Initial management of acute spinal cord injury

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Key points

Around 50% of patients with acute spinal cord injury (SCI) have an incomplete lesion; prevention of secondary injury is critical to future quality of life.

Patients with acute SCI often deteriorate in the first 72 h post-injury and must be carefully monitored.

Rapid sequence intubation is thought to be safe with in line stabilization acutely but succinylcholine is contraindicated from ~72 h to 6 months post-injury because of production of life threatening hyperkalaemia.

Do not sit patients up with acute high-thoracic SCI, they will breathe better lying flat and are also at risk of cardiovascular instability because of neurogenic shock.

Prevent avoidable complications: consider thromboprophylaxis, gastroprotection, and pressure area care (remove spinal board as soon as possible).

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A spinal cord injury (SCI) is defined as damage to the spinal cord caused by an insult resulting in the transient or permanent loss of usual spinal motor, sensory, and autonomic function. This article outlines the initial management of acute traumatic spinal cord injuries in adults. Approximately 14% of vertebral column fractures result in damage to the cord.[1] Of these, 50% will be incomplete, of whom 50% will walk by the time they leave hospital, if managed appropriately. Initial management is crucial to long-term survival and quality of life and is targeted at preservation of cord function together with prevention of avoidable complications.

While the annual incidence of SCI is relatively low (13 per million population in the UK and 35 per million in the USA), patients are often young (mean age 33) and years of good quality life are lost through severe morbidity and death. Patients who survive often have major psychological sequelae and physical dependency. In the USA, it is estimated each patient with a SCI costs US$320–985 000 in the first year of injury[2] and the lifetime cost of a young ventilated patient with SCI at US$5 million. Clinical outcome is determined by the level and degree of SCI, age, and associated complications.

Most of these patients will initially arrive in hospitals with no specific specialist spinal injury expertise and often exhibit significantly deranged physiology. Anaesthetists play a critical role in their early management. An understanding of the pathophysiology, treatment options, and potential risks in this patient group is essential to ensure avoidable damage to the cord does not occur and to prevent complications which may be life threatening or contribute to significant morbidity.

Anatomy

The vertebral column

The spinal canal and cord are protected by the vertebral column whose integrity depends on a series of ligaments joining together and strengthening the vertebrae (Fig. 1). The integrity and stability of the system may be functionally divided into three complexes, the anterior longitudinal ligaments with the anterior half of the intervertebral discs, the posterior longitudinal ligaments with the posterior half of the intervertebral discs, and the posterior complex of the ligamentum flavum, interspinous, and supraspinous ligaments joining the spinous and transverse processes. The posterior complex provides the most strength to the integrity to the vertebral column. If any two of these three complexes are disrupted, then the vertebral column is unstable.[1] Additional strength is given to the system by the inclination of the facet joints. The thoracic spine is also strengthened by the ribcage.

The commonest sites of injury occur where there is the greatest risk of conformational change where the spine changes in curvature and construction, namely the craniocervical, cervicothoracic, thoracolumbar, and lumbosacral junctions.[1]

The spinal canal

The spinal canal contains the spinal cord in a potential space filled with epidural fat and blood vessels. The size of this potential space varies considerably along the length of the canal. It is narrowest in the mid-thoracic region and unstable injuries here easily impact onto the cord, particularly retropulsion of bone fragments, leading to a high chance of complete cord injury.[3] In contrast, the upper cervical area has more space around the cord, particularly in the region of C1/2, and injuries at this level often do not produce associated cord injury.

The spinal cord extends from the brainstem to L1/2 in the adult and L2/3 in the neonate. Below this level, the cord terminates in the conus medularis, and the cauda equina, consisting of lumbosacral nerve roots. Injury at this level produces the cauda equina syndrome.

