the water hazards, these include farm dams, creeks, rivers, storage tanks and water troughs.

3. IDENTIFYING THE DANGERS AND HAZARDS

It is the responsibility of the adults on farms to recognise the potential dangers to the children around them, and the precautions that need to be taken to avoid the risk of death, injury and disease of those children.

Farm machinery, animals, chemicals, storage facilities and unsafe work environments all have the potential to expose young people to unnecessary risks to their health and safety. The key element to recognising potential dangers is **to identify the hazards associated with that danger**. An **assessment of the risks associated with those hazards** must be undertaken to gauge the severity of those risks.

S.A.F.E. - BE SAFE: SEE IT, ASSESS IT, FIX IT EARLY.

To do the job properly, you really have to go over familiar ground to look at your farm and your work areas.

A successful safety check of the workplace needs to consider the:

(a) nature of the work being performed (eg sowing, hay cutting, stock handling etc);

(b) types of machinery being used (eg tractors, slashers, motor bikes etc);

(c) types of animals present (eg horses, cattle, pigs etc);

(d) what chemicals are being used and how they are stored (eg fertilisers, herbicides, fuels, oils, solvents, veterinarian products etc);

(e) means of access to, exit from and security of the work areas (eg doorways, ladders, walkways, gates etc);

(f) types of storage facilities and access to them (eg dams, fuel tanks, silos etc);

(g) condition of paths, walkways, stairs, animal enclosures, gates, fences and doors;

(h) presence of septic tanks or pits, silos, field bins, disused mine shafts or wells, bores and irrigation channels etc.

A checklist can help to identify the hazards in your workplace. The industry has developed checklists specifically for each commodity and these are referred to in the accredited training programs mentioned later in this discussion document.

Like other lists in this guide, the **Hazard Identification Checklist** is not exhaustive but it is a starting point for the questions you need to ask yourself.

4. ASSESSING THE DANGER

Risk assessment is the next step in the process. The aim is to evaluate dangerous situations involving children and assess the level of risk so that appropriate prevention measures can be put in place.

When conducting an assessment, **it should be remembered that children are generally smaller and weaker than adults, they are more curious and have less awareness of dangerous situations.** Consequently, what may appear to be safe from an adult's perspective may be extremely dangerous to children.

The health and safety of children is at risk not only where they help in the farming activities but also simply because they may have access to the farm workplaces.

The assessment should apply to all children, whether involved in farming operations or at play. Also considered should be children who are likely to be visiting, crossing the property, walking on roadways or tracks that cross the property or even trespassing.

The assessment of danger should include:

(a) evaluating the likelihood, extent and type of exposure to the risks that may be associated with the danger; (eg. A child entering a chemical shed and drinking from a soft drink bottle containing a poison like brake fluid or a pesticide etc.);

(b) where necessary, obtaining information about the effects of those risks on a child's health and safety (eg Read the label on the original container or the material safety data sheet (MSDS) or contact the local health authority etc);

(c) evaluating the potential risk to a child's health and safety (eg very likely if poison is not stored as directed by the manufacturer etc);

Hazard assessment should be reviewed regularly, particularly following the introduction of new machinery and equipment, chemicals or work practices.

SEE IT, ASSESS IT, FIX IT, EARLY.

5. WHO IS MOST AT RISK

5.1 Toddlers or 0 - 4 year age group

- Toddlers are at great risk drowning in dams, creeks, rivers and channels. This is due mainly to their increased mobility, curiosity, lack of fear and their typical inability to swim.

- This age group is also at risk of being run over by machinery or vehicles. This generally occurs either because they are playing on or around machinery or fall off while riding as a passenger.

- Children in this age group are at greater risk of swallowing hazardous substances due to their curiosity. It is generally recognised that children under five years learn through curiosity, touching and putting things in their mouths.

- Toddlers who are inadequately supervised or not separated from the dangers are at greatest risk of injury or death.

5.2 Children 5 - 14 years

- Children in this age group are generally beginning to recognise dangers but continue to act before they think. They have an increased risk of being kicked or crushed by livestock and being entangled in or falling from machinery. This age group is easily influenced by older children and believe they will not suffer injury.

