CHAPTER 30
Craniocerebral and Spinal Trauma

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Craniocerebral Trauma

Introduction

Paediatric cranial injuries constitute a major portion of paediatric admissions and are the cause of the greatest number of deaths and chronic disabilities among children. Brain injuries are responsible for 7,000 paediatric deaths per year in the United States.[1] The figures are quite a bit higher in Africa, with various figures being quoted in different regions. Paediatric cranial injuries are a challenge to manage, requiring difficult decisions in a setting of limited resources. As in the developed countries, the socioeconomic impacts of head injury are enormous, including school failure, social maladjustment, and public liability.

Epidemiology

Adult cranial injuries are primarily diseases of young men, with a male-to-female ratio of 3–4:1. The sex ratio disparity is less in children; in all age groups, including infants, the boy-to-girl ratio is about 2 to 1. The great majority of cranial injuries in children are mild (86%).[2] Severe injuries show a bimodal age distribution; first, in infancy, due to a higher incidence of nonaccidental injuries, and second, in adolescence, due to road traffic accidents.[3–5]

Aetiology/Classification

The cause of head injuries based on mechanism of injury is classified into blunt or penetrating.

Blunt

Falls are the most common cause of paediatric blunt cranial injuries. Low-height falls rarely cause significant neurological morbidity. Falls from heights greater than four feet (1.2 meters), and falls from a caretaker's arms, however, may be associated with severe injuries, including contusions and depressed skull fractures.

Although motor vehicle accidents may account for a smaller percentage of all paediatric head injuries, they outweigh all other causes of serious head injury. The trauma may involve children as passengers, or as pedestrians and cyclists being struck by motor vehicles.

Crush injuries usually occur at home from falling objects, such as collapsed buildings, falling tables, televisions, and so on. They are characterised by skull fractures.

Birth injuries occur during delivery. Neonates may suffer cranial injuries such as cephalhaematomas, skull fractures, intracranial haematomas, and even brain injuries.

Penetrating

Penetrating head injuries involve falls unto playing objects such as pencils, nails, or sticks. Increasingly, though, penetrating cranial injuries are being seen from assaults, stab wounds, and gunshot wounds (Figure 30.1). Animal bites and horses' hooves are also common causes.

Inflicted injuries

Inflicted injuries may occur from child abuse, which includes beating, excessive shaking, and striking the head against hard surfaces.

Figure 30.1: Penetrating orbitocranial foreign body.

Classification of Head Injury by Pathology

Head injuries may be focal or diffuse, but both actually coexist to some degree. In severe head injury, diffuse predominates, but focal lesions carry a higher mortality rate.

In a diffuse injury, the alteration of mental status is out of proportion to computed tomography (CT) findings. Rotational acceleration/deceleration forces are usually responsible. Examples include concussion with transient loss of consciousness and diffuse axonal injury characterised by shearing at grey-white matter interfaces.

In focal injuries, scalp lacerations are of special importance when associated with skull fractures because the child is at risk of developing meningitis.[6] Basal skull fractures are suspected in the presence of raccoon eyes, Battles sign, haemotympanum, otorrhea, or rhinorrhea.

Depressed skull fractures (Figure 30.2), also called pond fractures, are the results of focal impacts. Intracranial haematomas, which may include epidural subarachnoid, intraventricular, and intraparenchymal, are also prominent, depending on the severity of the impact energy.

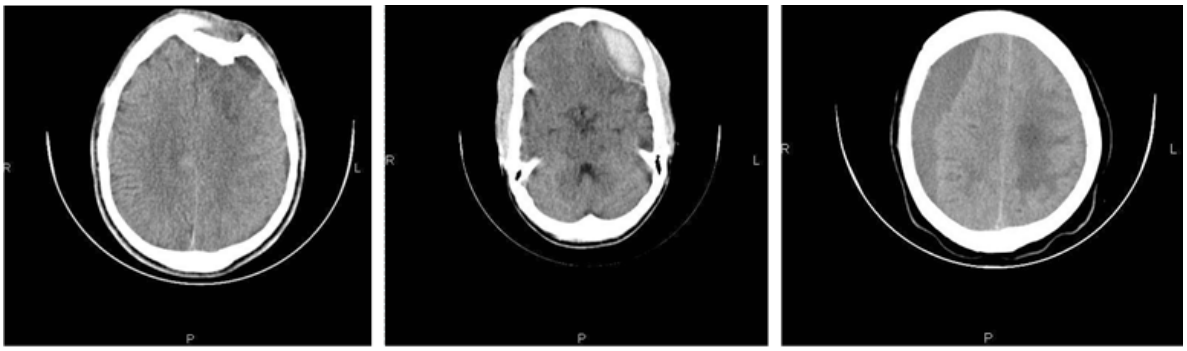
Pathophysiology of Brain Injury

Normal homeostasis

Cerebral blood flow is normally maintained at a constant level via autoregulation. Autoregulation is effective between systolic blood pressures of 50 and 150 mm Hg. Autoregulation may be lost following head injury, making the brain prone to ischaemia. Thus, hypotension should be avoided.

Intracranial hypertension

Intracranial hypertension is the end result of multiple intracranial processes that can be seen in trauma and which impair the cerebral blood flow. Cardiorespiratory compensatory processes produce hypertension



(A) (B) (C)

Figure 30.2: CT scans of (A) depressed skull fracture; (B) epidural haematoma; (C) chronic subdural haematoma.

and bradycardia, as well as irregular breathing—this is known as Cushing's triad. Therefore, in head injury, it is important to follow the cerebral blood flow (CBF). Because it is difficult to measure CBF directly, cerebral perfusion pressure (CPP) is used, which is calculated as: CPP = ICP – MAP, where ICP is intracranial pressure and MAP is the mean arterial pressure.[3,5]

Physiology of injury

Following the initial injury at impact, known as the primary injury, biochemical alterations occur, in particular, the release of glutamate, which is an excitatory neurotransmitter. This initiates a cascade of cytotoxic reactions, resulting in alterations in cellular energy metabolism, cerebral blood flow, transmembrane ion concentration gradients, free radical production, and cytokine release. Gross secondary changes, such as haematomas, cerebral oedema, hypotension, seizures, and hypoxia, further worsen the neurologic injury.

Clinical Features

History

Details of the mechanism of injury, such as distance of fall, the surface struck, and the velocity of striking objects, are important. In motor vehicular trauma, the speed of the vehicle and use of restraints should be determined. A careful history regarding immediate posttraumatic events, such as loss of consciousness, its duration, seizures, and vomiting, should be sought. In the older child, specific questions about neck pain, numbness, and weakness are asked. The possibility of child abuse should also be kept in mind.