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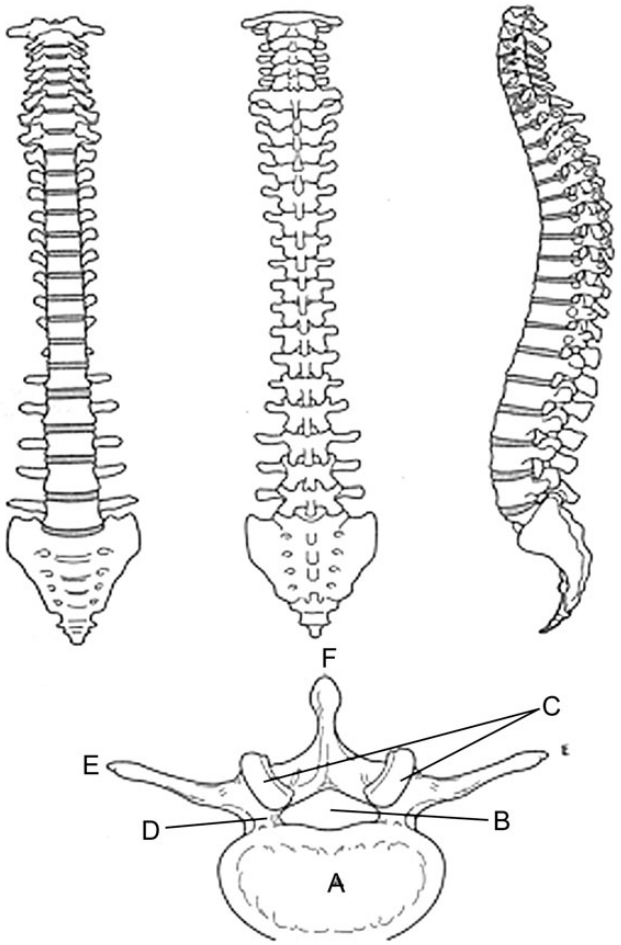

Fig 1 The anatomy of a typical vertebra: body (A), spinal foramen (B), facet joints (C), pedicle (D), transverse processes (E), and posterior spinous process (F). The facet joints are positioned to allow rotation in the cervical and thoracic but not the lumbar spine. (Reproduced with kind permission from the Taylor and Francis Group, *Trauma Care Manual*, 2nd Edn.[1] The vertebral column: anterior, posterior, and lateral view.)

The spinal cord

The function of the spinal cord is to allow both sensory and motor information to pass between the body and brain. The two major sensory pathways are the posterior columns and the spinothalamic tracts and the major motor pathway is the corticospinal tract (Fig. 2). The posterior column transmits sensory information for vibration, fine touch, and proprioception on the ipsilateral side of the body (fibres cross over in the medulla). The spinothalamic tracts carry pain, temperature, and coarse touch on the contralateral side of the body (fibres cross over to the contralateral side at 1–2 vertebral bodies above their level of sensory innervation). In brief, motor fibres from the cortex descend to the contralateral side of the body via the corticospinal tracts.

The arterial supply of the spinal cord is via the anterior and posterior spinal arteries (Fig. 3), branches of the vertebral, and posterior inferior cerebellar arteries respectively. Radicular arteries, including

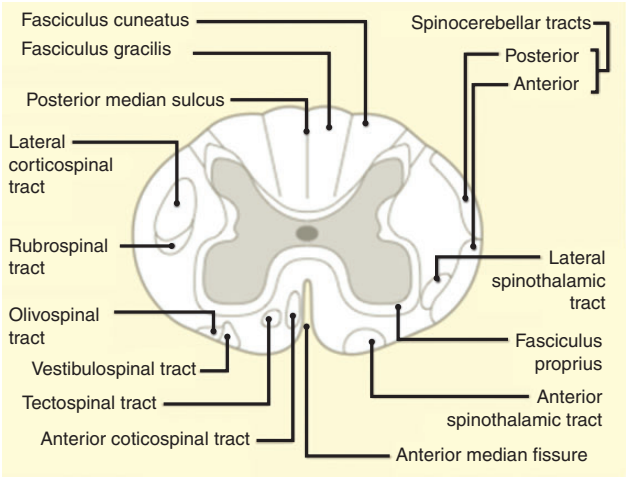

Fig 2 Cross-section of spinal cord showing ascending pathways on right and descending pathways on left. Reproduced with kind permission from Elsevier. *Anaesthesia and Intensive Care Medicine* 2005.[4]

