Part II – Neurological Disorders

CHAPTER 19
HEAD AND SPINAL INJURY

Dr William P. Howlett
2012

Kilimanjaro Christian Medical Centre,
Moshi,
Kilimanjaro,
Tanzania

BRIC 2012 

University of Bergen
PO Box 7800
NO-5020 Bergen
Norway

NEUROLOGY IN AFRICA

William Howlett

Illustrations: Ellinor Moldeklev Hoff, Department of Photos and Drawings, UiB

Cover: Tor Vegard Tobiassen

Layout: Christian Bakke, Division of Communication, University of Bergen

Printed by Bodoni, Bergen, Norway 

ISBN 978-82-7453-085-0

CONTENTS

CHAPTER 19

HEAD AND SPINAL INJURY

Trauma including head injury (HI) resulting in traumatic brain injury (TBI) and spinal injury is the leading cause of death and disability in 15-45 yr olds in high income countries. It is also a significant and increasing cause of morbidity and mortality in low and middle income countries, particularly in Africa. Road traffic accidents (RTAs) involving occupants and pedestrians account for the majority of cases and are increasing year on year. They are now a significant cause of disease burden in Africa. Head and spinal injuries occur as a consequence of the increasing numbers of vehicles, motorcycles, speeding, poor quality of the roads, and lack of enforcement of statutory safety regulations for driving and the lack of safety belts and helmets (Table 19.1). Other causes include falls and violence. Interpersonal violence is increasingly a cause of HI in some countries in Africa. The aim of this chapter is to present an overview of head and spinal injuries. The student should aim to know the increasing burden, clinical features, management and prevention of head and spinal injuries.

EPIDEMIOLOGY

The annual incidence of traumatic brain injury ranges from 150-500/100,000 per year depending on the individual country. It is estimated that 1-2% of high income populations live with a TBI disability. The incidence is high in some countries in Africa. In South Africa the mortality rate in TBI was reported to be 81/100,000 per year, with a >10% all case fatality rate. High risk groups for TBI include children, adolescents, young adults and the elderly, with males being affected 2-3 times more often than females.

Table 19.1 Main causes and risk factors for TBI in Africa

Cause	Risk factor	%	Risk
Road Traffic Accidents	speeding, driver sleepiness, lack of use of seat belts, poor roads, lack of law enforcement, alcohol, medications, drugs	>80	increasing
Falls	climbing trees, poor work place safety, age & co-morbidity	>10	static
Violence	urban poverty, unemployment, alcohol	<10	increasing

Pathology of head injury

The term HI includes injury to the brain, face, head and skull. Injury to the brain is called traumatic brain injury (TBI) and all grades can occur from mild to severe. Because the brain is a soft organ contained within a rigid box, any trauma to the skull will also cause movement of the brain inside the skull. This can result in TBI ranging from no visible signs in cases of mild head injury (concussion) to bruising, laceration, bleeding and oedema in severe head

injury. Bleeding from torn blood vessels may result in a haematoma, either intracerebrally (intracerebral haematoma, ICH), or in the extradural space (extradural haematoma, EDH) and in the subdural space (subdural haematoma, SDH). If the resulting volume is too large there may be herniation of the brain or brain stem through the tentorium or foramen magnum and coning and death will follow. In addition to the primary brain injury there may be secondary brain injury as a result of hypotension and hypoxia. The main causes of these are blood loss, lung injury and seizures. Secondary infection may arise as a result of penetrating injuries or a fractured skull.

Clinical diagnosis

A history of the cause and the circumstances of the injury should if possible be obtained either from the patient or a witness. It is important to enquire if there was any period of loss of consciousness and the current state of consciousness of the patient. The main steps in the examination of head injury are outlined below in Table 19.2. The Glasgow Coma Scale (GCS) uses a point system out of a maximum of 15 to evaluate the best eye opening, verbal and motor response (Chapter 9). An underlying fracture of the skull is suspected if there is a CSF leak, bleeding from the ears, bruising around the eyes or ears, deafness or on the basis of a significant head wound. The severity of a head injury resulting in TBI is graded in terms of the following: *current level of consciousness, any period of unconsciousness, memory loss, focal neurological deficit* and *skull fracture*. The current level of consciousness of the patient is the most important indicator of the severity and progress of head injury.

