

Critical Care of Spinal Cord Injury

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Study Design. Review article.

Objectives. To review the pathophysiology and management of the pulmonary and hemodynamic derangements that occur after acute spinal cord injury.

Summary of Background Data. Acute spinal cord injury is often associated with alterations in pulmonary and cardiovascular function that require treatment in the intensive care unit.

Methods. Review of published reports.

Results/Conclusion. Careful attention to the support of the pulmonary and cardiovascular systems can reduce the morbidity associated with acute spinal cord injury. Pulmonary function decreases markedly in the immediate postinjury period but improves in the subsequent weeks, allowing most patients with injury levels at C4 and below to be weaned from ventilatory support. Bradycardia and hypotension often accompany acute spinal cord injury, and management strategies are reviewed. The prophylaxis and diagnosis of thromboembolic disease are reviewed. [Key words: spinal cord injury, mechanical ventilation, shock, thromboembolic disease] **Spine 2001;26:S27–S30**

Acute spinal cord injury (SCI) is often associated with alterations in cardiopulmonary function that require management in the intensive care unit. Knowledge of the pathophysiology of these derangements is crucial for their successful management.

■ Respiration

The process of inspiration involves contraction of the diaphragm and the internal intercostal muscles that allow the chest cavity to expand. At high levels of ventilatory activity the accessory muscles are recruited to aid in this process. Expiration is largely passive but can be augmented by the forceful contraction of the muscles of the abdominal wall.

The diaphragm is supplied by the C3–C5 segments and so that injury above this level results in apnea and the need for immediate ventilatory support. With injuries at C5 and below, the function of the diaphragm is preserved, but ventilation is substantially compromised. The intercostal muscles are supplied by the thoracic segments, and in the acute period of injury there is flaccid paralysis of these muscles. As the diaphragm contracts and descends, the chest wall contracts rather than expands. The resulting loss of ventilatory function is striking: the forced vital capacity and maximal inspiratory force are acutely decreased by about 70%.^{20,23} This results in a breathing pattern consisting of shallow breaths.

The loss of the contribution of the abdominal muscles to expiration results in a similar decrease in the maximal expiratory force and thus a decreased ability to cough and clear secretions.²³

As spinal shock resolves and the paralysis of the intercostal muscles becomes spastic, the chest wall becomes rigid and no longer collapses with inspiration. There is a resulting improvement in ventilatory function. This improvement is mostly in inspiratory function, and by 5 months after injury the forced vital capacity and maximal inspiratory force are about 60% of predicted preinjury levels.^{20,23} The maximum expiratory force is 33% of the preinjury level.²³

■ Intubation

The altered ventilatory mechanics in the acute phase that result in shallow breaths can be compensated initially by an increase in the respiratory rate. The initial arterial blood gas is thus often normal or shows a mild hypoxemia.²³ Rapid, shallow breathing, however, is quite inefficient. This is because with shallow breathing there is still the need to move air in the dead space of the trachea and bronchi so that a larger percentage of each breath does not participate in gas exchange. The shallow breaths also promote a cascade of atelectasis whereby it becomes progressively difficult to inflate the lungs, especially in the face of diminished inspiratory force.

The decision making in proceeding with intubation involves some degree of judgment. About one third of patients with cervical injuries will require intubation, and the large percentage of these will require intubation within the first 24 hours after injury.⁹ Careful monitoring to look for signs of fatigue can help guide the decision. Serial measurement of vital capacity can be done at the bedside; if this value progressively declines or is <1 L, this should cause concern as should a rising respiratory rate or PCO₂. In general, it is best to proceed with intubation under controlled circumstances rather than waiting until it becomes an emergency.