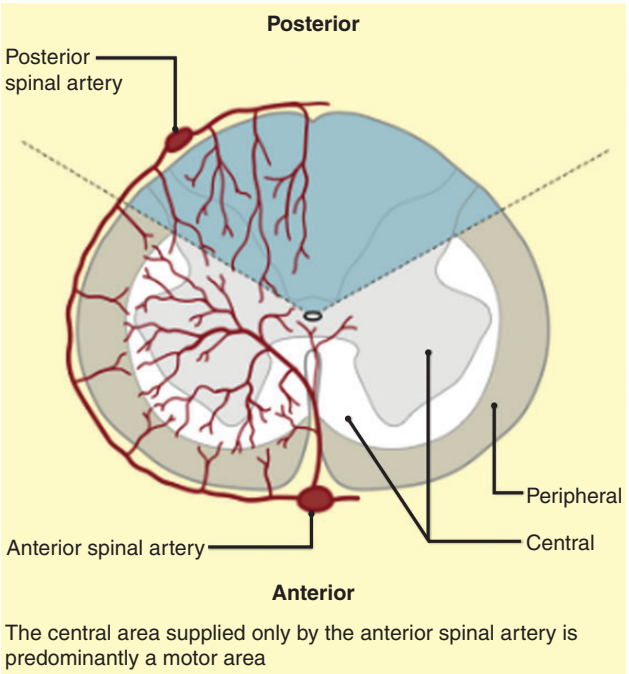

Fig 3 Blood supply to the spinal cord: horizontal distribution. (Reproduced with kind permission from Elsevier. Craven J. Spinal cord. *Anaesthesia and Intensive Care Medicine* 2004; **5**: 146[5].)

the arteries of Adamkiewicz also contribute especially in the thoracic region. The anterior spinal artery is particularly vulnerable to retropulsion of bone and disc fragments and may give increase to cord infarction in watershed areas, particularly at C6/7.

Injury at any level may produce a variety of clinical symptoms and signs depending on the nature of the insult to the cord and the

anatomical area damaged. This can range from mild paraesthesia to complete motor, sensory, and autonomic paralysis.

Incidence and mortality

There are ~1200 new cord-injured patients per year in the UK cared for in 12 specialist centres.[6] In-hospital mortality is quoted at 5–18% for acute lesions and is dependent predominantly on age and the level of injury. Associated higher mortality in age >60 is attributable to poor cardiorespiratory function, increased risk of complications, and higher incidence of high cervical injury.[3] Life expectancy post-injury also depends on injury severity, level of injury and patient's age and ranges from 1.5 years (ventilator dependent, any level, age 60) to 52.6 years (preserved motor function, any level, age 20).[2] Most patients who die in the chronic phase die of potentially avoidable complications such as thromboembolic disease and sepsis (chest, urinary tract infection, and pressure sore related) and the origins of some of these begin in the acute treatment phase. With correct treatment immediately after injury, cord damage may be minimized, and potential morbidity avoided (in particular pressure sores) so the life expectancy of a young patient with paraplegia approaches that of the normal population.

Aetiology

Traumatic SCI

The patterns of SCI vary between country, ethnic origin, age, and gender. It is more common in young men (80% male, mean age 33)[2, 3, 6] In the UK, ~40% are associated with vehicle accidents and only 3% with penetrating trauma or assault. Falls account for 40%[6] and are most common in the elderly, where they often occur falling at ground level in patients with known degenerative diseases of the spine.[3] Sport accounts for 11% of SCI in the UK: particularly horse riding, rugby, and diving.[6] SCI may also result from non-traumatic causes such as degenerative disease, infection, toxins, tumours, cysts, inflammation, disruption of spinal cord blood supply, and congenital abnormalities.[7] The incidence of penetrating SCI secondary to violence has increased especially in developing nations as a result of increased gun and knife crime.[8]

Pathophysiology

Primary injury

Initial damage results from direct cord compression, haemorrhage, and traction forces. The commonest mechanism in trauma is **subluxation** of the vertebral elements causing a pincer like direct damage to the cord. This can cause complete cord transection in extreme cases. **Hyperextension** injuries are common in the elderly. This can cause compression of the cord between the ligamentum flavum and anterior osteophytes. **Retropulsion** of bone or disc fragments, particularly in burst fractures, can cause either damage to the cord via direct cord compression or via impairment to the vascular supply, the anterior spinal artery being particularly vulnerable. Penetrating injury can also cause direct compression and vascular injury.[1,7,8]

