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# SEVERE HEAD INJURIES IN CHILDREN

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## Declaration

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# *CHAPTER 1*

# INTRODUCTION

Childhood head injury is best described as the silent epidemic. International studies have shown that trauma is the leading cause of death in children between the years of 5-15 in industrialized and some developing countries (74, 53). In South Africa injury kills more children over 4 years of age than all other diseases combined (34). Head injuries account for 25-80% of these childhood trauma related deaths (77).

In South Africa the public appears to be unaware of the trauma epidemic, and the importance of trauma as a major child health problem continues to go unrecognized. An accident continues to be thought of as a consequence of uncontrollable acts of God about which we are powerless. However, childhood accidents cannot be dismissed as random, unpredictable events; they occur when a vulnerable child meets an injurious agent in a hazardous or compromised environment (34). As such, injury must be regarded as a disease, with identifiable causative factors.

A severe head injury in a child is a sociological disaster that crosses all sociological boundaries, not only for the disability it causes the child, but also the load it places on the family, medical profession and the state. It encompasses all aspects of child health care and extracts an enormous toll on society as a whole (34).

Children are not small adults and they therefore respond differently to trauma and cannot be managed in the same way as adults. In fact, the response of a child to injury varies between an infant and an older child. Therefore it cannot be assumed that what holds true for adults can be extrapolated to children.

The aim of this dissertation is to review the current literature on severe head injuries in children, with particular respect to epidemiology, clinical features and investigation, pathophysiology, management and outcome. In addition a retrospective study was carried out on severe head injuries at Red Cross War Memorial Children's Hospital. Finally, the findings of this study are discussed in the light of the experience of other neurosurgical and trauma centres. In this way similarities as well as features peculiar to our setting can be identified with the aim of improving the understanding and management of severe head injuries in children in the Western Cape.

# *CHAPTER 2*

# EPIDEMIOLOGY

## *INTRODUCTION*

Head trauma is a major cause of mortality, morbidity and disability in children. However, the epidemiology of head injury in children is not well described because of the difficulties in identifying and classifying such injuries.

## *DEFINITIONS*

### Rates of Occurrence

The goal of epidemiology is to define the incidence and prevalence of health problems. Rates of occurrence of head trauma can be expressed as a mortality rate i.e. the number of deaths per 100 000 population per year or as the incidence rate i.e. the number of cases of head trauma per 100 000 population per year. The population will generally refer to the age group less than 14 years of age. Rates can also be expressed for subgroups e.g. sex, age, causes and severity of injury.

### Definition and Classification

Head trauma mortality rates often cannot be easily determined because many deaths occur outside hospitals and are classified by pathologists and coroners according to aetiologic agents rather than the anatomical systems involved. Thus mortality statistics may not differentiate between head injury and other causes of death.

Hospitals are the principal source of morbidity data. However, poor attendance at follow-up clinics severely limits the collection of morbidity data. An additional problem is the absence of national or local injury morbidity registers. However this is currently being addressed in South Africa by the Medical Research Council National Trauma Research Programme (J van der Spuy - personal communication).

In order to study the epidemiology of a health problem it is necessary to define a case. Such definitions are more meaningful if they are based on clinical criteria rather than broad diagnostic categories. Severe head trauma can be defined as a Glasgow Coma Scale of less than 9 following resuscitation (20, 85).

## *INCIDENCE*

Every year in South Africa 3 000 children under 15 years of age are permanently disabled from accidental injury (106). Over a 5 year period 57 468 patients were seen in Red Cross Children's Hospital trauma unit and in 17,1% of these head injury was the principal indication for admission. Although most of these injuries were minor, in-hospital mortality has been shown to be directly related to the presence and severity of head injury alone (73). In a study undertaken to describe the causes of unnatural death in children under 15 years of age in the Cape Peninsula, head injury alone was given as the cause of death at autopsy in 25,2% (77).

In the United States 200 000 children are hospitalized each year for the evaluation and treatment of head injury (40). According to Kraus (78) the annual brain injury rate of children in California was 185 per 100 000 children. Twelve of these head injuries were described as severe.

There is not always conformity in the description of a severe head injury because most studies tend to group all head injuries together. Therefore it is difficult to ascertain the exact rate of severe head injury in a population of children, although Klauber does state that the ratio of mild to severe injury is much higher in the paediatric age group than in adults. This again may reflect the difficulty in distinguishing mild from more severe head injuries, particularly in the very young (75), or the different aetiology of head injuries in children and adults.

## *MORTALITY*

Injuries have become the leading cause of childhood death in the United States (63, 115), accounting for 50% of all deaths in children aged 1 to 14 years (61). Almost 80% of paediatric patients with multiple trauma have an associated head injury (61), thus being the single most common site of injury. In the United Kingdom accidents constitute the greatest single cause of death between the age of 1 and 14 years and head injuries account for 40% of these (51).

In a Medical Research Council Report on Injury-related Deaths in South African Children 1981-85, injury accounted for 8% of deaths in children under the age of 15 years. The majority of all childhood deaths occurred under 5 years and were related to poor living standards. However injury was the leading cause of death between the ages of 5 and 14 years (accounting for 43%). Mortality rates for the country as a whole could not be calculated but in the case of whites, Coloureds and Asians, the injury death rates were 1,5 to 3,8 times higher than those in the United States of America (74). Although most data on injury-related childhood deaths describes the

aetiology rather than anatomical system involved, a study of non-natural deaths in children over a 15 year period in the Cape Peninsula found head injury alone to be given as the cause of death in 25,2% (77).

## *MORBIDITY*

Figures for the morbidity of severe head injuries are difficult to find. This is partially due to the difficulty with defining what constitutes a severe head injury, what constitutes morbidity, and in following up patients. The majority of patients sustain mild head injuries, and thus severe persistent disability as a result of head injury in childhood is uncommon. However, severe and fatal head injuries in children account for about 10% of the total (2), and an overall morbidity of 6% to 30% for all head injuries is quoted (61). In a study of recovery in children following severe head trauma in California, 73% of patients regained independence in ambulation and self care, 10% remained partially dependent in self care and achieved only limited ambulation, 9% regained consciousness but where totally dependent and 8% remained comatose (15).

## *SEX*

There is a male preponderance in children sustaining head injuries as a whole, as well as in those sustaining severe head injuries (2, 59, 70, 78, 132). The increased risk in boys is likely due to differences in exposure to risk, differences in behaviour, or a combination of the two.

Kraus and co-workers found that brain injury rates did not differ between male and female infants or children 1 to 4 years of age. There were, however, higher rates for boys and girls 5 to 14 years of age. The gender rates also varied for the different aetiologies with the male: female ratio for falls 1,7: 1, recreational activities (mainly cycling) 2,7:1 and motor vehicle accidents 2,1:1 (78).

## *AGE*

In the series of severe head injuries reported by Bruce et al. (21) the mean age was 7.2 years and in that reported by Humphreys et al. (65) the mean age was 7.5 years. Other authors have found the mean age to be younger at 5.1 years (46) and 4 years (54) respectively. In South Africa there is a higher reported incidence of non-natural deaths (77) as well as head injuries in children requiring admission to hospital (39) under the age of 6 years. The mean age of children in hospital in Cape Town for a head injury is 5.27 years as compared to 6.36 years in those delivered to the police mortuary (33).

The ratio of mild to severe head injury was much greater in the paediatric age group under 10 years of age than in patients older than 10 years (75). This may reflect the difficulty in distinguishing mild from severe head injuries, particularly in the very young. It is therefore likely that in most surveys of head injury, the admission rate of young children with mild head injuries compared to severe head injuries will be higher than that for older people. This may be attributable to parental anxiety or difficulties associated with the clinical assessment of small children.

Different age groups are characterized by different causes of head injury in children. Over 5 years of age pedestrian traffic collisions are the predominant cause of paediatric head injury (16, 87, 132, 140). However, in children under 5 years falls are the most common cause of head injury (2, 16, 87, 111, 132, 140).

Although the mortality rate in South Africa differs between population groups, there is no significant difference in age distribution (33).

## *SOCIAL AND ECONOMIC FACTORS*

In South Africa, population group correlates to a large extent with socioeconomic status. In addition, where apartheid legislation has influenced living conditions, one may expect to see a difference in the incidence and aetiology of head injuries amongst the different ethnic groups (33). There were significantly higher percentages of Black and Coloured children as compared to white children sustaining head injuries in the Cape Peninsula (39, 77).

In their analysis of childhood pedestrian injuries, Rivara and Barber found that socioeconomic factors influenced the incidence of injuries. Socioeconomic factors found to be significant were lower income households, children living in female-headed households, families living below the poverty level and crowded accommodation (117).

Children from deprived socioeconomic backgrounds were found to have a twofold to threefold greater risk of pedestrian injury compared with the more privileged. The increased risks are multifactorial but seem to be related to the child's environment. Economically deprived areas are likely to have high traffic volumes and density,

higher average and posted speed limits, fewer pedestrian control devices and children tended to play in the street due to lack of formal recreational areas (116).

Socioeconomic factors determine not only the type and quantity of hazards but also the quality of childcare and access to medical facilities (73). These socioeconomic factors have become related to race in South Africa as discussed earlier.

Theron states that poverty is associated with vulnerability and also noted that higher levels of socioeconomic adversity were found in patients with head injury than in a control group. An analysis of the psychosocial backgrounds of 6-14 year old children enlisted in a prospective study on paediatric head injuries in Cape Town found over-representation of the socioeconomically disadvantaged groups (135).

## *PHYSICAL ENVIRONMENT*

Most authors concur that head injuries in children peak in the afternoon and early evening when it is still light (2, 5, 77, 78). Knobel reported a peak in transport-related injuries between 16h00-20h00 (38%), whilst according to Craft, the commonest times for injury were mid-morning and mid-afternoon. This correlates with the times children are out of school and playing. Injuries sustained during these times are usually transport-related or due to falls.

Day of the week on which the injury occurred seems to vary from study to study. Craft (Newcastle, England) found the peak incidence on Thursday, and thought this may be related to the fact that Thursday is pay-day and also because many private practitioners take Thursday as their half-day resulting in more patients attending government hospitals. Knobel found that the incidence of transport-related injuries

peaked over the weekend; this may be related to the fact that children are outside playing and that there is an increased incidence of alcohol abuse by drivers over weekends. Other authors however, have found no variation with the day of the week (5, 77, 78). Day of the week may thus be influenced by regional and social trends.

Seasonal variation has been uniformly dominated by the summer months, peak incidence occurring in the Northern Hemisphere in July and August (2, 5, 78) and in the Southern Hemisphere in December and January (73, 77). In the summer months there are longer daylight hours as well as the traditional long summer holidays when children are out of school.

It has been uniformly found that pediatric head injuries occur predominantly in clear, warm weather (33). This corresponds to an increased number of children playing outdoors.

The most common site of the accident in children over 4 years was in the street, the site of transport-related injury (5, 32, 50, 73, 140). This predominance is even more marked in the lower socioeconomic groups (117). Atkins found that the majority of transport-related injuries occurred within 800m of the child's home. In the child less than 4 years of age, the head injury most commonly occurred in the home (50).

Frequent absence of a custodian at the time of injury has been found by Cumpsty and Theron. This may not necessarily be due to lack of awareness, but may be more related to socioeconomic circumstances (33, 135).

# *AETIOLOGY*

## Road Traffic Accidents

Road traffic collisions are responsible for the majority of head injuries in children in most studies (32, 51, 77, 105). Craft found that 33% of paediatric head injuries resulted from road accidents in Newcastle, England. The majority of fatal head injuries have been ascribed to road traffic accidents (2, 77), with 72% of fatal head injuries in children in Cape Town being transport-related (77). There is some variation with age; road traffic accidents are the most common cause of head injury in children over 4 years of age, but other causes are more common below this age (87).

The largest subgroup in this aetiologic category are pedestrians (39, 51, 106). Possible reasons for the high risk of neurotrauma in the paediatric age group are the child's poor perception of the hazards of the road, the need to cross roads on the way to school, friends and playgrounds and sadly in the poorer areas, the roads lack pavements, are poorly lit and act as playgrounds in the absence of safer alternatives (39, 106). Also, because of short stature, the child is often not visible to drivers over parked cars, and the head is hit directly by the oncoming vehicle.

The incidence of head injury in children as motor vehicle occupants is less than that of pedestrians. Of particular concern is the higher mortality in infants as motor vehicle occupants (111). The use of restraint devices may help to improve this situation. In January 1996 legislation was introduced in South Africa enforcing the use of seatbelts and child restraints.

Bicycle accidents have been found to be a common cause of head injury in a number of studies (2, 78), particularly in first world countries where bicycle ownership is high amongst children. Kraus found that three quarters of bicycle-related head injuries did not involve a motor vehicle collision but were caused by falls off a bicycle following impact with another object or loss of control (78). In South Africa, however, bicycles contribute very little to road death (73).

## Falls

Falls are a common cause of head injury and in a number of studies have been cited as the main cause of paediatric head injury (2, 10, 50, 59). In South Africa, falls are second to traffic-related accidents in the aetiology of paediatric neurotrauma (77, 39, 106). However, in children under a year of age, falls remain the most common cause of head injury (39, 52). This is consistent with the motor development of children at this age who are beginning to mobilise and often fall off elevated surfaces or out of attendants' arms.

In the first year and a half head trauma is more likely to occur from falls from heights, usually from the parents arms, the crib or the high chair. In children in the second or third year injury more often results from falls while running or playing (111). An important factor in falls in children under the age of three appears to be poor supervision (33, 39). An interesting observation is the low incidence of falls causing head injury in Black children in Cape Town. This may be attributable to the custom of mothers carrying their children strapped to their backs, keeping the child safeguarded from free-roaming (39).

## Assault / Abuse

In Cape Town this is the third most common cause of admission for head injury (39) in children. The majority of head injuries due to assault / abuse are caused by blunt instruments. Penetrating injury of the head is uncommon and is frequently associated with the child being used as a shield by one parent against the assault by the other parent. Penetrating injury is less often accidental e.g. the child falling on a piece of wire (39).

The head injury may be part of the battered baby syndrome with evidence of other injuries being present (e.g Caffey's syndrome with long bone fractures and an associated subdural haematoma).

## *CHAPTER 3*

# PATHOGENESIS AND PATHOPHYSIOLOGY

## *INTRODUCTION*

In order to improve the prevention and treatment of head injuries, a complete understanding of the mechanisms of causation of pathophysiology is essential (47). A child who suffers a head injury is nearly always a victim of some circumstance that could have been foreseen. Due to their small body size, relatively large head, soft pliable skull and incomplete myelination of the brain, children suffer more disastrous head injuries than adults (38).

Children are not small adults, and in fact the childs' response to injury varies with age. This is especially true of the central nervous system during the first three years of life, during which the process of maturation continues at nearly the intra-uterine rate. Changes in behaviour and neurological function which correlate with anatomical function are most striking in the first three years of life. Although the brain neuronal population at birth is equal to that of adults, glial cell proliferation has just begun and then continues well into the third year. Dendritic arborisation, and the formation of synaptic connections, occur primarily in the first two years of life and not in utero. Myelinization within the central nervous system progresses most rapidly in the first post-natal year, but continues during the first decade, and the increase in weight from about 25% of adult weight at birth to 70% of adult weight at 4 years of age is primarily an expression of myelination (111).

Dramatic changes in the brain's protective covering also occur in early life. At birth the cranial vault is pliable, being composed of independent floating plates. By one year of age the sutures have closed sufficiently to convert it into a solid and rigid skull although the anterior fontanelle may not ossify completely until 2 years of age (111). Perhaps the most striking observation concerning the fall in morbidity and mortality in paediatric head injury is that the age demarcation of good and poor outcomes is sharp, and this demarcation occurs approximately at the time of fontanelle closure. Thus it appears that the closure of the sutures in some way confers additional protection to the brain subjected to blunt trauma (111).

## *CLASSIFICATION OF HEAD INJURY*

In general terms, individual mechanisms of trauma produce very specific types of head injury. Several factors must be considered in determining which injury will occur from a particular type of mechanical insult. The nature, site, severity, and direction of mechanical force to the head are important. The manner in which the head responds to that force will determine which structures are injured and the extent of the injury. Finally, the total injury produced as a consequence of mechanical trauma, depends not only on the primary mechanical damage but also on the complex interaction of pathophysiological events that follow (47).

Brain injury occurs both at the time of impact (primary head injury) and as a result of secondary complications (46). Primary head injuries include three distinct types that may co-exist - skull fracture, focal injuries and diffuse brain injuries - each having its own distinct mechanical aetiology and pathophysiology. Secondary brain injury may

occur at any time after the initial impact. Impact damage is unavoidable but secondary brain injury caused by haematoma, brain swelling, brain shift, ischaemia and infection may be preventable (46).

## *MECHANISMS OF INJURY*

The types of mechanical loading of the head are numerous and complex (47).

