Cervical cord injury and critical care

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The majority of spinal cord injuries (SCI), involving previously healthy young adults, result from trauma; 37% occur after road accidents, 42% follow falls, 11% are associated with sports and recreational activities, and 3% after assault.1 There is a male predominance of 4:1.

Respiratory complications are the leading cause of death after cervical cord injury. Marked changes in respiratory physiology occur and recovery can be prolonged.

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Key points

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Respiratory system

The impact of SCI on respiratory function depends greatly upon the level of injury (Table 1).

Physiological changes after SCI

Normal inspiration involves the external intercostal muscles contracting to lift the ribcage, expanding the antero-posterior (AP) diameter of the chest. The diaphragm, the principle muscle of inspiration, performs three actions:

(i) as a pure piston contracting downward, increasing intrathoracic volume;
(ii) by flattening, it functions as a piston governed by Laplace’s law;
(iii) interacting through the zone of apposition with the lower ribcage, the abdominal contents act as a fulcrum to expand the lower ribcage.

After SCI, intercostal muscle function is lost, with consequent failure of AP expansion of the ribcage. More importantly, without intercostal contraction, as the diaphragm contracts, the chest wall is sucked in causing paradoxical chest wall movement.

Although innervation to the diaphragm is usually intact (lesions below C3), its function is also greatly affected post-SCI. Lost innervation to the lower thoracic segments causes the diaphragm to start at a more caudal position which increases its radius of curvature and, from Laplace, will reduce trans-diaphragmatic pressure on contraction. Additionally, as the diaphragm descends, due to lost abdominal muscle tone, abdominal contents are pushed out and fail to provide the fulcrum needed to expand the lower chest. The lower ribcage is pulled in while the abdomen is pushed out resulting in the ‘see-saw’ pattern of respiration seen. Finally, the diaphragm is pulled down by the weight of the abdomen, especially when upright, dramatically reducing apposition between diaphragm and chest wall.

The rapid shallow breathing which ensues is inefficient—a greater percentage of each breath ventilates dead space and more time is spent in inspiration, when contracting muscles receive proportionally less of their blood flow. Lost abdominal muscle activity results in a decrease in maximal expiratory force2 and a reduced ability to cough, clear secretions, and protect the airway. Atelectasis increases the load placed on already compromised inspiratory muscles and V/Q mismatching occurs. Alveolar hypoventilation is inevitable and respiratory failure very common.

Some of these changes can be reduced by maintaining patients in a supine position. When supine, the weight of abdominal contents pushes the diaphragm higher into the chest, increasing apposition with the ribcage, reducing radius of curvature, and restoring the fulcrum lost with higher abdominal compliance. Supine values of FVC and FEV1 are larger compared with values when seated, down to an injury level of T13 (Table 2).
Reduced compliance Due mainly to a loss of gas-containing alveoli secondary to atelectasis. Reduced lung volume compounds the problem by reducing surfactant production.

(ii) Quad cough. External upper abdominal and lower costal margin pressure is exerted in time with a cough after a vital capacity breath.

(iii) Insufflation–exsufflation device (e.g. CoughAssist®). A portable electric device that is very effective at clearing secretions. By causing a rapid change in pressure gradient from positive to negative, expiratory flow is increased and a cough instigated. It can be applied by a face mask or via a tracheal tube or tracheostomy and patient satisfaction is high.

**Mucolytics**

Nebulized N-acetylcysteine, oral carbocysteine, or rhDNase (pulmozyme) can help to loosen and clear secretions.

**Posture**

Frequent turning of patients is necessary to avoid pressure sore formation and to prevent atelectasis. Supine, rotating, and percussive beds are available to mechanize this. Early surgical stabilization allows for mobilization and removal of halo jackets (which reduce lung volumes), although early surgery increases the need for post-operative ventilation.

**Abdominal binding**

Abdominal binding and nursing patients supine can offset the decline in pulmonary function due to flaccid abdominal muscles.

