

# The Acute Cardiopulmonary Management of Patients With Cervical Spinal Cord Injuries

Timothy C. Ryken, MD, MS\*

R. John Hurlbert, MD, PhD,  
FRCS<sup>‡</sup>

Mark N. Hadley, MDS

Bizhan Aarabi, MD, FRCS<sup>¶</sup>

Sanjay S. Dhall, MD<sup>||</sup>

Daniel E. Gelb, MD<sup>#</sup>

Curtis J. Rozzelle, MD\*\*

Nicholas Theodore, MD<sup>‡‡</sup>

Beverly C. Walters, MD, MSc,  
FRCS<sup>§§§</sup>

\*Iowa Spine & Brain Institute, University of Iowa, Waterloo/Iowa City, Iowa; <sup>‡</sup>Department of Clinical Neurosciences, University of Calgary Spine Program, Faculty of Medicine, University of Calgary, Calgary, Alberta, Canada; <sup>§</sup>Division of Neurological Surgery and; <sup>\*\*</sup>Division of Neurological Surgery, Children's Hospital of Alabama, University of Alabama at Birmingham, Birmingham, Alabama; <sup>¶</sup>Department of Neurosurgery and; <sup>#</sup>Department of Orthopaedics, University of Maryland, Baltimore, Maryland; <sup>||</sup>Department of Neurosurgery, Emory University, Atlanta, Georgia; <sup>‡‡</sup>Division of Neurological Surgery, Barrow Neurological Institute, Phoenix, Arizona; <sup>§§</sup>Department of Neurosciences, Inova Health System, Falls Church, Virginia

## Correspondence:

Mark N. Hadley, MD, FACS, UAB  
Division of Neurological Surgery,  
510 – 20th St S, FOT 1030,  
Birmingham, AL 35294-3410.  
E-mail: mhadley@uabmc.edu

Copyright © 2013 by the  
Congress of Neurological Surgeons

**KEY WORDS:** Blood pressure augmentation, Cardiac instability, Intensive care unit, Respiratory failure

*Neurosurgery* 72:84–92, 2013

DOI: 10.1227/NEU.0b013e318276ee16

www.neurosurgery-online.com

## RECOMMENDATIONS

### Level III:

**M**anagement of patients with an acute cervical spinal cord injury in an intensive care unit or similar monitored setting is recommended.

- Use of cardiac, hemodynamic, and respiratory monitoring devices to detect cardiovascular dysfunction and respiratory insufficiency in patients following acute spinal cord injury is recommended.
- Correction of hypotension in spinal cord injury (systolic blood pressure < 90 mm Hg) when possible and as soon as possible is recommended.
- Maintenance of mean arterial blood pressure between 85 and 90 mm Hg for the first 7 days following an acute spinal cord injury is recommended.

## RATIONALE

The intensive care unit (ICU) setting has traditionally been reserved for critically ill patients who require aggressive medical care and exceptional medical attention. Most contemporary medical centers have multiple critical care units, each designed to provide discipline-specific observation and intensive care to patients in need. Select institutions have created Acute Spinal Cord Injury Centers and offer multidisciplinary care including ICU care to patients who have sustained acute spinal cord injuries (SCIs).<sup>1-10</sup> Several reports describe improved patient management and

lower morbidity and mortality following acute SCI with ICU monitoring and aggressive medical management.<sup>2-4,6-10</sup> Despite this interest in and commitment to more comprehensive care for the patient with an acute SCI, many traumatic SCI patients are not managed in an ICU setting, nor are they routinely monitored for cardiac or respiratory dysfunction. There exist divergent management strategies for acute SCI patients within regions, communities, even institutions, depending on the training and experiences of the clinicians providing care.

Respiratory insufficiency and pulmonary dysfunction are common after traumatic SCI, particularly when the injury occurs at cervical spinal cord levels.<sup>2,3,6,8,11-14</sup> Severely injured patients demonstrate marked reductions in expected vital capacity and inspiratory capacity and may experience relative hypoxemia, all of which contribute to global hypoxemia and can exacerbate spinal cord ischemia after acute injury.<sup>6,9,11-14</sup> It appears that the earlier cardiac and/or ventilatory/pulmonary dysfunction is detected, the more likely effective, often life-saving treatment can be initiated. It is for these reasons that the issues of early ICU care and cardiac and pulmonary monitoring for human patients following acute SCI have been raised.

Acute traumatic SCI is frequently associated with systemic hypotension. Hypotension may be due to hypovolemia, direct severe spinal cord trauma itself, or a combination of the two. The presence of hypotension has been shown to be associated with worse outcomes after traumatic injury, including severe head injury.<sup>1,4,14-17</sup> Although a prospective controlled assessment of the effects of hypotension on acute human SCI has not been performed, laboratory evidence suggests that hypotension contributes to secondary injury after acute SCI by further reducing spinal cord blood flow and perfusion.<sup>5,6,12,13,15-21</sup>

**ABBREVIATIONS:** ASIA, American Spinal Injury Association; ICU, intensive care unit; MAP, mean arterial pressure; SCI, spinal cord injury

Hypotension in animal models of SCI results in worse neurological outcome.<sup>9,11,17,22-25</sup> Several clinical series of human patients with acute SCI managed in an aggressive fashion with attention to blood pressure, oxygenation, and hemodynamic performance report no deleterious effects of therapy and suggest improved neurological outcome.<sup>9,11,17,22-25</sup> Despite these observations, many patients with acute SCI treated in contemporary practice are not routinely monitored in an ICU setting or treated with blood pressure augmentation after injury. For these reasons, the issues of routine blood pressure support and threshold levels of mean arterial pressure (MAP) maintenance following acute SCI have been raised.