An associated fracture may or not be present and is dependent on site of injury. Pickett and colleagues (n=151) reported an associated spinal fracture present in only 56% of patients with cervical SCI, compared with 100% in those with thoracic SCI, and 85% with lumbar SCI.[3]

Secondary injury

Within minutes after injury, secondary damage begins. Haemorrhage in the central grey matter occurs and axons and neuronal cell membranes become damaged.[7] These events lead to spinal cord oedema and subsequent spinal cord ischaemia. Loss of autoregulation with high thoracic lesions contributes to neurogenic shock. Untreated the resultant decrease in systemic arterial pressure may lead to further cord hypoperfusion and spreading penumbra of damage.[7]

Cord ischaemia extends bi-directionally, along the site of injury within hours and secondary SCI continues. Within the first 72 h, this may manifest as an ascending SCI level, which may lead to clinical deterioration.

Systemic effects, local vasomotor changes, the release of free radicals, intracellular electrolyte shifts, neurotransmitters, cord oedema, disruption of cell metabolism, and cell death are all thought to play their part in secondary injury[7,8] most of which have no specialist treatment but have resulted in the search for drug suppression of inflammation to limit secondary injury.[1]

Prevention of secondary cord damage may make a dramatic difference to the quality of life of a SCI patient. A tetraplegic with damage at C4, may present with diaphragmatic breathing but not have respiratory failure. If the cord damage extends only one level higher, phrenic nerve (C3–5) innervation will be lost, with resultant respiratory failure, and need for ventilation. If permanent, this patient will require long-term ventilation.

Neurogenic shock

Neurogenic shock is the interruption of autonomic pathways leading to hypotension and bradycardia (and hypothermia).[1,9] It is common in injuries involving cardiac sympathetics (T2–5) resulting in a decrease in systemic vascular resistance, decreased inotropism, and increased unopposed resting vagal tone.

Spinal shock

Spinal shock is the loss of reflexes below the level of SCI resulting in the clinical signs of flaccid areflexia and is usually combined with hypotension of neurogenic shock. There is a gradual return of reflex activity when the reflex arcs below redevelop, often resulting in spasticity, and autonomic hyperreflexia. This is a complex process and a recent four-phase classification to spinal shock has been postulated: areflexia (Days 0–1), initial reflex return (Days 1–3), early hyperreflexia (Days 4–28), and late hyperreflexia (1–12 months).[10]

Table 1 Definitions and clinical syndromes associated with SCI that have been agreed by the International Standards for Neurological and Functional Classification of SCI (adapted from[11]). ASIA, American Spinal Injury Association.

The ASIA impairment scale (modified from the Frankel Classification)	This is an internationally recognized scale to classify the clinical extent of SCI. Impairment is classified from A–E, the estimated incidence of each type of injury in brackets and bold
A=Complete	No sensory or motor function is preserved in the sacral segments S4–S5 (**45%**)
B=Incomplete	Preservation of sensory but not motor function below the neurological level and includes the sacral segments S4–S5 **(15%)**
C=Incomplete	Preservation of motor function below the neurological level. More than half of key muscles below the neurological level have a muscle grade of <3 (**10%**)
D=Incomplete	Preservation of motor function below the neurological level. More than half of key muscles below the neurological level have a muscle grade of ≥ 3 (**30%**)
E=Normal	Sensory and motor function is normal
Neurological level	The level of a SCI is the lowest level of the spinal cord with normal sensation and motor function on both sides of the body
Tetraplegia	This is attributable to a lesion or injury within the cervical spinal cord. There is incomplete or complete loss of motor (sensory function in the arms, the torso, pelvic organs, and the legs)
Paraplegia	This is attributable to a lesion or injury within the thoracic, lumbar, or sacral spinal cord. There is incomplete or complete loss of motor (sensory function of the torso, pelvic organs, and the legs). The level of injury will affect which of these are affected. Arm function is preserved
Anterior spinal artery syndrome	The anterior spinal artery runs as a single artery anterior to the cord and supplies the anterior 2/3 of the cord. Transection therefore produces sparing of the dorsal columns (Figs 3 and 4), resulting in paralysis and loss of pain and temperature with preservation of proprioception, fine touch, and vibration
Brown-Séquard syndrome	This is caused by lateral cord damage. It may occur because of osteophyte impaction on half of the cord producing sensorimotor damage at the level of the injury. There is ipsilateral loss of motor function, fine touch, proprioception and vibration and contralateral loss of pain, and temperature below this level
Cauda equina syndrome	Bladder and bowel dysfunction associated with upper motor neurone symptoms and signs in the legs caused by injury to the lumbosacral nerve roots
Central cord syndrome	Results from bleeding, infarction, or oedema to the central grey matter of the spinal cord. This is most common in the cervical region where it presents as upper motor neurone signs in the legs and mixed upper and lower motor signs in the arms with loss of pain and temperature sensation in the arms. Sacral nerve fibres are positioned laterally in the cord and the patient may demonstrate sacral sparing of sensation. This indicates incomplete cord damage and therefore offers the theoretical chance of some recovery of the cord
Posterior cord syndrome	Produces loss of vibration and proprioception. This is associated with damage to the posterior spinal artery and is very rare