**Preventing fatigue**

Work of breathing can be gauged by assessing patient comfort, trends in respiratory rate, changes in the ability and quality of speech, and monitoring for increasing 

\[ PaCO_2 \]

X-ray evidence of worsening volume loss, lobar collapse, and infection [together with white cell count (WCC), C-reactive protein (CRP), temperature, and sputum culture] require more aggressive treatment. Spirometry is an objective measure of respiratory reserve with FVC < 12–15 ml kg⁻¹, an indicator for assisted ventilation.

**Non-invasive ventilation**

Continuous positive airway pressure (CPAP) can increase functional residual capacity (FRC) thereby improving compliance and reducing work of breathing, but excessive CPAP can increase work of exhalation. Bilevel or pressure assisted non-invasive ventilation (NIV) is usually more beneficial, increasing minute ventilation and providing assistance to overloaded and failing muscles. NIV may be useful before surgery and as a means of offsetting intubation or as a step down after tracheal extubation or decannulation.

**Bronchoscopy**

This is the most direct way of clearing secretions, obtaining broncho-alveolar lavage specimens, and re-inflating collapsed lung segments.

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**Table 1** SCI level with likely effect on ventilation

<table>
<thead>
<tr>
<th>Level of injury</th>
<th>Effect on respiration</th>
<th>Clinical consequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1–3</td>
<td>Complete paralysis of all respiratory muscles</td>
<td>Apnoea and immediate death unless mechanical ventilation is applied. Ventilator-dependent unless a diaphragm stimulator is used.</td>
</tr>
<tr>
<td>C3–5</td>
<td>Varied impairment of diaphragmatic contraction (see text)</td>
<td>Ventilation often necessary in the acute stages. Vast majority will wean from mechanical ventilation depending upon functional descent of injury level, recovery of function in incomplete lesions, and improvement in respiratory mechanics over time</td>
</tr>
<tr>
<td>C6–8</td>
<td>Diaphragm and accessory cervical inspiratory muscles intact. Intercostals and abdominal muscles paralyzed</td>
<td>Expiration entirely passive. Secretion retention is a problem. Respiratory failure rarely seen unless co-existent chest/lung injury, pre-existing lung disease, or the need for surgery</td>
</tr>
</tbody>
</table>

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**Early respiratory management**

**Airway**

One-third of cervical SCI patients will require intubation at some point. Suxamethonium use should be restricted to within 48 h after injury to avoid excessive potassium release, and the unpredictable response to other neuromuscular blocking agents mandates neuromuscular monitoring. Any intervention before the neck is stable or fixed requires cervical in-line stabilization and log-rolling.

**Physiotherapy**

Physiotherapy reduces accumulation of secretions and hence load placed on fatiguing muscles. Physiotherapists can assist coughing by the following.

(i) Augmenting spontaneous cough. By moving the patient forward while seated, intra-abdominal pressure and expiratory flow are increased. Contraindicated in patients with other injuries or spinal instability.

(ii) Quad cough. External upper abdominal and lower costal margin pressure is exerted in time with a cough after a vital capacity breath.

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**Table 2** Lung volumes in cervical cord injury

<table>
<thead>
<tr>
<th>Spirometry</th>
<th>FEV1 and FVC. Maximal reduction immediately post-injury (33% of predicted). Improves initially up to 5 weeks (45% predicted), with more gradual improvement at 5 months (60% predicted)²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung volumes</td>
<td>As level of injury ascends, TLC is progressively reduced</td>
</tr>
<tr>
<td>Restrictive ventilatory impairment</td>
<td>FRC=RV+ERV. The reduction in FRC occurs at the expense of expiratory reserve volume, with a compensatory increase in RV. Loss of ERV means forced exhalation and coughing is severely impaired</td>
</tr>
<tr>
<td>Reduced compliance</td>
<td>Due mainly to a loss of gas-containing alveoli secondary to atelectasis. Reduced lung volume compounds the problem by reducing surfactant production⁴</td>
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Mechanical ventilation
Where there is an associated lung injury, for example, a contusion or underlying infection, traditional lung protective ventilation is preferred to avoid worsening lung damage. Where respiratory failure is mainly due to pure ventilatory failure (i.e. with a low FiO₂), large tidal volume (>20 ml kg⁻¹) ventilation has been advocated, as there is evidence that it lessens atelectasis and accelerates weaning.