- Motor bikes and all terrain vehicles (ATV) are recognised as a risk for these children. They are at an age where they enjoy the freedom and power of these vehicles but may not be large enough, strong enough or co-ordinated enough to adequately control them. Riding a motor bike, ATV or even a push bike without a suitably approved helmet, places them at a greater risk from injury or death.

- This age group is at risk from being smothered or becoming trapped when climbing into silos or other fodder storage areas such as hay stacks or field bins etc.

- Children between the ages of 10 and 14 years are at greater risk of injury or death as they are expected to undertake or are just becoming involved in farm related tasks such as driving tractors and other machinery or assisting in stock yards. **Generally these children have little experience in these tasks and will attempt to copy the example of an adult.**

6. RISK CONTROL

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Risks are controlled by taking the necessary steps to minimise the chance that a child will be harmed from a hazard. The following methods should be used:

- get rid of the hazard permanently;
- try a different option or product;
- place the hazard behind a barrier or mesh screen;
- administration (changing work practices);
- personal protection.

(Examples of these controls are given on the next page)

These have been placed in priority order - from the most preferred (providing a safe workplace) to the least preferred (providing some measure of safety for the person). Hazard control in your workplace should be based on this order of priority. Where it is not possible to eliminate the hazard altogether, the risk needs to be controlled and or minimised at the highest possible level (eg substitute instead of isolate or isolate instead of personal protective equipment etc).

Not all control methods require physical barriers or equipment which involve large costs. Training, education and supervision are also important.

In establishing controls, you should:

(a) select appropriate measures to control the hazard;

(b) ensure safety control methods are properly used and maintained;

(c) ensure appropriate information, instruction and supervision is given to children working in the workplace;

(d) ensure children in the workplace have received adequate information about the hazards and dangers present;

(e) **set a good example. If you work safely so will the children.**

Children should never be permitted to undertake any farm operation or activity unless the hazards and risks of those tasks have been identified, assessed and controlled.

Adults must ensure the children have received proper instruction, have understood those instructions and have the ability to undertake the tasks involved. They also need to be provided with an appropriate level of direct supervision when undertaking the tasks.

The value of instruction should not be underestimated, many children will not appreciate the significance of the dangers to their personal safety unless they have had detailed information regarding the dangers present.

7. CONTROL MEASURES

7.1 Elimination (To get rid of the hazard permanently)

- Elimination of all hazards that may be encountered on farms is not a likely option. However, there are some elimination options that should be considered, breeching the wall or the filling of a unused dams or the removal of water troughs that no longer serve a useful function, could be an option for the elimination of a potential drowning hazard that toddlers can encounter.

7.2 Substitution (Try a different option or product)

- Substitution to control the hazards for a toddler may involve the use of small water troughs for watering of the stock rather than a dam. In the 5 - 14 year age group, a child may be given a more suitable ATV or motor bike to ride which is smaller and more easily controlled by the child.

7.3 Engineering controls (Place the hazard behind a barrier or mesh screen)

- Where possible, mechanical control measures should be designed so that they operate without the presence of adults and are, as far as practicable, childproof. Safety features on machinery are designed to protect adults and may not provide adequate protection for children. Protective mesh or barriers must prevent the access of a person or limb coming into contact with the hazard, these physical barriers must be small enough to prevent children from injuring themselves.

- Silos and grain bins should only be accessible from fixed ladders that are not within the reach of children, if this is not possible, then the access should be fitted with a lockable barrier to prevent access. Any portable ladders must be stored safely to restrict the child's access to heights.