The previous medical evidence-based guideline effort by the Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons addressed the role of systemic blood pressure support and the role of the intensive care setting in 2 separate chapters.<sup>26,27</sup> The purpose of the current review is to update the medical evidence on the diagnosis and treatment of these issues since the original 2 medical evidence-based guidelines were published in 2002 and to address the following questions:

- Do patients with acute spinal cord injuries benefit from ICU cardiac, hemodynamic, and pulmonary monitoring and care?
- Does blood pressure management influence neurological outcome in patients with acute cervical SCI?

## SEARCH CRITERIA

A National Library of Medicine (PubMed) computerized literature search from 2000 to 2011 was undertaken using Medical Subject Headings in combination with “spinal cord injury”: medical management, nonoperative management, hypotension, spinal cord blood flow, respiratory insufficiency, pulmonary complications, and intensive care unit. Approximately 3500 citations were acquired. Non-English-language citations were excluded. Titles and abstracts of the remaining publications were reviewed, and relevant articles were selected to develop the guidelines. We focused on 4 specific topics concerning human patients with acute SCI: management in an ICU, cardiac instability, hypotension, and respiratory/pulmonary dysfunction. Additional citations were extracted from the reference lists of the remaining papers. Finally, members of the author group were asked to contribute articles known to them on the subject matter that were not found by other search means. Articles describing economics, epidemiology, anesthesia, monitoring techniques, penetrating cord injuries, nursing care, infectious or urologic complications, chronic complications, or remote SCIs were excluded. These efforts resulted in 11 articles, which form the foundation for this updated review. All studies provided Class III medical evidence. Twenty-seven articles are summarized in Evidentiary Table format (Table).

## SCIENTIFIC FOUNDATION

In 1976, Zäch et al<sup>10</sup> reported on a prospective medical management paradigm in the treatment of 117 consecutive acute

SCI patients in the Swiss Paraplegic Centre of Basel, Switzerland. All patients were treated in the ICU with central venous pressure monitoring and were administered dexamethasone 0.5 mg/kg for 4 days with a tapering dose through 10 days and volume expansion with Rheomacrodex 40 at 500 mL/d for 7 days. Patients were stratified by injury level, degree of deficit (Frankel grade), and time of admission after injury. The authors reported that 62% of cervical-level SCI patients they managed in this way improved at last follow-up, including 8 of 18 Frankel grade A patients, 2 by 2 grades and a third patient by 3 grades. No patient with a cervical injury worsened; 38% were unchanged from admission. Patients with thoracic T1-T10-level SCIs fared less well; 38% improved, none worsened, and 62% were without change, including 22 of 26 Frankel grade A patients. Two Frankel grade A patients experienced a complete recovery. Seventy percent of acute T11-L1-level SCI improved with this treatment paradigm, none worsened, and 30% were unchanged from admission. Of patients who arrived within 12 hours of injury, 67% were improved compared with their admission neurological examination. Of patients admitted between 12 and 48 hours of injury, only 59% improved. When admission occurred after 48 hours of injury, improvement was seen in only 50% of patients. The authors concluded that early transfer and “immediate medical specific treatment of the spinal injury” with attention to maintenance of acceptable blood pressure appeared to improve neurological recovery.

That same year, Hachen<sup>3</sup> reported a decade of experience with acute traumatic tetraplegia from the National Spinal Injuries Centre in Geneva. He described 188 acute SCI patients treated in an ICU setting following immediate transfer from the scene of the injury. The center reported a marked reduction in mortality rates following acute cervical SCI compared with annual statistics from 1966. Mortality for complete tetraplegia was reduced from 32.5% to 6.8% over the 10-year period. Mortality for patients with incomplete tetraplegia fell from 9.9% in 1966 to 1.4% in 1976. Most early deaths in the center’s experience were related to pulmonary complications. The likelihood of severe respiratory insufficiency was related to the severity of the cervical SCI. Seventy percent of patients with complete lesions experienced severe respiratory insufficiency in the center’s experience compared with 27% of patients with incomplete lesions. The improvement in mortality rates described was related directly to early monitoring and treatment of respiratory insufficiency in the ICU setting. Hachen stressed that facilities for continuous monitoring of central venous pressure, arterial pressure, pulse, respiration rate and pattern, and oxygenation-perfusion parameters must be available for all patients with neurological injuries following acute SCI, particularly those injuries above the C6 level.

In 1979, Gschaedler et al<sup>2</sup> described the comprehensive management of 51 patients with acute cervical SCIs in an ICU setting in Colmar, France. Forty percent of patients had multiple organ system injuries. They reported a low mortality rate of 7.8% and described several severely injured patients who made important neurological improvements, including 1 Frankel grade

A patient who improved to grade D and 2 Frankel grade B patients who changed to grade D. They cited early transport after injury and comprehensive intensive medical care with attention to and the avoidance of hypotension and respiratory insufficiency as essential to the improved outcomes their patients experienced.

McMichan et al<sup>14</sup> reported a prospective case series in 1980 of pulmonary complications identified in 22 patients with cervical-level acute SCI managed in an ICU setting. They compared their results with 22 historical controls with similar injuries. Institution of a new, aggressive pulmonary treatment paradigm resulted in zero deaths and fewer respiratory complications compared with those experienced by the retrospective group (9 deaths). They concluded that vigorous pulmonary therapy initiated early after acute SCI was associated with increased survival, a reduced incidence of pulmonary complications, and a decreased need for ventilatory support.