Weaning from mechanical ventilation
C1–3 tetraplegics who cannot be weaned are managed with domiciliary ventilation. Patients with an SCI of C4 and below are potentially weanable,2 but only once active pulmonary pathology has resolved and the chronic changes to pulmonary mechanics have commenced. After SCI, intercostal muscles are initially flaccid, but over the ensuing weeks, spasticity develops and the chest wall becomes more rigid with less tendency to collapse with inspiration. Individuals with cervical SCI continue to experience compromised expiratory muscle function, but the action of the clavicular portion of pectoralis major muscle, by compressing the rib cage and increasing intrathoracic pressure, can contribute to an improved cough.8

Long-term respiratory management
Tracheostomies
Tracheostomies have clear advantages, especially when weaning may take weeks. Patients find them comfortable, and they minimize laryngeal damage from prolonged intubation, have less dead space compared with a tracheal tube, and are associated with fewer respiratory infections.2 Tracheostomies interfere with surgical anterior fixation.

Weaning strategies
After conversion to pressure support (PS) ventilation and in the absence of mandatory ventilation, two common weaning strategies exist:

(i) PS weaning, where incremental reductions in the level of PS are carried out until ultimately patients are converted to high flow or CPAP breathing.

(ii) T-piece weaning (progressive ventilator-free breathing) or ‘sprint weaning’. Once PS levels reach 12–15 cm H₂O, rather than reducing PS further, patients breathe without PS either on a high flow circuit (with or without CPAP) or simply through a heat and moisture exchange device (HME), for a predetermined time every hour. The duration free of PS is gradually increased (sometimes by as little as a few minutes per hour) until they can attain full or partial independence from mechanical assistance. It is important that during ‘sprinting’ patients are rested at night to prevent exhaustion.

Studies, albeit involving small numbers of patients, suggest that T-piece weaning is as effective or better when compared with PS weaning with both superior to synchronized intermittent mandatory ventilation (SIMV) weaning.2, 9

Speaking
Establishing communication is essential for the wellbeing of SCI patients. Although cuffed tracheostomies allow mouthing of words, speech is possible by using fenestrated tracheostomies and deflating the cuff. During this time, CPAP and PS values will not be achieved, and respiratory effort will increase. Therefore, speech must be factored into any weaning programme. Increasing PS before cuff deflation can compensate for the leak while speech is occurring and prolonging inspiratory time and increasing PEEP may improve speech quality. A one-way Passy-Muir speaking valve may be used, again with the cuff deflated, to permit air drawn in via the tracheostomy to be exhaled upwards through the larynx.4

Exercise training
Exercise training (upper and lower body exercises) results in increased maximal oxygen uptake, increased muscle strength, and improved muscle co-ordination.10

Diaphragmatic pacing
Diaphragmatic pacing can restore inspiratory muscle function in tetraplegics.4 Upper airway obstruction induced or worsened by diaphragmatic pacing often necessitates permanent tracheostomy. Over time, the diaphragm can fatigue (i.e. pacing fails altogether) and the procedure is not without risk (surgical implantation may require a thoracotomy). It is very expensive and only offered by specialist centres.

Cardiovascular system
Neurogenic shock
Hypotenension occurs with lesions above T6 due to loss of sympathetic autonomic function and unopposed parasympathetic function. Vasoconstrictor tone is lost and venous pooling occurs. Loss of cardiac accelerator fibres results in bradycardia and patients are unable to increase cardiac output by changes in heart rate. Although the duration of neurogenic shock is variable, recovery tends to be incomplete and postural hypotension can be a persistent problem.