Static loading occurs slowly with gradual application of force to the head, usually taking more than 200 milliseconds to develop. It usually occurs in situations where the head undergoes a gradual crush. Sufficient force results in multiple fractures of the skull vault or base, but brain injury is characteristically not seen until the skull deformity is severe enough to cause distortion and compression of the brain itself, at which time serious or fatal brain injury occurs (47). This mechanism of injury is rare.

Dynamic loading is the more common mechanism of injury and the forces act in less than 20 milliseconds in most cases. The length of time of loading is the critical factor in determining the type of injury produced. Two types of dynamic loading occur - impulsive and impact loading. Impulsive forces occur when the acceleration or deceleration forces are applied to the head without any impact to the head itself. Thus there is no impact to the head itself and no contact forces occur. The injury is therefore caused solely by the inertial forces applied to the head and brain. Impact loading is the more frequent type of dynamic loading and is the result of a combination of contact and inertial forces. Whether contact or inertial factors predominate, is determined to a large extent by the characteristics of the object that strikes the head (mass, surface area, velocity and hardness of the impacting object).

Strain is the proximal cause of tissue injury, whether induced by inertial or contact forces. Strain is defined as the amount of deformation a tissue undergoes as a result of a mechanical force being applied (47). The type of injury that occurs is determined by the type and location of the induced strains, and by the ability of the tissue to withstand those strains. Three types of strain can occur - compression, tension and shear (47). The three principle tissues involved in head injury - bone, vascular tissue, and brain tissue - vary considerably in their respective tolerances to deformation. Bone is considerably stronger than brain or vascular tissue and more force is therefore required to produce injurious levels of strain. Since brain is virtually incompressible, and since it has a very low tolerance to tensile and shear strain, the latter two types of strain are the usual types of brain damage. The same applies to vascular tissue (47).

There are four mechanisms that appear to be the major cause of brain damage in head injured patients. These are: 1) mechanical injury, particularly to axons; 2) haemorrhage into the brain parenchyma; 3) oedema that develops around a contusion or haematoma; and 4) ischaemia produced by an expanding mass or brain swelling (80).

## *PRIMARY BRAIN INJURY IN HEAD TRAUMA*

### Primary Damage to the Cranium

Skull fractures may be linear, depressed or basilar. The occurrence of skull fractures depends on the material properties of the skull, the magnitude and direction of impact, the size of the impacted area, and the thickness and strength of the skull in various areas. The local inbending that occurs when an object strikes the skull

produces a compression strain on the outer table and a tensile strain on the inner table. Since bone is weaker during tension than during compression, sufficient inbending causes a fracture to originate in the inner table. It will then propagate along the lines of least resistance from the impact site. The deformation may either occur at the site of the blow or remote from it and if of sufficient magnitude, results in mechanical failure of the bone. Linear fractures occur with a force applied over a wide surface area, and if the impact is great enough, subsiduary fracturing produces a stellate fracture. Acceleration injuries may be superimposed if substantial head motion occurs after impact. Depressed fractures are similar to linear fractures except that more contact forces are present, usually because of a smaller impact surface, thus exceeding the elasticity of the skull and allowing skull perforation to occur. Basilar fractures result from: 1) direct impact to the base of the skull, 2) energy transmitted to the skull base from facial or mandibular impact, or 3) remote effects from skull impact resulting in stress waves or change in skull shape (47).

Skull fractures can occur without serious consequences to the patient, or they may be associated with devastating injury to the brain. Nevertheless, a skull fracture indicates an impact of considerable force and patients with a skull fracture have a higher incidence of parenchymal injury and intracranial haematoma (47).

The location and nature of the skull fracture may suggest additional complications. If the fracture line extends through the squamous portion of the temporal bone, the possibility of an extradural haemorrhage from laceration of the middle meningeal artery is increased. Fractures extending through the base of the skull may be associated with leakage of cerebrospinal fluid, and the development of meningitis.

Rarely such basal fractures also injure the pituitary stalk causing transient or (less commonly) permanent diabetes insipidus. Cranial nerves may also sustain direct injury if they are entrapped in the line of fracture as they course through bony canals (40). The likelihood of intracranial infection is increased when there is a fracture which extends through the base of the skull, or there is disruption of the natural anatomical defences to infection, such as a tear in the dura in a depressed skull fracture.

### Contusion and Laceration

Contusions deep to the site of impact are due to local tissue strains arising from local bending of the skull, which then exceeds the pial vascular and brain cortical tissue tolerances. Superficial focal areas of vascular disruption and cortical damage remote from the site of impact occur principally because of inertial effects (contra-coup). Intermediate coup contusions refer to vascular disruptions of the brain surface that are not adjacent to the skull. It is likely that they are due to strain concentrations from impact-generated stress waves, or from inertia-generated brain movements. Intracerebral haematomas are essentially contusions in which larger, deeper vessels have been disrupted.

The terms "contusion" and "laceration" are used to describe respectively the bruising or tearing of cerebral tissue, frequently accompanied by parenchymatous haemorrhage. The contusion or laceration is usually directly beneath the site of impact, but the lesion may be remote from the site of direct trauma. Often in severe head injuries there may be multiple sites of injury. The poles and undersurface of the frontal and temporal lobes are most frequently injured (40).

In contrast to what is seen in adults, the largest number of cerebral lacerations and contusions in children are secondary to depressed skull fractures. Cerebral lacerations following contra-coup lesions are rare in children and carry a very poor prognosis (136).

### Diffuse Axonal Injury

In acceleration-deceleration injuries of the head, large shearing forces are applied to the brain tissue resulting in stretching and tearing of the neural elements, particularly axons (fig. 1). Particularly when shearing strains are very large, there is extensive destruction of axons in the white matter of the cerebral hemispheres and brainstem accompanied by prolonged coma. These pathological alterations of the white matter were first described by Strich and are called Strich lesions or diffuse white matter shearing injury. Lesser degrees of shearing injury almost surely occur in patients with less severe neurological deficits (41, 80).

The degree and location of axonal damage in all likelihood determine the severity of injury and the degree of recovery. Critical factors in the amount of axonal damage are the magnitude, duration and onset rate of the angular acceleration, as well as the direction of head motion (47).

Microscopic evidence of neuronal damage is determined by the duration of patient survival and on the severity of the injury. After a few days retraction balls and microglial clusters are seen in the white matter. If the patient survives five weeks or more after injury then appropriate staining demonstrates Wallerian degeneration of the long tracts and white matter of the cerebral hemispheres (46).

### Penetrating Injuries

Low velocity penetrating injuries occur when the child is struck by or falls on an object. The child usually remains conscious and the focal nature of the brain injury is reflected in the neurological signs. Missile injuries may cause both focal and generalized damage (51).

## *SECONDARY INJURY IN HEAD TRAUMA*

Most of the pathological consequences of head injury other than mechanical damage result to a greater or lesser degree from an insufficient supply of oxygen to brain cells (80). This may result from a systemic insult or compression from an extracerebral or intracerebral haematoma, and uncontrolled intracranial hypertension from brain swelling.

### Systemic Insults

Serious head injuries are frequently aggravated by hypoxia and hypotension in the patient with multiple trauma. These systemic insults are the most common causes of secondary brain damage in patients with head injuries and contribute significantly to mortality and morbidity (46). Moreover, the injured brain appears to be more sensitive to these derangements.

Hypoxia may result from aspiration of gastric contents, airway obstruction or thoracic trauma. It can adversely effect the other processes of secondary injury. Both hypoxia and hypercarbia lead to cerebrovascular vasodilation with consequent increases in intracranial pressure and exacerbation of mass effect.

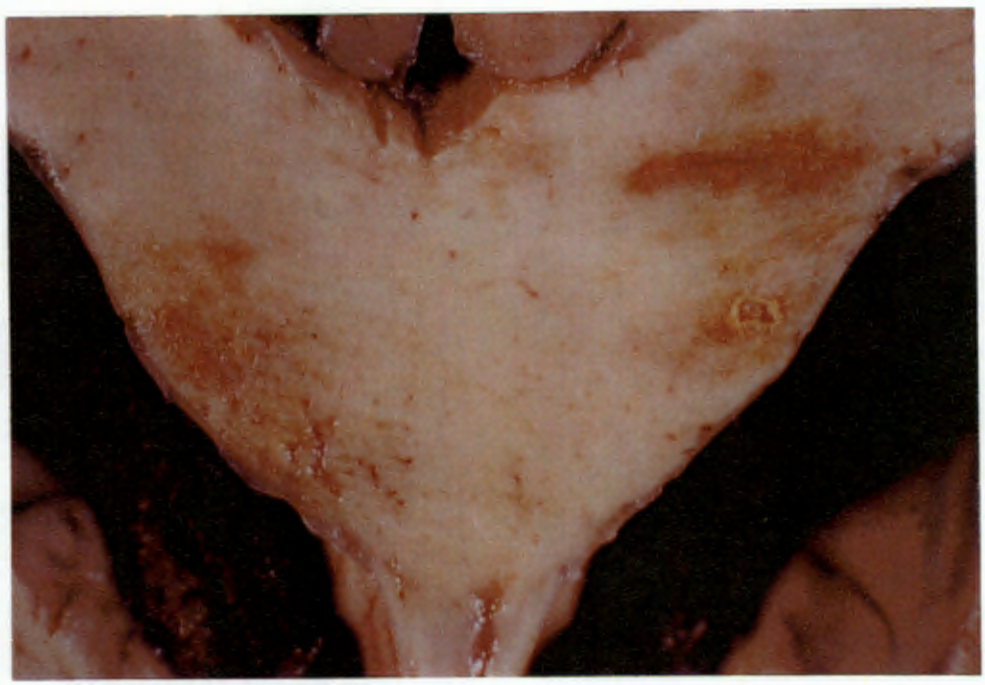


**Figure 1: Post mortem specimen showing haemorrhages in the corpus callosum in a patient who sustained a diffuse axonal injury.**

Hypotension in a head injured child is usually due to associated injury. Hypotension in adults with severe head injury has clearly been shown to cause secondary brain damage and thus increase mortality and morbidity (109). The pathophysiology of this is unclear, but is probably related to ischaemic damage caused by a reduction in cerebral perfusion pressure. With brain injury there is loss of autoregulation in the cerebral vasculature and oxygen delivery is determined by the mean arterial pressure. If hypotension occurs, the delivery of oxygen to the injured brain cells is decreased and ischaemic injury results, aggravating primary injury.

Piguela studied the effects of hypoxia and hypotension on children who sustained severe head injuries and concluded that hypoxia alone did not appear to alter the outcome, but that hypotension alone or associated with hypoxia had a deleterious effect on both mortality and morbidity. This implicates hypotension as the more critical factor in the oxygen delivery cascade, at least in children. This suggests that the oxygen extraction capabilities of the brain are significant and are able to prevent damaging ischaemia even in the presence of hypoxia. Hypotension, however, severely impedes oxygen delivery to the brain and can cause ischaemic insult, even in the absence of hypoxia (109).

### Traumatic Haematomas

In children, intracranial haematomas occur, both in the extradural and subdural sites, although they are less common than in adults (51, 111). Haemorrhages developing between the calvarium and cerebral surfaces will compress the underlying brain, resulting in underlying brain shifts and ischaemia. If the haemorrhage results from

arterial bleeding, the sequential course of the resulting neurologic syndromes will be more rapid than if the bleeding is venous in origin (40).

***Extradural Haematoma***

Histological examination of bones and dura mater in children has shown rich diploic and dural vascularization which accounts for the relative frequency of this complication in children, with an incidence of 2-4% (28, 41, 99, 129). It is thought that the initial impact with inbending or fracturing of the cranium causes separation of the dura from the inner table and injures dural arteries, dural veins, venous sinuses or diploic channels. Extradural bleeding is usually accompanied by a fracture. However, in children, a fracture may be absent in more than a quarter of cases. This is attributable to the reactive plasticity of the child's skull and looseness with which the dura mater is attached to the overlying calvarium (40, 129).

***Subdural Haematoma***

Subdural haematoma results from inertial loading with high strain rates, in which tissue stresses tend to be concentrated at the brain surface, causing injury to vascular elements. They are commonly associated with parenchymal injury (46), including diffuse axonal injury, as the mechanisms of injury are similar. In some patients impact may rupture bridging veins or occasionally arteries resulting in a pure subdural haematoma. Contusions and lacerations involving the frontal or temporal lobes may bleed into the overlying subdural space resulting in an associated subdural haematoma.

In children there is a marked fall in the frequency of subdural haematoma following closure of the skull sutures. The vast majority of subdural haematomas occur in

children under two years of age, and the incidence is very low in the subsequent decade (11). How suture closure protects against formation of subdural haematoma is not at all clear. Possible reasons are that the subdural haematoma may accumulate more easily in an expansile cavity, or differences in the bonding of arachnoid to dura at the sagittal sinus may play a part (111).

***Cerebral Swelling***

Traumatic brain swelling is a common finding in head trauma. Diffuse brain swelling is a very frequent post-traumatic lesion in children suffering acceleration-deceleration intracranial injuries (31). Diffuse brain swelling occurs approximately twice as often in children as in adults (1). The proportion of cases complicated by diffuse brain swelling in severe head injury ranges from 5-41%, but this lesion may also be seen in patients with no or minimal disturbance of consciousness (79). It has been stated that this could be a pathophysiological response of the brain to trauma that seems to be virtually unique to children and adolescents (20, 130). Many believe that diffuse swelling represents a physiological response to trauma and it may be related to the mechanism rather than the degree of injury, because some studies show that swelling is seen in patients with a wide spectrum of injury severity (19, 79).

Vascular engorgement appears to be the most satisfactory explanation for brain swelling, although the causes of such engorgement remain obscure (19, 20, 79, 101, 130, 142). The presence of hyperaemia is substantiated by cerebral blood flow measurements, and in autopsies done on patients dying immediately after trauma, two common findings are moderate increase in brain weight and dilated vessels (142).

Several authors have demonstrated that brainstem stimulation can increase cerebral blood flow without increasing cerebral metabolism (19, 130). Locus ceruleus stimulation may change both cerebral blood flow and cerebral capillary permeability, probably via central vascular aminergic pathways (19, 130). Thus, acute vascular changes may be precipitated by alterations in reticular formation or locus ceruleus function.

Increased cerebral blood volume can occur in head injury when systemic blood pressure is elevated and there is loss of autoregulation in the cerebral vasculature. One hypothesis to explain the phenomenon of hyperaemia is that vasodilatation of the cerebral arterial bed occurs following injury, resulting in increased cerebral blood flow and cerebral blood volume. The systemic arterial blood pressure, however, is not usually much elevated at the time of increased cerebral blood flow. The increased intracranial blood volume reduces intracranial compliance by forcing cerebrospinal fluid out of the ventricles and subarachnoid spaces, producing a characteristic picture of cerebral swelling (142). However, if autoregulation were defective, one would expect an elevated blood pressure to be associated with increased intracranial pressure or a passive systemic arterial pressure/intracranial pressure couple (19).

Another postulated mechanism of cerebral swelling is that chemicals released by the trauma into the CSF reach the 3$^{rd}$ or 4$^{th}$ ventricle and then trigger sudden vasodilatation (19).

Diffuse bilateral swelling may be a different phenomenon from the unilateral swelling commonly seen in association with an intracranial haematoma where the predominant

mechanism may be ischaemic oedema. Patients with diffuse swelling who deteriorate may have developed true oedema superimposed on hyperaemia (79).

***Brain Shift***

Any discrete expanding intracranial mass will usually produce brain shift and distortion together with raised intracranial pressure, which ultimately lead to herniation of brain out of the intracranial compartment in which the mass originates. Broadly speaking, the faster a mass expands, the higher the intracranial pressure relative to the amount of brain distortion produced.

***Infection***

The development of infection can lead to secondary injury following head trauma. The most common sources of infection following head trauma are base of skull fractures, depressed skull fractures, penetrating wounds and operative procedures for trauma. Infection may be in the form of meningitis, osteitis, extradural / subdural empyema or brain abcess, all of which can increase the mortality and morbidity of brain injury.

## Metabolic Response to Brain Injury

One metabolic response to head injury is a state of hypermetabolism. Elevation of the metabolic rate could be a catecholamine-mediated response to neural injury. Arterial concentrations of epinephrine and norepinephrine have been shown to be increased in severe head injury, and oxygen consumption has been found to be increased in proportion to the catecholamine elevation (22). Other factors that could effect the metabolic rate in head injury are temperature, specific dynamic action and increased muscular tone. The cause of elevated metabolic rate in head injured patients is likely

to be multifactorial and determined by the hormonal response to head injury, to management variables such as feeding, and to secondary effects of head injury such as muscular tone and motor response (29).

While patients with head injuries are initially hypermetabolic, this usually resolves by the 7$^{th}$ day after injury, and a pattern consistent with progressive starvation is noted. The hyperdynamic cardiovascular state shows a clear peak by day 3 post injury and a subsequent trajectory towards normal. This persistent elevation may represent a response mediated by injured brain tissue but does not appear to reflect systemic hypermetabolism (36).