Classification of injury

A SCI affects the functioning of the spinal cord at the level of injury. There are a number of associated definitions and clinical syndromes (Table 1). It is important to discriminate between complete and incomplete injury. Up to 80% of patients with incomplete paraplegia will stand by 12 months, 50% will walk out of hospital within 12 months, and patients may show neurological improvement up to 2 years after injury.

Initial management of the SCI

The immediate resuscitation phase follows the basic principles of 'ABC'.

SCI in polytrauma is common (30%) and needs to be considered.[12] One-third of patients with acute SCI may also have associated major injuries or other spinal fractures[8,12,13] and injuries to the head, chest, abdomen, pelvis, lower limb, and upper limb all need to be excluded.[1,3,13] Some may be life threatening and will require urgent attention; preserving spinal alignment at all times is imperative.

Extraction and transport

Spinal immobilization is indicated if a patient has sustained an injury with a mechanism compatible with spinal damage. Any patient with spinal pain or tenderness, neurologic deficit, depressed level of consciousness, drug or alcohol intoxication, or a painful distracting injury should be immobilized at the scene with a hard collar. They should be transferred to hospital with surrounding head blocks on a spinal board. Patients must be 'log rolled' off the board, ideally within 30 min after arrival in hospital. The spinal board is a transport device only and its prolonged use is associated with pressure sores. SCI patients should continue to have spinal immobilization according to specialist advice and be 'log rolled' for transfer at all times until spinal fixation.

Airway

Patients should be well oxygenated at all times to prevent secondary cord damage and a low threshold for intubation is required. Patients with high cervical lesions around C3–5 (diaphragmatic innervation) are especially at risk of deterioration, with vital capacities $<30\%$ of predicted and 80% of all cervical cord injuries require ventilation at some point, most within the first 48 h. Patients with SCI are also at increased risk of regurgitation and pulmonary aspiration of gastric contents because of paralytic ileus and loss of gastroesophageal sphincter tone.

In the acute setting, a rapid sequence induction (RSI) should be used. Before induction the patient's hard collar and head blocks should be removed and manual in line cervical spine stabilization (MILS) should be initiated by a second operator. Cervical spine movement should be minimized during laryngoscopy, especially flexion, which is thought to be more dangerous to the cord than extension.[14] Applying cricoid pressure with the posterior part of the

hard collar still in place (with MILS) with the assistant's second hand supporting the posterior part of the hard collar may further decrease cervical movement as this does not necessitate removing the whole collar.[15] The hard collar and head blocks should be replaced after intubation.