7.4 Administrative controls (Rules, out of bounds areas and adult supervision)

- There should be strict and enforced rules in place on what children are allowed to do and where they are allowed to play or go on the farm. **Adults must practise safety around the farm environment, thereby setting a good example for the children.** Children look at adults as a role model, this plays a very large part in children learning the right way to complete a task. Adults need to consider and anticipate lapses in concentration by children, even the best child may not always be perfect. Where children could be exposed to identified hazards they should have specific instructions, the extent of these instructions will depend on the complexity and risks involved and the child's age and experience. **Children should not be allowed in or near hazardous areas, operations or activities and be kept clear, or at a safe distance, when their presence is unnecessary.**

8. EXAMPLE OF CHILDSAFE FARMING

Farms and agricultural workplaces **need to be made safe - and kept that way.** Farm injury can happen at any time, in the most unexpected ways. Fortunately a large percentage of injuries are preventable in advance.

Over the next few pages we discuss some of the areas where your workplace/farm could be particularly vulnerable to accidents, and introduce childsafe farming ideas which will go a long way towards ensuring the protection of your children, your neighbours children and their friends.

8.1 Danger Areas

Many accidents involve children being run over or crushed by vehicles. Drivers need to be aware that a child could unexpectedly be in a position of danger, and should follow systems of work designed to safeguard children.

Those in charge of the farm should instruct their children (if they are conducting any of the following activities), employees and visitors in the following precautions:

(a) Before getting in the vehicle, the driver should check that a child, who may not be visible from the driving position, is not near the wheels or liable to be crushed against a fixed object such as a building.

(b) Drivers should ensure that their vehicles are driven at a safe speed. Speed restrictions should be obeyed. Drivers should be able to pull up within the distance which they can see to be clear. This is particularly important when approaching blind corners.

(c) When parking, the parking brake should be applied firmly, any hydraulic equipment lowered to the ground, the engine stopped, the power-take-off (PTO) lever put into neutral and the starter key removed or a kill switch activated.

(d) Any machinery or equipment specifically designed for work purposes (eg cherry pickers, and tree planting equipment, front end loaders) must not be used to transport passengers unless properly designed restraints are available.

Access to work areas is another major cause of injuries and fatalities to children. So far as is reasonably practicable, all doors, fences, guards, covers and other protective equipment preventing access to hazardous areas, operations, plant or substances should be locked and secured to prevent access by children.

A child must **NOT** be permitted to enter places where suffocation or death may occur. Measures must be undertaken to prevent access to:

(a) A fruit, grain or other store rooms likely to contain low levels of oxygen, poisonous gases, explosive gases or cold atmosphere.

(b) A silo or storage bin where a risk exists of becoming trapped and suffocated from grain.

(c) Any other place where low levels of oxygen, poisonous gases, explosive gases or cold atmosphere may exist or develop (eg septic pits, above or underground tanks, old cellars etc or areas that have very poor ventilation).

Children should be prevented from playing in such areas and checks made to make sure that they are not there.

Where dangerous and or hazardous work is done in the open, children should be kept at a safe distance. Some examples of dangerous or hazardous work include:

- rotary mowing;
- forage harvesting;
- rotary cultivating;
- grain handling;
- pruning, cutting and trimming;
- manure spreading;
- winching, towing and tree pulling.

Very dangerous types of work include:

- chain or circular saw operating;
- tree felling;
- blasting operations;
- post-hole digging.

The following machinery or equipment should be considered as potentially fatal to children:

(a) tractors (falls from or trapped under); and unguarded power take-off equipment;

(b) towed or self-propelled harvesters and processing machines (run over or trapped under);

(c) trailers, tankers or feeding equipment with built-in conveying, loading, unloading or spreading mechanisms (clothing trapped in mechanism);

(d) power driven machines incorporating any built-in cutting, splitting, grinding or crushing mechanism (run over or trapped in);

(e) chemical applicators (poisoning);

(f) rotary hoes and post-hole diggers, and machines incorporating power-operated soil engaging parts (auger entangling clothing); unguarded rotating parts;

(g) ditch digging and drainage equipment (clothing trapped in mechanism, struck by backhoe boom etc);

(h) harvesters and balers (run over or trapped in);

(i) front-end loaders, fork-lift trucks/tractors, and similar machines (run over);

(j) grain conveying equipment (becoming entangled or crushed in mechanism);

(k) powered feed preparation equipment (auger or PTO entangling clothing);

(l) tillage equipment (run over or trapped under).