Table 19.2 Summary of main steps in the examination of patient with head injury

Clinical finding *(examine for the following)*	**Comments**
STEP 1 level of consciousness	**most important of all assessments** *(repeat at regular intervals, usually every 15 mins)*
STEP 2 signs of HI	
lacerations, bruising or skull fractures **injury to cervical spine**	may be absent in TBI check neck & limbs
signs of basal skull fracture *csf leak from nose/ear, usually clear* *bleeding around eyes & subconjuctiva* *bleeding from external ear* *bruising over the mastoid (Battle's sign)*	 confirm by testing fluid for glucose no posterior limit indicates fractured skull exclude laceration of external ear may take 48 hours to develop
STEP 3 focal neurological signs	
pupillary response to light	pupil may dilate on same side in expanding lesion
limb weakness	asymmetrical limb withdrawal to deep pain indicates focal brain lesion
eye movements	observe for spontaneous eye movements
cranial nerve lesions **other observations**	may indicate a skull fracture BP may rise & pulse rate fall in ↑ICP

Imaging

All head injuries should be considered for imaging. Imaging includes plain X-rays of skull and spine, CT of the head and occasionally MRI of the brain and spinal cord. Indications for urgent CT include depression/loss of consciousness, focal neurological signs, seizures, fractured

skull and CSF leak. CT may reveal evidence of fractures, intracranial haematomas and cerebral contusion (Fig. 19.1).

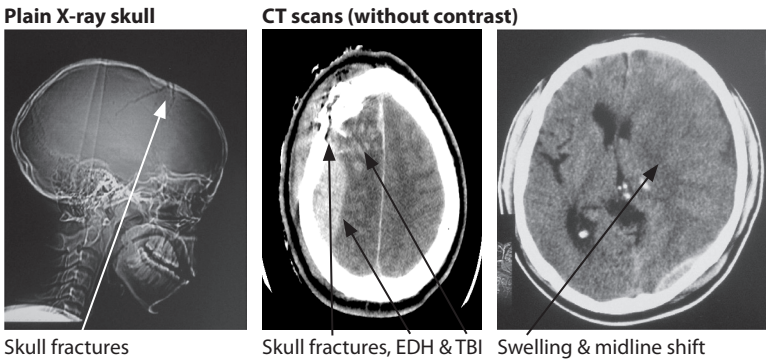


Figure 19.1 Traumatic brain injury

DIFFUSE AXONAL INJURY (DAI)

This occurs from the shearing of axons as a result of a high impact closed HI. It is usually associated with an immediate LOC which persists after the injury. Imaging initially by CT is normal in >50-70% of cases and this may need to be repeated after 24/48 hours before showing haemorrhages and cerebral oedema. MRI is more sensitive than CT in the early stages of DAI. Management includes sedation, ventilation and measures for intracerebral pressure monitoring and control.

Classification

The classification or grading of HI is based on the initial history, clinical evaluation and GCS. This is made in order to identify and treat patients at risk. It is important to remember that the majority of cases of HI do not result in TBI. Of those that do, TBI is graded as mild if the GCS at assessment is >12, or the period of unconsciousness is <30 minutes. The majority, 85-90% of all TBIs are classified as mild. TBI is classified as moderate if the current GCS is 9-12 or there is persisting coma for >30 mins or there is a skull fracture but no evidence of brainstem malfunction (Table 19.3). TBI is categorised as severe if unconsciousness is greater than 24 hours or current GCS ≤8 or there are focal neurological signs or intracranial haematoma or failing brain stem function. Patients with moderate and severe TBI represent about 10-15% of all cases.

Table 19.3 Traumatic brain injury, summary of main clinical features

Grade	GCS	%	CT signs present*	Mortality %	Morbidity %	Disability
mild	13-15	85-90	in one third	1	50 (minor)	none/minor
moderate	9-12	5-10	in two thirds	2-5	60	disabled
severe	3-8	3-5	in all**	20-50	75	severely disabled

** includes fractured skull, contusion, bleeding, swelling*
*** except in diffuse axonal injury (DAI) when the initial CT of head may be normal*

Key points

- main causes of HI are RTAs, falls & violence
- obtain a history of the cause of the head injury
- assess the level of consciousness
- examine for signs of head and neck injury& for neurological deficits
- TBIs are graded as mild, moderate and severe
- grade is the most important predictor of outcome

Management

The medical management of brain injured patients is complex. It involves 4 main sites/stages: *pre hospital care, transport to hospital, in hospital care* and *rehabilitation*. Any delay or failure at any of these stages results in increased mortality and morbidity.

Trivial and mild

Trivial head injuries and mild TBIs account for the majority of TBIs and most patients may be discharged home after initial examination with a warning about possible complications. The risk of complications for mild TBIs is low <1%. However patients with mild TBI and other clinical features of head injury should be admitted for 24 hours for closer neurological observations, care, possible CT scanning and a re-evaluation later. This group has an increased risk (1-5%), of serious neurological deterioration. The main indications for a CT scan in mild TBI include *alteration in level of consciousness, confusion, a history of transitory loss of consciousness, post traumatic amnesia, the presence of focal neurological signs* and *a possible skull fracture*. A skull X-ray should be performed if a CT scan is unavailable.

Moderate and severe injuries

This group represents 10-15% of all TBIs. Moderate and severe head injuries should all have a CT scan of the head, be admitted to an intensive care unit for observation and care and considered for any possible neurosurgical intervention.