Physical assessment

Observation of the mildly head injured child provides a great deal of information. The level of consciousness is determined. Examination of the head and scalp are done. Scalp abrasions, lacerations, and haematomas are carefully examined. The skull is palpated for areas of tenderness and fractures without inflicting pain. In older children with moderate to severe injuries, age-specific behavior is a great guide to neurological assessment. They may appropriately respond to noxious stimuli by grimacing, crying, or exhibiting a facial expression of distress. Palpation of an open fontanelle provides a good idea of intracranial pressure.

Assessment of injury severity

The Glasgow Coma Scale (GCS) is a good measure of acute injury severity and has been modified using age-appropriate parameters as indicated in Table 30.1. The table shows the best score achievable by a normal child for each parameter at various age groups.

Laboratory Assessment

Infants and small children can develop acute anaemia with relatively little blood loss. Haemogram and baseline serum electrolytes levels and blood gasses are assessed.

Radiological Assessment

Skull x-ray

A skull x-ray is useful as an initial assessment tool, particularly in Africa, where CT scans are not readily available. Skull fracture sites may herald potential intracranial pathologies. The x-ray may also show other pathologies, such as pneumocephalus (Figure 30.3), and linear fractures parallel to the slice plane, which may be missed by a CT scan.[7,8]

CT scan

A CT scan is the most useful tool for acute assessment of traumatic head injury. Bony and parenchymal lesions are usually well seen. Haematomas are clearly seen and can easily be categorised based on age. [9]

Magnetic resonance imaging

Magnetic resonance imaging (MRI) offers superior resolution in visualising small lesions, such as is seen in diffuse axonal injury, but is not as widely available and affordable as the CT scan. It is also not an investigation of choice in terms of skull fractures and intracranial haematomas.[10,11]

Cranial ultrasound

Cranial ultrasound (US) is usually a bedside technique used to monitor intracranial collections and ventricular size following trauma. This useful tool is underutilised for the child with an open fontanelle, largely as a result of lack of experience by radiologists and unavailability of US to the neurosurgeons.[10]

Management

Initial management

Adequate resuscitation and stabilisation must be given priority. The airway is the highest management priority. A child with severe head injury will require control of the airway with intubation. This helps to prevent secondary injury from hypoxia and hypercarbia.[12] The cervical spine must be assumed to be unstable until proven otherwise by plain radiographs later. Meanwhile, the breathing, circulation, and the stabilisation of vital signs are then attended to. It is the postresuscitation GCS score that is useful.

A focused neurological examination is performed to determine life-threatening intracranial pathology and assess the child's baseline neurological level; the papillary examination and the GCS score are most important for this purpose. Efforts are made to look for lateralising signs, such as hemiparesis, pupillary dilatation, facial nerve palsy, and so on. The next priority in a child who is unresponsive is to assess brainstem function by means of the corneal and gag reflexes. Corticosteroids and routine administration of anticonvulsants are not recommended.

Measures to treat raised intracranial pressure

Where ICP can be monitored, the treatment threshold for raised ICP is 20–25 mm Hg.[13]

Table 30.1: Paediatric (Adelaide) Scale.

Normal score at age	Normal eye opening	Score	Normal motor response	Score	Normal verbal response	Score	Total
0–6 months	Spontaneous	4	Flexion	3	Cries	2	9
6–12 months	Spontaneous	4	Localises	4	Vocalises	3	11
1–2 years	Spontaneous	4	Localises	4	Words	4	12
2–5 years	Spontaneous	4	Obeys	5	Words	4	13

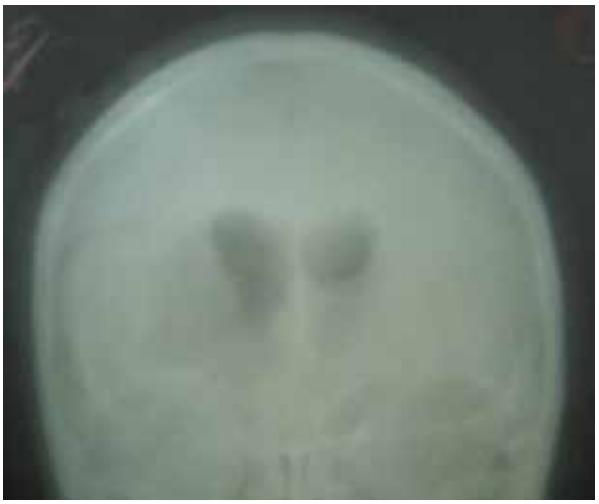


Figure 30.3: Skull x-ray showing intraventricular pneumocephalus.

Positioning

Elevation of the head of the bed to 15–30 degrees, when not contraindicated, optimises arterial flow and venous drainage.

Oxygenation

Oxygenation leads to improved cerebral oxygenation and a reduction in cerebral oedema.

Mannitol

Mannitol is very effective and can be life saving. It reduces blood viscosity and acts as an osmotic diuretic. It is given only after adequate volume resuscitation. The dose is 0.5–1g/kg body weight over 20 minutes (bolus). However, because of its rebound effect, it should be administered only when the patient is being prepared for an indicated surgery.

Controlled hyperventilation

Hyperventilation has a very rapid effect and is aimed at reducing the $PaCO_2$ to about 40 mm Hg. Lowering the $PaCO_2$ further is associated with a risk of cerebral vasoconstriction, leading to cerebral ischaemic injury.

Surgery

Intracranial haematomas in children are treated more aggressively.

Specific head injuries

Scalp injuries

In young children, blood loss from scalp laceration can lead to shock and should be promptly controlled. Scalp loss is managed with skin grafts or rotational flaps.[6]

Skull fractures

Linear, diastatic, and stellate fractures occur from focal contact forces. They may occur over a venous sinus with resultant tear haemorrhage. A CT scan is advised in the setting of stellate fractures due to the high incidence of underlying contusion.

Ping-pong fractures are managed most often nonoperatively because they often resolve spontaneously.

Depressed skull fractures are treated with elevation when indicated.

Fractures at the base of the skull are common in children, but cerebrospinal fluid (CSF) leaks often resolve spontaneously. Persistent leaks, however, require surgical intervention. Accompanying cranial nerve deficits may require surgical decompression and corticosteroid.

