Cervical Spinal Cord Injury and the Need for Cardiovascular Intervention

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Hypothesis: The level of cervical spinal cord injury (CSCI) can be used to predict the need for a cardiovascular intervention.

Design: Retrospective review. Data included level of spinal cord injury, Injury Severity Score, lowest heart rate, and systolic blood pressure in the first 24 hours and intensive care unit course. The level of CSCI was divided into high (cord level C1-C5) or low (cord level C6-C7). Neurogenic shock was defined as bradycardia with hypotension. Statistical analysis was performed with the t test and the χ² test.

Setting: Level I trauma center.

Patients: The patients studied were those with quadriplegia who experienced a CSCI and were admitted to the hospital between December 1, 1993, and October 31, 2001.

Interventions: Pressors, chronotropic agents, and pacemakers.

Main Outcome Measure: Use of a cardiovascular intervention in the presence of neurogenic shock.

Results: Eighty-three patients met the criteria for CSCI and quadriplegia, 62 in the high (C1-C5) and 21 in the low (C6-C7) level. There was no significant difference between the 2 groups in mean±SD age (38.2±17.8 vs 34.7±15.6 years; P=.43), mean±SD Injury Severity Score (35.7±17.5 vs 32.5±11.2; P=.44), mean±SD admission base deficit (-0.7±3.6 vs 0.7±2.7; P=.06), or mortality (12 [19%] of 62 patients vs 2 [10%] of 21 patients; P=.29). Neurogenic shock was present in 19 (31%) of the 62 patients with high CSCI and in 5 (24%) of the 21 patients with low CSCI (P=.56). There was a marked difference in the use of a cardiovascular intervention between those with a high and those with a low CSCI: 15 (24%) of 62 patients vs 1 (5%) of 21 patients (P=.02). Two patients with C1 through C5 spinal cord injuries required cardiac pacemakers.

Conclusions: There was no significant difference in the frequency of neurogenic shock by injury level. Patients with a high CSCI (C1-C5) had a significantly greater requirement for a cardiovascular intervention compared with patients with lower injuries (C6-C7).

Arch Surg. 2003;138:1127-1129

Cervical Spinal Cord Injury (CSCI) is associated with dysfunction of the sympathetic nervous system and cardiovascular deficits, including severe bradycardia, asystole, and loss of peripheral vascular tone. These effects have been replicated in animal studies. Direct myocardial injury and a decrease in contractility (dP/dt [ie, change in pressure per change in time]) may also contribute to hypotension.

Patients with a CSCI may develop neurogenic shock, characterized by bradycardia and hypotension from decreased peripheral vascular resistance and cardiac output. These changes are related to increased vagal tone, decreased sympathetic input, and possibly changes in the heart itself. Pharmacologic and electrical interventions (ie, pacemakers) may be necessary in these patients if fluid resuscitation alone cannot maintain adequate tissue perfusion. Failure to adequately treat neurogenic shock may result in further ischemic injury of an already compromised nervous system (secondary injury). Aggressive treatment with fluids and pressors, and appropriate invasive monitoring (arterial and central venous access), is paramount.

To our knowledge, no study has specifically examined whether the level of CSCI can be used to determine which patients are most susceptible to neurogenic shock and the requirement for cardiovascular interventions (CVIs), namely, the administration of pressors, chronotropic...
agents, and/or cardiac pacing. This study was performed to test the hypothesis that the level of CSCI can be used to predict the likelihood of requiring a CVI.

**METHODS**

The records of quadriplegic patients who experienced a CSCI at University Medical Center, a level I trauma center, between December 1, 1993, and October 31, 2001, were reviewed. Level of spinal cord injury, age, sex, Injury Severity Score; admission base deficit; mechanism (blunt vs penetrating), associated injuries, hemorrhage, previous medical conditions, and in-hospital mortality were noted. The lowest heart rate and systolic blood pressure in the first 24 hours after injury and during the remaining intensive care unit course were recorded. All resuscitations followed Advanced Trauma Life Support guidelines. Initial hypotension was treated with crystalloid, followed by packed red blood cells as appropriate and control of hemorrhage. Resuscitation was guided by response to therapy and the end points of systolic blood pressure of 90 mm Hg or higher, no further need for transfusion, and correction of base deficit. Pressors and chronotropic agents were used only when hypotension was refractory to intravenous fluid administration, and were titrated to keep the systolic blood pressure at 90 mm Hg or higher. All patients who began treatment with pressors and chronotropic agents underwent placement of central venous and/or pulmonary artery catheters and invasive arterial monitoring.

The level of CSCI was divided into 2 categories: high (cord level C1-C5) and low (cord level C6-C7). C5/C6 was chosen as the level of division between the 2 categories because of innervation of the phrenic nerve, which terminates at the C5 level. Previous researchers used this level as the line of demarcation because of the respiratory pathophysiologic features that may contribute to neurogenic shock. Bradycardia was defined as a heart rate of less than 50 beats/min. Hypotension was defined as a systolic blood pressure lower than 90 mm Hg. Neurogenic shock was defined as the presence of simultaneous bradycardia and hypotension. The use of pressors (dopamine or phenylephrine hydrochloride, dobutamine hydrochloride, epinephrine hydrochloride, norepinephrine bitartrate, and phenylephrine hydrochloride), chronotropic agents (atropine sulfate), and cardiac pacing was recorded. Use of 1 or more of these treatments constituted a CVI. Patients with a CSCI from blunt trauma were given intravenous methylprednisolone sodium succinate. Patients with a CSCI who experienced penetrating trauma did not receive corticosteroids.