Ledsome and Sharp<sup>11</sup> measured pulmonary function in 16 patients with complete cervical SCI and compared initial values with those obtained in the same patients at 1, 3, and 5 weeks and 3 and 5 months after injury. In their 1981 report, they noted profound reduction in forced vital capacity (FVC) and expiratory flow rate immediately after injury. Patients with an FVC < 25% of expected had a high incidence of respiratory failure requiring ventilator support. This was especially true of patients with injuries at C4 or above. FVC was significantly increased at 5 weeks after injury and doubled at 3 months regardless of the level of cervical cord injury. Importantly, hypoxemia ( $PO_2 < 80$  mm Hg) was identified through blood gas analyses in 74% of patients who did not require ventilator support despite adequate alveolar ventilation ( $PCO_2$  normal; low FVC). The authors attributed this to a ventilation perfusion imbalance occurring immediately after acute SCI. Systemic hypoxemia responded to treatment with supplemental oxygen in most patients.

Piepmeyer et al<sup>5</sup> identified cardiovascular instability following acute cervical SCI in 45 patients they managed in an ICU setting in New Haven, Connecticut. Twenty-three patients had Frankel grade A injuries, 8 had grade B, 7 had grade C, and 7 had grade D. They discovered a high incidence of cardiovascular irregularities in these patients and identified a direct correlation between the severity of cord injury and incidence and severity of cardiovascular problems. Three patients returned to the ICU setting during the 2-week observation period of the study because of cardiac dysfunction despite a period of initial stability. Twenty-nine of the 45 patients had an average daily pulse rate of < 55 bpm, and 32 had episodes during which their pulse rate was < 50 bpm for a prolonged period of time. Hypotension was common after acute SCI in their series, but most patients responded well to volume replacement. However, 9 patients required vasopressors ranging over a period from hours to 5 days to maintain systolic pressure > 100 mm Hg. Cardiac arrest occurred in 5 patients (11%). All had Frankel grade A injuries. Three arrests occurred during endotracheal suctioning. The authors found that the first week after injury was the timeframe during which patients were most vulnerable to cardiovascular instability. Patients with the most

severe neurological injuries were most likely to experience cardiovascular instability after acute SCI regardless of autonomic function. They concluded that careful monitoring of severely injured acute SCI patients in the ICU setting reduces the risk of life-threatening emergencies.

In 1984, Tator and colleagues<sup>8</sup> described their experience with 144 patients with acute SCI managed between 1974 and 1979 at a dedicated SCI unit at Sunnybrook Medical Centre in Toronto, Ontario, Canada. They compared their results with a cohort of 358 SCI patients managed between 1948 and 1973 before the development of the acute care SCI facility. All 144 patients managed from 1974 to 1979 were treated in an ICU setting with strict attention to the treatment of hypotension and respiratory failure. Their medical paradigm was developed on the principle “that avoiding hypotension is one of the most important aspects of the immediate management of acute cord injury.” Hypotension was “treated vigorously” with crystalloid and transfusion of whole blood or plasma for volume expansion. Patients with respiratory dysfunction were treated with ventilatory support as indicated. They reported a reduced mean time from injury to admission and treatment (5 hours) compared with their 1948 to 1973 experience (> 12 hours). Neurological improvement was observed in 41 of 95 patients (43%) managed under the aggressive ICU medical paradigm. Fifty-two patients (55%) demonstrated no improvement. Only 2 patients (2%) deteriorated. The authors reported lower mortality, reduced morbidity, shorter length of stay, and lower cost of treatment compared with the 1948 to 1973 experience a result of this aggressive ICU strategy. They cited improved respiratory management in their ICU as one of the principal factors responsible for reduced mortality and credited the avoidance of hypotension, sepsis, and urologic complications for reduced morbidity after injury. These improved outcomes were realized despite the fact that 28% of the acute SCI patients they treated had additional injuries that increased their risk of morbidity and mortality.

In a 1987, Lehmann et al<sup>28</sup> reported on 71 acute SCI patients managed in an ICU at Yale/New Haven Medical Center. Patients were admitted within 12 hours of SCI and stratified by level and severity of neurological injury (Frankel scale). Patients were excluded if they harbored comorbidities such as head injury, diabetes mellitus, preexisting cardiac disease, or a history of cardiac medication use. All were monitored; hypotension was aggressively treated. The authors found that all patients with severe cervical SCIs (Frankel grades A and B) had prolonged bradycardia defined as heart rate < 60 bpm lasting at least 1 day. Thirty-five percent of Frankel grade C and D patients also demonstrated prolonged bradycardia. Only 13% of thoracic and lumbar SCI injuries had this finding. Marked bradycardia (< 45 bpm) was frequent in patients with severe cervical SCI (71%) and less common in patients with more mild cervical (12%) and thoracolumbar (4%) SCI. Sinus node slowing was profound enough to produce hemodynamic compromise and systemic hypotension necessitating bolus injections of atropine or placement of a temporary pacemaker in 29% of the severe cervical SCI

patients. Episodic hypotension unrelated to hypovolemia was identified in 68% of the severe cervical injury group, requiring the use of intravenous pressors in half. Five of 31 patients (16%) in the severe injury group experienced a primary cardiac arrest, three of which were fatal. All 5 patients had Frankel grade A SCI. There were no significant cardiac rate disturbances or spontaneous episodes of hypotension beyond 14 days of injury. The authors concluded that potentially life-threatening cardiac arrhythmias and hypotension regularly accompany acute severe injury to the cervical spinal cord within the first 14 days of injury. These events were not solely attributable to disruption of the autonomic nervous system. Detection and treatment were best accomplished in an ICU setting.