Evaluation of moderate and severe TBIs

The first priority is to check that the airway is clear and that breathing and circulation are adequate. This may involve inserting an airway and ensuring that oxygen saturation is >95%. Circulation is maintained by starting emergency intravenous fluids and ensuring that ≤2 litres is given in the first 24 hours unless the patient is hypovolaemic for other reasons (Chapter 9). The next step is the evaluation of the extent and seriousness of the injuries. Many patients, in particular RTA cases, have concomitant injuries of the chest, abdomen, spine and extremities.

All patients with HI and altered consciousness should be assumed to have a neck injury and be immobilized until proven otherwise. This means a general evaluation in addition to measuring the level of consciousness, examining for neurological deficits and for evidence of head and spinal cord injury, and any skull or vertebral fractures. (Table 19.3) Check with family or friends for any history of any predisposing risk factors for head injury including loss of consciousness, stroke, seizures, alcohol and drugs. The GCS together with vital signs, pupil size and reaction to light should be checked every 15 minutes in unstable patients and then at 1-4 hourly intervals appropriate to the patient's condition. Special attention should be paid to avoiding the risk factors for secondary brain injury; this includes maintaining adequate blood

pressure and oxygen saturation. If the patient's consciousness is deteriorating then mannitol 1 gm/kg/iv is given stat followed by iv boluses of 1-200 ml of 20% mannitol depending on the clinical situation. This may be helpful as a short term measure to reduce intracranial pressure. Prophylactic antibiotics and antiepileptics are indicated for all severe head injuries. Indicators of a poor prognosis are: *coma for >6 hours, fractured skull, non reacting pupils* and *focal neurological deficits*. The main indications for neurosurgical intervention are extradural and subdural haematoma, depressed skull fracture and occasionally intracerebral haemorrhage.

Key points

- 4 main stages, prehospital, transport, in hospital and rehabilitation
- aim is to prevent secondary complications of TBI
- brain damage is prevented by adequate evaluation & care
- this involves ABC & preventing seizures & infection
- CT head scan indicated in all moderate & severe TBI
- main indications for surgery are extradural & subdural haematomas and some ICH

OUTCOME

TBI accounts for up to half of all traumatic deaths with over half of these deaths occurring at the scene of the accident or on the way to hospital. The possible outcomes of TBIs are summarized in Table 19.3 and in the Glasgow Outcome Scale (Table 19.4). The initial grade of TBI after the primary injury is the most important predictor of outcome.

Table 19.4 Glasgow Outcome Scale

Classification	Description
dead	
persistent vegetative state	awake but unaware
severely disabled	conscious but dependent
moderately disabled	independent but disabled
good recovery	may have minor sequelae

Mortality

The overall case fatality rate (CFR) for all TBI is over 10%; however this varies from less than 1% in mild to 2-5% in moderate and 20-50% in severe TBI. The CFR for severe TBIs with coma lasting longer than 6 hours is >50%.

Morbidity

Most patients with mild TBI make a good recovery but about half have minor sequelae e.g. headache, lack of concentration etc which usually clear after a variable period of months to years. There is also increasing evidence that repeated episodes of mild TBI can lead to dementia and/or depression with lesions deep in the brain identified at post-mortem. In contrast, the majority of surviving patients with moderate and severe TBI will be permanently disabled from the onset. This is the leading cause of neurological disability in young persons (<40 yrs) worldwide. Moderate disability is 3-4 times more common than severe. The range of moderate and severe disabilities includes *personality changes, memory loss, dysphasia, paralysis* and *epilepsy*. Persistent vegetative state is uncommon. Over 90% of patients reach their maximum recovery

by 6 months although some patients may continue to recover for years. Over 10% of patients with severe TBI develop epilepsy within 5 years of the injury.

REHABILITATION

Most patients with mild head injury make a good recovery without requiring any special measures or rehabilitation. Patients with moderate or severe head injury benefit most from rehabilitation. This may involve a considerable period of hospitalization, *usually >3 months* together with extensive inpatient and later outpatient rehabilitation while waiting for recovery of neurological function and healing of injuries. Recovery of function reaches a peak in about 90% of patients within the first six months after injury but may continue in some for years.

Post concussion syndrome

Post concussion syndrome may follow after mild or moderate head injuries. Patients can be disabled by recurrent symptoms including headaches, dizziness, poor concentration, impaired memory, fatigue and depression. These usually subside after a few months but can continue for as long as 6 months to 3 years. There are no abnormal neurological findings and no abnormalities on neuroimaging of the brain. Management is conservative and supportive.