Growing skull fractures are peculiar and are characterised by a pulsatile scalp swelling overlying an enlarged bony defect with an associated underlying leptomeningeal cyst. They are repaired via cyst drainage, dura graft, and autologous skull graft.

Haematomas

Subgaleal haematomas can be very massive in children to the extent of inducing hypovolaemic shock.

Prompt recognition and evacuation of *epidural haematomas* via a craniotomy leads to a good and rapid neurological recovery.

Acute *subdural haematomas* carry a high mortality. The recommended surgery is craniotomy and evacuation. Chronic subdural haematomas are approached via burr-hole evacuation.

Intraparenchymal haemorrhage may be large enough to cause neurologic deficit or midline shift, and surgical evacuation is indicated in such children as they tend to recover from neurologic deficits.

Intraventricular haematoma is managed with external ventricular drainage.

Penetrating injuries

In the presence of a protruding penetrating foreign body, the offending object should *not* be removed instantaneously. Following clinical evaluation, a plain radiograph in two views and a brain CT scan should be requested. Appropriate consultation should be sought, such as from an ophthalmologist for orbitocranial injuries. Appropriate broad spectrum antibiotics and tetanus prophylaxis are to be instituted. The aim of surgery is removal of the foreign body in a controlled condition in the operating room.[14] In the absence of a retained foreign body, the goal of surgery is to debride the tract and repair the dura and bony defects.

Basic Neurosurgical Procedures

Burr-hole evacuation of chronic subdural haematoma

1. The patient is positioned supine, head turned laterally and elevated 15 degrees, with shoulder support.
2. The site of surgery is shaved and cleaned, and the patient is then draped.
3. A vertical incision is made over the site of the haematoma.
4. Usually, the first burr hole is temporal, 2.5 cm above the zygomatic arch, just anterior to the ear.
5. Scalp bleeding is controlled with cautery and self-retaining mastoid retractors.
6. The periosteum is incised and retracted.
7. A burr hole is made with a drill.
8. The dura is coagulated and incised in a cruciate fashion with a size 11 blade.
9. The haematoma is evacuated with gentle suction and irrigation, taking care not to injure any bridging vessels. Any bleeding point is controlled with bipolar cautery.
10. The subdural space is irrigated with normal saline.
11. A subdural drain may be left in place for 24–48 hours.
12. The incision is closed.

Craniotomy for trauma

1. The patient is positioned supine, head turned laterally, and elevated 15 degrees, with shoulder support.

2. A large trauma flap ("question mark") incision is made, starting anterior to the ear, above the tragus, extended rostrally above the pinna, turned posteriorly circling around the occipitoparietal area, and turning anteriorly to end frontally behind the hairline.

3. Haemostasis is secured by using Riney clips or artery forceps.

4. When necessary, the temporalis muscle is dissected by using monopolar diathermy after raising the scalp flap.

5. A series of burr holes are placed encircling the area of the haematoma, connected by using a foot plate on a power drill or a Gigli saw.

6. An epidural haematoma should now be exposed and is evacuated with gentle suction and irrigation; bleeding vessels are controlled with diathermy.

7. The dura is "tented" to prevent re-collection, and the bone flap is replaced.

8. The scalp flap is closed in layers over a closed drain.

9. In the case of acute subdural haematoma, the dura is incised, the haematoma evacuated, and haemostasis is controlled.

10. The bone flap is replaced immediately and postoperative measures instituted to control the brain swelling.

Postoperative Complications

Postoperative complications include re-collection, wound infection, and dehiscence. Re-collection of haematoma is managed by re-evacuation.

Prognosis and Outcome

Children have better outcomes compared to adults for the same type and severity of injury.[15] Mortality is age dependent—it is highest in infants and declines until about age 12 years, and then increases in the teens. In terms of morbidity, the youngest children have the worst outcome.[16] Factors that influence the prognosis include low GCS at 72 hours posttrauma, extracranial trauma, acute hypoxia, elevated raised intracranial pressure, and duration of coma.[17] The Glasgow Outcome Scale (GOS; Table 30.2) gives a reproducible outcome, which means different patients with similar GOS scores from different centres will have similar outcomes.

Table 30.2: The Glasgow Outcome Scale.

Score	Meaning
5	Good recovery
4	Moderate disability
3	Severe disability
2	Persisten vegetative state
1	Death

Complications of Head Injury

Intracranial haematoma

A majority of patients who die from head injury have an intracranial haematoma that has caused brain shift and compression. This complication may be present at the time of presentation or it may develop later. The haematoma may be in the epidural, subdural, or subarachnoid space. It may also be intraventricular or within the brain parenchyma. The patient presents with a deteriorating level of consciousness and localising signs or features of rising intracranial pressure. A brain CT scan or MRI will accurately localise the haematoma; it will also show the size of the clot and whether there is a midline shift. Evacuation of the clot through an appropriately sited burr hole or a craniotomy is life saving. A small haematoma can be managed nonoperatively.

Intracranial infections

Intracranial infections are associated with very high mortality and morbidity if not treated energetically. Meningitis can occur within a few days after head injury. The predisposing factors are open skull fractures, penetrating injuries, fractures into air sinuses, and skull base fractures with CSF otorrhoea or rhinorrhoea. The patient usually has a headache, restlessness, vomiting, photophobia, and seizures. There is high-grade fever, rigors, neck stiffness, and a positive Kernig's sign.

A CSF sample is taken through lumbar puncture for cultures and biochemical analysis. An empirical intravenous antibiotic is commenced. Intravenous (IV) benzyl penicillin (50-75 mg/kg every 6 hours) and IV chloramphenicol (1 gm every 6 hours) are effective. Cerebral abscess may develop following meningitis or when there is gross contamination from compound fractures. If the abscess is large, there may be features of raised intracranial pressure. Evacuation of the abscess with appropriate antibiotic cover is essential.

CSF leakage and fistula

The most common cause of CSF fistula is trauma. Skull fractures and associated arachnoid tears can lead to the development of CSF leakage and fistula. The incidence of posttraumatic rhinorrhea in closed head injury is 2–3%. CSF fistula occurs in 8.9% of penetrating head trauma. Posttraumatic CSF leaks are seen commonly in penetrating injuries and compound fractures of skull bones, paranasal sinuses, middle ear, and mastoid air cells. Basilar fractures are notorious for development of CSF fistula. Diagnosis is usually obvious with copious CSF leakage. A blotting paper test can be done by the bedside to show the double ring sign. A glucose test is positive in CSF as against mucus. β_2 transferin is specific to CSF. An x-ray usually shows a fracture at the base of the skull or opacity in the paranasal sinus. A CT scan is diagnostic. It may show associated pneumocephalus. In difficult cases, a dye test (metrizamide or Iohexol injected intrathecally) can be used to locate the site of the CSF leakage. Treatment is usually conservative. The use of an antibiotic is controversial unless when leakage. Treatment is usually conservative. The use of an antibiotic is controversial unless there are signs of meningitis. Most cases can be managed nonoperatively. Persistent fistulae are repaired via craniotomy.