Wolf et al<sup>29</sup> in 1991 described their experience with bilateral facet dislocation injuries of the cervical spine at the University of Maryland in Baltimore. Fifty-two patients with acute cervical trauma were reviewed who received ICU care, volume resuscitation, invasive monitoring, and hemodynamic manipulation to maintain mean blood pressure > 85 mm Hg for 5 days. Thirty-four patients had complete neurological injuries, 13 had incomplete injuries, and 5 patients were intact. The authors attempted closed reduction within 4 hours of patient arrival to their center and performed early open reduction on patients who could not be reduced by closed means, including closed reduction under anesthesia. All but 3 patients underwent surgery for stabilization and fusion. The authors reported neurological improvement at discharge in 21% of complete SCI patients and in 62% of patients with incomplete cervical SCI. No intact patient deteriorated. Only 52% 1-year follow-up was provided. The authors concluded that their protocol of aggressive, early medical and surgical management of patients with acute SCI improved outcome following injury. Treatment in the ICU setting, hemodynamic monitoring with maintenance of MAP, and early closed or open decompression of the spinal cord were linked to a reduction of secondary complications.

Levi and coworkers<sup>4</sup> treated 50 acute cervical SCI patients in the ICU at the University of Maryland in Baltimore according to an aggressive management protocol that included invasive hemodynamic monitoring and volume and pressor support to maintain a hemodynamic profile with adequate cardiac output and mean blood pressure > 90 mm Hg. Their 1993 report described 31 patients with Frankel grade A injuries on admission, 8 patients with Frankel grade B injuries, and 11 patients in Frankel C and D grades. Eight patients had severe hypotension at the time of admission (systolic blood pressure < 90 mm Hg), whereas 82% of patients developed volume-resistant hypotension requiring pressors within the first 7 days of treatment. This was 5½ times more common among patients with complete motor injuries. The authors reported that the overall mean pulmonary vascular resistance index for the 50 patients they studied was less than the normal range, and it was less than the normal value in 58% of patients. Half of their acute SCI patients had a lower-than-normal systemic vascular resistance index. No patient with a complete motor deficit (Frankel grades A and B) and marked

pulmonary vascular resistance index/systemic vascular resistance index deficits experienced neurological recovery at 6 weeks. Forty percent of patients managed by protocol including several with complete injuries had some degree of neurological function improvement, 42% remained unchanged, and 9 patients died (18%). There was minimal morbidity associated with invasive hemodynamic monitoring. The authors concluded that hemodynamic monitoring in the ICU allows early identification and prompt treatment of cardiac dysfunction and hemodynamic instability and can reduce morbidity and mortality following acute SCI.

Vale et al<sup>9</sup> reported their results in 1997 from a prospective case series in which aggressive medical resuscitation and blood pressure management were performed on 77 patients with acute SCI treated at the University of Alabama in Birmingham. All patients were managed in the ICU with invasive monitoring (Swan Ganz catheters and arterial lines) and blood pressure augmentation to maintain MAP > 85 mm Hg for 7 days after injury. They reported 10 patients with complete cervical SCI (American Spinal Injury Association [ASIA] grade A), 25 with incomplete cervical injuries (ASIA grades B, C, and D), 21 patients with complete thoracic SCI, and 8 patients with incomplete thoracic-level SCI (grades B, C, and D). The average admission MAP for ASIA A cervical patients was 66 mm Hg. Nine of 10 patients required pressors following volume replacement to maintain an MAP of 85 mm Hg. Fifty-two percent of incomplete cervical SCI patients required pressors to maintain MAP at 85 mm Hg. Only 9 of 29 patients with thoracic-level SCI required the use of pressors. The authors reported minimal morbidity with the use of invasive monitoring or with pharmacological therapy to augment MAP. At 1-year follow-up (mean, 17 months), neurological recovery was variable and typically incomplete. Three of 10 cervical ASIA A patients regained ambulatory capacity, and 2 regained bladder function. Incomplete cervical SCI patients fared better. Twenty-three of these patients regained ambulatory function at 12 months of follow-up, only four of whom had initial examination scores consistent with ambulation. Twenty-two of 25 patients (88%) regained bladder control. Thirty-one of 35 cervical SCI patients and 27 of 29 thoracic-level SCI patients were treated surgically. The authors statistically compared selection for and timing of surgery with admission neurological function and compared surgical treatment, early and late, with neurological outcome and found no statistical correlation. They concluded that the enhanced neurological outcome identified in their series after acute SCI was optimized by early and aggressive volume resuscitation and blood pressure augmentation and was in addition to and/or distinct from any potential benefit provided by surgery.

In 2001, Vitaz et al<sup>30</sup> described a clinical pathway for SCI management developed in multidisciplinary fashion and compared the results before and after implementation. Thirty-six patients in the study group were compared with 22 control patients. Study group patients had 6.8 fewer ICU days, 11.5 fewer hospital days, 6 fewer ventilator days ( $P < .05$ ), and a lower

rate of complications. The authors concluded that the use of a clinical care pathway for SCIs resulted in improved patient care and fewer complications. Despite the prospective comparison, the groups were not comparable and the study was considered to provide Class III medical evidence.

Aito<sup>31</sup> prospectively assessed the incidence of complications associated with acute SCI on the basis of the type of facility in which the acute care of the traumatic SCI was provided. In their 2003 publication, nearly all of the described complications they identified occurred in patients not initially admitted to a specialized SCI unit, including respiratory complications, deep-vein thrombosis, pulmonary embolism, trophic skin changes, heterotopic ossification, and urinary complications. The authors concluded that prevention of complications during the acute phase after SCI is best accomplished by early admission to a specialized multidisciplinary SCI unit.

Como et al<sup>32</sup> characterized the need for mechanical ventilation in patients with acute cervical SCI and neurological deficits. Their 2005 study included 119 patients, of whom 45 (37%) had complete SCI. Twelve patients (27%) had injury levels from C1 to C4. Nineteen (42%) had a C5 injury level, and 14 (31%) had an injury level of C6 or below. Eight of the complete injury patients died (mortality, 18%). All patients with complete SCI at the C5 level and above required a definitive airway and tracheostomy. Of patients with a complete SCI at C6 or below, 79% required intubation and 50% eventually required tracheostomy. From these results, the authors recommended consideration of early intubation for patients with complete SCI, especially for patients with injuries at the C5 level or above.