Once the decision has been made to proceed with intubation, it is clearly preferable that, if at all possible, practitioners who have considerable experience in airway management should perform or supervise the procedure. Two large series have demonstrated the safety of the use of orotracheal intubation with manual in-line traction in the setting of acute cervical injury.^{11,26} Control of the airway is often facilitated by the use of pharmacologic paralysis. Succinylcholine is an excellent agent because of its rapid onset and short half-life. However, it should not be used in the setting of spinal cord injuries of >4 days duration because of the risk of precipitating hyperkalemia.¹⁹

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■ Pneumonia

Respiratory complications are a leading cause of death in patients with SCI, and the majority of these are due to pneumonia.^{1,5,6} Ventilator-associated pneumonia (VAP) is a consequence of intubation and mechanical ventilation; the risk of its occurrence increases by 1–3% per day of intubation.^{3,9} VAP that occurs in the first 4 days of intubation is usually caused by *Streptococcus pneumoniae* or *Hemophilus influenzae*. VAP that occurs after 4 days is usually the result of gram-negative bacilli, especially *Pseudomonas aeruginosa* or *Staphylococcus aureus*. The mortality attributed to VAP has been reported to be 27% overall and 43% in cases due to *P. aeruginosa*.⁸

Effective treatment of VAP is dependent on accurate diagnosis, but this can be a difficult diagnosis to establish with certainty. The presence of alveolar infiltrates or air bronchograms on chest radiographs have a high sensitivity but a low specificity. Fever and leukocytosis are common in critically ill patients and can be due to infections other than pneumonia or numerous noninfectious causes. Intubated patients will often have copious amounts of pulmonary secretions. There has been considerable controversy about the relative accuracy of cultures obtained by qualitative methods such as tracheal suctioning or quantitative methods such as bronchoscopy. If the diagnosis of VAP is missed or delayed, serious consequences may ensue. If antibiotics are given in the absence of pneumonia, the selection of resistant organisms is risked. In an attempt to help clarify these issues, the American College of Chest Physicians recently convened an expert panel to establish recommendations for the diagnosis of VAP. The recommendations of this panel were that at least two of the following clinical signs should be present for the diagnosis of VAP to be considered: temperature >38 C or <36 C, leukocytosis or leukopenia, purulent secretions, and hypoxemia. If the chest radiograph demonstrates alveolar infiltrates or air bronchograms, then a strategy to select an antibiotic could be based on the results of sampling of tracheal secretions. The panel determined that there was insufficient evidence to favor a quantitative over a qualitative method of identification of pathogens.¹⁵

Antibiotics chosen should cover the suspected organisms until final culture results are available. If *P. aeruginosa* is suspected, double coverage with an antipseudomonal β -lactam agent and an aminoglycoside is appropriate to help prevent the development of resistance.

■ Weaning From Mechanical Ventilation

There is a steady improvement in ventilatory mechanics over the weeks following SCI as the chest wall becomes spastic, and this allows for the large percentage of patients with C4 levels of injury and below to be weaned from mechanical ventilation.³⁰ The indications that it

may be appropriate to begin weaning are a rise in the forced vital capacity, resolution of any pulmonary infection, an inspired oxygen fraction of <50%, and a minute ventilation <10 L. Although the time course for this to occur varies, it is >2 weeks before weaning can begin in more than half of patients.⁹

Although there has been a trend toward early surgical stabilization to allow for mobilization, it must be noted that quadriplegics have better pulmonary mechanics in the supine position than when upright.^{7,10} The explanation for this paradoxical association to positioning is that when quadriplegics are placed in the upright position, the paralyzed abdominal musculature allows the abdominal contents to descend and overdistend the diaphragm to an inefficient starting position for contraction. If quadriplegics are returned to the supine position, the abdominal contents push the diaphragm into a more efficient position for contraction. The use of abdominal binders can partially offset the decline in pulmonary function in the upright position.¹⁰

A variety of ventilator strategies may be used as part of the weaning process including T-piece trials, continuous positive airway support, and pressure support, but it is not clear that one is superior or allows more rapid ventilator independence.^{9,22} The use of pressure support ventilation has become more common in recent years, allowing for slow titration of the amount of ventilatory support.