Key points

- most patients with mild TBI eventually make a full recovery
- patients with moderate and severe TBI have high mortality & morbidity rates
- patients with moderate and severe TBI benefit most from rehabilitation

EXTRADURAL HAEMATOMA (EDH)

Extradural haematoma is a rapid collection of arterial blood occurring over minutes to hours in the extradural space as a result of a temporal/parietal skull fracture or a serious head injury. EDH results from a traumatic tear in the middle meningeal artery. The bleeding shells the dura mater from the inner table of the skull compressing the brain. The main cause is HI arising from RTA.

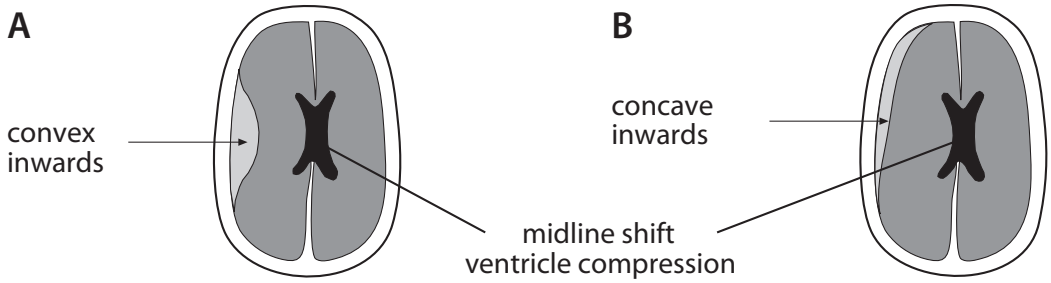
Clinical features

In EDH, characteristically there is a *lucid interval* between the patient waking up from the acute head injury and then becoming unconscious again. This lucid period is variable ranging from minutes to over 24 hours. It should be suspected whenever there is a sudden decline in the level of consciousness of a patient with a very recent head injury. The site of the extradural haematoma is on the same side as the underlying skull fracture and the pupillary dilatation.

Diagnosis

The diagnosis is suggested by the clinical presentation, neurological findings and a skull X-ray may show an underlying fracture. In EDH the CT scan of head shows on the affected side an area of increased density *biconvex inwards* with midline shift in severe cases (Figs. 19.2 & 3). However obtaining the CT scan should not be a reason to delay surgery, as any delay will inevitably result in the death of the patient.

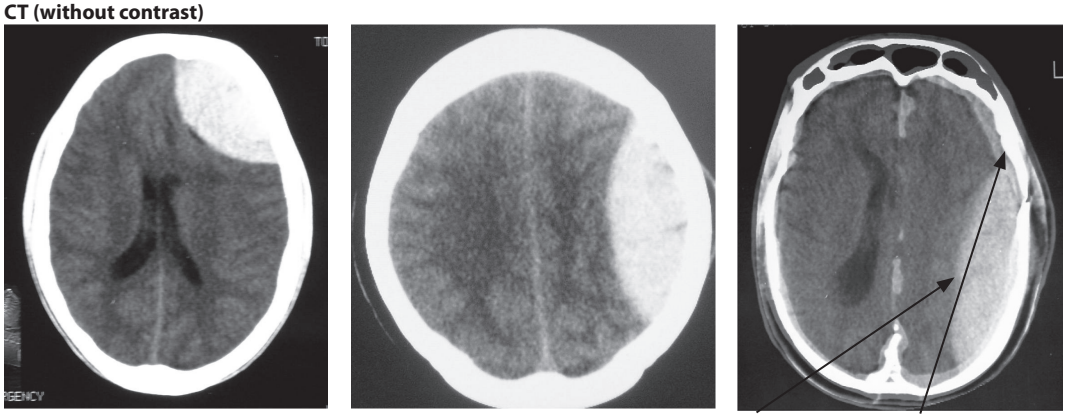
CT scans (without contrast)



A Extradural haematoma B Subdural haematoma

Figure 19.2 Illustrations of extradural and subdural haematoma

CT (without contrast)



EDH (left) EDH (left) EDH (left) & small SDH

Figure 19.3 Extradural haematoma

Management

This is a true surgical emergency and management is by emergency craniotomy in order to establish urgent ligation of the bleeding vessel and surgical drainage of the EDH. Emergency resuscitation and a burr hole may provide temporary relief before the craniotomy.

Prognosis

Prognosis depends on the preoperative state of the patient. The presence of fixed dilated pupils is not a contraindication for surgery. If GCS ≤ 8 the mortality is >30%.

Key points

- EDH results from a meningeal artery tear outside the dura
- main cause is a fractured skull secondary to a RTA
- blood accumulates within minutes to hours in the extradural space
- lucid interval of minutes/hours followed by rapid onset of FND & LOC
- management is urgent craniotomy to ligate the bleeding vessel & drain the EDH

SUBDURAL HAEMATOMA (SDH)

Subdural haematoma (SDH) is a collection of blood which lies in the space between the dura and arachnoid layers of the meninges. It arises from an injury which causes rupture of bridging veins between the meningeal layers and can be *acute* or *chronic*.