Berly and Shem<sup>33</sup> in 2007 reported on acute respiratory management following acute SCI. They found that respiratory complications were frequent and were the most common cause of morbidity among acute SCI patients (36% of total complications). Respiratory failure was the most common cause of mortality in their series, cited in 86% of deaths following acute SCI. Ventilatory failure occurred on average 4.5 days after acute SCI. The authors concluded that the incidence of respiratory complications can be significantly reduced by transfer of acute SCI patients to an SCI center. Hassid et al<sup>34</sup> reviewed nearly 55 000 Level I trauma patients and identified a subgroup of 186 patients with isolated acute cervical SCI. They reported that early intubation for acute complete SCI patients is mandatory. They favor close observation of incomplete SCI patients and immediate airway intervention should the patient manifest any evidence of respiratory failure.

Guly et al<sup>35</sup> found an incidence of neurogenic shock (systolic blood pressure < 100 mm Hg and heart rate < 80 bpm) of 19.3% (95% confidence interval, 14.8-23.7) in a series of 490 patients with acute SCI. In 2006, Franga et al<sup>36</sup> described an incidence of cardiovascular instability of 17%, including bradyarrhythmias requiring permanent pacemaker placement among 30 acute complete cervical SCI patients. Neumann et al<sup>37</sup> performed a retrospective review of mortality following SCI. They found that Glasgow Coma Scale score < 9, the need

for vasopressors to support mean blood pressure, and mechanical ventilation were predictors of mortality among acute SCI patients. All of these investigators favor ICU care for monitoring and treatment of acute SCI patients, particularly those with more severe injuries.

Macias et al<sup>38</sup> evaluated the importance of admission to a specialized trauma center on the incidence of paralysis in patients with acute SCI. Their 2009 review included 4121 patients diagnosed with traumatic SCI treated at 100 trauma centers and 601 other local and regional medical facilities. Mortality was 7.5%, and the incidence of paralysis, based on the reported discharge diagnosis, was 16.3%. A designated trauma center provided the initial care in 57.9% of the patients (n = 2378). Multivariate analysis determined that the incidence of paralysis was significantly lower at designated trauma centers compared with local and regional hospitals without trauma center designation (adjusted odds ratio, 0.67; 95% confidence interval, 0.53-0.85; *P* = .001). There was no significant difference in the incidence of mortality between the 2 types of facilities. The authors concluded that early admission to a designated trauma center significantly reduces the incidence of paralysis following acute SCI.

Berney et al<sup>39</sup> described their experience with the pulmonary/ventilatory care of 114 acute SCI patients. They described a clinical pathway (classification and regression tree) to assist in clinical decision making regarding airway management in patients following acute cervical SCI. The following variables were considered crucial in predicting the need for aggressive airway management: FVC, the volume of pulmonary secretions, and gas exchange. The use of these variables in their regression tree analysis allowed accurate prediction of the need for airway management in > 82% of their patients and predicted extubation success in the vast majority of ventilated patients (8.7% extubation failure rate).

In a subsequent publication, the same group performed a literature review on respiratory complications associated with acute cervical SCI.<sup>40</sup> They identified 21 studies including 1263 patients that described definitive protocols for the respiratory management of acute cervical SCI. Although the majority of the reports were case series, the authors discovered that mortality (adjusted risk ratio = 0.4; 95% confidence interval, 0.18-0.61), the incidence of respiratory complications (adjusted risk ratio = 0.36; 95% confidence interval, 0.08-0.58), and the requirement for a tracheostomy (adjusted risk ratio = 0.18; 95% confidence interval, -0.05 to 0.4), were all significantly reduced when care givers/institutions used a respiratory protocol in the management of acute SCI patients. Specifically, the use of a clinical pathway reduced the duration of mechanical ventilation by 6 days (95% confidence interval, -0.56 to 12.56) and ICU length of stay by 6.8 days (95% confidence interval, 0.17-13.77).

## SUMMARY

Patients with acute cervical SCI frequently develop hypotension, hypoxemia, pulmonary dysfunction, and cardiovascular instability, often despite initial stable cardiac and pulmonary function. These