There are some clear advantages to tracheostomy in patients undergoing prolonged mechanical ventilation. Tracheostomy is usually more comfortable for the patient as the irritation of the endotracheal tube against the posterior pharyngeal wall is eliminated. There is less dead space ventilation with tracheostomy, and in patients with marginal pulmonary mechanics this can be important. There is some evidence that the use of early tracheostomy may be associated with lower rates of pneumonia in trauma patients.²⁵ Furthermore, it is possible to allow for periods of mechanical ventilatory support to alternate with spontaneous breathing without having to manipulate the airway. However, tracheostomy carries surgical risks such as bleeding, infection, and tracheal injury. A concern is that the site of tracheostomy is proximate to the incision site commonly used for anterior cervical stabilization, and the period of time that should separate these procedures to allow healing of tissue planes is unclear but 2 weeks is often used.

The use and timing of tracheostomy must therefore be individualized based on an estimate of the length of time ventilatory support will be required and what type of surgical stabilization is planned. Respiratory failure is more common in complete *versus* incomplete injuries,² and the average length of time of ventilator dependence is strongly related to the level of injury: 65 days for patients with C1–C4 levels, 22 days in patients with C5–C8 levels, and 12 days for patients with thoracic injuries.¹⁷

Table 1. Vasoactive Agents

Agent	Common Dosage Ranges	Comments
Dopamine ($\mu\text{g}/\text{kg}/\text{min}$)	1–10	Has primarily α -adrenergic effects at low doses and primarily β -adrenergic effects at higher doses; a commonly used agent in SCI
Dobutamine ($\mu\text{g}/\text{kg}/\text{min}$)	5–15	Most prominent effect is augmentation of cardiac performance but may lower systemic blood pressure so generally a less useful agent in SCI
Epinephrine ($\mu\text{g}/\text{min}$)	1–8	Both α - and β -adrenergic effects, may promote arrhythmias
Norepinephrine ($\mu\text{g}/\text{min}$)	1–20	Has some α -adrenergic but predominately β -adrenergic effects; a useful agent for blood pressure support, especially in cervical and high thoracic SCI
Phenylephrine ($\mu\text{g}/\text{min}$)	10–100	Exclusively β -adrenergic agent; should be used with caution in cervical SCI because of potential for reflex bradycardia

■ Hemodynamic Instability

Acute SCIs, especially those occurring in the cervical region, are often associated with hemodynamic instability. This is because of interruption of the sympathetic fibers that exit the spinal cord in the thoracic region and consequent unopposed parasympathetic outflow, which can result in cardiac arrhythmias and hypotension.

The most common arrhythmia seen is bradycardia, although supraventricular tachycardia and ventricular tachycardia can also be seen. Arrhythmias appear to be most common within the first 14 days after injury and are more common and more severe in more severe injuries.²¹

The hypotension seen is due to loss of vasoconstrictor tone in the peripheral arterioles and subsequent pooling of blood in the peripheral vasculature. The first line of treatment for this is volume resuscitation. If the infusion of 1–2 L of intravenous fluid fails to bring the blood pressure into the normal range, consideration should be given to the placement of a pulmonary artery catheter. The problem that can arise is that as extra volume is infused and the venous return increases, there needs to be an increase in cardiac output. Because the cardiac accelerator fibers have often been interrupted in cervical and upper thoracic lesions, the heart is not able to increase the cardiac output by increasing the heart rate but must rely on an increased stroke volume that may not be attainable. A pulmonary artery catheter allows the peripheral resistance and cardiac output to be measured directly and vasopressor therapy guided. The choice of a vasopressor should be an agent that has both α - and β -adrenergic actions, such as dopamine or norepinephrine,

to counter the loss of sympathetic tone and provide chronotropic support to the heart (Table 1). An agent with purely α -adrenergic action such as phenylephrine does not provide the needed chronotropic influence to the heart.