Acute SDH

An acute SDH immediately follows a high energy head injury with just a short delay of hours or at most days between the injury and the onset of symptoms. Clinically this can lead to a rapid clinical deterioration due to the accumulation of blood with an immediate loss of consciousness and progressive or fluctuating decline in GCS. The source of the haemorrhage in acute SDH is both arterial and venous from contused cerebral cortex and blood vessels. If left untreated this may eventually lead to transtentorial herniation, coning, hemiparesis, coma and death. Management is by emergency surgical drainage usually involving a craniotomy. Cerebral swelling is common and may require decompression surgery. The prognosis is poor with 50-70% mortality.

Chronic SDH

A chronic SDH is a late complication which can follow any head injury but may occur after a relatively minor low energy or non reported head injury particularly in the elderly, in those on anticoagulants and in persons prone to falling e.g. alcoholics. The injury may be so minor that it is not remembered. Very occasionally chronic SDH is spontaneous without previous history of trauma. In chronic SDH there is a slow accumulation over weeks or months of venous blood over one or occasionally both cerebral hemispheres.

Clinical features

The clinical presentation is one of non specific gradual fluctuating drowsiness, confusion, and headache and only later followed by the onset of a focal neurological deficit, usually hemiparesis and progressing to alteration in level of consciousness, seizures and sometimes coma. The finding of a progressive hemiparesis or a recent onset confusional or dementia type illness is a feature in Africa because of late hospital presentation.

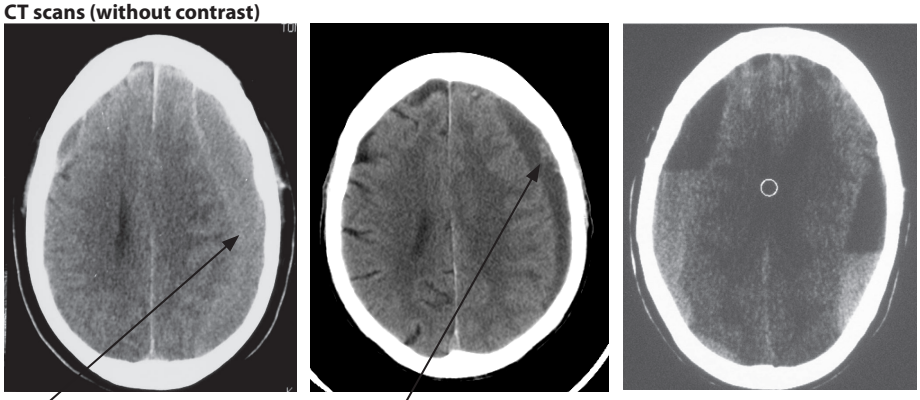
Imaging

The diagnosis of SDH is either confirmed or else newly discovered by a CT of the head. The SDH shows as a *crescent shaped concave inwards* area of increased density spreading round the surface of the cerebral hemisphere with or without an accompanying mid line shift (Figs. 19.2 & 4). Approximately 10-12 days after injury the appearance of SDH on CT becomes isodense and is then more difficult to notice. After about 3 weeks post HI the SDH becomes hypodense on non contrasted CT and is then more obvious.

Management

The management of chronic subdural haematoma depends on the size and clinical state of the patient. Small subdurals without focal neurological signs can be managed conservatively and these may resorb spontaneously. Larger subdurals with changing levels of consciousness and focal neurological signs need surgical drainage and evacuation. CT indications for surgery include *cortical compression, midline shift* and *hydrocephalus*. Drainage is established via burr holes in the skull with or without a surgical drain. This procedure is carried out by most general surgeons in Africa. Steroids, dexamethasone 4 mg qds may be helpful in the early stages.

CT scans (without contrast)



SDH (left) isodense SDH (left) hypodense SDH (bilateral, old)

Figure 19.4 Subdural haematoma (chronic)

Prognosis

The outcome is generally good in all age groups but 10-15% of patients may require a second drainage procedure and a subdural empyema occurs in <1%.

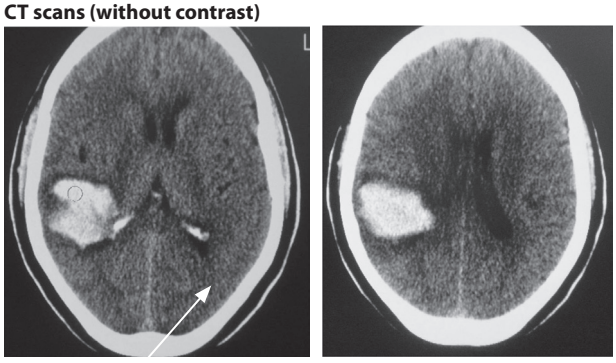
Key points

- in SDH there is usually a history of HI
- acute SDH occurs within hours or days of HI
- chronic SDH occurs within weeks/months of HI
- presentations include headache, confusion and coma, seizures & hemiparesis
- SDH is usually confirmed or diagnosed by CT of head
- management is mostly by surgical drainage

Intracerebral haematoma (ICH)

This arises from severe contusional HI or vascular injury. Common sites include the cerebral cortex, cerebellum and subarachnoid space (Fig. 19.5). Management depends on the severity, clinical condition and evidence of mass effect. Craniotomy and surgical evacuation is indicated in presence of focal neurological signs and decreasing GCS.