Pneumocephalus

A fracture involving the paranasal sinuses could lead to intracranial gas collection, known as pneumocephalus. When the gas is under pressure, it is called tension pneumocephalus, which is a surgical emergency. The gas may collect in the epidural, subdural, or subarrhacnoid spaces. It may also be, within the brain parenchyma, or in the ventricular system. Intracranial infection with gas-forming organisms also cause pneumocephalus. The patient presents with headache, vomiting, dizziness, alteration in level of consciousness, and CSF leakage. An x-ray may show intracranial gas. A CT scan shows hypodense (very dark) areas. Treatment is conservative if the gas collection is small and there is no mass effect, as the gas will resolve with time. Tension pneumocephalus must be urgently evacuated. Craniotomy and repair are done for persistent CSF leakage.

Cranial nerve palsy

Any of the cranial nerves may be injured, depending on the magnitude and location of trauma. The most commonly affected nerves are olfactory, optic, oculomotor, trochlear, abducent, facial, and vestibulocochlear nerves. Healing is usually spontaneous after a variable period of time.

Posttraumatic hydrocephalus

Hydrocephalus occurs following trauma with associated subarachnoid haemorrhage. It is a communicating hydrocephalus; the patient may present with the triad of dementia, gait disturbance, and urinary incontinence. Lumbar puncture yields CSF under normal pressure. Diagnosis is made by CT or MRI. Symptoms can be remediated by CSF shunting.

Posttraumatic seizures

Seizures that occur in the first 7 days of injury are termed early posttraumatic seizures, and those that occur after 1 week are late posttraumatic seizures. The incidence of early posttraumatic seizures is 1–5%. It can precipitate adverse events as the result of an elevation of intracranial pressure, alteration in blood pressure, and changes in oxygenation. The estimated incidence of late posttraumatic seizure is 10–13% within 2 years after head injury. The risk of seizure is higher in patients with acute intracranial haematoma, open depressed skull fractures, parenchymal injury, seizures within the first 24 hours of injury, GCS <10, penetrating brain injury, history of significant alcohol abuse, and cortical haemorrhagic contusion on CT scan. Treatment with anticonvulsants is started early. IV phenobarbitone (5–10 mg/kg in 3 divided doses) or IV phenytoin (15 mg/kg loading dose; at a rate of 1-3 mg/kg per minute is given to control convulsion, and continued orally for 18-24 months).

Fat embolism

Fat embolism may occur especially in the presence of multiple injuries. Symptoms include drowsiness, confusion, epilepsy, and irritability. Dyspnoea, tachypnoea, and tachycardia may also occur. Petechial haemorrhage over the base of the neck and upper chest appears after 48–72 hours. Treatment is by measures aimed at protecting the brain from anoxia (i.e., proper care of the airway, tracheostomy, and oxygen therapy).

Posttraumatic (concussion) syndrome

This syndrome is a collection of symptoms that is considered as a sequel of a mild head injury. It can also be seen in patients recovering from severe head injury. The symptoms include headache, dizziness, lightheadedness, visual disturbances, and anosmia, as well as memory impairment, loss of intellectual ability, depression, anxiety, disruption of sleep/wake cycles, photophobia, and personality changes. The treatment of the condition is supportive. Recovery follows a variable course.

Carotico-cavernous fistula

This fistula is between the intracavernous part of the internal carotid artery and the cavernous sinus. The patient complains of noise in the head and has pulsating exophthalmos, which is usually unilateral. A continuous to-and-fro murmur is synchronous with the pulse and audible on auscultation of the eyeball. This murmur is abolished by compression of the carotid artery in the neck.

Posttraumatic headache

Posttraumatic headache is a common complaint. It may be caused by intracranial haemorrhage, increased intracranial pressure, skull fractures, CSF leaks, and infections. Treatment of the primary cause is essential; analgesics and bed rest are supportive.

Posttraumatic aneurysm

Posttraumatic aneurysms comprise less than 1% of intracranial aneurysms. Most are false aneurysms. They commonly arise from closed head injury and penetrating trauma. They present with delayed intracranial haemorrhage. The patient may present with recurrent epistaxis, progressive cranial nerve palsy, or severe headache. A CT scan shows intracerebral and subarrhacnoid haemorrhage. Angiography can demonstrate the site of the aneurysm. Although cases of spontaneous resolution have been reported, direct treatment is usually recommended by clipping, coiling, or trapping.

Posttraumatic hypopituitarism

Posttraumatic hypopituitarism follows penetrating trauma or closed head injury with or without a basilar skull fracture. The patient may have deficiency of the growth hormone, gonadotropin, corticotrophin, or reduced TSH. Some patients will develop diabetes insipidus.

Prevention

The greatest majority of paediatric head injuries are preventable. The mortality and morbidity from motor vehicle accidents could be reduced by 50% with proper use of child restraints and responsible driving. Enforcement of legislation for observation of speed limits, use of seatbelts and restraints, and wearing motorcycle helmets are said to have worked well in the developed world. Parental attitudes and sibling behavior influence a child's attitude immensely.

Children playing by the roadside, street hawking, and engaging in activities such as tree climbing to obtain a means of livelihood should be discouraged. Legislation should be enacted and enforced to contain child abuse.

Spinal Cord Injury

Introduction

Spinal cord injuries remain one of the most devastating of all survivable trauma. Paediatric spinal cord injury is relatively uncommon, accounting for 5–7% of all spinal injuries.[18,19] The peculiar developing anatomy and biomechanics of the child's spine make the management of spinal cord injury in children distinct. A good number of affected children will need supportive care for life. The availability of only a few specialised centres in Africa compounds the problem of adequate care for these children.