## *CHAPTER 4*

# CLINICAL EVALUATION

## *INTRODUCTION*

With severe head injury due to any cause, prompt evaluation, recognition of incipient complications and appropriate treatment are essential if the outcome is to be favourable. While the great majority of children seen in accident and emergency departments will have sustained relatively minor injuries to the nervous system, a proportion, will have suffered an injury which has the potential for creating irreparable damage to the brain if not properly evaluated and treated. The problem of identifying those children at risk after head trauma is further compounded by the even greater frequency of apparently trivial head injuries which often go unreported or undertreated, but occasionally leads to catastrophe.

Awareness of the rapidity with which complications can evolve may lead to fear and uncertainty, and impede the methodical evaluation of the patient. All too frequently, recently injured patients will be sent for tests such as skull x-rays before an adequate clinical evaluation has been completed. Without accurate baseline clinical information, subsequent examinations of the patient are rendered more difficult and early recognition of developing complications may be delayed.

## *CLINICAL ASSESSMENT*

At primary survey, the child's heart rate, blood pressure, respiratory rate and level of consciousness should be assessed rapidly, with close attention being paid to vital

ventilatory and circulatory functions. Impaired circulation, hypoxia and hypercapnia, if not life-threatening, may all compromise cerebral homeostasis and can elevate intracranial pressure further by increasing cerebral oedema and intracranial blood volume. Rising systemic blood pressure associated with slowing of the heart rate and irregular breathing, usually implies rising intracranial pressure. Rapid heart rate with marked hypotension and irregular respiration may reflect brainstem dysfunction, as occurs with occipital fractures involving the foramen magnum. These alterations may lead to fulminant pulmonary oedema but these same findings in an injured child should also raise suspicion of occult haemorrhage, ruptured viscus, aspiration, sepsis, or fat embolization. In the infant, bleeding into the subdural or extradural space may be of such magnitude as to lower the haematocrit significantly. In such circumstances, the associated increase in intracranial pressure should draw attention to the probable site of bleeding. In older children the volume of blood lost into the intracranial cavity is usually insignificant. Rapid increases in intracranial pressure in the absence of intracranial haemorrhage all too frequently follows head trauma in children, and has been related to the rapid development of cerebral swelling (40).

The level of consciousness must be carefully assessed on initial examination. Immediate coma after head injury is usually due to primary damage to neural pathways, whereas deterioration in the level of consciousness is usually due to a developing complication or extension of the basic pathological process (51).

Careful assessment of pupillary size and responsiveness to stimuli can yield important signs of increasing intracranial pressure. A dilated pupil, poorly reactive or unreactive to light, most often indicates compression of the ipsilateral third nerve

caused by herniation of the mesial portion of the temporal lobe through the incisura of the tentorium from increased intracranial pressure. While this may be due to cerebral swelling as well as intracranial haematomas, the surgically remediable lesion should be kept foremost in mind. This pupillary finding is often accompanied by paresis of the occulomotor nerve on the same side and contralateral, ipsilateral, or bilateral body weakness or intermittent decerebrate posturing. Other causes of pupillary dilatation, unilateral and bilateral, include the use of mydriatics, eye trauma and seizures. Bleeding into the subarachnoid or subdural spaces may be suggested by retinal or preretinal haemorrhages. Well-developed papilloedema is usually not seen in the first 48 hours post-injury. Spontaneous pulsations of the retinal veins usually reflect normal intracranial pressure, particularly if the systemic blood pressure is not elevated, and may thus be a reassuring sign in the head injured child.

Focal neurological signs developing after a head injury may be due to direct trauma to the nervous system (126), or may be secondary to raised intracranial pressure. Cranial nerve palsies may be due to basilar fractures or to direct injury to the nerve from penetrating trauma. Assessment of brainstem function in severe head injuries should include testing of the cranial nerves as well as ventilatory drive. Focal signs may be due to primary injury such as a contusion, but in clinically silent areas there may be injury without any clinical features.

Whenever possible, serial examinations by a single observer are strongly recommended. Only in this way can subtle deterioration in the neurological condition over a period of time be appreciated. Alterations in mental status, including increased difficulty in arousing a patient and mounting agitation almost invariably

imply an extension of the primary pathological process. Developing focal or lateralizing neurological findings or alarming changes in the vital signs, should alert the examiner to the presence of a progressive lesion (40) or onset of a secondary brain injury.

It is likely that adults and children do not have similar clinical signs in the presence of intracranial injury. The same high yield criteria used for skull x-rays in adults and children demonstrated a decrease in the sensitivity of the criteria for detecting possible skull fractures in children (118). In addition, clinical signs do not adequately predict brain injury as seen on a CT scan (41, 118, 129).

## *ASSESSMENT OF LEVEL OF CONSCIOUSNESS*

The Glasgow Coma Scale has been widely accepted and used as an objective means of neurological assessment in patients with head injuries (133), and has also allowed precise comparison of head injured patients from one centre to another (111). However it is of limited use in children under 3 years of age. Even in older children who are neurologically unimpaired but perhaps unco-operative, the Glasgow Coma Scale may not be accurate. A normal infant will not speak or obey commands. Normally a neonate cannot respond vocally to or locate painful stimuli. Only the eye-opening responses can be measured in the standard manner. Consequently, attempts have been made to develop a coma scale that accurately measures levels of consciousness in children .

A paediatric modification of the Glasgow Coma Scale used in the Adelaide Children's Hospital since 1977 takes neurological immaturity into account (56, 114).

| ADULT COMA SCALE | | PAEDIATRIC COMA SCALE | |
|---|---|---|---|
| EYES OPEN | | | |
| spontaneously | 4 | | |
| to speech | 3 | As in adult coma scale | |
| to pain | 2 | | |
| none | 1 | | |
| BEST VERBAL RESPONSE | | | |
| orientated | 5 | orientated | 5 |
| confused | 4 | words | 4 |
| inappropriate words | 3 | vocal sounds | 3 |
| incomprehensible sounds | 2 | cries | 2 |
| none | 1 | none | 1 |
| BEST MOTOR RESPONSE | | | |
| obeys commands | 5 | | |
| localizes pain | 4 | | |
| flexion to pain | 3 | As in adult scale | |
| extension to pain | 2 | | |
| none | 1 | | |

It has been assumed that during the first six months of life, the best vocal response is normally a cry, although some infants do make vocal responses during this period. The normal score expected at this age is therefore 2. Between 6 and 12 months, the healthy infant makes noises; the expected normal score is 3. After 12 months,

recognizable words are expected and the normal score is 4; orientation, defined as awareness of being in hospital, is expected by 5 years. Motor responses are recorded as in the original 5 point adult scale. However, this system recognizes that before the age of 6 months, the best normal motor response is usually flexion (score 3), whilst between 6 and 24 months of age the infant will usually locate pain but not obey commands (score 4). Thus the normal aggregate score will be as follows; birth - 6 months 9, >6 -12 months 11, >1 - 2 years 12, >2 - 5 years 13, >5 years 14. The scales are arbitrary but accord reasonably well with standard developmental screening tests (114).

At Children's Memorial Hospital in Chicago the Children's Coma Scale was developed, modifying the best verbal portion of the Glasgow Coma Scale (48). This scale uses the same 3 - 15 scoring method, so that children can be compared to adults. The eye opening response was thought to be applicable to patients of any age. However, in assessing a best motor response in infants care is required; a child under 6 months may still have reflex physiological responses and may flex or withdraw all extremities following any painful stimulation but fail to localize pain. A more obvious problem is the verbal response in infants; smiling or orientation to sound or verbal stimulus, following objects or, in general, interacting appropriately with the environment as a subscore of 5 (orientated). When the child had consolable cries and was able to interact appropriately, a subscore of 4 was given; a subscore of 3 applies when a child has inconsistently consolable cries, moans or is unable to interact. When a child cries inconsolably and is unable to interact, a subscore of 2 is given; a subscore of 1 is when a child has no response (50).

CHILDREN'S COMA SCALE (MODIFIED GLASGOW COMA SCALE)

EYE OPENING

4 spontaneous

3 reaction to speech

2 reaction to pain

1 no response

BEST MOTOR RESPONSE

6 spontaneous

5 localizes pain

4 withdraws in response to pain

3 abnormal flexion in response to pain

2 abnormal extension in response to pain

1 no response

BEST VERBAL RESPONSE

| GCS subscore | | CCS subscore |
|---|---|---|
| orientated | 5 | smiles, orientated to sound follows objects |
| confused | 4 | consolable crying, appropriate interaction |
| inappropriate words | 3 | inconsistently consolable, moaning |
| incomprehensible sounds | 2 | inconsolable, irritable, restless |
| no response | 1 | no response |

A Children's Coma Score was developed by Raimondi (111).

CHILDREN'S COMA SCORE

OCULAR RESPONSE

4 pursuit

3 extra ocular muscles intact, reactive pupils

2 fixed pupils, extra ocular muscles impaired

1 fixed pupils and extra ocular muscles paralyzed

VERBAL RESPONSE

3 cries

2 spontaneous respiration

1 apnoeic

MOTOR RESPONSE

4 flexes and extends

3 withdrawal from painful stimuli

2 hypertonic

1 flaccid

The maximal score assignable is 11 and the minimal 3. This scoring system is primarily for infants.

## *SEIZURES*

Although children have a greater incidence of early post-traumatic seizures than adults, their susceptibility to late seizures was less whether the injury was severe, moderate or mild (3). Early seizures have been defined as those occurring in the first

week post trauma. Early seizures post-trauma have an incidence of 2.1 - 4.6% overall whereas in children this incidence is 6.5 - 15% (3, 56).

Regardless of the definition of early seizures, the majority of early seizures in all studies occur within the first 24 hours of injury. Some patients experience convulsions at the time of injury, so-called impact seizures. It is not clear whether this type of episode represents a seizure in the true sense of the word or rather convulsive activity which may be the manifestation of a normal brainstem response following deafferentation of cortical and subcortical inhibiting mechanisms. One study of paediatric head trauma patients reported 0.92% of all cases to suffer immediate seizures (56). These immediate episodes have been felt to have little predictive value in determining subsequent course, and there has been no definite proof that early seizures correlate with a higher incidence of late seizures (56). Raimondi has shown that children with focal or generalized seizures on admission have a poorer outcome than those who do not (111). The management of seizures is dicussed in Chapter 5.

# *CHAPTER 5*

# MANAGEMENT OF SEVERELY HEAD INJURED CHILDREN

## *INTRODUCTION*

If ever there was a condition better prevented than cured, it is a head injury in childhood. Head injury usually implies brain injury, and damage to the brain cannot be repaired. A child's brain is incompletely developed at birth and its increase in size is due to enlargement of existing structures as well as increasing sophistication resulting from dendrite growth, synapse production and myelination. The mature brain is said to contain 10 billion neurones, each having approximately 60 000 connections. A large proportion of these connections or synapses develop in the first 7 years of life, during which period any insult to the brain would disturb this subtle and delicate process (57).

Before discussing management, a major pathophysiological concept must be re-emphasized. This is the delineation between primary and secondary injury. The primary injury is the immediate response of the nervous system to a traumatic injury. These immediate effects occur before any medical intervention is possible and therefore, cannot be reversed. The secondary head injury is the result of progression of events triggered by the insult, resulting in further brain damage. The common pathway of secondary injury is limitation of cellular oxygen and glucose delivery. Such injury is produced by hypoxemia, hypercarbia, arterial hypotension, intracranial hypertension or infection. All of the mechanisms producing secondary injury are

potentially treatable or preventable, and it is to this end that early therapy is directed (17).

Early diagnosis of intracranial injury and aggressive surgical and medical management result in significant reduction in mortality and morbidity (20). It has been suggested that children who survive their head injury long enough to reach hospital have insufficient primary brain injury to cause death and that active management to control secondary injury will be associated with a satisfactory recovery in 80% of cases (20), however this may be an overly optimistic view.

## *INITIAL MANAGEMENT*

In the emergency room, the first steps in resuscitation are as always, attention to the ABC's (airway, breathing and circulation). The importance of extracranial insults, both in promoting diffuse swelling and in leading to subsequent deterioration and poor outcome, emphasizes the need for prompt and effective initial resuscitation. It underlines the priority of identifying and treating extracranial injuries that might lead to hypoxia or hypotensive insults superimposed on severe initial primary brain damage (79, 107, 109).

A patent airway and normal gas exchange in the respiratory system may have a profound effect on cerebral blood flow, metabolism, and intracranial pressure. Obstruction of the airway not only triggers the secondary complications of atelectasis and infection, but will also produce a gross abnormality in the gaseous exchange with resultant perfusion of the cerebral vascular system by arterial blood with a high carbon dioxide content. This, in turn, will increase cerebral blood flow leading to increased

cerebral swelling, and intracranial hypertension. In addition, restlessness or struggling of the individual with an obstructed airway will raise intrathoracic pressure and ultimately, intracranial venous pressure. Careful examination of the nasopharynx for foreign bodies, vomitus, and secretions should be carried out and mechanical clearance performed immediately. The chest should be examined to exclude a tension pneumothorax and / or haemopneumothorax. Insertion of an endotrachial tube or the performance of a cricothyroidotomy should be done as soon as possible in the emergency room if required (60). All patients with a admission Glasgow Coma Score of 7 or less require endotracheal intubation. Patients with coma scores of greater than 7 may or may not require intubation depending, on their ability to protect their airway and the presence of facial injuries. Non-cuffed tubes which allow a small air leak are used in children up to approximately ten years of age. Depending on the level of consciousness, sedation or even paralyzing agents may be required before intubation to prevent elevation of intracranial pressure during the procedure.

Shock should generally not be ascribed to head injury. There are a few exceptions to this statement: (a) newborn infants with severe haemorrhage as a result of basal fractures; (b) infants with epidural haematoma; (c) children with enlarged cerebrospinal fluid space who have a functioning shunt system and sustain intracranial haemorrhage; (d) terminal head injury with failing vital centers; (e) haemorrhage from extensive scalp lacerations (17, 60). These are uncommon exceptions and the general rule stands. Clinical shock is thus indicative of injury elsewhere in the body and scrupulous examination of the trunk and extremities should be carried out immediately to eliminate the possibility of haemorrhage within these

areas. Stabilization of heart rate and blood pressure, and ensuring an adequate airway are necessary before inter-hospital transport of the patient, should this be necessary.

During the initial period of resuscitation an intravenous catheter is inserted and blood drawn for a full blood count, blood grouping, electrolytes and clotting studies. An indwelling urethral catheter is inserted. An arterial line may be inserted for monitoring of systemic arterial pressure and blood gas analysis. If no shock is present and the only injury appears to be to the head, 0.9% normal saline is given intravenously at a rate equal to two thirds of the child's normal daily requirement by body weight. If the patient is shocked, balanced salt solution is infused to raise the mean arterial blood pressure above 60 mm Hg. Blood is given as soon as it is available. A central venous line may also be inserted. Care must be taken not to over-infuse a patient as this can aggravate cerebral swelling, particularly if there is loss of autoregulation. Once the patient has been stabilized and a thorough clinical evaluation been carried out, the patient can be sent for further investigations.

The goal of fluid administration in severe head trauma patients is to maintain cerebral homeostasis with special attention to specific features of brain injury such as decreased cerebral perfusion pressure (CPP), impaired cerebral blood flow (CBF) autoregulation, alterations of blood brain barrier (BBB), development of intracranial hypertension and cerebral oedema. The choice of fluid for the acute resuscitation of children with severe head trauma should take into consideration the specific characteristics of the BBB. The size of the transendothelial pores in the intact capillary membrane makes the latter impermeable to proteins and sodium ions. Water equilibrates across the capillary membrane between the cerebral extracellular space

and intravascular compartment according to both osmotic and oncotic pressures on both sides. Transcapillary osmolality difference is a greater driving force for water diplacement than colloid osmotic pressure gradient, so when the BBB is intact, a small reduction in plasma osmolality may result in a greater increase in cerebral water content than a more important reduction in intravascular oncotic pressure (58).

It has been postulated that different severities of brain injury produce different degrees of disruption of the BBB and that less severe alterations render the BBB permeable to isotonic solutions with preservation of impermeability to larger molecules. This hypothesis suggests that the decrease in colloid oncotic pressure which follows large infusions of crystalloid may be an important factor in formation of brain oedema in the regions of partially disrupted BBB. When the BBB is completely disrupted, it becomes permeable to both ions and large molecules, and vasogenic oedema driven by hydrostatic forces develops regardless of oncotic and osmotic changes (58).

The debate over the use of crystalloids or colloids in severely head injured patients remains unresolved. Although it has been reported that a large reduction in colloid oncotic pressure following crytalloid infusion contributes to the formation of cerebral oedema, most studies examining the effects of crystalloids involved the use of Ringer's lactate, which is not an isotonic but a hypotonic solution. Massive infusion of Ringer's lactate reduces plasma osmolality and results in the movement of water into the cerebral parenchyma with a consequent increase in intracranial pressure. In contrast, where isotonic crystalloid solutions have been compared with colloids, no significant difference in cerebral water content was observed (58).