CT scans (without contrast)



ICH & intraventricular extension

Figure 19.5 Intracerebral haematoma

SPINAL INJURY

Spinal injury arises mostly from road traffic accidents (RTAs) and falls and is a major cause of death and disability in Africa. It is often associated with HI and multiple trauma. Males in the age group 20-40 years are the main risk group affected. In Africa boys and adolescent males are frequently affected as a result of falls from trees and more recently RTAs. Females are also affected because of falls whilst carrying heavy loads on their heads. Early detection and immobilization are critical to avoid secondary damage.

Clinical features

The cervical and the lumbar spine are the most common sites of injury with the cervical spine being more frequently affected. Paraplegia and quadriplegia are the main neurological disorders resulting from spinal injury. Spinal injuries anywhere from the cervical spine down to the level of the first lumbar vertebra inevitably involve the spinal cord whereas injuries below L1 involve the cauda equina. In spinal cord injury there may be an initial period of *spinal shock* which can persist for *days* to *weeks* (usually 1-2 weeks) before the characteristic spasticity and upper motor neurone signs develop. Spinal shock is characterized by flaccid paralysis with no reflexes or sensation below the level of spinal cord injury. In contrast paraplegia resulting from spinal injury affecting the cauda equina remains permanently flaccid with characteristic lower motor neurone signs. Bladder and bowel dysfunction occurs with both types. The completeness of the lesion is the most important factor in determining prognosis and management. Incomplete lesions may recover to a variable extent.

Spinal stability

The stability of the spine at the level of injury plays a crucial role in further management. Instability is defined as the loss of ability of the spine to maintain normal alignment under normal loads. Instability increases the risk of further spinal cord damage. The Denis three column model (Table 19.5) is used to classify spinal stability.

Table 19.5 Denis three column model of the spine

anterior column	anterior one-half of the vertebral body and annulus fibrosus & the anterior longitudinal ligament (ALL)
middle column	posterior one-half of the vertebral body & annulus fibrosus & posterior longitudinal ligament (PLL)
posterior column	pedicles, laminae, spinous processes & ligaments

Spinal injuries are classified as stable when the interspinous ligaments are intact and as unstable when ligaments are disrupted. The spine is unstable if ≥2 columns on the Denis three column model are disrupted. The mechanism of injury helps to determine the degree of stability and type of cord injury. A shearing or hinge injury typically results in unstable spinal injury with cord involvement, whereas a compression injury results in stable fractures. Stable spinal injury occurs mostly without cord involvement. Typically the former arises from RTAs, whereas the latter arises from an object falling on to the head. However a severe or burst fracture from whatever cause results in an unstable spine and possible spinal cord injury.

The initial examination should be targeted at checking for local injury in the spine, particularly in the neck and looking for paralysis of limbs, being careful to avoid aggravating existing injuries by any movement. It is critical to emphasise *that the spine must be stabilized in any patient with evidence or suspicion of spinal injury before being moved.* This is done by means of

using a *hard cervical collar*, putting the patient *lying on a back board* and using the *logrolling method* when turning the patient.

Key points

- RTAs and falls are the main causes of spinal injury
- neck and lower back are the sites most frequently affected
- spine should be stabilized in patients with spinal injury before being moved
- spinal shock lasting >24 hours is a bad prognostic sign
- death or paraplegia are the two main outcomes