Since cardiac output is the product of heart rate and stroke volume, preserving stroke volume is essential. Neurogenic shock is initially managed by fluid resuscitation and hypervolaemia is poorly tolerated. Induction of anaesthesia and institution of intermittent positive pressure ventilation (IPPV) will exacerbate hypotension. If fluid resuscitation alone is inadequate, vasoactive drugs should be used to achieve a mean arterial pressure of 80 mm Hg within the first 72 h post-injury to optimize cord perfusion (and prevent secondary ischaemia). α-Agonists (phenylephrine and norepinephrine) are preferred, but bradycardias may occur in...
conjunction with α-agonists. Epinephrine can be used; however, glycopyrrolate or atropine usually suffices to increase heart rate.

**Thromboembolism**

Vessel wall damage, stagnant blood flow, and increased blood viscosity (Virchow’s triad) all may occur in SCI. However, the principal cause of deep vein thrombosis (DVT) in SCI is loss of mobility and vasodilatation resulting in venous pooling and stagnant blood flow. Pulmonary embolism (PE) may ensue. The risk of DVT is increased three-fold in SCI; lower in the first 72 h and increasing thereafter.2

Preventative measures should always be undertaken as early mobilization is seldom an option. Prophylaxis is provided by combining graduated compression stockings (GCS) with low-molecular-weight heparin (LMWH). Heparin prophylaxis is inadequate when used alone.2 Where heparin is contraindicated, GCS should be combined with intermittent pneumatic compression. In addition, femoral i.v. access contributes to venous stasis and should be avoided. Individuals who develop a DVT or PE should be fully anticoagulated. A vena caval filter should be used to prevent PE formation in patients with proven DVTs, in whom anticoagulation is contraindicated or where PEs have occurred despite anticoagulation. Those with a PE and hypotension or large V/Q abnormalities should be thrombolysed. Prophylaxis should continue for 3 months and, during convalescence, warfarin (international normalized ratio (INR) 2–3) can be substituted for LMWH.

**Sympathetic hyperreflexia**

Sympathetic hyperreflexia is a life-threatening condition triggered by somatic or visceral stimuli below the level of the injury, classically bladder or rectal distension. Individual patients will be unaware of this stimulus because of sensory loss. Hyperreflexia manifests after 4–6 weeks, once neurogenic shock has resolved and reflexes have returned. Hyperreflexia is common in lesions above T6 (above the splanchnic sympathetic outflow) and rare with lesions below T10. Sympathetic hyperreflexia can develop suddenly and without warning.

Triggering stimuli generate an ascending sensory nerve impulse (spinthalamic and posterior columns) that stimulates the sympathetic nervous system in the spinal cord (intermediolateral gray matter) below the level of the injury. The normal descending inhibitory mechanisms generated by the vasomotor centres cannot pass below the level of injury and sympathetic outflow continues unchecked. This results in profound vasoconstriction below the level of the injury with compensatory baroreceptor-mediated vasodilation above the level of the injury. The vasomotor centre triggers parasympathetic (vagal) mediated bradycardia. The clinical picture is one of malignant hypertension with reflex bradycardia. Above the level of the injury, the parasympathetic system cause headaches, sweating, and nasal congestion, while the sympathetic system predominates below the lesion with resultant skin blotching, goose pimples, and cool peripheries. Patients become restless and agitated and may become unconscious, develop seizures, strokes, and potentially die.

Risk assessment and prevention is the mainstay of management. In the event of hyperreflexia, precipitating stimuli must be identified and removed and pharmacological management, in the form of vasodilators (α-blockers) used to control increases in arterial pressure. Pressure sores, hip fractures, peptic ulcers, and urinary retention can all trigger hyperreflexia and may not be immediately apparent as the precipitant. If surgery is required, blocking ascending nerve impulses by using neuraxial local anaesthetics can prevent sympathetic hyperreflexia.

**Gastrointestinal (GI) system**

**Delayed gastric emptying and paralytic ileus**

Delayed gastric emptying and paralytic ileus are common after SCI and may last for 2–3 weeks. Vomiting may occur and hypokalaemia can result. Pulmonary aspiration is a definite risk, especially where patients are nursed supine rather than at 30°. Gastric distension will further restrict inspiration.