8.2 Visiting Children

Children at risk are not only those who live or work on a farm. Owners should take precautions against the possibility of children straying into potentially dangerous areas and situations.

Physical barriers and/or warning notices should be provided where appropriate and practicable.

When school parties visit farms, it is important that farmers and teachers discuss beforehand the arrangements for the visit to ensure that the children are not put at risk.

Where public visiting activities are offered - for example, "bed-and-breakfast on the farm" or "pick-your-own" invitations - appropriate steps and supervision should be adopted to prevent injuries to children.

8.3 Visiting Workers' Responsibilities

Visiting workers such as building or other contractors, or delivery drivers, should be made aware (for example, by use of signage) of the need for particular care as they may not be used to the presence of children in close proximity to work areas.

8.4 Safe Keeping

Everyone involved in farming activities needs to keep their eyes open to ensure that risks to children are minimised.

Everyday housekeeping measures such as:

(a) Fences, gates and walls should be kept in good repair;

(b) Ladders should be stored in a safe place when not in use, for example, hung securely from a wall **out of reach of children**;

(c) Heavy items, such as wheels detached from tractors, should be secured or laid flat to eliminate the risk of them toppling onto a child;

(d) Keys should be removed from machinery when not in use or a kill switch activated;

(e) Sheep dips should be emptied and secured by covering them when not in use;

(f) Empty cans or drums previously containing agricultural or veterinary chemicals should be disposed of in accordance with the manufacturer's instructions or industry code;

(g) Veterinary products should be stored in a secured area.

Doors, fences, guards, covers and other protective devices for high risk areas should be kept locked or in a position to deny access by children. This is particularly important when the work area is unattended. Portable ladders should be removed and fixed ladders made inaccessible to children.

Where children have access to work areas, safe means of entry and exit should be provided. In particular, all stairways should be safely constructed and maintained, wells, bore holes and any grain or slurry pit should be adequately guarded with child proof mesh when unattended.

Access to water hazards, such as tanks, ponds, streams and dams, irrigation channels and drains, wells, and other waterways should be assessed and made safe, the use of boats should be assessed for the appropriate safety equipment.

Areas where there are hay stacks, bales, pallets, bags or stacked timber, should be assessed. Reasonable steps should be taken to ensure that they cannot crush or trap children.

Where environmental conditions in work areas may be harmful to children's health, particularly where excessive noise or hazardous dusts or substances may occur, children should not be present unless appropriate protection is provided.

It should be noted, that due to the poor fit, personal protective equipment designed for adults will not protect children.

Then there are the preventive measures required by legislation, including:

(a) Machinery needs to be guarded appropriately in accordance with the Occupational Health, Safety and Welfare Regulations, 1995. If it is necessary to remove guards from machinery - for instance when it is being repaired - steps must be taken to ensure it can't be started. **A guard designed to protect an adult is not necessarily an effective safeguard for children.** Children have been known to squeeze through or behind guards designed to protect an adult;

(b) All electrical installations and equipment should be installed and maintained so as to minimise the risk of electrical shock or fire, eg Residual Current Devices (RCD) on all portable electrical equipment (Occupational Health, Safety and Welfare Regulations, 1995);

(c) Agricultural chemicals should be kept in their original containers, correctly labelled and safely stored (Agricultural Chemicals Act, 1955);

(d) Fuels and highly dangerous substances should be safely stored (Dangerous Substances Act, 1979);

(e) Firearms should be safely kept in accordance with the appropriate legislation.