**TABLE. Evidentiary Table: Cardiopulmonary Management<sup>a</sup>**

Citation	Description of Study	Evidence Class	Conclusions
Berney et al, <sup>39</sup> <i>Spinal Cord</i> , 2011	Prospective observational study of a clinical pathway for airway management in 114 patients with acute cervical spine injury	III	Forced vital capacity, the volume of pulmonary secretion, and gas exchange were predictive of airway management on 82.3% occasion with an 8.7% extubation failure rate.  The authors conclude that a clinical pathway of respiratory management was useful in clinical decision making.
Berney et al, <sup>40</sup> <i>Spinal Cord</i> , 2011	Systematic review of acute respiratory management of cervical SCI in the first 6 wk after injury	III	The authors have demonstrated that a clinical pathway with a structured respiratory protocol is effective in reducing respiratory complications, ventilator time, and intensive care unit length of stay.  Class III as the majority of articles included are case series.
Casha and Christie, <sup>41</sup> <i>Journal of Neurotrauma</i> , 2010	Systematic review of intensive cardiopulmonary management following acute SCI	III	Because of the high incidence of cardiopulmonary complications, acute SCI patients should be managed in monitored unit.  There is Class III evidence supporting the maintenance of MAP > 85 mm Hg for a period extending up to 1 wk following acute SCI.
Ploumis et al, <sup>42</sup> <i>Spinal Cord</i> , 2010	Systematic review of the evidence supporting a role for vasopressor support in acute SCI	III	No statistical difference in neurological improvement with vasopressor support with an MAP of < 85 mm Hg and those with MAP < 90 mm Hg.  The authors conclude that there is no gold standard on vasopressor support and that cervical cord injuries require vasopressors more frequently than other SCIs ( $P < .001$ ).
Neumann et al, <sup>37</sup> <i>Journal of Trauma</i> , 2009	Retrospective study of risk factors for mortality in traumatic cervical SCI	III	Independent predictors for mortality were Glasgow Coma Scale score < 9 and vasopressor use.  The authors conclude that Glasgow Coma Scale score < 9, mechanical ventilation, and vasopressor use were predictors of mortality.
Guly et al, <sup>35</sup> <i>Resuscitation</i> , 2008	Database review to determine the incidence of neurogenic shock in patients with isolated SCI	III	Incidence of neurogenic shock in cervical cord injuries was 19.3% (95% confidence interval, 14.8-23.7) vs in 7% (95% confidence interval, 3-11.1) in the thoracic or 3% (95% confidence interval, 0-8.85) in the lumbar spine cord.
Hassid et al, <sup>34</sup> <i>Journal of Trauma</i> , 2008	Database review of 54 838 consecutive Level I trauma patients	III	Respiratory complications in SCI are frequent.  Early intubation is mandatory for complete SCI patients. For incomplete patients, close observation for any evidence of respiratory failure should prompt immediate airway intervention.
Berly and Shem, <sup>33</sup> <i>Journal of Spinal Cord Medicine</i> , 2007	Retrospective review of respiratory management during the first 5 d after SCI	III	Morbidity and mortality following acute SCI were 36% and 83%, respectively, with ventilatory failure occurring an average 4.5 d following injury.

(Continues)

**TABLE. Continued**

Citation	Description of Study	Evidence Class	Conclusions
			The authors recommend transfer to a center specializing in acute management of SCI to reduce the number of respiratory complications.
Franga et al, <sup>36</sup> <i>The American Surgeon</i> , 2006	Retrospective evaluation of recurrent asystole resulting from high cervical SCIs	III	5 of 30 (17%) patients with complete cervical SCIs required placement of permanent cardiac pacemakers for recurrent bradycardia/asystolic events.
Como et al, <sup>32</sup> <i>Journal of Trauma</i> , 2005	Retrospective review evaluating the need for mechanical ventilation following cervical SCI in the presence of neurological deficit	III	The authors recommend considering early intubation, particularly with a complete injury at C5 or above.
Vitaz et al, <sup>30</sup> <i>Journal of Spinal Disorders</i> , 2001	Prospective comparison of patients treated with and without a clinical pathway for treatment of acute SCI	III	The authors demonstrate that the use of a clinical care pathway for SCIs resulted in improved patient care and fewer complications.
Lu et al, <sup>12</sup> <i>Spine</i> , 2000	Retrospective review of apnea in 36 acute SCI patients	III	Delayed apnea most likely in acute SCI patients with severe, diffuse acute SCI. Apnea most likely within first 7-10 d.
Bötel et al, <sup>18</sup> <i>Spinal Cord</i> , 1997	225 acute SCI patients treated in ICU; only 87 admitted within 24 h of injury	III	Significant numbers of multiply injured and head-injured patients. The percentage of complete injuries not recorded. Improved outcome when admitted to ICU early after injury
Vale et al, <sup>9</sup> <i>Journal of Neurosurgery</i> , 1997	Prospective assessment of 77 acute SCI patient treated in ICU, aggressive hemodynamic support, MAP > 85 mm Hg	III	Improved outcome with aggressive medical care, distinct from potential benefit from surgery at 1-y follow-up.
Levi et al, <sup>20</sup> <i>Neurosurgery</i> , 1993	50 patients treated in ICU, aggressive medical treatment, MAP > 90 mm Hg	III	Improved outcome with aggressive hemodynamic support at 6 wk after injury.
Tator et al, <i>Paraplegia</i> , 1993 <sup>16</sup>	201 acute SCI patients, ICU care, hemodynamic support compared with 351 prior patients	III	Less severe cord injuries resulting from immobilization, resuscitation, and early transfer to ICU setting.
Wolf et al, <sup>29</sup> <i>Journal of Neurosurgery</i> , 1991	52 patients with locked facets reduced within 4 h, ICU care, MAP > 85 mm Hg, 49 operated on: 23 on day 1, 26 delayed (mean, day 8.7)	III	Closed reduction 61%
			52% 1 year follow-up In general, improved neurological outcome with hemodynamic therapy.
Lehmann et al, <sup>28</sup> <i>Journal of the American College of Cardiology</i> , 1987	71 consecutive acute SCI patients, ICU care, monitoring of cardiac/hemodynamic parameters	III	Bradycardia, 100%; hypotension (< 90 mm Hg systolic), 68%. Life-threatening bradyarrhythmias, 16% incidence related to severity of SCI.
Reines and Harris, <sup>6</sup> <i>Neurosurgery</i> , 1987	123 cases, acute SCI patients in ICU, aggressive pulmonary treatment	III	Respiratory insufficiency major cause of morbidity and mortality after ASCI. Aggressive ICU care, pulmonary treatment reduce incidence.
Piepmeyer et al, <sup>5</sup> <i>Central Nervous System Trauma</i> , 1985	45 ASCI patients, all managed in ICU setting with cardiac, hemodynamic monitoring	III	Cardiac dysrhythmia, hypotension, and hypoxia common in first 2 wk after ASCI. Incidence related to severity of injury.
Bose et al, <sup>1</sup> <i>Neurosurgery</i> , 1984	28 patients with acute SCI, 22 managed in ICU setting	III	Improved neurological outcome at discharge for group 2 but better scores initially. Group 1 with intrinsic cord injury vs Group 2 compression on myelo and/or instability.
	Group 1: medical treatment		
	Group 2: medical/surgical treatment		

(Continues)