Associated maxillofacial trauma, blood or vomit in the upper airway, airway oedema secondary to direct trauma, or access problems such as cervical immobilization devices including halo traction can all make intubation difficult. Difficult airway equipment including a fibre-optic bronchoscope should be immediately available and awake fibre-optic intubation (AFI) may also be considered. The spine must be immobilized if AFI is undertaken. No individual airway technique is superior, it is more important to avoid hypoxia and use familiar methods. Despite direct and indirect intubation techniques and cricoid pressure all being associated with spinal movement, this movement is unlikely to result in neurological injury providing reasonable care is taken.[16]

It must be remembered that after 72 h post-injury succinylcholine should be avoided as it may precipitate life threatening hyperkalaemia. Acute denervation causes acetylcholine receptors to spread beyond motor end plate of the neuromuscular junction, increasing receptor exposure to succinylcholine, an effect that wears off at ~6 months. Gastric emptying remains reduced so a modified RSI (or AFI if airway or access limited) may be considered in the weeks after initial injury.[17]

Breathing

The effect on breathing is dependent on the site of injury (see Table 2). Cord injury above T1 removes intercostal function and respiration will be entirely diaphragmatic. Lesions involving the phrenic nerves (C3–5) will impair diaphragmatic function and if the damage is above C3 the patient will be permanently ventilator dependant unless there is partial recovery of the cord.

Any patient with cervical cord injury or those demonstrating signs of high cord injury with inability to cough or diaphragmatic breathing should be monitored acutely in a critical care setting as they may deteriorate neurologically especially in the first 72 h post-injury and again early intubation should be considered.

It is important to remember that in the acute phase high cord-injured patients will have better respiratory function lying flat as the diaphragm has a greater excursion in inspiration as it is pushed into the chest by abdominal contents, whereas if sitting up the diaphragm is pulled down by abdominal contents impeding further excursion in inspiration. Patients should, therefore, not sit up for the first few days after injury and thereafter only gradually as intercostal paralysis develops and the chest no longer collapses in during inspiration preserving the smaller tidal volumes produced by diaphragmatic descent.

It is important that early assessment of respiratory function, vital capacity, pulmonary recruitment, and passive limb movements by an experienced spinal injury physiotherapist is carried out.

Cardiovascular

In the minutes after cord injury a massive release in catecholamines occurs, particularly after high cervical injury producing dramatic hypertension and tachycardia. Following this phase, paralysis of sympathetic tone leads to hypotension. This is attributable to a combination of vasodilatation, decreased inotropism, and bradycardia (if above T5 and cardiac sympathetics are involved). Bradycardia occurs because of unopposed vagal tone and intermittent atropine or glycopyrrolate may be required, especially before vagally stimulating procedures such as laryngoscopy or tracheal suctioning as the incidence of asystole in severe cervical injury may be as high as 20%.

Classically, the patient presents with hypotension and bradycardia, but they may be warm and vasodilated. Systolic arterial pressure will often settle at 80–90 mm Hg but this is unpredictable. Hypotension may be fluid resistant and most patients with high cord injury require vasopressor support. Overtransfusion of fluid may lead to pulmonary oedema as capillary integrity of the pulmonary circulation may be impaired as a result of the catecholamine surge. Invasive pressure monitoring should be used for cord injuries associated with neurogenic shock, i.e. above T4. The neurogenic shock phase lasts from 24 h to several weeks.

Early catheterization is essential not only to act as a marker of renal perfusion but also to avoid bladder overdistension that may

Table 2 Level of injury and effect on respiratory function

Level of injury	Nerve denervation	Vital capacity (% of normal)	Effect on cough	Need for ventilation
Above C3	Loss of phrenic nerves. Total diaphragmatic paralysis if bilateral C3 injury	<10%	Absent	Immediate and long-term ventilation
Between C3–C5	Partial phrenic nerve denervation/weakness/ paralysis of diaphragm	10–30%	Weak/ineffective	Ventilation required in 80% within 48 h. Older patients may need long-term ventilation. Role of non-invasive ventilation in weaning
Above T8	Loss of inspiratory intercostal muscles/abdominal muscles	30–80%	Normal to weak cough	Short-term ventilation sometimes required because of impaired sputum clearance. If respiratory co-morbidities, may occasionally still need long-term support
Below T8	Loss of abdominal muscles and lower expiratory intercostals forced cough may be weak especially in older patients	80–100%	Normal to weak cough	Short-term ventilation occasionally required because of weak cough and impaired sputum clearance

precipitate bradycardia. Consider supra pubic catheterization if priapism is present.