No child should be permitted to operate, ride on or in, or be in the vicinity of machinery before the child and the machinery are assessed to ensure:

(a) The child is properly supervised by a responsible and capable adult, taking into account the age, maturity, experience and capabilities of the child, and that the child has the size, strength and reach to operate all of the controls with ease;

(b) All controls are conveniently accessible for safe operation by the child when seated in the driving seat or standing in the normal control position;

(c) The controls which operate any power take-off devices, hydraulic equipment or engine-stopping devices are clearly marked to show the effect of their operation;

(d) The machine is maintained so as to be safe for the child when driving it; for example, the brakes have been checked and operate correctly;

(e) The terrain over which the machine is to be driven is free from hazards such as steep slopes or excavations;

(f) The child has received adequate instruction in the safe operation of the particular make and model of machine being driven and on the work to be undertaken and can demonstrate an adequate ability in doing so;

(g) There are no other children present on or about the machine unless they are involved in the work and have been similarly assessed;

(h) There is no person present on any trailer or other equipment being towed, other than for the purposes of work, instruction or supervision.

Children should only ride on power-driven machinery or associated equipment:

(a) where they are actively involved in the work being performed (such as hay carting);

(b) for the purposes of training;

(c) for education and recreation (such as hay rides).

If a child is assessed as capable of operating machinery or equipment, direct supervision should be provided during its operation.

A child should only be permitted to drive, operate, assist in the operation of, clean or maintain any other machinery after the risks have been assessed, appropriate safeguards introduced, and adequate instruction and supervision provided.

Children rely on grown-ups to keep them safe and sound. Protection of our energetic, adventurous kids is a privileged responsibility for all adults, particularly those who live and work in the potentially dangerous environment of our farming industry.

We know how youngsters want to get into everything. How they love to ride on the tractor, to help out with the work on the farm and tend the animals. Some farm equipment/buildings and structures can have a fatal attraction.

Health and Safety hazards for adults on farms are also hazards for children. However, the risk of injury from these hazards is far greater. The age of the child plays a significant part in determining the level of risk from these hazards.

Of all fatal farm accidents in South Australia in the past eight years, 25% have involved the death of children. In addition, many children are seriously injured every year. Between 1986 and 1992 in South Australia, farm accidents accounted for 82% of the 201 childhood hospital admissions from workplace injuries and 73% of the 37 children admitted with machine injuries. In fact, the rural industry has the worst record for causing fatal injury of all industries in this State.

The most common causes of child death and injury are machinery (including tractors and agbikes), animals, chemicals and drowning.

This guide is designed as a practical help for busy farmers and other agricultural workers who need to reduce the everyday dangers that their own children, and their children's friends face about the farm.

It can be done in three simple stages - identifying the dangers, assessing the risk and introducing measures to control those risks.

8.5 Child Care as an Option

Child care is an option which will isolate the child from the hazards. Unfortunately, it has been a difficult option in the past due to the unique nature of farming conditions. One option is that child care schemes should be set up for each community by that community. It must take into account needs such as before and after school care, care in the family home, especially for people such as dairy farmers who are busy in the early morning and late afternoon, occasional care where children are minded while parents are at a special event and long day care for parents who work on-farm and off-farm.

9. SUMMARY

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Farms can be an extremely dangerous place for children. Some of the dangers include:

- TRACTORS - the majority of deaths to children result from falls from tractors.
- SILOS - binned grain can turn into quicksand, particularly when unloading from the bottom. Children have also 'drowned' in grain while a silo is being filled; even an empty silo can contain 'bad air'.
- FARM DAMS - children can drown in the most shallow of dams.
- ELECTRICAL - boxes, wiring without safety switches and tools in poor condition.
- HEIGHTS - anywhere high enough to make a fall serious.

The only way to make any of these areas safe for children, is to make these areas inaccessible.

Have you ever set down a container of pesticide or similar for 'just a few seconds' - and ended up leaving it there for several hours or even days? Half-hearted safety measures just aren't good enough! Some practical advice that should be considered:

- Practice Safety by setting a good example - when children start to help on the farm, they will learn by following your example. If you work safely, so will they.
- Make work areas "off limits or out of bounds" - until children are old enough to begin helping, they should not be allowed in areas where work is underway. It is impossible to operate a machine and watch for children at the same time. When children do start to help with the farm activities, adults must keep in mind the child's limited strength and experience when conducting these activities.

Farmers are known for their practical approach to solving problems quickly. This practical approach needs to be directed to farm safety and especially child safety on farms.