Management is aimed at symptom control, establishing an early bowel regime with regular use of laxatives and establishing early feeding. A nasogastric (after base of skull injury has been excluded) or orogastric tube is necessary to minimize stomach distension and possible aspiration, and prokinetic agents (metoclopramide and erythromycin) are used to promote feeding. Nasojejunal feeding can sometimes resolve difficulties when gastric feeding has failed.

**Gastric stress ulceration**

Gastric stress ulceration is often seen and thought to be secondary to unopposed vagal activity below the level of the injury. H2-antagonist prophylaxis is essential until enteral feeding, known to reduce ulcer incidence in ventilated patients, is established. Proton pump inhibitors may be more effective.

**Constipation**

Constipation is often problematic as sensation of defecation is lost. SCI patients are routinely commenced on laxatives and should receive daily rectal examinations to avoid and identity faecal impaction. Abdominal distension and pseudo-obstruction occur commonly. Physical obstruction should be excluded by history, examination, and radiological investigations [abdominal X-ray (AXR) and computerized tomography (CT)] together with a surgical opinion. A flatus tube or neostigmine (2 mg i.v. bolus) can resolve pseudo-obstruction.
Metabolic considerations

Temperature regulation

Vasodilatation causes aberrant temperature regulation and hypothermia with the patient temperature equilibrating with environmental temperature. Core and peripheral temperature must be monitored and warming devices used when necessary. If fluid resuscitation is required, fluids need to be warmed. An inability to sweat below the injury can impair heat loss and iatrogenic hyperthermia can occur.

Hyperglycaemia

Hyperglycaemia is known to aggravate ischaemic neurological injury. SCI patients will frequently be hyperglycaemic due to the stress response. Good glycaemic control needs to be maintained. Concurrent use of steroids will also induce hyperglycaemia.

Steroids and acute spinal cord injury

The NASCIS 2 and 3 trials recommended high-dose methylprednisolone (30 mg kg$^{-1}$) in non-penetrating SCI. However, these trials have been criticized. High-dose steroids are not without side-effects and may increase mortality and morbidity. NASCIS 3 showed those treated with steroids had an increased incidence of severe sepsis and severe pneumonia with no discernable improvement in neurological function. Steroids are no longer universally used in the UK.

Other considerations

Psychological aspects

Psychological disturbances are more likely to occur in ventilated patients. Depression, anxiety, and confusion are common in this group and will benefit from early intervention including liaison psychiatry. Lack of dignity due to an inability to self-care together with sexual dysfunction all contribute to depression. Rehabilitation, as well as providing physical benefit, helps individuals rediscover identity and obtain psychological independence. Early referral to regional spinal injury services is important.

Pain management

Acute pain is almost always a feature after SCI. Mismatched sensory and motor loss occurs and patients may have additional injuries above the level of sensory loss. Pressure sores are common (including occipital sores); surgery is often necessary; frequent turning and the inability to scratch may trigger pain. The WHO analgesic ladder should be used for acute somatic pain.

Chronic neuropathic pain is a common problem and occurs together with muscle spasm. Both can be difficult to treat. Pain affects quality of life, reduces sleep, and exacerbates depression. Hyperaesthesia and allodynia are common and pain is not limited to areas with preserved sensation but can occur in areas devoid of sensation. Neuropathic pain is often opioid-resistant. Gabapentin and amitriptyline are effective for neuropathic pain but need to be commenced as early as possible. Oral ketamine may be beneficial and in the intensive care unit setting can be given i.v.

Spasticity

Spasticity is common and can be sporadic in nature. Contractures can result from spasticity and are both painful and decrease function, compromise posture, and reduce functional capacity. The initial treatment of spasticity should include conservative measures such as stretches, splinting, and casting, and good pain control increases cooperation with such interventions. Medication is often necessary; baclofen, dantrolene, and gabapentin have all been used to reduce and improve spasticity. More invasive treatments include botulinum injections and intrathecal baclofen. Severe contractures may need to be released surgically, but contractures can recur unless supported by preventative treatments.

References


Please see multiple choice questions 8–12