In recent years there has been much interest in the use of hypertonic saline solutions for fluid resuscitation in patients with severe head trauma. The rationale for the use of hypertonic saline is that the restoration of blood pressure, cardiac output, and oxygen transport can be obtained with much smaller volumes of infusate. A decrease in cerebral water content and a significantly lower intracranial pressure have also been reported with infusion of these solutions (123, 58). During the infusion of hypertonic saline, it has been postulated that water is extracted not only from the uninjured cerebral parenchyma but also from cerebrovascular endothelial cells and erythrocytes. Reduction of intracranial pressure and improvement in cerebral compliance decrease the tissue pressure around cerebral capillaries, and this mechanism, together with the decrease of endothelial cell volume, increases the diameter of capillary lumen. This decrease in capillery lumen as well as the decrease in erythrocyte size are probably responsible for the improvement of CBF and cerebral oxygen delivery which are observed following the use of hypertonic solutions. However concern has been expressed over the effect of hypertonic fluid on cerebral oedema in the injured part of the brain where the BBB is disrupted and where water and sodium may pass freely from the capillaries into the cerebral parenchyma enhancing oedema formation (123).

Cervical spine x-rays are necessary in all severely head injured patients, except those due to penetrating injury and missiles. Skull x-rays provide useful information about fractures, but the patient who manifests rapidly increasing intracranial pressure will benefit little from routine x-rays, and the delay may influence the outcome to a major degree. In subdural or extradural haematoma and acute cerebral swelling, x-ray examination should be delayed until the primary problem has been dealt with (60). X-

rays of the other possible sites of injury are also done as well as a chest x-ray to confirm the position of the endotracheal tube.

Computerised Tomographic (CT) scanning is the definitive diagnostic test since it reveals the whole range of pathology from skull fractures to diffuse impact injury (17). The use of CT head scanning in children with severe symptomatic head trauma has been shown to have a definite effect in reducing mortality and morbidity (15, 61, 99). The use of CT scan in both the initial evaluation and follow-up of patients with symptomatic head injury may prevent a missed or delayed diagnosis of significant intracranial injury. Magnetic Resonance Imaging (MRI) has not been shown to disclose additional surgical lesions, although estimate volumes of lesions on MRI are frequently greater than with CT scan (84).

At this stage, based on the clinical findings, CT scan and associated injuries, the further management of the patient can be decided on, be it surgical, medical or a combination of both.

## *SURGICAL MANAGEMENT*

The outcome from severe head injury can be markedly improved by early evacuation of intracerebral haematomas (80, 89). The prime indication for immediate surgical intervention in patients with head injury is the presence of subdural or extradural haematomas, and occasionally, a significant intracerebral haematoma. Compound penetrating injuries of the skull do not have the same urgency as a subdural or extradural haematoma, but they should be operated on as soon as possible.

All patients referred for immediate surgery should receive intravenous mannitol ( 1 gm / kg body weight ) en route to the operating room (7). A wide craniotomy is carried out, exposing the haematoma and usually the frontal and anterior temporal lobe on the appropriate side. The source of the haematoma in an extradural haemorrhage is usually the middle meningeal artery or the overlying fracture, whereas in a subdural haematoma, the source is either a bridging vein or cortical bleeding that has spread into the subdural space. After decompression by removal of the clot and any necrotic brain tissue the bone flap is replaced. At this stage an intraventricular or subdural catheter can be placed for post-operative monitoring of intracranial pressure. Burr-hole placement for evacuation of an acute haematoma is ineffective. If CT scan shows a thick extracerebral haematoma with midline shift, the decision to operate is unequivocal. When CT scan shows a thin extracerebral lesion in a patient with clinically severe head injury, the decision is more difficult. Generally, if there is midline shift then craniotomy is performed and the haematoma evacuated; the reason for this being that the haematoma is frequently larger than it appears on CT scan, and the haematoma or oedema underlying the haematoma may exert a mass effect causing midline shift. The oedema may worsen within the first 48 hours post injury and thus increase the mass effect (98). An even more difficult decision is that regarding the role of decompressive craniotomy for intraparenchymal haematomas; although there is no clear-cut indication, the majority of surgeons will undertake surgery if the haematoma is causing significant shift and is in an accessible site. However, the outcome in patients with intracerebral haematomas is often poor, even with surgery (98). Occasionally, ventriculostomy is required for patients who have developed acute hydrocephalus due to intraventricular haemorrhage or a posterior fossa haematoma.

Cerebrospinal fluid (CSF) fistula from an anterior or middle base of skull fracture carries the risk of developing meningitis in 11-25% of cases, with a cumulative risk of 85% over 10 years, and the time of the meningitis can be many years after the injury (35). Repair of the fistula is normally carried out if the patient develops meningitis, if the CSF leak persists more than 2 weeks or reccurs. The literature has not shown prophylactic antibiotics to be beneficial; indeed this stategy may be harmful as the use of antibiotics may eradicate the normal flora and allow overgrowth of resistant bacteria that result in infections which are less amenable to treatment (35, 4).

## *MEDICAL MANAGEMENT*

Most of the pathological consequences of head injury other than mechanical damage have as their common denominator an insufficient supply of oxygen to brain cells, and the most common and important complications are brain ischaemia caused by compression from an extracerebral or intracerebral haematoma, and uncontrollable intracranial hypertension from brain swelling. Therefore the outcome of a child with a severe head injury is related to the successful medical management and prevention of secondary brain injury (80, 89).

Hyperaemia and vasodilatation seem to be a common response of the paediatric brain to trauma. When hyperaemia occurs in isolation with minimal underlying damage, control of the hyperaemia and maintenance of normal intracranial pressure may prevent death and permit rapid, complete recovery. When hyperaemia occurs in the

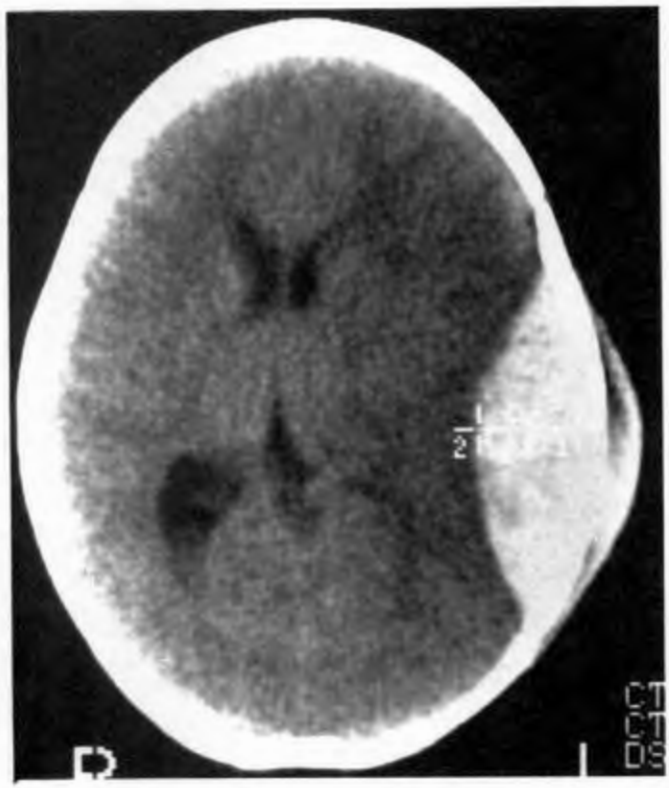


**Figure 2: Left parietal extradural haematoma in a nine month old infant who fell from a height.**

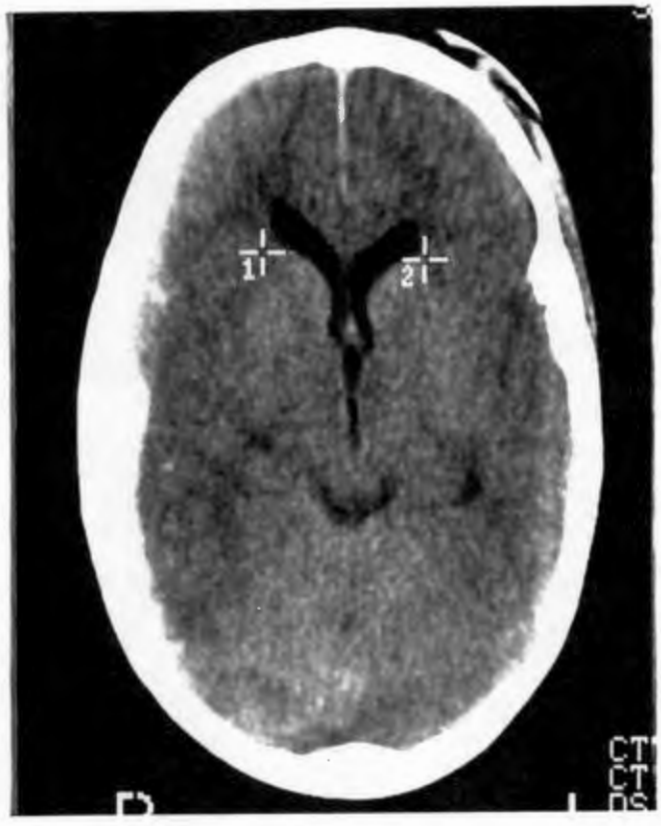



**Figure 3: An 8mm thick right subdural haematoma in a three year old child involved in a pedestrian motor-vehicle accident.**

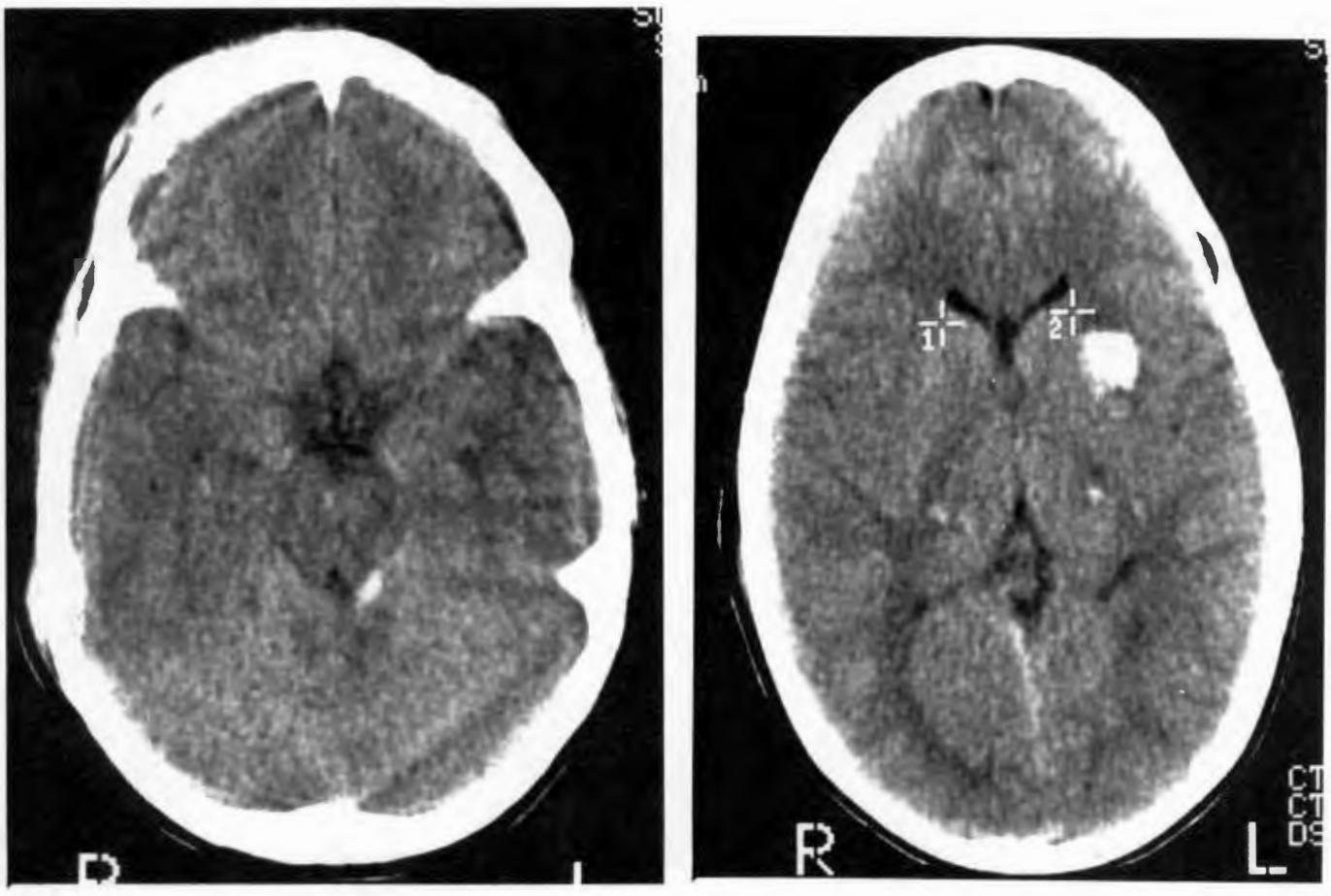



**Figure 4: Brainstem and basal ganglia contusions in a seven year old child who sustained a diffuse axonal injury following a pedestrian motor-vehicle accident.**

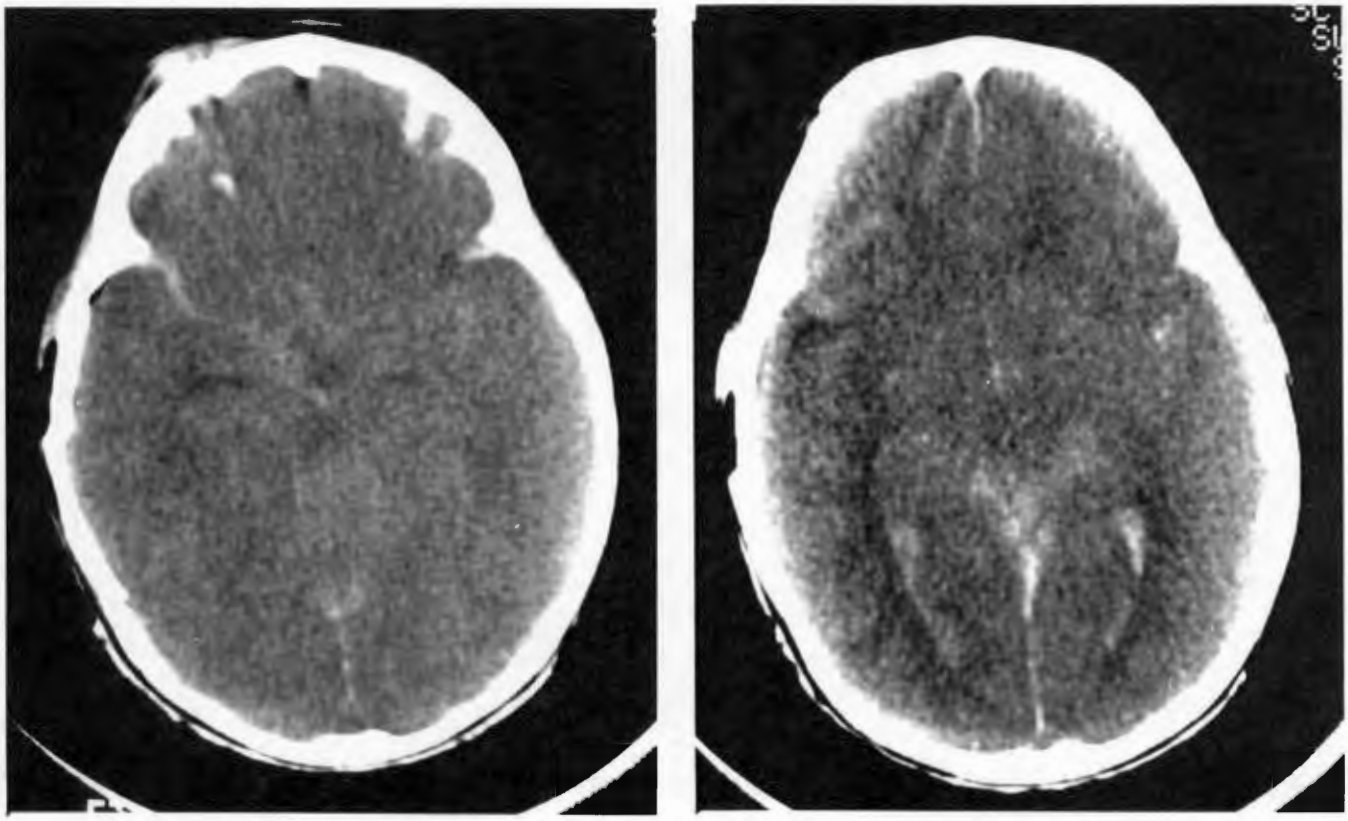


**Figure 5: Features of severe brain swelling as well as diffuse axonal injury (gliding contusions and intraventricular haemorrhage) in a three year old child involved in a motor-vehicle collision as a passenger. The outcome from this type of injury is exceedingly poor.**