The Injury Severity Score is an expression of anatomic injury developed for comparison of trauma care that correlates with outcome. The admission base deficit has been shown to be an excellent index of shock, perfusion, and outcome for patients who experience trauma.

**RESULTS**

Eighty-three patients met the inclusion criteria for CSCI and quadriplegia during the study period, with 62 in the high injury (C1-C5) category and 21 in the low injury (C6-C7) category. There was no significant difference in age, Injury Severity Score, admission base deficit, or mortality between the groups (Table). There was no significant difference in the presence of neurogenic shock between the C1 through C5 group and the C6 through C7 group (19 [31%] of 62 patients vs 5 [24%] of 21 patients; P = .56). However, there was a significant difference between the 2 groups in the need for a CVI: 15 (24%) of the C1 through C5 group required an intervention for neurogenic shock, whereas only 1 (5%) of the C6 through C7 group needed an intervention (P = .02). In addition, the only 2 patients requiring permanent pacemakers were in the higher cervical level injury group. None of the patients with C6 through C7 injury required a pacemaker.

Dobutamine was used in 2 patients. One patient was a 69-year-old woman with a history of congestive heart failure, and the other was a 90-year-old man with a low cardiac index. Both patients met invasive criteria for inotropic support, and both also required a vasoconstrictive agent (dopamine or phenylephrine hydrochloride [Neo-Synephrine]).

Cervical spinal cord injury can cause significant changes in pulse, blood pressure, cardiac output, and rhythm. Within seconds to minutes after injury, there is a systemic pressor response with a widened pulse pressure seen in animals and humans, resulting from short-term outflow of sympathetic activity and adrenal hormones. This pressor response quickly abates, and is followed by neurogenic shock with bradycardia and hypotension. Preganglionic sympathetic nerve fibers exit the spinal cord at the first through fourth thoracic level. Parasympathetic outflow, however, is carried by the vagus nerve, which originates in the medulla. Therefore, a CSCI may completely transect spinal cardiac and vasomotor sympathetic fibers from above while the parasympathetic fibers remain intact. We chose C5/C6 as the border between high and low cervical injuries because phrenic nerve innervation terminates at C5. Piepmeier et al and Lehmann and colleagues invoke hypoxia as a culprit in the exacerbation of bradycardia and cardiac arrest in patients with cervical cord injuries. Piepmeier et al further explain that hypoxic patients with normal cervical cord function will increase their heart rate with the sympathetic outflow that normally accompanies hypoxic-induced tachypnea (pulmonary inflation reflex). This respiratory-related sympathetic outflow, which normally overrides vagal parasympathetic effects on the heart, seems to be blocked by cervical cord injuries that compromise
ventilation (ie, at C5 and above). Lehmann et al explain this phenomenon as well in ventilator-dependent patients undergoing endotracheal suctioning. The mechanical respiratory embarrassment that accompanies high (C1-C5) injuries may also contribute to the specific cardiovascular complications associated with those injuries.

Lehmann et al12 reviewed the presence of bradycardia, hypotension, and cardiac arrest and the need for atropine and pressor therapy by severity of CSCI, but not by level within the cervical spine. These researchers found that 71% of patients with a severe CSCI who had Frankel scores of A (complete loss of neurologic function below the level of injury) and B (no motor function but sensory function below the injury level) experienced bradycardia, with a heart rate lower than 45 beats/min. In addition, 68% of the patients with a severe CSCI were quadriplegic, with hypotension, cardiac arrest, and the need for pressors. In our study, a significant difference was found between the high and low injury groups and the need for pressors. The difference in findings may be explained by the difference in definition of high vs low. Piepmeier et al did not include the C5 level in the high group, which could account for this difference. In both previously mentioned studies,11,12 bradycardia and hypotension were examined separately, not together as one entity of neurogenic shock. Both studies showed that severity of CSCI correlates with cardiovascular complications, especially bradycardia. Our study showed that the level of CSCI with quadriplegia could define a subgroup of patients who are more likely to require CVIs. There was no significant difference in age, Injury Severity Score, admission base deficit, and mortality between the high and low CSCI groups in our study. The 2 groups differed only in the need for a CVI.

There was a trend toward a higher mortality in the higher CSCI group (12 [19%] of 62 patients vs 2 [10%] of 21 patients). This is consistent with previous studies,19,20 in which persons with injuries above C5 have had a higher mortality.

In conclusion, although there was no significant difference in the frequency of neurogenic shock (bradycardia with hypotension), patients with a high CSCI (C1-C5) had a significantly greater requirement for CVIs, including pacemakers, compared with patients with injuries at a lower level (C6-C7). Patients with C1 through C5 CSCIs are more likely to require a CVI regardless of age or concomitant injuries. This difference may affect allocation of intensive care unit resources between the 2 groups.

Accepted for publication May 3, 2003.

This paper was presented as a poster at the 74th Annual Meeting of the Pacific Coast Surgical Association; February 16, 2003; Monterey, Calif; and is published after peer review and revision.

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