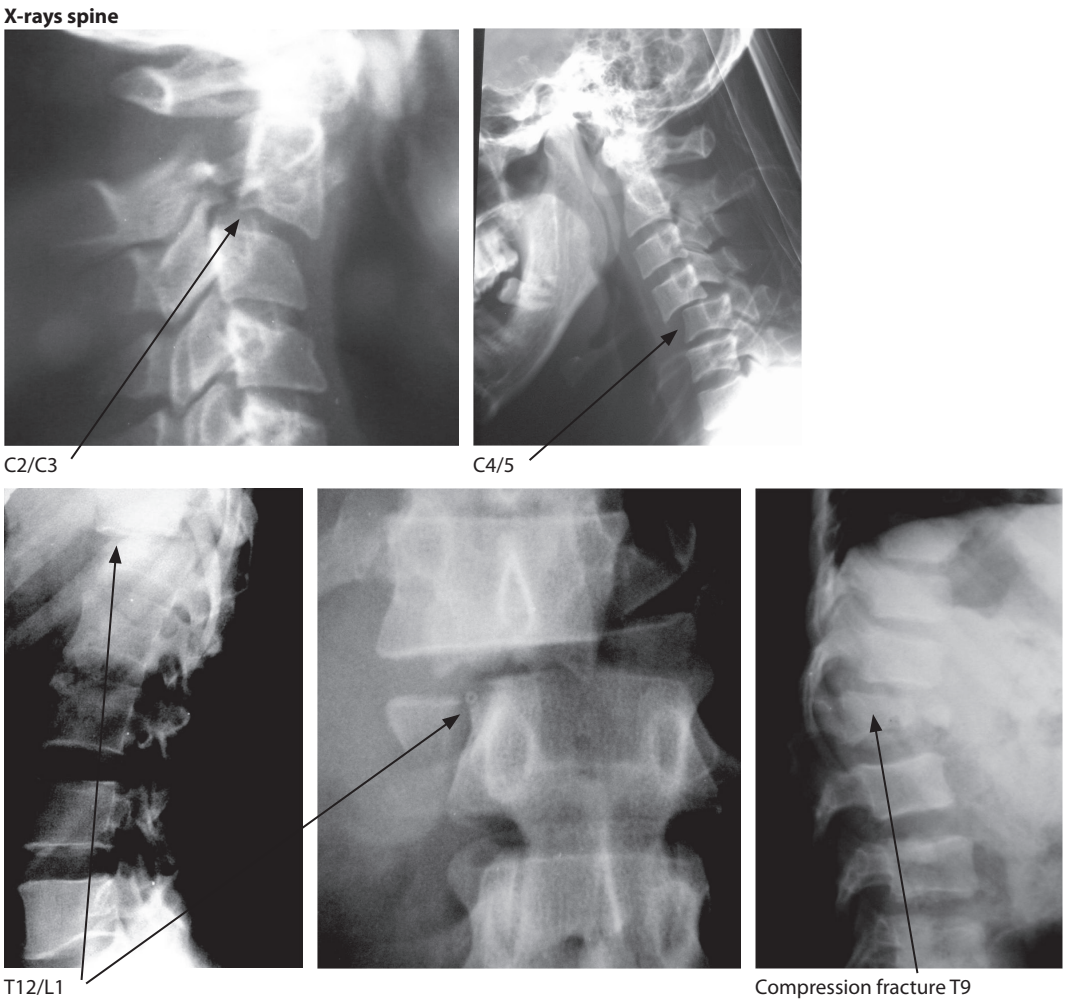


Figure 19.6 Fracture dislocation of spine

Imaging

Management depends on the site and stability of the injury. Straight X-rays of the spine will confirm obvious fractures and dislocations (Fig. 19.6). X-rays, AP and lateral of the cervical spine should visualize C1 down to the upper border of T1. CT scan may reveal fractures not visible on plain X-rays.

Management

In principle, an unstable cervical fracture risks further damage to the spinal cord and requires immobilization before being moved or mobilized. This can be done by operative fixation or skull traction with a Halo or plaster jacket immobilization for 12 weeks. If a cervical fracture is stable without cord injury, then a cervical collar is used. Thoracolumbar fractures are managed along the same general principles, although internal fixation is used less frequently.

General management includes support of arterial oxygenation and adequate spinal cord perfusion pressure. The use of high dose steroids in acute cord injury is still common practice although its value is controversial. Methylprednisilone in doses of 2 grams intravenously, if given within 8 hours of the spinal cord injury and continued over the first 24 hours may have a protective role but this it is not proven and may be actually deleterious. An up to date reference should be consulted before recommending their use. A urinary catheter should always be inserted. Very special attention from the onset should be paid to the prevention of respiratory complications, bedsores, urinary tract infections, DVT and limb contractures. Long term rehabilitation of paraplegia is already discussed in chapter 10.

Outcome

Patients with high cervical cord injuries seldom survive even with ventilatory support. Patients with a lesion above C7 remain dependent on others for continuous care. Patients with a lesion below C7 may be able to learn to transfer to wheelchair independently, while patients with lower spinal cord or cauda equina injuries gain complete wheelchair independence. Respiratory failure, infections and bedsores are the main complications of spinal cord injuries. Long initial periods of hospitalization of typically 3-6 months are usual, and mortality rates in Africa while recovering in hospital are frequently in the order of 20-30%.

Key points

- imaging is essential in the management of spinal injuries
- acute management includes stabilization of fractured spine & respiratory support
- scrupulous attention is needed to prevent bedsores and infection
- mortality rates are 20-30% and survivors need assistance with mobilization

PREVENTION

The main cause of head and neck injuries are RTAs. These are increasing at an alarming rate in Africa. They are mostly preventable by reducing both speed and dangerous driving. Measures to achieve these include adequate enforcing of traffic safety regulations, speed limits, road bumps, alcohol checks and ensuring that motor cyclists and cyclists wear helmets. Other causes of HI such as falls, work related accidents and violence are also partially preventable. General measures for prevention include better education and informed legislation.