Epidemiology

Paediatric spinal injuries peak from June to September in the West due to extracurricular activities during the summer holidays. About 1–13% of spinal cord injuries occur in children 1–15 years of age, with 60–75% occurring in older children aged 10–15 years. The male-to-female ratio in paediatric spinal cord injury varies with age, being 1.1–1.3:1 for ages 0–9 years, to 2.3–2.5:1 for ages 15–17 years. Most spinal cord injuries occur in the cervical spine (42%), thoracic (31%), and lumbar (27%).[20] In children younger than 9 years of age, 67% of the cervical spine injuries occur between the occiput and C2 due to the higher level of the fulcrum for maximal flexion.

Aetiology

The cause of spinal cord injuries varies with age. Pedestrian-vehicle accidents and falls account for 75% of the injuries in the 0–9 year age range.[21] Motor vehicle accidents account for about 40%, and in the 15–17 year age group, motor vehicle accidents account for more than 70%. Other causes include sporting activities and motorcycle and bicycle accidents, which tend to occur in the older child.

Pathophysiology

The mechanisms of spinal cord injury in children are similar to those seen in adults. They include hyperflexion, rotation, hyperextension, axial loading, flexion rotation and shearing forces. The initial injury, either concussive or compressive, leads to immediate death of neural cell bodies in the local central grey matter. Subsequently, secondary damage occurs, initiated by the release of inflammatory mediators such as glutamate and free oxygen radicals. Oedema and spinal cord infarction result. Apoptotic changes in neurons and glial cells are now evident.[22]

Paediatric spinal traumas commonly cause ligamentous injury and facet capsule rupture. In the cervical region, there could be avulsion and epiphyseal separation of basal synchondrosis of the odontoid into the body of C2. There could be a split in the cartilaginous end plate, particularly of the growing zone. Fractures of the vertebral bodies and disc herniation are uncommon in children.[23]

Epidural, intradural, or intramedullary haematomas also occur following trauma.

Clinical Presentation

The area of spinal cord damage and nerve root involvement determine the clinical presentation. In complete spinal cord injuries, there is a loss of voluntary nervous function below the level of injury. There is an initial temporary phase of spinal shock, with loss of all reflexes below the injured segment that may last for minutes or days. About 3% of patients with complete injuries on initial examination will develop some recovery within 24 hours. In incomplete spinal cord injuries, some nervous function is present in the form of some muscle power or sensation below the level of injury; these injuries carry a better prognosis for recovery. Frankel grading is used to categorise spinal cord injuries, as shown in Table 30.3.

Table 30.3: Frankel grading of spinal cord injuries

Class	Functional status	Description
A	Complete	Total motor and sensory loss
B	Sensory only	Sensory sparing
C	Motor useless	Motor sparing of no functional value
D	Motor useful	Motor sparing of functional value
E	Recovery	No functional deficit

The various spinal cord syndromes include:

- *Anterior cord syndrome*: Damage to the spinothalamic and corticospinal tracts with resultant predominant motor weakness.
- *Brown–Sequard's syndrome*: Hemicord injury with ipsilateral motor weakness and loss of proprioception and contralateral loss of pain and temperature below the level of injury.
- *Central cord syndrome*: Injury to the central portions of the cervical spinal cord with resultant predominant motor affectation of the upper limb.
- *Conus medullaris syndrome*: Injury towards the end of the spinal cord results in a mixed upper motor neurone and lower motor neurone dysfunction.

Spinal cord injury without radiographic abnormality (SCIWORA) is a unique type of spinal cord injury common to children characterised by posttraumatic neurological deficits with normal plain radiographs or tomographs. It occurs mostly in children younger than 8–10 years of age. The mechanism of occurrence is thought to be vascular or ischaemic in origin, resulting in spinal cord infarction.

Investigations

Radiographic evaluation is done after adequate resuscitation. A lateral plain x-ray is the most informative and may show fractures, subluxation, or angulation of the spine. Soft tissue swellings may indicate ligamentous injury. In suspected odontoid fractures, an open mouth view can be done for the older child. In infants, a CT scan is recommended. At least 75% of patients with spinal cord injury have injury to the vertebral column and thus some degree of radiographic abnormalities. Therefore, initial plain films are indispensable.

Dynamic studies can be done to search for occult instability in the older cooperative child with neck pain but no neurologic deficit.

CT scans and MRI could further elucidate the extent of the injury (Figure 30.4).

Radiographic signs of cervical spine trauma include:

- soft tissue in retropharyngeal space >22 mm (child not crying);
- displaced prevertebral fat stripe;
- tracheal deviation and laryngeal dislocation;
- vertebral malalignment;
- loss of lordosis;

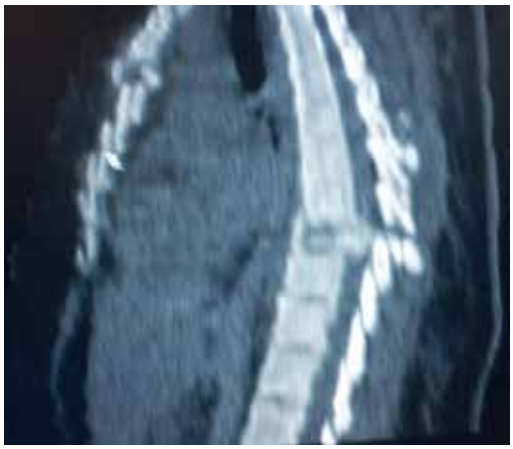


Figure 30.4: CT scan, saggital reconstruction. Slide shows retropulsed thoracic vertebra into spinal canal.

- acute kyphotic angulation;
- widened interspinous space;
- axial rotation of vertebra;
- discontinuity in contour lines;
- abnormal joints;
- atlanto-dental interval of more than 5 mm;
- narrow or widened disc space; and
- widening of apophyseal joints.

Management

The goal of management of spinal cord injuries is to prevent further injury and reduce neurological deficits.

Initial management and evaluation

Ideally, initial management and evaluation are commenced at the scene of the injury. In most African settings, however, prehospital management is not well established, and the initial management is usually commenced at the receiving hospital. The initial management includes resuscitation, immobilisation, constant monitoring, and assessment of the injured child.[24]

Resuscitation

The main causes of death of in a child with spinal cord injury are aspiration and shock, and so the "ABC" of life support is commenced. Early airway control with endotracheal intubation and oxygen administration may be indicated in respiratory insufficiency. Manual in-line immobilisation of the cervical spine is mandatory during intubation. Hypotension accompanied by bradycardia may be present due to autonomic paralysis. Therefore, adequate hydration with systolic blood pressure maintained at or above 90 mm Hg prevents shock.[25] Volume resuscitation suffices, but occasionally ionotropes such as ephedrine may be indicated.