TABLE. Continued

Citation	Description of Study	Evidence Class	Conclusions
Tator et al, <sup>8</sup> <i>Canadian Journal of Surgery</i> , 1984	144 acute SCI patients, ICU care, hemodynamic support, compared with prior series	III	Improved neurological outcome, less mortality with early transfer, avoidance of hypotension, and ICU care.
Ledsome and Sharp, <sup>11</sup> <i>American Review of Respiratory Disease</i> , 1981	Reassessment of pulmonary function in acute SCI patients, comparison over time	III	Reduced vital capacity, flow rates, and hypoxia after ASCI. Incidence related to severity of SCI. Marked improvement in pulmonary functions 3 mo after injury.
McMichan et al, <sup>14</sup> <i>Journal of the American Medical Association</i> , 1980	Prospective study of pulmonary complications in 22 acute SCI patients compared with 22 prior patients managed with aggressive ICU care	III	No deaths in series vs 9 of 22 deaths in prior group. ICU care and vigorous pulmonary therapy improves survival, reduces complications.
Gschaedler et al, <sup>2</sup> <i>Paraplegia</i> , 1979	51 acute SCI patients managed in ICU, aggressive medical treatment, avoid hypotension	III	Improved morbidity and mortality with early transfer, avoidance of hypotension, respiratory insufficiency.
Hachen, <sup>3</sup> <i>Journal of Trauma</i> , 1977	188 acute SCI patients managed in center's ICU, aggressive treatment of hypotension, respiratory insufficiency	III	Reduced morbidity and mortality with early transfer, attentive ICU care and monitoring, and aggressive treatment of hypotension and respiratory failure.
Zäch et al, <sup>10</sup> <i>Paraplegia</i> , 1976	117 acute SCI patients at Swiss Center, ICU setting, aggressive blood pressure, volume therapy: Rheomacrodex × 5 d, dexamethasone × 10 d	III	Improved neurological outcome with aggressive medical treatment. Better outcome for early referrals.

<sup>a</sup>ICU, intensive care unit; MAP, mean arterial pressure; SCI, spinal cord injury.

complications are not limited to patients with complete SCI. Life-threatening cardiovascular instability and respiratory insufficiency may be transient and episodic and may be recurrent in the first 7 to 10 days after injury. Patients with the most severe neurological injuries appear to have the greatest risk of these life-threatening events. Class III medical evidence indicates that ICU monitoring allows the early detection of hemodynamic instability, cardiac disturbances, pulmonary dysfunction, and hypoxemia. Prompt treatment of these events in patients with acute SCI reduces cardiac- and respiratory-related morbidity and mortality.

Management in an ICU or other monitored setting appears to have an impact on neurological outcome after acute cervical SCI. Retrospective studies consistently report that volume expansion and blood pressure augmentation performed under controlled circumstances in an ICU setting are linked to improved ASIA scores in patients with acute SCI compared with historical controls. Class III medical evidence suggests that the maintenance of MAP at 85 to 90 mm Hg after acute SCI for a duration of 7 days is safe and may improve spinal cord perfusion and ultimately neurological outcome.

## KEY ISSUES FOR FUTURE INVESTIGATION

The length of stay in the ICU setting necessary to provide optimal management of patients with acute SCI is unknown. The available

evidence suggests that most untoward and potentially life-threatening cardiac and respiratory events occur within the first 2 weeks of injury. Patients with less severe acute SCIs may require less time in a monitored setting than those patients with more severe injuries. Class II medical evidence is needed to guide treatment recommendations in these areas.

The issue of whether or not blood pressure augmentation has an impact on outcome following human SCI is important and deserves further study. If augmentation of MAP is determined to be of potential benefit, the most appropriate threshold levels of MAP and the length of augmentation therapy need definition. These questions may be best analyzed in a multi-institution prospective cohort study or a properly designed multi-institution retrospective case-control study.

## Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

## REFERENCES

1. Bose B, Northrup BE, Osterholm JL, Cotler JM, DiTunno JF. Reanalysis of central cervical cord injury management. *Neurosurgery*. 1984;15(3):367-372.
2. Gschaedler R, Dollfus P, Mole JP, Mole L, Loeb JP. Reflections on the intensive care of acute cervical spinal cord injuries in a general traumatology centre. *Paraplegia*. 1979;17(1):58-61.
3. Hachen HJ. Idealized care of the acutely injured spinal cord in Switzerland. *J Trauma*. 1977;17(12):931-936.