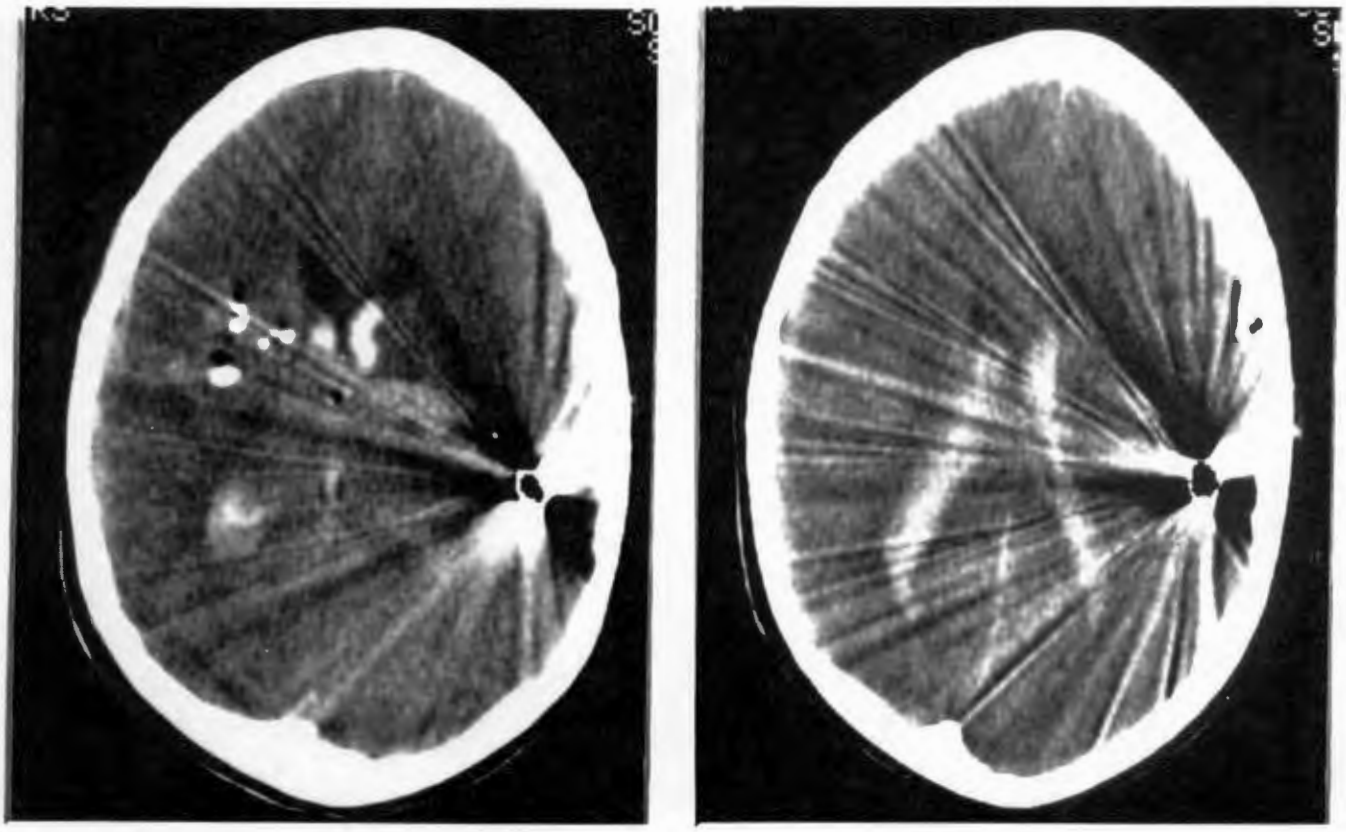


**Figure 6: Gunshot injury in a ten year old child. The entrance wound is in the right parietal area and the bullet traversed the lateral ventricles and both cerebral hemispheres. This type of injury is fatal.**

face of diffuse cerebral injury, the hyperemia and vasodilatation can be controlled for the first 24 - 48 hours. Thereafter, multifocal brain oedema may occur, and secondary increases in intracranial pressure are found. If these are controlled good recovery can occur, but if not controlled, poor outcome or death are likely (20).

Once adequate ventilation and circulation are ensured, control of intracranial pressure becomes the major challenge. Frequent pressure waves even if not high, may abolish or compromise cerebral vascular pressure autoregulation, and therefore are best prevented. Similar episodes of elevated intracranial pressure, whether induced by coughing, decerebration or seizures may interfere with autoregulation. Each episode of increased intracranial pressure may also produce tentorial or foramen magnum herniation with sudden obliteration of cerebrospinal fluid pathways. Consequently, a marked decrease in intracranial compliance occurs so that intracranial pressure previously well tolerated, now produces brainstem compression, distortion and malfunction. While these changes can be more easily appreciated when a focal mass (e.g., extradural haematoma) is present, the same progressive herniation of cerebral tissue, decrease in cerebrospinal fluid spaces and severe brainstem distortion can occur with diffuse increase in brain bulk due to swelling or oedema (17).

Intracranial hypertension has been documented in 50 - 86% of paediatric patients with severe head injury (20, 89, 97). The objectives of intracranial pressure control are the maintenance of intracranial pressure at less than 20 mm Hg, and the maintenance of cerebral perfusion pressure at 50 mm Hg. Perfusion pressure is calculated as the difference between mean arterial pressure and intracranial pressure. Reduction of

intracranial pressure from levels above 20 mm Hg can usually be attained through the use of hyperventilation, osmotic agents, diuretics or barbiturates.

All patients with severe head injuries must be managed in an intensive care unit. The child must be in the head-up position to decrease venous stasis and dural sinus pressure and with the head in the midline position to avoid constriction of venous outflow via the jugular veins.

### Monitoring Intracranial Pressure (ICP)

Efficacy of therapy for raised ICP can be judged only by measuring intracranial pressure, and intracranial pressure is the only real-time guide to intracranial events (97). The high incidence of intracranial hypertension in severely head injured patients is considered by Marshall as enough reason for all severely injured patients to be monitored for ICP (89). Miller has found ICP monitoring to be of prognostic value in head injury where there has been diffuse cerebral injury. ICP readings on admission provide a valuable guide to expected outcome, and the higher the ICP is on admission in such patients, the greater the risk of persistent or recurrent hypertension which is associated with a poor outcome (97).

Bruce only monitors ICP in patients with a Glasgow Coma Score of 3 or 4. In patients with a Glasgow Coma Score of 5 - 8 he believes that the hyperaemia is controlled by hyperventilation and that the patients will recover after 24 -48 hours. In patients with Glasgow Coma Scores of 3 - 4 the ICP remains elevated for 1 - 5 days despite continued hyperventilation and he therefore feels that monitoring of intracranial hypertension is required (20).

ICP monitoring is also important in post- operative monitoring of patients who have had an intracranial haematoma removed. A sudden rise in intracranial pressure post-operatively may suggest re-accumulation of the haematoma. However, in 60% of patients who have had an intracranial haematoma successfully removed, the ICP may still be elevated (89).

ICP may be monitored in the extradural or subdural space, or via an intraventricular catheter. The ventricular catheter also offers an opportunity to control the intracranial pressure by cerebrospinal fluid drainage (97).

The main risks of ICP monitoring are intracranial haematoma induced by the placement of the subarachnoid screw or ventricular catheter, or intracranial infection (97). Although ICP monitoring is useful in documenting intracranial hypertension as it occurs, it does not identify children likely to experience sudden life threatening elevations of ICP (127).

### Ventilation

Ventilation of a child with a severe head injury takes one of two forms: (a) ventilation to maintain normal blood gases and thus prevent secondary injury due to hypoxia; (b) hyperventilation to decrease the PaCO2 to a level of 20 to 25 mm Hg. Acute hyperventilation, through its effect on extracellular pH, produces vasoconstriction which reduces cerebral blood volume and cerebral blood flow, resulting in a decrease in intracranial pressure.

There is persistent controversy regarding whether patients should be hyperventilated after traumatic brain injury. The theoretical advantages for hyperventilation are cerebral vasoconstriction for ICP control and reversal of brain and cerebrospinal fluid

acidosis (97). Hyperventilation, however, may be associated with complications which mitigate against its routine use in severe head injury (113). Muizelaar has shown that the outcome at 3 and 6 months in hyperventilated patients was significantly poorer in patients with motor scores of 4 and 5 (102). The effect of hyperventilation on cerebrospinal fluid pH and arteriolar diameter is only short lived and may be counterproductive after 24 hours; prophylactic hypocapnia can compromise cerebral oxidative metabolism (113). When sustained hyperventilation becomes necessary for intracranial pressure control the additional need for intravenous tromethamine as a buffer should be considered, as it counteracts the loss of bicarbonate from the cerebrospinal fluid (102).

In children, as in adults, cerebral blood flow can be normal or sub-normal shortly after head injury. Under these circumstances, hyperventilation intended to cause cerebral vasoconstriction may be harmful because it may induce cerebral ischaemia. Although head injury is a recognized cause of loss of reactivity of the cerebral circulation, this is relative rather than absolute, and the blood vessels of recently head injured children show some degree of vascular response to changes in arterial blood gases and in the perfusion pressure. This suggests that measurements of cerebral blood flow or flow velocity are important if appropriate treatment is to be offered. Unless such measurements are available and show true hyperaemia, it may be wise to avoid profound hyperventilation in head injured children (96). Thus when hyperventilating children, it is valuable to insert a jugular bulb catheter so that the central venous PO2 (JVPO2), cerebral arteriovenous differences for O2 and cerebral blood flow may be monitored.

When a child is being ventilated, no sedation may be necessary, depending on the restlessness as well as the tolerance of the patient to ventilation. Alternately, the patient may be sedated or even paralyzed with muscle relaxants.

### Mannitol and Other Diuretics

There is some controversy over the use of mannitol for the treatment of raised intracranial pressure in children. Bruce advised against the use of mannitol in head injuries on the basis that osmotic diuretics would aggravate hyperaemia (59). However, the experience of other authors indicates that mannitol does not increase the intracranial pressure (9, 94, 101). Mannitol is administered as a bolus or rapid iv infusion in a dose of 0.5 to 1.5 gm per kg of body weight. This will usually effect a reduction of intracranial pressure within 5 to 10 minutes. The duration of effect is variable, but the mannitol injection should not be repeated at less than 3- to 4- hour intervals since the effectiveness of the drug depends entirely on the sudden increase of intravascular osmolality and this change is reduced if the drug is administered continuously or at frequent intervals (94). Consequently the use of mannitol "buys time" and this is why it is often used in patients with intracranial haematomas awaiting surgery. Too rapid administration of large quantities can cause pulmonary oedema and produce further cerebral hypoxia. It is mandatory to ensure adequate cardiopulmonary and renal function prior to the use of mannitol.

Renal tubular diuretics have been used in conjunction with mannitol or instead of them. In fact, Bruce advocated their use in the place of mannitol for hyperaemia (60). The renal tubular diuretics are presumed to remove excess circulating fluid, decrease cerebrospinal fluid production, and possibly have some positive effect on glial cells.

Another agent that has been used effectively is glycerol. This may be administered orally or intravenously. The theoretical advantage of glycerol is that it is metabolized by the liver rather than being excreted entirely by the kidney. It thus not only contributes to the patient's caloric intake but also has a less severe dehydrating effect. Glycerol administered through a nasogastric tube is rapidly absorbed and has an effect within 30 minutes. However, there is evidence that barbiturates may impair the absorption of glycerol from the gastrointestinal tract, and glycerol is therefore not recommended if barbiturate therapy is being used (94).

### Steroids

Studies using both low and high dose steroids have shown no benefit in terms of improving mortality or morbidity (30, 37, 49, 69). Steroids are also thought to prolong the metabolic abnormalities associated with the initial phase of head injury, and predispose to a higher incidence of gastric haemorrhage and hyperglycaemia (37, 49).

### Barbiturates

Barbiturate-induced coma has been advocated to treat intracranial hypertension when other methods fail. Pentobarbital may have less effect on systemic arterial pressure but also has less effect on intracranial pressure. Because of its effect on blood pressure, the loading dose of pentobarbital should preferably be given slowly over a period of 1 hour. This initial dose should be 10 to 15 mg per kg of body weight, and the blood level is maintained at approximately 3 to 5 mg per dl. This may be maintained by a continuous infusion of 1 to 2 mg per kg per hour (94).

Barbiturates are thought to have a twofold beneficial effect: reduction of intracranial pressure as a result of decreased vascularity, and reduction of cerebral tissue metabolism and substrate requirements (94). Marshall has claimed that high dose barbiturates have improved the outcome after severe head injury (89). Shapiro and Marmarou found a greater improvement in the pressure - volume index in patients who received barbiturates and mannitol than in those who received mannitol alone (127).

### Fluid Balance and Metabolism

Two thirds of normal daily maintenance volume is given using half strength dextrose in normal saline. Hypo-osmolar fluids are avoided and an effort is made to maintain serum osmolality around 300 mOsm. Routine dehydration for head injuries having been recommended in many early texts, has proven to be an unnecessary and even dangerous procedure (60).

Children will frequently develop inappropriate ADH secretion in the first 24 - 48 hours after head injury. The first indication may be a sudden dramatic fall in serum sodium over hours with elevation of intracranial pressure, decreased urine output, high specific gravity and falling Pa02. Even with early fluid restriction, this complication may persist and frequent electrolyte and serum and urine osmolality determinations are required over the first 48 hours. Since hourly urine outputs are part of routine monitoring, a falling urine output should be the earliest alerting factor. If shock requiring vigorous fluid replacement has been present or multiple trauma is present with significant third space loss, it can be difficult to clinically separate whether the falling urine output is due to too little or too much circulating fluid.

Arterial blood gas analysis may show some reduction in Pa02 due to fluid overload and incipient pulmonary oedema, "shock lung" or neurogenic pulmonary oedema. If lung perfusion is inadequate because of decreased circulating volume, a drop in Pa02 occurs because of ventilation perfusion mismatch in the lungs. Serum and urine osmolalities and electrolytes should differentiate between hypo- and hypervolaemia, but it may be difficult to differentiate between hypervolaemia due to simple fluid overload and hypervolaemia due to inappropriate ADH secretion. If chemical studies do not clearly delineate the fluid and circulating volume state, it may be necessary to insert a central venous line or Swan-Ganz catheter to measure the state of the circulation. In children with fluid and circulatory problems in whom intracranial pressure is elevated, the early insertion of a central line or Swan-Ganz catheter can be an invaluable aid to fluid management. When the syndrome of inappropriate ADH has been diagnosed, fluid restriction is the most effective form of management with the replacement of insensible losses only.

Diabetes insipidus (DI) may be seen following head trauma. It occurs as a result of two separate mechanisms. In younger children between birth and 4 years of age, DI occurs in association with basilar skull fractures which cross the pituitary fossa. This is likely to either be due to injury to the pituitary stalk or to the gland itself. This lesion may be associated with optic nerve or chiasm injury. It is possible for stalk injury to occur purely as a result of acceleration / deceleration injury and vascular infarction of the pituitary without actual skull fracture. The onset of DI is usually 24 hours or more post injury. While the occurrence of DI signifies a major traumatic insult, it is reversible and recovery is possible. The second situation in which DI

occurs is in patients with cerebral death and intracranial pressure equal to systemic arterial pressure. Pituitary ischaemic infarction occurs followed by DI. The treatment of DI in the former situation can be quite difficult. If large quantities of fluid lost in the urine are replaced with dextrose-containing solution, marked elevation of serum glucose can occur, particularly if steroids have been used. This may lead to severe hyperosmolality and renal failure. The osmotic shifts produce marked elevation of intracranial pressure, even if the glucose levels have been lowered by using insulin. When fluid replacement exceeds 2 - 2.5 maintenance volumes, it is safer to use vasopressin. This can be given by slow intravenous infusion, bolus intravenous doses, or nasal application. The use of a vasopressin infusion obviates the wide swings in serum sodium and osmolality that can be induced by single injections of vasopressin (17).

During the first 24 - 72 hours after head trauma (even in the absence of abdominal trauma) gastro-intestinal stasis is common. Enteral feeding may be commenced after 48 hours via a nasogastric tube. One has to be alert for the development of diarrhoea because of the high fat content of feeds given by this route (60).