Nasogastric tube decompression of the stomach is instituted because gastric distention can interfere with respiration or lead to gastric mucosal ulceration.

The loss of sympathetic tone may also lead to urinary retention and hypothermia. An indwelling urethral catheter is passed, and attention paid to the temperature of the child with constant monitoring.

Immobilisation

The entire spine of the child with suspected spinal injury should be immobilised. Whole-body braces usually are not readily available, so the cervical spine is immobilised with collars, particularly in the older child. Infants can be immobilised with sand bags or intravenous fluid bags secured at both sides of the head, with the head taped to the board.

Evaluation

Following resuscitation, a detailed history should be taken as soon as possible, including mechanism and time of injury, severity of injury, the first aid given, and mode of transportation. Examination should include all motor functions of the major muscle groups as well as a rectal examination to assess sphincteric tone. Sensory functions, reflexes, and motor functions of the diaphragm and intercostal muscles should be assessed.

Treatment

Medical

High-dose methylprednisolone administration within 8 hours of injury is said to be beneficial to long-term outcomes. The patient is given 30 mg/kg bolus over 15 minutes, followed by a 45-minute pause. Maintenance infusion of 5.4 mg/kg per hour over 23 or 47 hours is given. However, the efficacy has not been fully evaluated in children younger than 13 years of age. Gastric erosion is prevented by the use of H_2-receptor antagonists such as ranitidine.

Attention is paid to the prevention of pressure ulcers, chronic urinary tract infection, and contracture and deformities of the limbs.

Cervical injury

Besides collars, bracings immobilise the cervical spine (Table 30.4). Cervicothoracic orthosis (CTO) incorporates a body vest to immobilize the cervical spine and includes the Guilford brace, sterno-occipito-mandibular Immobilisation (SOMI), and Yale brace.

Table 30.4: Recommended bracing for various cervical spine injuries.

Condition	Recommended brace
Cervical strain	Philadelphia collar
Jefferson fracture stable unstable	Cervicothoracic orthosis Halo
Odontoid fracture type I types II & III	Cervicothoracic orthosis Halo
Hangman's fracture stable unstable	SOMI Halo
Flexion injuries mid cervical (C3-C5) low cervical (C5-T1)	SOMI, cervicothoracic orthosis Cervicothoracic orthosis
Extension injuries mid cervical (C3-C5) low cervical (C5- T1)	Halo, cervicothoracic orthosis Halo

Traction

Skull traction is aimed at reducing cervical fracture or dislocation, maintaining normal alignment, immobilising the spine, and decompressing the spinal cord and nerve roots. It also facilitates bone healing. Traction includes Crutchfield tongs, Gardner-Wells' tongs, or halo traction. The traction weight should be increased slowly under the guidance of an image intensifier to achieve reduction. Three pounds per cervical vertebral level is recommended (but not more than 10 pounds should be used in children younger than 14 years of age).

Thoracolumbar injury

Perhaps the most popular theory in terms of spinal stability is the three-column theory of Dennis. In this model, the anterior column includes the anterior longitudinal ligament, anterior portion of disc, and vertebra. The middle column incorporates the posterior portion of disc and vertebra, posterior longitudinal ligament, and the pedicle. The posterior column includes the posterior ligamentous complex and arch. The rib cage-sternum complex serves as a fourth column of support unique to the thoracic spine.

Damage to more than one column of the spine renders it unstable. Thoracolumbar spine instability can be categorised into (1) first-degree instability, which is mainly mechanical; (2) second-degree instability, in which there is neurological instability; and (3) third-degree instability, in which there is both neurological and mechanical instability.Those with stable and first-degree instability can be managed with bed rest for 1–6 weeks followed by ambulation in an orthosis (e.g., thoracolumbar sacral orthosis (TLSO) or Jewett brace) for 3 to 5 months. Second- and third-degree instability may require instrumentation.

Surgery

Operative management of spinal cord injury aims at decompression and stability. Emergency decompression has been associated with neurological deterioration, although it is indicated in incomplete lesions. Other indications are as follows:

- progressive neurological deterioration;
- complete spinal block (on MRI or myelogram);
- bone fragment within the spinal canal;
- cervical root compression;
- compound fracture or penetrating spinal trauma;
- acute anterior cord syndrome; and
- nonreducible, locked facet causing compression.

Complications of Spinal Cord Injury

Respiratory complications

Respiratory insufficiency is common in patients with injuries of the cervical cord. If the neurological lesion is complete, the patient will have paralysed intercostals muscles and will have to rely on diaphragmatic respiration. Partial diaphragmatic paralysis may also be present ab initio or after 24–48 hours if ascending posttraumatic oedema develops. In thoracic spine injuries, there may be associated rib fractures, haemopneumothorax, ventilation perfusion, mismatch, and so on.

Patients need to be nursed in the recumbent position even after spinal stabilisation to ensure that diaphragmatic excursion is not compromised. Regular chest physiotherapy and respiratory function monitoring should be done.

A patient whose respiratory function is initially satisfactory after injury but then deteriorates should regain satisfactory ventilatory capacity once spinal cord oedema subsides. Artificial ventilation should therefore not be withheld.

Cardiovascular complications

Haemorrhage from associated injuries is the most common cause of posttraumatic shock and must be treated vigorously. In traumatic quadriplegia, the thoracolumbar (T1–L2) sympathetic outflow paralysis gives rise to hypotension and bradycardia. Pharyngeal suction and tracheal intubation stimulate the vagus, and in high spinal cord injuries, these can produce bradycardia and cadiac arrest. Hence, atropine and glycopyrronium should be used before such procedures or when heart rates fall below 50 per minute.

Cardiac arrest from sudden hyperkalaemia following the use of depolarising agents such as suxamethonium is a risk in these patients between 3 days and 9 months after injury. Hence, nondepolarising agents are preferred.

Thromboembolism

Newly injured quadriplegics or paraplegics are at risk of thromboembolism. Antiembolism stockings and anticoagulants must be started immediately once medical contraindications and head injury are ruled out.

Bladder complications

After severe cord injury, the urinary bladder is initially acontractile, and if untreated this leads to acute urinary retention. A Foley catheter should be passed.

Gastrointestinal tract complications

Paralytic ileus is a common accompaniment of severe spinal cord injury

and should be treated with intravenous fluids, nil per os (NPO), and nasogastric tube decompression. Uncommonly, ulceration, haemorrhage and perforation may occur. Therefore, proton pump inhibitors or H_2-receptor antagonists should be started immediately.