Neurological imaging and examination

Examination

Signs that may indicate spinal injury (especially in the patient with the depressed level of consciousness) include:

- Diaphragmatic breathing,
- Hypotension without obvious cause,
- Bradycardia,
- Priapism,
- Flaccid areflexia (e.g. in legs but tone in arms), and
- Loss of pain response below a level.

A thorough evaluation should be undertaken as soon as possible and should include assessment of motor function, sensation, respiratory function, reflexes, and anal tone on log rolling (sacral sparing). It should also document whether the injury is complete or incomplete and whether a clinical syndrome is suspected. A standardized form has been produced by The American Spinal Injury Association (ASIA) to assist in this assessment (http://www.asia-spinalinjury.org/elearning/ISNCSCI_Exam_Sheet_r4.pdf).

Imaging

Plain X-rays have largely been superseded by computerized tomography (CT) and magnetic resonance imaging (MRI). Early total body CT is crucial in excluding other life threatening injuries in trauma patients with a cord injury to exclude occult haemorrhage as usual physiological response to shock is impaired. Therefore, neurogenic shock may mask other injuries such as ruptured spleen/fractured pelvis, etc. and the patient may also be unable to describe pain below the level of injury. Spinal reformatting of CT images will aid immediate assessment of vertebral column, assist in spinal clearance, and give some information on cord integrity (Fig. 4).

Early MRI has a role in investigating cord integrity and guiding early surgery (e.g. epidural haematoma identification). It is a balance of risk *vs* benefit in this unstable group, ventilated patients, and those on vasopressors or inotropes should be carefully monitored by experienced personnel if MRI is undertaken.

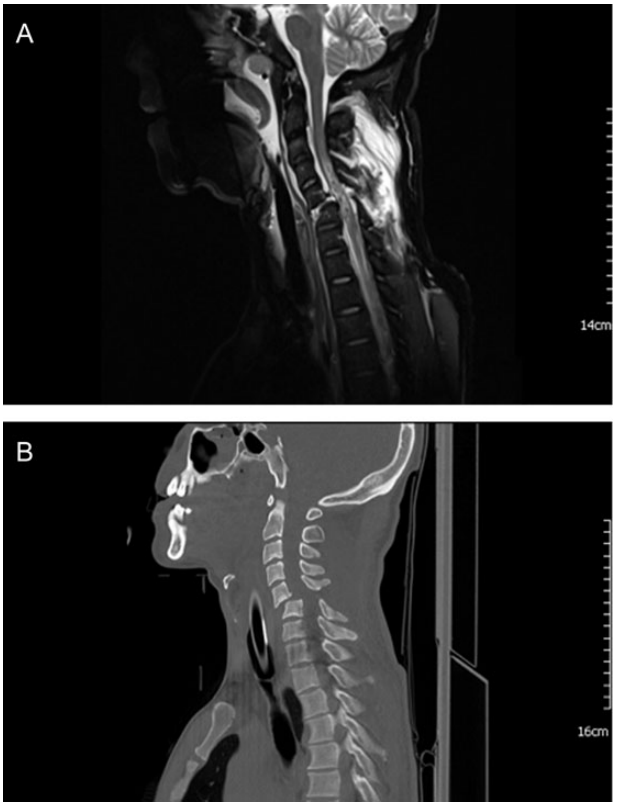

Fig 4 (A) MRI of cervical spine showing C5/6 bilateral facet displacement with disc disruption and extensive soft tissue disruption. Anterior displacement of C5 vertebrae on C6 vertebrae is demonstrated. There is cord swelling and contusion and a focal intramedullary haematoma at the level of C5/6, the cord oedema extends form C2/3 to C7. (B) CT of the same patient showing marked anterior subluxation of C5 on C6 and bilateral facet lock of C5/6. This CT is consistent with an unstable hyperflexion injury.