**Seizures**

Some controversy remains about the indications for anti-convulsant therapy in children with severe head injuries. The argument for anti-convulsant therapy is that a seizure may cause an elevation in ICP, and if ICP is already high, this further rise may have severe consequences. If anti-convulsant medication is not routinely given to children (as is the case in many units) the indications for the use of anti-convulsants are: (1) recurrent seizures, (2) seizures after the first hour post trauma, (3) evidence of

cerebral laceration at surgery, (4) evidence of severe cortical contusion on CT scan, (5) prior history of epilepsy and current prophylactic medication (17).

In children with a Glasgow Coma Score of 6 or more, phenytoin sodium is the anticonvulsant of choice. In patients with a Glasgow Coma Score of 5 or less, phenobarbitone is usually given as such patients are likely to require barbiturates to control intracranial pressure (17). If the patient develops status epilepticus, the addition of sodium pentothal may be necessary.

## *REHABILITATION*

In the last decade, emergency medical services and short term care facilities have improved their ability to care for the injured child. Because of these improvements, more children survive trauma despite severe nervous system injuries and multiple system injuries. Many of these children will suffer permanent disabilities. However, disabilities can be minimized through attention to rehabilitation needs during the early care phase and throughout the child's return to life in the community (119).

Rehabilitation is concerned with restoration of function to the highest level permitted by an organic deficit or deficits. When permanent damage to the central nervous system does not allow complete recovery, therapeutic efforts are concentrated on developing appropriate methods of functional substitution (100).

The goals of rehabilitation change with the passage of time as signs and symptoms evolve from the initial stage of head trauma through successive phases of recovery or chronic disability. Although each stage calls for different strategies of intervention, from an overall viewpoint the purpose of treatment is four-fold: (1) to avert

preventable complications which arise as a result of immobilization, disuse and neurologic dysfunction and which can delay or interfere with maximal utilization of eventual functional return, (2) to augment the use of abilities which are regained as the result of recovery of the central nervous system, (3) to train the patient in methods of adaptive compensation for impaired or lost function, and (4) to alleviate the effect of chronic disability on growth and development (100).

Only about 10% of persons with new disabilities have access to rehabilitation facilities. An integral system of care is needed in which injured children are evaluated during their short -term care phase, their disabilities are defined and quantified, and a rehabilitation plan is developed to gain maximum levels of function (119).

# *CHAPTER 6*

# OUTCOME FOLLOWING HEAD TRAUMA IN CHILDREN

## *INTRODUCTION*

Outcome prediction is of great importance in the early management of severe head injury (15). In South Africa, availability of trained staff and intensive care facilities are limited because of these circumstances it is even more important to identify patients with severe head injuries who have the potential for a good functional outcome, and who would benefit from such limited resources.

The severity of head injury may be measured by a combination of the neurological examination, the CT scan, the monitoring of intracranial pressure in selected cases, and an understanding of the mechanism of the injury involved. The outcome in any individual child will be dependent on all of these plus a factor reflecting the original potential of the child. In children the brain is a developing organ and its potential is unknown. What may be accepted as a good result after injury may still represent a significant loss of function for the child had the injury not occurred. Cognitive function may also appear more disturbed because one is looking mainly at the ability to acquire new information as opposed to the situation in the adult where one is comparing the recovery process of information already learned. For each of the pathological injuries that may occur following head injury, the outcome may be quite different, despite a uniform initial clinical picture. This most probably reflects the role of primary as opposed to secondary brain injury. For example, a child with a

poor Glasgow Coma Score and an extradural haematoma may have a much better result than one with a similar score following diffuse impact injury (18).

Soon after injury, brain damage may well appear more serious than it actually is, owing to extracranial factors such as shock, hypoxia and associated injuries. It is wise, therefore, to defer any prognostic statement until at least 6 hours after injury when resuscitation is complete and by which time many of the more severely brain-damaged patients will already have died. Early prediction often tends to be unduly optimistic because complications may occur in patients whose outlook initially appeared hopeful. For this reason a bad outlook will always be more reliably predicted than a good one (67). In adults the Glasgow Outcome Scale has been developed to provide an objective measurement of physical and mental handicap following head injury (66). However, in children, particularly the very young, it is difficult to apply such a scale as their development is still in its early stages and the pre-injury potential of the child is not known.

It has been believed for many years that children and infants tolerate head injuries better than adults (71, 88, 95). Death rates for childr9n with head injuries range from 8 - 33% which is less than the 36 - 49% for adults (7, 8, 15, 72, 81, 86, 88, 90, 105, 107, 136). Severe disability in paediatric patients surviving a severe head injury has been quoted as 6 - 9% (15, 72). However, most studies have compared the entire paediatric age group with adults and no distinction has been made between infants and older children. Also, the outcome is often not assessed in terms of the neuropsychological sequela which may be devastating in a child, and Brink identified the age group of less than 10 years as having a worse intellectual outcome for a given coma duration

(71). This has been supported by Levin who has shown that the paediatric brain demonstrates greater resilience to focal damage but decreased resilience to diffuse damage (83).

## *CLINICAL PREDICTORS*

### Age

Prognosis is worst at the extremes of age. In adults, older patients with severe head injuries have a poorer prognosis (68, 138). Infants appear to have a poorer outcome than older children (88, 111). The reason for this is unknown although the age demarcation of good and poor outcomes in children appear to correlate with fontanelle closure. One of the reasons for poorer outcomes in infants may be related to the higher incidence of subdural haematomas in this group. Suture closure may protect against the formation of a subdural haematoma, but the mechanism is not clear. Other possible reasons for the higher mortality in infants with head injuries may be related to either greater shearing forces in the less myelinated immature brain, or to differences in the ability to autoregulate vascular perfusion.

### Depth and Length of Coma

In numerous adult studies the Glasgow Coma Scale has been found to be a predictor of outcome with an accuracy of over 80% (14, 26, 27, 42, 75, 90, 98, 103, 105, 125). The post-resuscitation score is much more closely linked to outcome than the initial score; it has, however, not been possible to define a Coma Score that would always predict non-survival (42). Choi developed a prediction tree for severely head injured patients using a recursive splitting technique which divides patients into subgroups

with varying degrees of risk. The best prognostic factors were age, motor score and pupillary response (42). The Glasgow Coma Scale is therefore a method of immediate assessment of the severity of injury and a predictor of immediate and long term outcome. Although the score is often influenced by factors other than the head injury, it is nevertheless a very strong indicator of the relative severity of injury and of the likely risk to the patient of either death or permanent disability (75).

The Glasgow Coma Score (or Child Coma Score) has also been found to be an important predictor of outcome in children (21, 50, 64, 72). Bruce stated that children with a score of 3 or 4 were often associated with a poor outcome. Hahn found that 85% of children aged under 36 months and 75% over 36 months with a Child Coma Score of 3 or 4 had a poor outcome (50).

The length of coma is also an important predictor of outcome (72, 82). However, there does not seem to be agreement on the length of coma that correlates with a poor outcome. Heiskanen showed that children in a coma lasting 2 weeks are rarely able to succeed even moderately well at school, and more than half the patients were unable to attend a normal school; even coma lasting a few days may lead to deterioration in mental capacity (57). Brink identified a favourable prognosis for recovery of motor function if duration of coma is less than 3 months (15). Boyer felt that children who remained unconscious for up to 2 months after injury had a good potential for recovery with 27 - 43% achieving independence in the tasks of daily living (13).

## Focal Neurological Signs

The most reliable focal signs in the prediction of outcome following severe head injury include pupillary responses, abnormal motor patterns, and spontaneous and reflex eye movements, including the oculocephalic eye reflex (14, 25, 26, 27, 45, 81, 98, 103, 125). The combination of abnormal motor response, impaired or absent oculocephalic responses, and absence of the pupillary light reaction has a strongly adverse prognostic significance and 76% of these patients die (7).

In children the oculovestibular reflex and bilateral fixed dilated pupils have the most reliable correlation with poor outcome (50). Bruce found the worst prognostic sign to be flaccidity, with 33% of these patients dying or remaining in a persistent vegetative state (21). In infants the ocular examination appears to be the most reliable. Raimondi found that all the infants in his series who had bilateral nonreactive pupils and impaired or absent caloric reflexes, died. He also found hemiparesis to be the most important lateralizing sign associated with poor outcome (111).

## Associated Injury

The presence of multiple trauma involving the abdomen or chest is associated with significantly higher incidence of adverse outcomes (12). In a study of children who sustained multiple trauma with an associated head injury, a poor outcome was found to be 2.5 times as common as in those with isolated head injury (93). The combination of severe head injury and multiple trauma predisposes the patient to additional blood loss, with possible development of shock and further compromise of pulmonary and immune function. Patients with severe chest injury are particularly at risk, inasmuch as controlled ventilation is more difficult and less effective in that

setting. Thus shock, hypoxaemia, sepsis or combinations of these factors are more likely to develop and are possibly more refractory to critical care when severe neurologic and multiple trauma are combined (92, 93, 108, 109, 125). An alternative way of looking at this is that 86% of all deaths following multiple trauma are due to head injury (139). The Paediatric Trauma Score and Innsbruck Coma Scale are both methods that have been developed for predicting survival following head injury associated with multiple trauma (6, 112, 134).

## *PATHOLOGY AND RADIOLOGY*

### Intracranial Pressure

Raised intracranial pressure has been shown by a number of researchers to be associated with increased mortality (50, 97, 107). Intracranial pressure greater than 40 mm Hg in children with Child Coma Scores of 3-5 is invariably fatal. However, over 60% of children with intracranial pressure less than this may have a good outcome (97). Miller found that even moderately raised intracranial pressure (> 20 mm Hg) predicted a higher morbidity in over 50% of patients if an intracranial mass lesion or diffuse brain injury was present (97).

### Mass Lesions

There appears to be general agreement that patients with intracranial mass lesions, with the exception of extradural haematomas, have a poorer outcome than those with diffuse brain injury (7, 81, 105). Patients with mass lesions tend to be worse neurologically than those with diffuse brain injury, with a significantly higher percentage of patients showing impairment of pupillary and oculomotor function and

abnormal motor responses. Also the mortality rates quoted in reports dealing with intracranial mass lesions are all high, ranging from 45% to 90% (7). It appears that one of the main reasons that infants have a poorer outcome than older children is the high incidence of subdural haematomas in the younger group. The answer may be found, at least in part, in the association between fontanelle closure and marked fall in frequency of subdural haematoma. Only 4.9% of the closed-fontanelle population, as opposed to 32% of the open-fontanelle population, had a subdural haematoma (111).

### Diffuse Cerebral Swelling

Diffuse brain swelling occurs approximately twice as commonly in children as in adults. A higher mortality rate has been found in these children ( 53% ), being three times that of the children without diffuse brain swelling (1). Diffuse brain swelling, characterized on CT scan by absent or slit-ventricles and cisterns, is the most common finding in children with acute brain injury, and has a good prognosis. However, diffuse cerebral swelling in association with features of diffuse axonal injury has a very poor prognosis (31).

### CT Scan

The importance of the classification of brain injury pathology using CT scan (and more recently Magnetic Resonance Imaging) for predicting outcome, has become established with different CT scan appearances being associated with different outcomes (91, 137). CT scan features particularly associated with a poor outcome include: 1) obliteration of the perimesencephalic cisterns, 2) subarachnoid haemorrhage, 3) diffuse axonal injury, and 4) acute subdural haematoma (64, 125).

However, CT scan criteria alone are are not nearly as accurate prognostically as when they are used together with clinical criteria (103).

## *Metabolic and Electrophysiological*

### Evoked Potentials and Electroencephalogram

The prognostic use of evoked potential measurement has been studied in several populations. By the end of the 1980's evoked potential monitoring of post-traumatic coma suggested that evoked potentials had similar or slightly better predictive value than clinical scales, were resistant to the effects of depressant drugs, and that somatosensory potentials were more useful than brainstem auditory evoked potentials in predicting outcome (42). In recent studies which compared evoked potentials with clinical signs in predicting outcome following severe brain injury, it has been shown that evoked potentials provide useful prognostic information in sedated or paralyzed patients. However, when the neurological examination is feasible, the benefits of evoked potentials do not justify the effort in data collection (42).

In a study on the use of EEG in predicting outcome in severely head injured patients, Rae-Grant proposed a new scale classifying EEG findings into dichotomous categories. He found that the reliability of the new scale was high in predicting outcome at discharge as measured by the Glasgow Outcome Scale (137).

### Cerebral Blood Flow

A number of studies have correlated cerebral blood flow and arterio-venous oxygen difference with Glasgow Coma Scale and outcome. Bouma found a correlation between cerebral blood flow and motor response in the first 8 hours post injury.

Arterio-venous oxygen was highest soon after admission and declined over time. Cerebral blood flow was also found to be associated with Glasgow Outcome Scale at follow-up (42). Robertson found mortality to be highest in patients with low cerebral blood flow. No difference was found between normal and high cerebral blood flow groups. Good recovery was highest in patients with high cerebral blood flow and lowest in the low cerebral blood flow group. Low cerebral blood flow was related to outcome independent of age, Glasgow Coma Scale, cerebral metabolic rate or cerebral perfusion (42).

### Metabolic Changes

Researchers have found a number of metabolic changes in patients who have sustained severe head injuries, but the relevance of these findings to the clinical situation has not been substantiated.

Lam found that glucose levels were higher in comatose patients and were associated with poorer outcomes when measured 10 days post injury (42).

Hamill has shown circulating catecholamines to be excellent endogenous and readily identifiable markers that appear to reflect the extent of brain injury and that may predict the likelihood of recovery (54).

# *CHAPTER 7*

# A RETROSPECTIVE STUDY OF SEVERELY HEAD INJURED CHILDREN ADMITTED TO RED CROSS CHILDREN'S HOSPITAL

## *INTRODUCTION*

Severe head injury is an unnecessary cause of morbidity and mortality in children. The treatment of severe head injury in children is, at best, far from satisfactory and permanent disability or death is common (39). The result is often immense human suffering and financial cost.

Much has been documented on the epidemiology, pathogenesis, clinical picture, treatment and outcome of head injury in children, but this work has been largely confined to descriptions of circumstances in developed countries. Local factors often vary in a developing country such as South Africa and it is worthwhile comparing the situation in this country with those in developed countries to identify the differences in aetiology, prognosis and outcome and to evaluate the quality of our management. It is also important to identify patients who have the potential to do well, and would benefit from utilization of resources.

There have been a number of studies on head injuries in South African children (39, 74, 77, 135), but none has dealt specifically with severe head injuries. The aim of this

study is a descriptive analysis of aetiology, pathophysiology and factors influencing outcome in severely head injured children admitted to Red Cross Children's Hospital.

## *MATERIAL AND METHODS*

This retrospective study was limited to severely head injured children under 14 years of age admitted to Red Cross War Memorial Children's Hospital (RCCH) over the 4 year period from January 1990 to December 1993. All head injuries were managed by the Trauma Unit and Department of Neurosurgery, University of Cape Town, at this hospital. During the period under study, 102 severely head injured children were admitted to the hospital.

The term "severe head injury" was applied to patients with a Glasgow Coma Scale of 8 or less, following resuscitation. For children less than 3 years of age the initial assessment was made using the Child's Coma Scale and a severe head injury was defined as a score less than 6. However, once the child had an endotracheal tube inserted the Glasgow Coma Scale could be accurately applied to all the children with a severe head injury. A potential criticism of applying the Glasgow Coma Scale to young children is that their speech maturation may prohibit interpretation of examiner's verbal commands. However, this potential restriction is inapplicable to children with "severe" head trauma, for by definition their Glasgow Coma Scale could be as high as 9 (E:2+M:5+V:2) and not require any verbal skills (64). Once an endotracheal tube has been inserted the vocal score is effectively excluded from the assessment with the Glasgow Coma Score being followed by a "T" to indicate the artificial airway.

All children with severe head injuries underwent resuscitation in the trauma unit and once stabilized an emergency CT scan was performed. Patients with operative lesions underwent immediate surgery and then were transferred to the intensive care unit if ventilation was required. Children who did not have surgically amenable lesions on CT scan were transferred directly to the intensive care unit. Management in the intensive care unit consisted of ventilation, clinical monitoring, measurement of arterial oxygen and carbon dioxide, fluid restriction and anti-convulsant therapy. Repeat CT scan was undertaken in all patients who did not show a significant improvement within 24 hours.

Outcome was measured using the Glasgow Outcome Scale (66). In this study a good outcome was defined as a normal neurological state or a disability, whether the child was independent or not. A poor outcome was defined as death or a persistent vegetative state. In adult studies a good outcome is usually defined as a normal neurological state or a situation in which the patient is independent despite any degree of neurological deficit (66). In this series it was decided to include in the "good" result category those patients with a persistent neurological deficit and who were not independent for the following reasons: (1) children are not independent of parental care, and thus it becomes more difficult to assess whether they will be independent when they mature, (2) children are neurologically immature and thus there is considerable potential for development even after severe head injury. It is also difficult to predict the exact degree of recovery that may occur in the months immediately following the injury, (3) the potential for children to recover from a severe head injury may be greater than that of adults (20), and (4) the recovery process

may be extended over an indefinite period of time (76) . With this approach, it is accepted that there will be a number of cases who do not make a good recovery as defined by the Glasgow Outcome Scale in the long term, but the children who have the potential to improve to an adequate level of function by the time they reach adulthood are not treated as patients doomed to a poor quality of life.