Skin and pressure areas

The patient must be turned every 2 hours manually or automatically, and all pressure areas must be padded to prevent pressure sores.[26]

Joints and limbs

Joints must be passively moved and splinted if necessary to prevent contractures.

Autonomic dysreflexia

Autonomic dysreflexia is seen particularly in patients with cervical injury above the sympathetic outflow. It occurs after spinal shock and usually is due to distended bladder or detrusor-sphincter dyssynergia. Distended bladder causes reflex sympathetic overactivity below the level of the spinal cord lesion, causing vasoconstriction and severe hypertension. The carotid and aortic baroreceptors respond via the vasomotor centre with increased vagal tone and bradycardia, but these stimuli cannot pass distally through the injured cord.

Patient suffers headache, profuse sweating, and flushing above the level of the cord lesion. Without prompt treatment, intracranial haemorrhage may occur.

Another cause of autonomic dysreflexia includes urinary tract infection.

Treatment is by removing the cause, sitting the patient up, and administering nifedipine or glycryl trinitrate; spinal or epidural anaesthetics are used occasionally.

Hyponatraemia

Hyponatraemia is usually caused by fluid overload, diuretic usage, and the sodium-depleting effect of some drugs such as carbamazepine and inappropriate ADH secretion. It is treated by treating sepsis, fluid restriction, and administration of frusamide with potassium supplements.

Hypercalcaemia

Hypercalcaemia is caused by prolonged immobility and manifests with constipation, abdominal pain, and headache. Treatment involves hydration, achieving diuresis, and the use of sodium etidronate or disodium pamidronate.

Para-articular heterotopic ossification

New bone is often deposited in soft tissue around paralysed joints. Best treatment is surgical excision after 18 months when the bone is matured.

Spasticity

Spasticity is seen only in patients with an upper motor neurone lesion whose intact spinal reflex arcs below the level of the lesion are isolated from the higher centres. It enhances the tendency to contractures.

Aggravating factors are detected and treated, pain is managed, and spastic muscles are passively stretched. Oral baclofen is usually helpful. In intractable cases, however, the use of butulinum toxin, motor point injection, intrathecal baclofen pump, tenotomy, neurectomy, or Intrathecal block may be employed.

Urologic complications

After spinal cord injury, dysfunctional voiding patterns may soon emerge. These are associated with serious sequelae. Therefore, as part of early management, intermittent catheterisation, tapping and expression, indwelling catheterisation, suprapubic cystostomy, or intermittent self-catheterisation can be used. Later management may involve augmentation cystoplasty, neuromodulation and sacral anterior root stimulation (SARS), or intermittent self-catheterisation (Mitrofanoff's technique).

Prognosis and Outcome

The neurological examination and age of the patient are the most critical prognostic factors for short- and long-term recovery. Children with complete lesions rarely improve, whereas those with incomplete but severe lesions improve with time but hardly regain normal function. Only children with mild to moderate deficits can hope for full recovery. Mortality in the acute setting is 20%.

Prevention

The majority of spinal cord injury is caused by pedestrian-motor vehicle accidents and falls. Enforcement of traffic rules cannot be overemphasized. Prevention of children from engaging in activities that are detrimental, such as climbing trees and unprotected heights or depths must be ensured.[27,28]

Ethical Issues

Those who survive spinal cord trauma develop life-long disabilities. Treatment is often not curative. The decision to operate must be carefully discussed with the family, and the prospective multidisplinary mode of subsequent management should be instituted.

Evidenced-Based Research

Table 30.5 is a review of paediatric severe head injuries that compares two age groups. Table 30.6 is a review of paediatric spine fractures that compares parameters of the injuries.

Table 30.5: Evidence-based research.

Title	Severe head injury in children: early prognosis and outcome
Authors	Zuccarello M, Facco E, Zaampieri P, Zanardi L, Andrioli GC
Institution	Department of Neurosurgery and Institute of Anesthesiology and Intensive Care, University Hospital, Padova, Italy
Reference	Child's Nervous System 1985; 1:158–162
Problem	Identifying indicators for early prognosis and outcome in children with severe head injury.
Intervention	Controlled hyperventilation and bolus infusion of hypertonic (20%) mannitol, surgical removal of any mass lesions.
Comparison/ control (quality of evidence)	Sixty-two children with severe head injury were divided into two groups: infants aged <36 months (24.2%) and children aged 36 months–14 years (75.8%). The study was limited to patients who remained in coma for at least 6 hours.
Outcome/ effect	The difference between good and poor results in patients with GCS 4 or less and those with score of 5 points or better was significant (p < 0.001). There was a correlation between the best motor response and outcome. Of flaccid patients, 85% did poorly or died, whereas 69% of those who withdrew in response to pain did well (p<0.001). The presence or absence of brainstem dysfunction (assessed on basis of pupil reaction and oculocephalic reflex) was statistically related to good or poor result but oculocephalic reflex was considered to be the most indicative (p<0.001). The necessity for assisted ventilation at admission was associated with a less favourable outcome (p<0.001). An intracranial haematoma was not associated with a worse outcome. The authors observed that recovery was almost complete when the duration of coma was less than 2 weeks, with 93% of patients moderately disabled or with a good recovery. There was high incidence of poor outcome in those with coma lasting >2 weeks (p = 0.0002). Overall mortality was 32%.
Historical significance/ comments	This study provides a useful insight into the early prognostic factor in children with severe head injury. Clinical features available soon after injury that are important indicators of treatment and outcome are identified.

Table 30.6: Evidence-based research.