The resolution of shock and restitution of tissue perfusion is a clear goal: this can be demonstrated by adequate urine output, resolution of systemic acidosis, and normal mentation. The blood pressure and cardiac output that will achieve this will vary from patient to patient.

The appropriate end point for spinal cord perfusion is less clear. Given that there is evidence for ischemic and vascular factors on secondary SCI, the appropriate blood pressure to perfuse the injured spinal cord is an important question. Unfortunately, there is little information on this subject and no clear way to measure spinal cord perfusion. Vale et al treated a series of patients with acute SCI with fluid and vasopressors to achieve a mean arterial pressure of 85 mm Hg for a minimum of 7 days and reported favorable neurologic outcomes.²⁷ In this uncontrolled series the mean arterial pressure chosen was arbitrary and the optimal value is unknown.

■ Thromboembolic Disease

Patients with spine and spinal cord injuries are clearly at high risk for venous thromboembolism. A recent meta-analysis of risk factors for the development of venous thromboembolism in trauma patients demonstrated that spine fractures increased the risk by twofold and that SCI increased the risk by threefold. Conversely, no such association could be clearly demonstrated for the widely assumed risk factors of head injury, pelvic fracture, and long bone fracture.²⁹

Given this substantial risk, there is a clear logic to begin prophylaxis in the acute phase in the intensive care unit. The methods available for prophylaxis are mechanical devices, such as external pneumatic compression devices or compression stockings, and anticoagulants, such as heparin, low molecular weight heparin, or warfarin. Mechanical devices carry no clear risk but are not on their own sufficient prophylaxis in patients with spinal injury.¹⁴ Anticoagulants are effective in preventing venous thromboembolism in patients with spinal injuries but carry the risk of bleeding.

There is often reluctance to start anticoagulants acutely in the setting of a spinal fracture because of concern for promoting bleeding at the injury site or the need for surgery. Studies of patients with spinal injuries have shown that without prophylaxis the risk of the development of deep vein thrombosis is quite low in the first 72 hours after injury.¹⁴ In this initial period the use of mechanical devices is a safe option. After this period mechanical devices can be continued, but consideration should be made of starting anticoagulation. Standard mini-dose heparin twice a day does not appear to provide adequate prophylaxis in patients with spinal injuries.²⁴

Adjusted-dose heparin to raise the partial thromboplastin time to $1.5\times$ control is effective but has a significant bleeding risk.¹³ Low molecular weight heparin appears to be safe and effective.¹⁶ The length of time for prophylaxis to continue should be 8 weeks, less if the patient becomes ambulatory.¹²

The diagnosis or exclusion of deep vein thrombosis on clinical grounds is notoriously inaccurate. The use of compression B mode ultrasound is a safe and reliable means of diagnosis and has largely replaced venography and ¹²⁵I scanning.

The diagnosis of pulmonary embolism (PE) has always been difficult. The pulmonary angiogram remains the reference standard for the diagnosis, but concerns over availability, cost, and complications have limited its routine use. For many years ventilation/perfusion (V/Q) scans have been widely used to help establish the diagnosis, but the result is often indeterminate. Recently, spiral computed tomography (CT) of the chest has started to replace V/Q scanning in many situations. This technique can identify clot in the segmental and subsegmental branches of the pulmonary arteries. Comparison with pulmonary angiography suggests that the sensitivity is 94% and the specificity is 96%.²⁸ In patients with suspected PE randomized to V/Q scan or spiral CT, the diagnosis was able to be established in a larger percentage of patients with spiral CT.⁴ In the subset of patients who present with massive PE associated with hemodynamic compromise, the use of echocardiography can often demonstrate clot in the proximal pulmonary artery.

The mainstay of treatment of patients with PE who are hemodynamically stable is anticoagulation with heparin or low molecular weight heparin followed by warfarin. If anticoagulation is contraindicated because of concerns for bleeding, a vena cava filter is an alternative. In patients with massive PE and hemodynamic compromise, the use of thrombolytic therapy appears to reduce mortality over anticoagulation.¹⁸

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