The results were then analyzed retrospectively.

## *RESULTS*

There were 102 patients who satisfied the criteria for severe head injury. This included all patients who arrived at the Red Cross War Memorial Children's Hospital with a severe head injury, including those who did not survive resuscitation. The initial assessment by neurosurgical staff was done on average 2.8 hours post injury. The average length of outpatient follow-up was 7.5 months, but if fatalities were excluded, then the average length of follow-up was 17 months.

### Age and Sex

There were 57 males and 45 females who sustained severe head injuries. The children were divided into groups according to age: 0-1 year; >1-3 years; >3-7 years; >7-13 years. The older childen of school-going age made up the majority of patients (fig. 7).

### Aetiology

The majority (83%) of children with severe head injuries treated at our hospital were the victims of pedestrian motor vehicle accidents. Falls accounted for 11%, and the remaining 6% were victims of passenger motor vehicle accidents, bicycle accidents and assaults (fig 8).

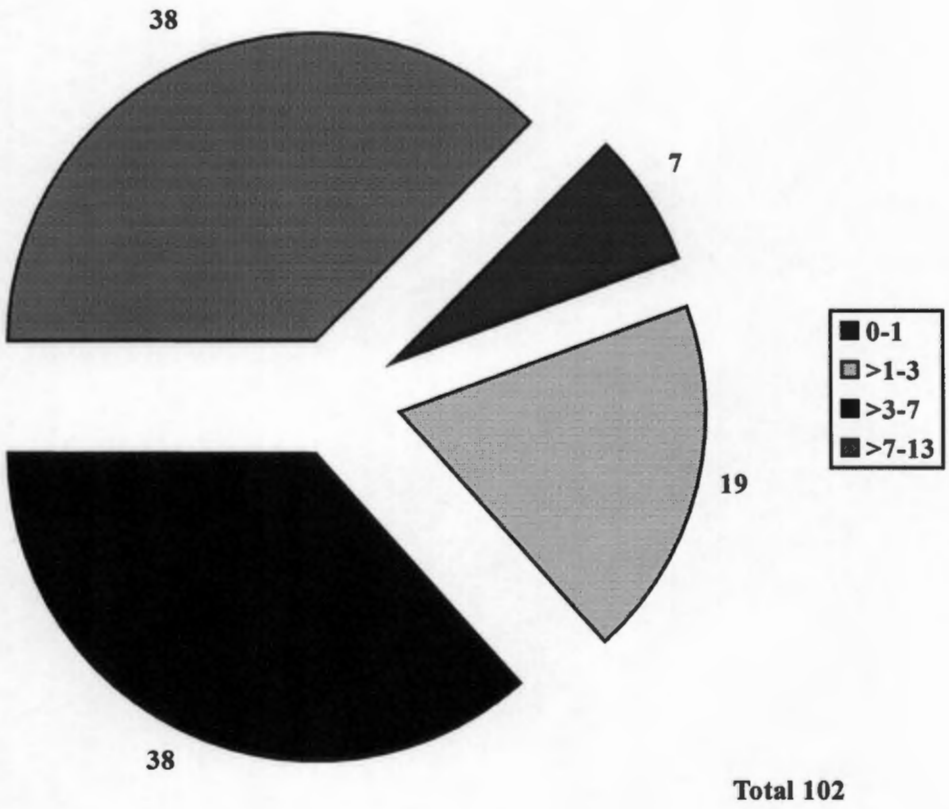



**Fig 7: Age distribution.**

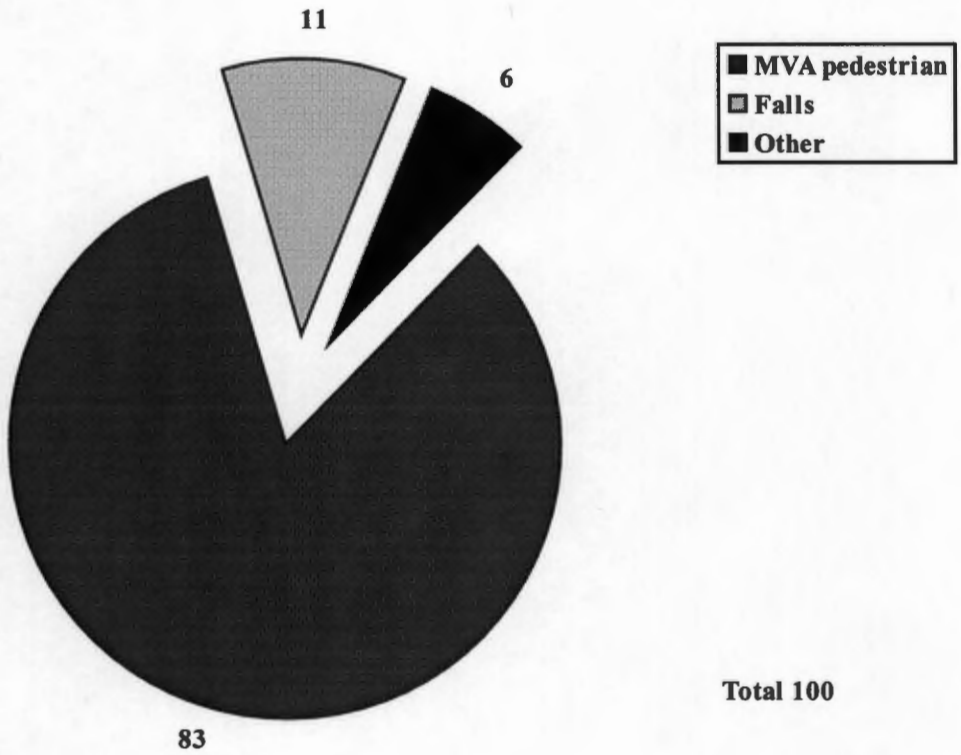



**Fig 8: Mechanism of injury.**

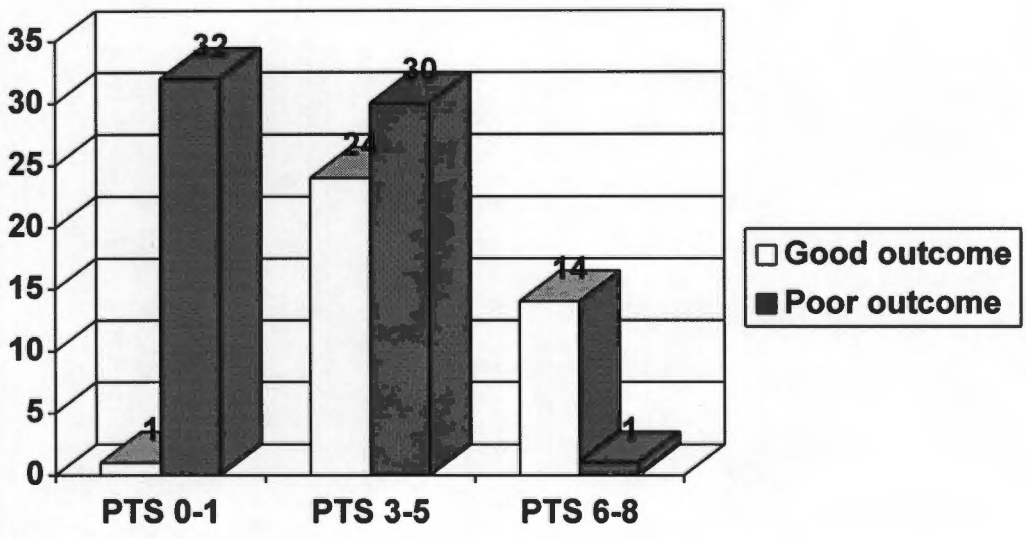



**Fig 9 : Paediatric trauma score (PTS) compared with neurological outcome as defined by the Glasgow outcome score (page 74).**

**Associated Injuries**

There were 37 patients who sustained an injury to at least one other organ system in addition to the head injury. Seventeen of these patients had multiple injuries involving 3 or more organ systems; musculoskeletal injuries occurred in 12 children; and abdominal and chest injuries were found in 4 cases each. The outcome of patients with associated injuries was poor in 26 cases (70%). The Paediatric Trauma Score (134) was also applied to all the patients and the outcome compared with the admission GCS (see fig. 9 ).

## Operative Procedures

Nine children underwent neurosurgical operations, and 6 of these were for intracranial haematomas (fig. 10). The 4 patients who survived following surgery had sustained extradural haematomas. Children who died had a subdural haematoma (1), extradural haematoma (1), a depressed skull fracture in association with a severe head injury (1) and 2 missile injuries (fig. 11).

## Outcome

Of the 102 patients reaching hospital, 58 (57%) died. Using the Glasgow Outcome Scale (GOS) to assess outcome, 66 patients (65%) had a poor outcome and 36 children (35%) a good outcome. However, in children under 3 years of age there was a worse outcome with 18 (75%) of 24 patients having died.

## Outcome Vs Glasgow Coma Scale

The outcome of the patients based on their Glasgow Coma Scale (GCS) following resuscitation was analyzed and the patients were divided into 2 groups: those with a GCS of 5-8 and those with a GCS of less than 5.

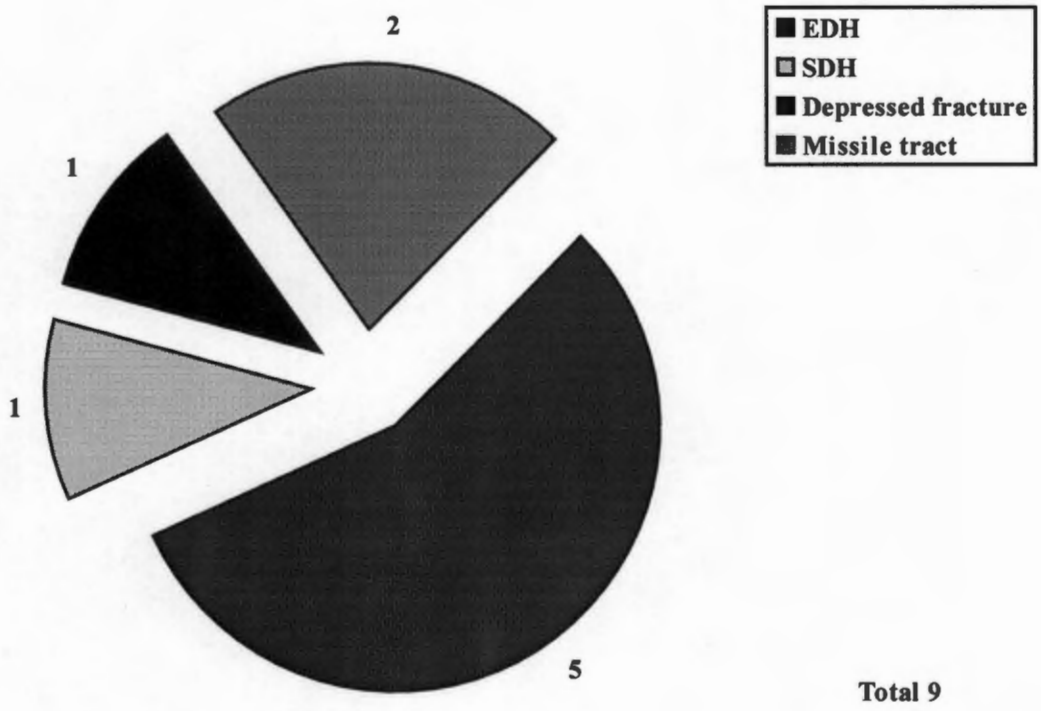



**Fig 10: Operative procedures.**

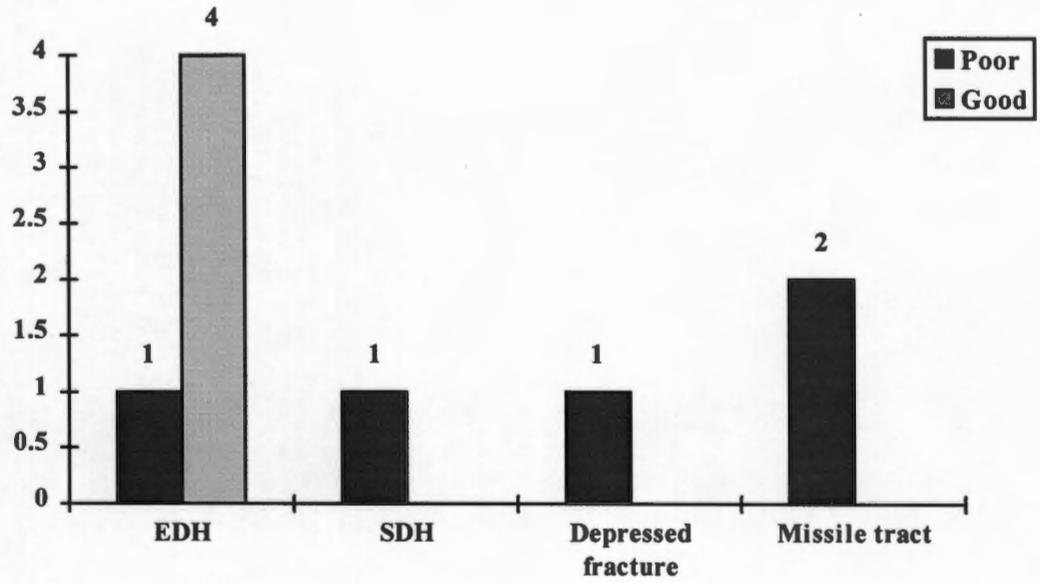



**Fig 11: Outcome of operative procedures.**

There were 40 children with a GCS of less than 5 following resuscitation. Twenty of these patients died prior to a CT scan being done; 12 of them were isolated head injuries and 8 had sustained polytrauma. Of 20 patients who underwent a CT scan, 17 were shown to have diffuse cerebral swelling and 3 had features of diffuse axonal injury (fig 12). All the children who had a GCS of less than 5 following resuscitation died (100%). The single patient from this group with a good outcome had 2 seizures immediately prior to examination and thus was effectively excluded as the GCS rapidly improved post-ictally.

Sixty-two children had a GCS of 5 or more following resuscitation and 60 underwent a CT scan. Twenty-nine of these patients had diffuse cerebral swelling on CT scan and all the intracranial haematomas in this series (9) occurred in this group (fig. 13). A good outcome was obtained in 38 patients (62%).

## Radiology

The pathology as seen on the CT scan was compared between the group of patients who had a good outcome and those who had a poor outcome. Of the 36 patients who had a good outcome, 12 (33%) had diffuse swelling on the CT scan. However, in the 44 patients who had a poor outcome but survived to undergo a CT scan, 34 (77%) had diffuse cerebral swelling (fig. 14). Therefore diffuse cerebral swelling was found in both the poor and good outcome group but it was twice as common in the patients who had a poor outcome.