Title	Pediatric spine fractures: a review of 137 hospital admissions
Authors	Carreon LY, Glassman ST, Campbell MJ
Institution	Leatherman Spine Center, Louisville, Kentucky, USA
Reference	J Spinal Disorders Techniques 2004; 17: 477–482
Problem	In children with spinal injury, prevention of further neurologic damage and deformity, as well as good potential for recovery, makes timely identification and appropriate treatment of the injury critical.
Intervention	Decompression, fusion, instrumentation.
Comparison/ control (quality of evidence)	The 137 patients were divided into three groups for analysis: 0–9 years of age (36 patients), 10–14 (49 patients) and 15–17 (52 patients). This allowed for comparison of age with mechanism of injury, injury pattern and level, incidence of cord injury, treatment, and outcomes.
Outcome/ effect	Thirty-six (1%) of 3,685 injured children aged 0–9 years sustained spine injury, compared to 49 (3%) of 1,609 injured children aged 10–14 years, and 52 (5%) of 921 injured children aged 15–17 years ($p < 0.001$). Motor vehicle accidents were the most common cause of injury across all ages, followed by falls, sports, and pedestrian accidents. The incidence of multilevel and noncontiguous injuries in the different age groups was not significantly different. Twenty-four patients (19%) had spinal cord injury; 21 (87%) were complete cord injuries, and 3 (13%) were incomplete. Cord injury was more common in the 0–9 year age group. Four of five patients with spinal cord injury without radiographic abnormality (SCIWORA) were in the 0–9 age group and had complete neurologic injuries. Young children with cervical injuries were more likely to die than older children. Fifty-three percent had associated injuries. Eighteen percent underwent decompression, fusion, and instrumentation. Two patients developed scoliosis. The complication rate in surgical patients was higher than in patients treated nonsurgically and in polytrauma patients.
Historical significance/ comments	This retrospective clinical case series has presented important and useful data from a large series of paediatric patients with spine injuries from a single regional trauma center.

Key Summary Points

Craniocerebral Trauma

1. Paediatric head injury is common in our environment.
2. Motor vehicle accidents cause the severest form of injury.
3. Child abuse is increasingly becoming recognised as a cause of paediatric head injury.
4. Prompt resuscitation and cervical spine protection are key to survival.
5. Scalp bleeding may easily cause anaemia because of small intravascular volume.
6. The postresuscitation GCS score is an important prognostic factor.
7. Intracranial haematomas are aggressively managed.
8. Education and enforcement of legislation on vehicle safety rules are important in preventive strategies.

Spinal Cord Injury

1. Paediatric spinal cord injury presents an enormous challenge, not only to the neurosurgeon but to the health and economic resources of any nation.
2. The care of the spinally disabled child is far from ideal in our environment.
3. Late presentation is the rule in most African settings, precluding those who would have benefitted from the institution of early treatment modalities.
4. Prompt resuscitation and optimal fluid administration limit further cord injury.
5. More personnel, facilities, and dedicated centres for spinal care are in arrears, needing urgent attention.

References

1. Winn HR, ed. Paediatric head injury In: Youman's Neurological Surgery, 5th ed. Saunders, 2004.
2. Tindall GT,Cooper PR, Barrow DL, eds. Head Injury in the Paediatric Patient. In: The Practice of Neurosurgery, Version 4.18.4; Williams and Wilkins, 1996.
3. Basso A, Previglioano I, Duarte,JM, Ferrari, N. Advances in Management of Neurosurgical Trauma in Different Continents. World J Surg 2001; 25:1174–1178.
4. Shokunbi MT, Solagberu BA. Mortality in Childhood Head Injury in Ibadan. Afri J Med Sci 1995; 24:159–163.
5. Solagberu BA, Adekanye AO, Ofoegbu CP, Udoffa US, Abdur-Rahman LO, Taiwo JO. Epidemiology of trauma deaths. West Afr J Med 2003; 22:177–181.
6. Legbo JN, Shehu BB. Managing scalp defects in Sub Saharan Africa. East Afri Med J 2004; 81:87–91.
7. Thanni L. Evaluation of guidelines for skull radiography in head injury. Niger Postgrad Med J 2003; 10:231–233.
8. Obisesan AA, Bohrer SP. The uses and abuses of skull x-ray in head injury. Niger Med J 1979; 9:65–69.
9. Nagy KK, Joseph KT, Krosner SM, et al. The utility of head computed tomography after minimal head injury. J Trauma 1999; 46:268–270.
10. Umerah B. Cerebral angiography in Central Africa. Diagn Imaging 1981; 50:225–228.
11. Otieno T, Woodfield JC, Bird P, et al. Trauma in rural Kenya. Injury 2004; 35:1228–1233.
12. Muhammad I. Management of head injuries at the ABU Hospital, Zaria. East Afr Med J 1990; 67:447–451.
13. Brain Trauma Foundation AAON. Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. Paediatr Crit Care Med 2003; 4:S1–S75.
14. Bullock MR, Chestnut R, Ghajar J, et al. Guidelines for the surgical management of traumatic brain injury. Neurosurgery 2006; 58(3 Suppl.):S1–S60.
15. Shokunbi MT, Olurin OI. Childhood head injuries in Nigeria: causes, neurologic complications and outcome. West Afr Med J 1994; 13:38–42.
16. Mwang'ombe NJ, Kiboi J. Factors influencing the outcome of severe head injury at Kenyatta National Hospital. East Afr Med J 2001; 78:238–241.
17. Bahloul M, Chelly H, Ben Hmida MS, et al. Prognosis of traumatic head injury in south Tunisia: a multivariate analysis of 437 cases. J Trauma 2004; 57:255–561.
18. Winn HR, ed. Paediatric vertebral column and spinal cord injuries, In: Youman's Neurological Surgery, 5 ed. Saunders, 2004.
19. David P. Spinal cord injury in children. In: Principles and Practice of Paediatric Neurosurgery. Thieme, 1999, Pp. 955–969.
20. Brockmeyer DL. Spinal cord and spinal column injuries in children : current management options. In: Paediatric Neurosurgery for the General Neurosurgeon. Thieme, 2002, Pp. 36–48.
21. Hamilton MG, Myles ST. Paediatric spinal injury: review of 174 hospital admissions. J Neurosurg 1992; 77:700–704.
22. Rauzzino MJ, Grabb PA, Hadley MN. Paediatric vertebral column and spinal cord injuries. Persp Neurolog Surg 1997; 8:27–60.
23. Scher AT. Trauma of the spinal cord in children. S Afr Med J 1976; 50:2023–2025.
24. Adeolu AA, Shokunbi MT, Malomo AO, Komolafe EO, Adeloye EA. Cervical spine injury in children with head injury in Ibadan, Nigeria. Afr J Paediatr Surg 2005; 1:76–79.
25. Mayuri G, Maharajh J. Case report of cranio-cervical dislocation in a child. S Afr Radiographer 2006; 44(2):18
26. Chen Y, DeVivo MJ, Jackson AB. Pressure ulcer prevalence in people with spinal cord injury: age-period-duration effects. Arch Phys Med Rehabil 2005; 86:1208–1213.
27. Odeku EL, Richard DR. Peculiarities of spinal trauma in Nigeria. West Afr Med J 1971; 20:211–225.
28. Rhodes N. Focus on parents: teen's perceptions of parental safety messages. Workshop presentation at the 2007 Lifesavers Conference. Chicago, IL, March 2007.