Key points

- head and neck injury is a leading cause of death in Africa
- young adult males are a particularly high risk group
- main causes are RTAs and falls
- measures for primary prevention are now urgently needed

Selected references

Alexander T, Fuller G, Hargovan P, Clarke DL, Muckart DJ, Thomson SR. *An audit of the quality of care of traumatic brain injury at a busy regional hospital in South Africa*. S Afr J Surg. 2009 Nov;47(4):120-2, 4-6.

Benatar SR, Fleischer TE, Peter JC, Pope A, Taylor A. *Treatment of head injuries in the public sector in South Africa.* S Afr Med J. 2000 Aug;90(8):790-3.

Bruns J, Jr., Hauser WA. *The epidemiology of traumatic brain injury: a review*. Epilepsia. 2003;44 Suppl 10:2-10.

Casey ER, Muro F, Thielman NM, Maya E, Ossmann EW, Hocker MB, Gerardo CJ. *Analysis of traumatic injuries presenting to a referral hospital emergency department in Moshi, Tanzania*. Int J Emerg Med. 2012 Jun 8;5(1):28.

de Villiers JC. *Head injuries in South Africa*. S Afr J Surg. 1984 Feb-Mar;22(1):51-6.

Draulans N, Kiekens C, Roels E, Peers K. *Etiology of spinal cord injuries in Sub-Saharan Africa*. Spinal Cord. 2011 Dec;49(12):1148-54.

Fielingsdorf K, Dunn RN. *Cervical spine injury outcome--a review of 101 cases treated in a tertiary referral unit.* S Afr Med J. 2007 Mar;97(3):203-7.

Hart C, Williams E. *Epidemiology of spinal cord injuries: a reflection of changes in South African society.* Paraplegia. 1994 Nov;32(11):709-14.

Jennett B. *Epidemiology of head injury*. J Neurol Neurosurg Psychiatry. 1996 Apr;60(4):362-9.

Le Roux AA, Nadvi SS. *Acute extradural haematoma in the elderly.* Br J Neurosurg. 2007 Feb;21(1):16-20.

Leucht P, Fischer K, Muhr G, Mueller EJ. *Epidemiology of traumatic spine fractures*. Injury. 2009 Feb;40(2):166-72.

Lindsay Kenneth W, Bone Ian, *Neurology and Neurosurgery Illustrated,* Churchill Livingstone 4th edition 2004.

Neurological Disorders: *Global burden of neurological disorders*: WHO 2006

Nantulya VM, Reich MR. *The neglected epidemic: road traffic injuries in developing countries.* BMJ. 2002 May 11;324(7346):1139-41.

Nwadinigwe CU, Iloabuchi TC, Nwabude IA. *Traumatic spinal cord injuries (SCI): a study of 104 cases.* Niger J Med. 2004 Apr-Jun;13(2):161-5.

Omoke NI, Chukwu CO, Madubueze CC, Oyakhiolme OP. *Outcome of road traffic injuries received in the emergency room of a teaching hospital, Southeast Nigeria*. Trop Doct. 2012 Jan;42(1):18-22.

Seleye-Fubara D, Etebu EN. *Pathology of death from severe head injuries in Rivers State: a study of sixty eight consecutive cases in five years*. Niger J Med. 2011 Oct-Dec;20(4):470-4

Solagberu BA. *Spinal cord injuries in Ilorin, Nigeria*. West Afr J Med. 2002 Jul-Sep;21(3):230-2.

Tagliaferri F, Compagnone C, Korsic M, Servadei F, Kraus J. *A systematic review of brain injury epidemiology in Europe*. Acta Neurochir (Wien). 2006 Mar;148(3):255-68; discussion 68.

Umaru H, Ahidjo A. Pattern *of spinal cord injury in Maiduguri, North Eastern Nigeria. Niger* J Med. 2005 Jul-Sep;14(3):276-8.

Watters DA, Sinclair JR. *Outcome of severe head injuries in central Africa*. J R Coll Surg Edinb. 1988 Feb;33(1):35-8.

Wilson DA, Garrett MP, Wait SD, Kucia EJ, Saguda E, Ngayomela et al. *Expanding neurosurgical care in Northwest Tanzania: the early experience of an initiative to teach neurosurgery at Bugando Medical Centre*. World Neurosurg. 2012 Jan;77(1):32-8.

Winkler AS, Tluway A, Slottje D, Schmutzhard E, Hartl R. *The pattern of neurosurgical disorders in rural northern Tanzania: a prospective hospital-based study*. World Neurosurg. 2010 Apr;73(4):264-9.