As a farmer, you need to anticipate what could go wrong - then - take action to stop an unwanted event from happening that could kill or seriously maim a child on your property.

Safety is not a time-consuming task. Once established, a secure environment offers lasting protection. It is the least we can do - for our children.

Safety on the farm is your business because you are the one in control.
The safety of children is your direct responsibility.

10. HAZARD IDENTIFICATION CHECKLIST

(These are only suggested headings for a checklist)

(a) what types of work are being carried out in the workplace?

(b) what types of machinery are being used or stored in the workplace?

(c) what types of animals are in the workplace?

(d) List the types of chemicals being used or stored in the workplace, including:

- herbicides
- fungicides
- solvents
- other chemicals
- insecticides
- fuels
- veterinary chemicals

(e) Does the work process use electricity?

(f) Does the work process generate any hazard such as:

- dust
- fumes
- mists
- flying bits of material
- smoke
- gases
- vapour
- noise which could damage hearing

(g) What are the means of access to and exit from work areas?

(h) What is the condition of the following?

- animal enclosures
- fences
- paths
- stairs
- gates
- doors
- walkways

(i) List the types of storage facilities, including:

- dams
- silos
- fuel tanks
- field bins or other storage bins

(j) Are there tanks / pits or other dangerous areas, such as:

- septic tanks
- bores
- disused mine-shafts or wells
- water storage tanks

Please note that the checklist provided is not exhaustive and is included as a guide only.

11. INFORMATION

Occupational health and safety information and other useful information is available from the following sources:

WorkCover Corporation	(08) 8233 2222
Department for Industrial Affairs - Regional Offices:	
Adelaide Central	(08) 8362 0044
Adelaide Northern	(08) 847 4066
Adelaide Southern	(08) 8276 7088
Berri	(085) 95 2199
Mount Gambier	(087) 35 1199
Port Pirie	(086) 33 0919
Whyalla	(086) 48 8151
Primary Industries South Australia	(08) 8226 0222
Health Commission SA	(08) 8226 6000
Department for Environment and Natural Resources	(08) 8204 9322
Environment Protection Authority	(08) 8204 2000
Women's and Children's Hospital - Health Information Centre	

11.1 Local Community Organisations

These include:

Farm Safety Action Group	Local Council
SA Country Women's Association	Local Library
Child and Youth Health	

11.2 Employer Groups

South Australian Farmers Federation	(08) 8232 5555
SA Employers Chamber of Commerce and Industry	(08) 8373 1422

and other industry associations can provide health and safety information and advice to their members.

11.3 Union Members

Union members should contact their local union office or the United Trades and Labour Council (08) 8212 3155 for advice and information.

11.4 Industry Training Program

Farmsafe Australia, in conjunction with the SA Farmers Federation, has been developing, on a national basis, a training program for farmers on Managing Farm Safety. The program will commence during 1997. Up to 20 guidance notes (with number 7 devoted to children on farms) will form part of this comprehensive program which will be widely available within the industry. Interested farmers should contact the SAFF or Farmsafe representative for more details. WorkCover commends and supports the industry for this initiative.

11.5 Product Manufacturers and Suppliers

Manufacturers, suppliers and re-sellers of machinery, equipment and chemicals should provide health and safety information at the time of supply. Make sure you ask for them when you purchase products for use at the workplace.

11.6 Private Consultants

There are some private consultants who specialise in farm safety.

12. USEFUL REFERENCES

12.1 South Australian Occupational Health and Safety Legislation

Copies of the Occupational Health, Safety and Welfare Act, 1986, regulations and approved codes of practice, and other South Australian legislation, can be purchased from the WorkCover Bookshop at 100 Waymouth Street, Adelaide (08) 8233 2504.

12.2 Standards Australia

A large number of publications relevant to occupational health and safety are available from Standards Australia. 63 Greenhill Road, Wayville, Adelaide (08) 8373 4140.

12.3 Farmsafe Australia

Australian Agricultural Health Unit
PO Box 256
MOREE NSW 2400

Telephone: (067) 529 203
Facsimile: (067) 526 639