Neuroprotection strategies in SCI

There are no magic bullets in protecting the cord, but avoidance of hypoxia, hypotension, and hypercarbia are crucial in the days after injury. Some specific areas of investigation include:

Vasopressor support

Early vasopressor support has been advocated to ensure adequate spinal cord perfusion pressure and reduce secondary cord injury. There is weak evidence from historical cohort studies that a mean arterial pressure of >85 mm Hg for 5–7 days may be associated with a better functional outcome,[9] but given potential complications of prolonged inotropic support it seems pragmatic to also target arterial pressure to age of the patient and presence of co-morbidities such as hypertension, ischaemic heart disease, renal insufficiency, etc. Given fluid resistant arterial pressure in some patients and predisposition to pulmonary oedema it has been recommended that if >2000 ml of fluid is required acutely, further fluid should be target based with invasive monitoring and inotropes started. There is little evidence for choice of inotrope, although vasoconstriction appears logical as first line.[9]

Therapeutic hypothermia

A number of animal studies showing a potential benefit in functional recovery have been reviewed; however, human studies are limited and at present cooling is not recommended.[18]

Steroids

The high-profile NASCIS II study recommended use of steroids demonstrating a small reduction in the level of injury in those treated early with high dose methylpredisolone, and this has recently

been ratified in a review.[19] However, concerns over the mortality and rates of sepsis in the steroid group have led to them not being recommended by the British Association of Spinal Cord Injury Specialists and are not routinely used in the UK or in the USA.[17]

Surgery

Stabilization, open or closed reduction, and surgical decompression must be considered to relieve direct pressure on the cord and prevent secondary injury. Urgent stabilization should be considered in patients with any deterioration in neurology.[16] Early surgical decompression has been shown to reduce length of stay and days of mechanical ventilation but not injury level.[20,21]

Other considerations

Thromboprophylaxis

Fatal pulmonary embolus occurs in 3% of SCI patients and rates of deep vein thrombosis and non-fatal PE are 90 and 10%, respectively. These patients are at high risk both because of immobility and increased thrombogenicity secondary to trauma. The use of anticoagulants should be restricted in the first 48–72 h because of risk of bleeding around the cord and intermittent calf compression devices or graduated compression stockings should be used instead. Standard prophylactic low molecular weight heparin should be started after 72 h. If anticoagulant use is contraindicated early insertion of an IVC filter may be considered.[17]

Gut protection

Unopposed vagal activity increases gastric acid and therefore rates of peptic ulceration. A routine use of prophylactic H_2 antagonists or proton-pump inhibitors for 6 weeks significantly reduces duodenal ulceration from an incidence of $\sim$20 to $<$5%.

Nutrition/glycaemic control

Despite normal bowel sounds and increased gastric acid secretion, unopposed vagal activity may also lead to a gastroparesis. Feeding patients with high cord lesions may lead to nausea, vomiting, risk of aspiration, and abdominal distension, further impairing respiration. However, early enteral feeding decreases mortality in polytrauma patients and it is usual practice to feed all intubated patients within 24 h. Glycaemic control is essential to avoid both hypo- and hyperglycaemia.

Pressure areas

Pressure sores are devastating for cord-injured patients leading to prolonged immobilization or severe sepsis. These usually develop in the first few days after admission to hospital and are a result of immobility, poor perfusion of the skin, hypoxia, and leaving patients on spinal boards. Appropriate mattresses and good nursing care are essential to reduce pressure sores and early spinal fixation will allow earlier mobilization.

The future

Current research is not only focusing on secondary injury prevention, but also investigating more novel ways into the repair, remodelling and remyelination of damaged neurones, the regeneration of lost connections within the spinal cord, and replacement of lost nerve cells.[22] The use of anti-oxidants, anti-inflammatory agents, apoptosis-blocking drugs, calpain-inhibitors (a calcium-activated enzyme involved in post-traumatic axonal damage mediation), and naloxone and thyrotropin releasing hormone as opioid antagonists thereby inhibiting injury-induced endorphin release have all been considered.[23] The use of stem cells, gene therapy and application of electrical stimulation to the damaged spinal cord are being investigated. The creation of a spinal neurone from stem cells heralds potential advances for the future.

Conclusions

Given the nature of physiological derangement exhibited by SCI patients, the role of the anaesthetist is crucial in optimizing outcome for this high-risk group of patients. Good quality early treatment and prevention of associated complications can make a dramatic difference to mortality and in particular the patient's functional outcome.

Declaration of interest

None declared.

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Please see multiple choice questions 29–32.