4. Levi L, Wolf A, Belzberg H. Hemodynamic parameters in patients with acute cervical cord trauma: description, intervention, and prediction of outcome. *Neurosurgery*. 1993;33(6):1007-1016; discussion 1016-1017.
5. Piepmeier JM, Lehmann KB, Lane JG. Cardiovascular instability following acute cervical spinal cord trauma. *Cent Nerv Syst Trauma*. 1985;2(3):153-160.
6. Reines HD, Harris RC. Pulmonary complications of acute spinal cord injuries. *Neurosurgery*. 1987;21(2):193-196.
7. Tator CH. Vascular effects and blood flow in acute spinal cord injuries. *J Neurosurg Sci*. 1984;28(3-4):115-119.
8. Tator CH, Rowed DW, Schwartz ML, et al. Management of acute spinal cord injuries. *Can J Surg*. 1984;27(3):289-293, 296.
9. Vale FL, Burns J, Jackson AB, Hadley MN. Combined medical and surgical treatment after acute spinal cord injury: results of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. *J Neurosurg*. 1997;87(2):239-246.
10. Zäch GA, Seiler W, Dollfus P. Treatment results of spinal cord injuries in the Swiss Paraplegic Centre of Basle. *Paraplegia*. 1976;14(1):58-65.
11. Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis*. 1981;124(1):41-44.
12. Lu K, Lee TC, Liang CL, Chen HJ. Delayed apnea in patients with mid- to lower cervical spinal cord injury. *Spine (Phila Pa 1976)*. 2000;25(11):1332-1338.
13. Mansel JK, Norman JR. Respiratory complications and management of spinal cord injuries. *Chest*. 1990;97(6):1446-1452.
14. McMichan JC, Michel L, Westbrook PR. Pulmonary dysfunction following traumatic quadriplegia: recognition, prevention, and treatment. *JAMA*. 1980;243(6):528-531.
15. Amar AP, Levy ML. Pathogenesis and pharmacological strategies for mitigating secondary damage in acute spinal cord injury. *Neurosurgery*. 1999;44(5):1027-1039; discussion 1039-1040.
16. Dolan EJ, Tator CH. The effect of blood transfusion, dopamine, and gamma hydroxybutyrate on posttraumatic ischemia of the spinal cord. *J Neurosurg*. 1982;56(3):350-358.
17. Sandler AN, Tator CH. Effect of acute spinal cord compression injury on regional spinal cord blood flow in primates. *J Neurosurg*. 1976;45(6):660-676.
18. Bötzel U, Gläser E, Niedeggen A. The surgical treatment of acute spinal paralysed patients. *Spinal Cord*. 1997;35(7):420-428.
19. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury: results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial: National Acute Spinal Cord Injury Study. *JAMA*. 1997;277(20):1597-1604.
20. Levi AD, Tator CH, Bunge RP. Clinical syndromes associated with disproportionate weakness of the upper versus the lower extremities after cervical spinal cord injury. *Neurosurgery*. 1996;38(1):179-183; discussion 183-185.
21. McMahon D, Tutt M, Cook AM. Pharmacological management of hemodynamic complications following spinal cord injury. *Orthopedics*. 2009;32(5):331.
22. Hall ED, Wolf DL. A pharmacological analysis of the pathophysiological mechanisms of posttraumatic spinal cord ischemia. *J Neurosurg*. 1986;64(6):951-961.
23. Tator CH. Experimental and clinical studies of the pathophysiology and management of acute spinal cord injury. *J Spinal Cord Med*. 1996;19(4):206-214.
24. Tator CH, Duncan EG, Edmonds VE, Lapczak LI, Andrews DF. Complications and costs of management of acute spinal cord injury. *Paraplegia*. 1993;31(11):700-714.
25. Tator CH, Fehlings MG. Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. *J Neurosurg*. 1991;75(1):15-26.
26. Management of acute spinal cord injuries in an intensive care unit or other monitored setting. In: Guidelines for the management of acute cervical spine and spinal cord injuries. *Neurosurgery*. 2002;50(3 suppl):S51-S57.
27. Blood pressure management following acute spinal cord injury. In: Guidelines for the management of acute cervical spine and spinal cord injuries. *Neurosurgery*. 2002;50(3 suppl):S58-S62.
28. Lehmann KG, Lane JG, Piepmeier JM, Batsford WP. Cardiovascular abnormalities accompanying acute spinal cord injury in humans: incidence, time course and severity. *J Am Coll Cardiol*. 1987;10(1):46-52.
29. Wolf A, Levi L, Mirvis S, et al. Operative management of bilateral facet dislocation. *J Neurosurg*. 1991;75(6):883-890.
30. Vitaz TW, McIlvoy L, Raque GH, Spain DA, Shields CB. Development and implementation of a clinical pathway for spinal cord injuries. *J Spinal Disord*. 2001;14(3):271-276.
31. Aito S. Complications during the acute phase of traumatic spinal cord lesions. *Spinal Cord*. 2003;41(11):629-635.
32. Como JJ, Sutton ER, McCunn M, et al. Characterizing the need for mechanical ventilation following cervical spinal cord injury with neurologic deficit. *J Trauma*. 2005;59(4):912-916; discussion 916.
33. Bertly M, Shem K. Respiratory management during the first five days after spinal cord injury. *J Spinal Cord Med*. 2007;30(4):309-318.
34. Hassid VJ, Schinco MA, Tepas JJ, et al. Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. *J Trauma*. 2008;65(6):1328-1332.
35. Guly HR, Bouamra O, Lecky FE. The incidence of neurogenic shock in patients with isolated spinal cord injury in the emergency department. *Resuscitation*. 2008;76(1):57-62.
36. Franga DL, Hawkins ML, Medeiros RS, Adewumi D. Recurrent asystole resulting from high cervical spinal cord injuries. *Am Surg*. 2006;72(6):525-529.
37. Neumann CR, Brasil AV, Albers F. Risk factors for mortality in traumatic cervical spinal cord injury: Brazilian data. *J Trauma*. 2009;67(1):67-70.
38. Macias CA, Rosengart MR, Puyana JC, et al. The effects of trauma center care, admission volume, and surgical volume on paralysis after traumatic spinal cord injury. *Ann Surg*. 2009;249(1):10-17.
39. Berney SC, Gordon IR, Opdam HI, Denehy L. A classification and regression tree to assist clinical decision making in airway management for patients with cervical spinal cord injury. *Spinal Cord*. 2011;49(2):244-250.
40. Berney S, Bragge P, Granger C, Opdam H, Denehy L. The acute respiratory management of cervical spinal cord injury in the first 6 weeks after injury: a systematic review. *Spinal Cord*. 2011;49(1):17-29.
41. Casha S, Christie S. A systematic review of intensive cardiopulmonary management after spinal cord injury. *J Neurotrauma*. 2011;28(8):17-29.
42. Ploumis A, Yadlapalli N, Fehlings MG, Kwon BK, Vaccaro AR. A systematic review of the evidence supporting a role for vasopressor support in acute SCI. *Spinal Cord*. 2010;48(5):356-362.