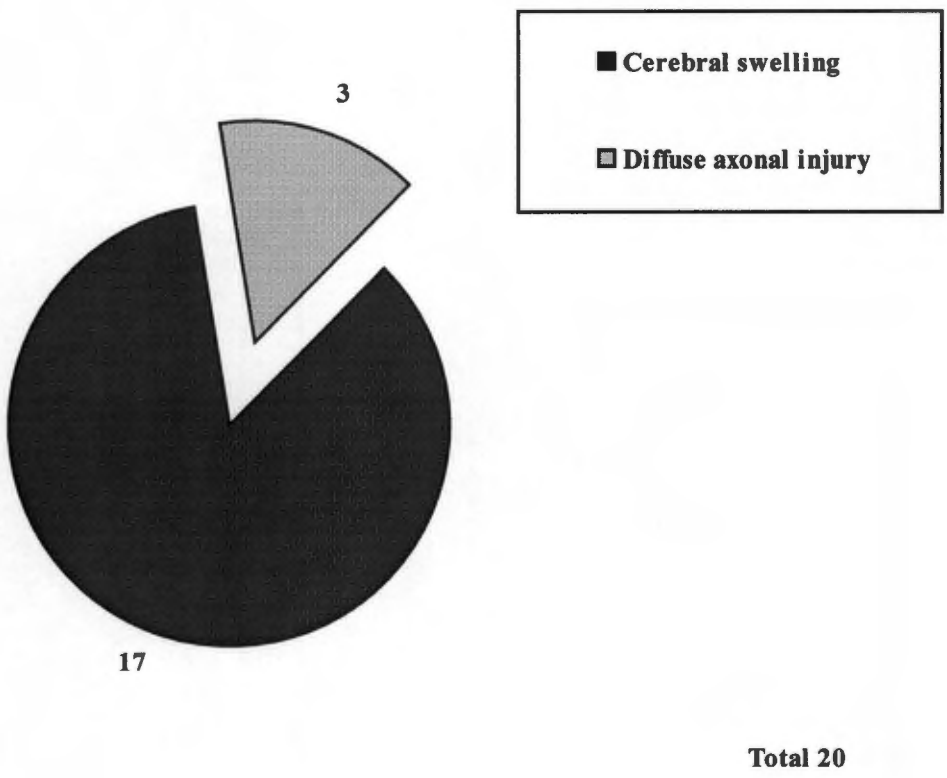
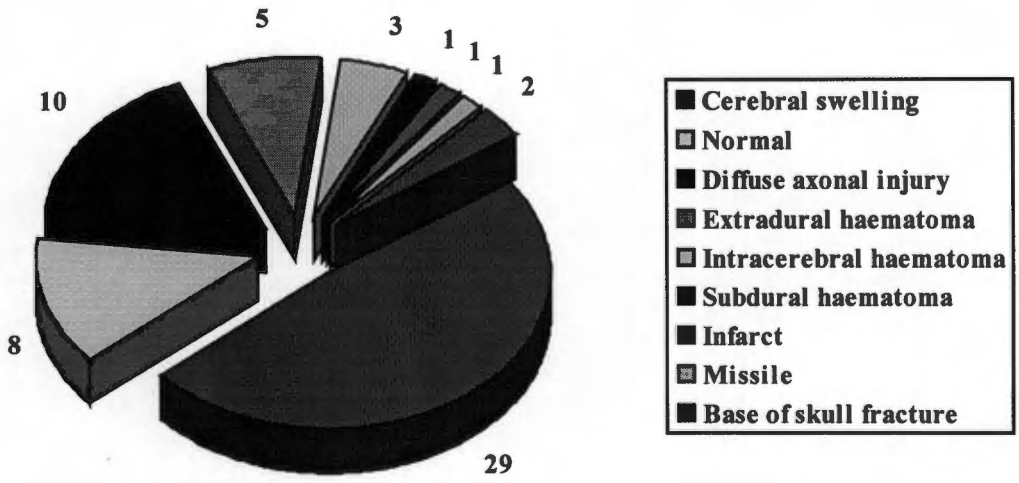



**Fig 12: CT-scan findings in children with a Glasgow Coma Scale less than 5.**




**Total 60**

**Fig 13: CT-scan findings in children with a Glasgow Coma Scale of 5 and above.**

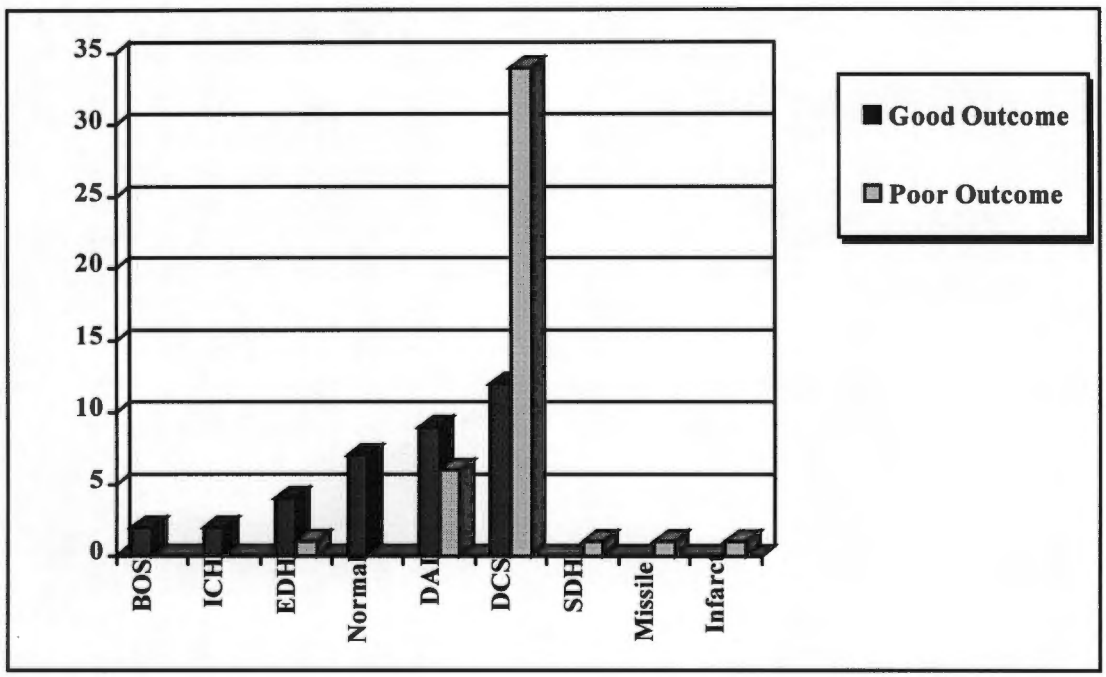



**Fig 14: CT-scan vs outcome of children.**

# *SUMMARY OF RESULTS*

The results of this study of severe head injuries in children can be summarized as follows:

1. Severe head injuries occur more commonly in the older child

2. The is a slightly higher incidence of males than females

3. The majority of children with severe head injuries are victims of pedestrian road traffic collisions (83%)

4. There is a very high mortality (57%) and poor outcome in 65% of survivors

5. Intracranial haematomas are uncommon (9%)

6. Factors associated with a poor prognosis are

    a) associated extracranial injury and a PTS of 5 or less

    b) age of less than 3 years

    c) GCS less than 5 following resuscitation

7. Diffuse cerebral swelling is found in 77% of patients with a poor outcome, but is also found in one third of children with a good outcome.

# *CHAPTER 8*

# DISCUSSION

## *INTRODUCTION*

This chapter is limited to a discussion of points that were thought to be of importance in the Red Cross Children's Hospital study of severe head injuries in children, and will examine how local conditions and factors compare to those highlighted in similar studies. This will hopefully offer a better understanding of the clinical situation in Cape Town, and also identify areas where further research would be worthwhile.

## *AGE AND SEX*

There was a small predominance of boys over girls with severe head injuries, and this is in keeping with the majority of studies (2, 59, 70, 78, 132). That the majority of severely head injured children were older is not surprising. The older child is more independent and is beginning to explore the world without the constant supervision of adults, resulting in an increased time spent away from protective custody in a hostile environment that he has not yet fully learned to judge or cope with.

## *MECHANISM OF INJURY*

In developed countries falls are the most common cause of head injuries (50, 59, 115), although in the poorer socioeconomic areas pedestrian motor-vehicle injuries may predominate (115).

In Cape Town 83% of severe head injuries were caused by pedestrian motor-vehicle accidents, and this is thought largely to be a socioeconomic phenomenon with the majority of patients coming from the poor areas (135). The specific reasons are poor parental supervision often due to both parents working and care being delegated to an older child (2); lack of play areas such as parks and public open spaces; fewer traffic and pedestrian control devices; higher traffic volumes and density; lack of pavements and street lighting; and lack of education in road safety.

In Cape Town the majority of head injuries are sustained in Coloured and Black children and this relates to the poorer socioeconomic circumstances of these two groups (39, 77).

## *MORTALITY AND MORBIDITY*

The mortality rate in this study was 59%, and altogether 68% of the children had a poor outcome. There is no consensus in the literature regarding the expected mortality for severely head injured children with reported mortality ranging from 6-60%, although the most frequently reported mortality statistics occur within a range of 25-30% (85). Luerssen, in a review of outcome studies in children who had sustained severe head injuries put forward a number of reasons for this wide variation (85). There is a variation in the numbers of patients available for analysis and the smaller the number studied the greater the likelihood that differences in mortality are due to random variation. Furthermore, paediatric head injuries are a relatively rare occurrence and large numbers are usually not available from a single institution unless the study is conducted over a long period of time during which diagnostic modalities

and treatment may have changed. The majority of studies are conducted on all children with head injury and do not differentiate between mild and severe head injuries, therefore the mortality is often biased toward the patients with a better outcome. In addition, there is variation in the definition of what constitutes a severe head injury, with some authors defining a GCS of 3-7, and others 3-8 as a severe head injury resulting in variations in mortality. Another factor is the "6 hour rule". The use of the 6 hour rule to assess severity after 6 hours post admission, results in lower mortality as it excludes early deaths. The variation in the age range of reported head injury is also important as the different age groups even within the paediatric population will have different outcomes and a large number of patients within a specific age group will bias the results towards that of the dominant age group.

Within the Red Cross Children's Hospital study there are a number of possible reasons which may account for the high mortality rate. This study includes only those children who sustained a severe head injury and consequently is going to be biased toward a poor outcome. In Cape Town the vast majority of severe head injuries are admitted directly to the trauma unit at Red Cross Children's Hospital and thus there is no triage of patients at other hospitals and selection of patients with a more favourable prognosis. All patients who arrived at this hospital with a severe head injury were included in the study no matter what the length of survival or whether they survived the resuscitation. Consequently a large number of patients were included with head injury so severe that survival was impossible. Another factor thought to be important is that childhood head injuries tend to occur in daylight when transportation of patients to hospital is more rapid; this results in some children reaching hospital

despite having fatal and unsalvagable head injuries who would otherwise have been declared dead at the site of the accident. The fact that pedestrian road traffic accidents are responsible for 83% of severe head injuries in this region is another reason for the high mortality rate as the forces involved are considerable and there is a high incidence of associated injuries.

## *INTRACRANIAL HAEMATOMAS*

There were 6 patients with intracranial haematomas in this study. This is in keeping with the findings of a low incidence of intracranial haematomas occurring in children as compared with adults described in the literature (21, 51, 111). In children extradural haematomas have a frequency of 2-4% (28, 41, 99, 129, 110); in this series 5 patients (5%) had an extradural haematoma. Four of the children with an extradural haematoma had a good outcome and one patient died; the quoted mortality rate in published series of extradural haematomas varies from 0- 17% (41, 110) and the outcome in our very small number of cases is similar. A single patient sustained a subdural haematoma and this child died. This is in keeping with both the rarity and poor prognosis of childhood subdural haematomas which seem to be largely confined to children under 1 year of age (111), with the associated mortality rate in this age group being similar to that in adults (86).

## *DIFFUSE CEREBRAL SWELLING*

Diffuse cerebral swelling on CT scan was present in both children who had a good outcome (33%) as well as those with a poor outcome (77%), although it was more frequently present in the latter group. The fact that one third of patients in the good

outcome group had cerebral swelling suggests that cerebral swelling cannot be used in isolation to predict outcome, Bruce has stated that diffuse cerebral swelling may be a normal pathophysiological response to trauma in children and may be associated with a good prognosis. This was true in 33% of the patients with a good outcome in this series. Aldrich found that the incidence of cerebral swelling was twice as common in children as in adults (31) and our results confirm that diffuse cerebral swelling is a common finding in children with a severe head injury. In addition Aldrich found that the mortality rate of children with diffuse cerebral swelling was three times that of children without diffuse cerebral swelling (31); in our series diffuse cerebral swelling also occurred in three times more patients with a poor outcome.

What may be of significance is that 10 of the 34 patients with diffuse cerebral swelling also had radiological features of diffuse axonal injury, whereas none in the good outcome group did. Cordobes in his series, showed that patients with features of both diffuse axonal injury and diffuse cerebral swelling presented with deep and prolonged coma and only 8% made a good outcome, while more than 50% died (31). The local results concur with the literature in that there appear to be 2 distinct groups of patients with diffuse cerebral swelling: in the one group, diffuse cerebral swelling appears to be a "normal" pathophysiological response of the paediatric brain to trauma and is associated with a favourable prognosis; the second group of children have sustained a shearing component to their injury as well, resulting in diffuse axonal injury with an associated poor prognosis. It is difficult to distinguish these two groups as diffuse axonal injury is not always clearly demonstrated on CT scan. However Cordobes also found that when using intracranial pressure monitoring, the

children who had diffuse cerebral swelling with a more benign clinical course had intracranial pressure which was normal or only moderately elevated, while patients with diffuse cerebral swelling associated with diffuse axonal injury were in a poor neurological condition with marked intracranial hypertension and an unfavourable outcome (31).

## *OUTCOME*

The factors associated with a poor outcome in this series were: associated injury; age less than 3 years; and a Glasgow Coma Scale of less than 5 following resuscitation.

### Associated Injury

Twenty-six (70%) of the 37 patients who sustained injuries to other organ systems had a poor outcome. Associated injury, particularly patients sustaining multiple trauma, is a well established cause of a poor prognosis in the head injured patient (12, 108, 109, 125, 139), and poor outcome was 2.5 times more common in children who had multiple trauma in association with a head injury (93). The PTS has been found to be an effective predictor of injury severity and mortality (134) and this was true in our study where the lower scores (less than 5) was accompanied by an increased insidence of poor outcome. The increased mortality rate is thought to be related to shock, hypoxaemia and sepsis or a combination of all three resulting in devastating secondary injury to the already compromised brain.

### Age

At Red Cross Children's Hospital 18 (70%) of the 24 children under 3 years of age had a poor prognosis, and all died. It has been believed for many years that children

tolerate head injury better than adults (86, 125). However, it is now apparent that age within the paediatric group is a major factor affecting the mortality rate in head injured patients (87). However, this effect is less apparent in the more severely injured patients who do badly regardless of age.

Bruce and Berger found no correlation between age and outcome in the paediatric age group (9, 21). Humphreys stated that age is a reliable indicator of outcome within the paediatric age group. He found that children less than 5 years old accounted for two thirds of the deaths from severe head injury (64). Raimondi stated that children under 1 year have a poorer outcome than the older child and attributes this to the higher incidence of subdural haematomas in this group, the fact that the fontanelle has not closed and that the brain is still immature and incompletely myelinated (111). Thus the Red Cross Children's Hospital study is in agreement with the general belief that the younger children have the poorest outcome.

### Level of Consciousness

The Glasgow Coma Scale (or Child Coma Score in infants) has been shown to be a good predictor of survival and outcome in both adults and children(14, 21, 26, 27, 42, 50, 64, 72, 75, 90, 98, 103, 105, 125). All the children in this study who had a Glasgow Coma Score of 3 or 4 died (100% mortality).

Bruce found that children with a GCS of 3 or 4 had a poor outcome (21) and Hahn found that children with a Child Coma Score of 3 or 4 had a poor outcome in 85% if they were less than 36 months old, and 75% if they were more than 36 months (50). Humphreys feels that initial GCS is a reliable indicator of outcome with 15 of the 17 deaths from head injury in his series occurring in children with a GCS of 7 or less

(64). These figures are very similar to those of the Red Cross Children's Hospital study.

The effect of an early post-traumatic seizure on the initial assessment of the head injured child must be considered as patients can be assessed to be worse than they really are. In this study there was 1 patient who was noted to have had a post-traumatic seizure and who was initially assessed as having a GCS of 4 but who then rapidly improved. CT scan was normal.

CT scan alone is more a diagnostic tool than a predictor of outcome (103) as is evidenced by the presence of diffuse cerebral swelling in both the poor and good outcome groups in our study. However, when used in combination with clinical data, such as the GCS, the prognostic prediction value is improved (53, 64). This was previously noted by Bruce who showed that CT scan findings of diffuse cerebral swelling were related to the GCS of his patients, and all those patients with a GCS of 8 and more with cerebral swelling made a good recovery (21).

## *CONCLUSION*

Review of the literature and the results of Red Cross Children's Hospital study leaves little doubt that a severe head injury in a child is a situation best avoided. The primary injury sustained at the time of impact is irreversible and management is primarily aimed at preventing secondary injury.

The findings of the Red Cross Children's Hospital study are in general similar to those of other published series. The age and sex distribution, incidence of intracranial haematomas, predictors of outcome and radiological findings are consistent with the

literature. In addition the management of patients is comparable to other centres. However there are 2 factors that differ from well-documented findings. The first is the high mortality rate of 57% which falls within the range of reported mortality but is higher than the reported average of 25-35%. The postulated reasons for this have already been discussed, but the two most important factors are the inclusion of all patients, no matter what the length of survival, and the fact that the majority of severe head injuries in our patients were the result of road traffic accidents where the force of injury is exceedingly high and results in a more severe injury than, for example, a fall. Not only is this a reason for the high mortality rate but it is most importantly one of the factors that can be prevented.

Therefore the most important factor from the Red Cross Children's Hospital study that is likely to make a significant difference to the high toll from severe head injuries in children, is better prevention of pedestrian road traffic accidents. Thus the majority of research in this region should be in prevention of childhood head injury to build on the work of Cumpsty (39) and others. With the data obtained a strategy that goes way beyond the medical management needs to be formulated which takes into account work performed in other centers as well as characteristics peculiar to this region.

There is general agreement that serious injury is the result of factors that place a child in an "at risk" situation, and, with the identification of causative factors, such injury should be amenable to preventative measures (140). The Child Accident Prevention Foundation of Southern Africa, established with the objectives of research, education and soliciting legislative measures, provides us with the opportunity to prevent childhood injury instead of picking the pieces up afterwards.

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