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# The Cause of Neurologic Deterioration After Acute Cervical Spinal Cord Injury

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**Study Design:** A retrospective review was performed to identify patients at risk for secondary neurologic deterioration after complete cervical spinal cord injury.

**Objective:** To examine the causes of early neurologic deterioration in patients with complete spinal cord injury at a regional spinal cord injury center.

**Summary of Background Data:** After complete spinal cord injury, neurologic deterioration occurs in a subgroup of patients. Despite anecdotal reports, no study has clearly identified the subgroups at highest risks.

**Methods:** One hundred eighty-two patients with complete spinal cord injury were identified among 1904 consecutive patients with acute spinal trauma evaluated from March 1993 through September 1999. Parameters analyzed included demographics, mechanism of injury, American Spinal Cord Injury Association (ASIA) level on admission and during hospital stay, onset of ascension, blood pressure, hemoglobin, febrile episode, heparin administration, and the timing of operation and traction. Radiographs of patients with ascending complete spinal cord injury were reviewed with attention to fracture type and neurologic and vascular injuries.

**Results:** Twelve of 186 patients with ASIA Grade A (6.0%) complete spinal cord injury had neurologic deterioration during the first 30 days after injury. No patients with penetrating injuries had deterioration. A significant association between death and ascension was observed. The onset of ascension of the injury could be categorized into three discrete temporal subsets. Early deterioration (less than 24 hours) was typically related to traction and immobilization. Delayed deterioration (between 24 hours and 7 days) was associated with sustained hypotension in patients with fracture dislocations. Late deterioration (more than 7 days) was observed in a patient with vertebral artery injuries.

**Conclusion:** Delayed neurologic deterioration in complete spinal cord injury (ASIA A) is not rare. Specific causes were identified among discrete temporal subgroups. Management of complete spinal cord injury can be improved with recognition of these temporal patterns and earlier intervention. [Key Words: ascension, deterioration, cervical spine, spinal cord injury, neurologic loss]
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Traumatic cervical spinal cord injury (CSCI) typically occurs in young patients and results in significant economic costs including hospital stay, rehabilitation, loss of employment, and productivity. These injuries can vary from partial to complete loss of upper and lower limb function. Because each cervical segmental level provides a unique functional capacity, subsequent loss of an additional motor segment may cause an alteration in functional outcome. For example, a functionally independent patient may subsequently need assistance for basic transfers or a patient previously breathing independently may need permanent ventilatory support.

Despite early recognition, rapid transport, cardiopulmonary resuscitation, spinal immobilization, and administration of intravenous methylprednisolone in the setting of an acute CSCI, secondary neurologic deterioration occasionally occurs.[15,17,25] The pathogenesis for delayed neurologic deterioration is not clearly understood. However, improvements in noninvasive neuroimaging now allow detailed sequential imaging of the spinal cord during these episodes. This study identifies and classifies the causes of secondary neurologic deterioration in patients with CSCI treated at a level one regional spinal cord injury center.

## Methods

From March 1993 through September 1999, 1904 consecutive patients with acute, penetrating, and blunt spine injuries were evaluated at the Delaware Valley Regional Spinal Cord Injury (SCI) Center at Thomas Jefferson University Hospital, and 182 of these patients had complete cervical spinal cord injuries classified as American Spinal Injury Association Grade A (ASIA A). On admission, patients were independently examined by the spinal cord injury team comprising a neurosurgeon, orthopedic surgeon, and physiatrist who determined and graded the neurologic function using the ASIA classification.[3] Intravenous methylprednisolone (Solu-Medrol Pharmacia & Upjohn, Kalamazoo, MI) was administered using the protocol from the second National Acute Spinal Cord Injury Study (NASCIS II).[7] Imaging studies were obtained. Open and/or closed operative treatments were provided if indicated. All patients had routine serial neurologic examinations performed during their hospital stays, during rehabilitation, and at set postinjury intervals.

The designation of "ascending" CSCI was applied to patients having loss of motor function rostral to the initial level of spinal cord injury within 30 days of the injury. Plain radiographs obtained after worsening were reviewed to identify the presence of subsequent spinal displacement. Magnetic resonance imaging (MRI) and angiography (MRA) were obtained

**Table 1. Neurologic Deterioration After Cervical Spinal Cord Injuries**

| Group | Age | Sex | Mech | Injury | Asc. Time | Ascend | M. Score | Change | OR | Time OR | Fever | Fracture |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Early | 76 yr | M | MVA | Intact | <12 hrs | C6 ASIA A | Dead (3 mo) | Dead | No | No | No | C67 Sub |
| Early | 38 yr | M | MVA | C5 ASIA A | <12 hrs | C3 ASIA A | Dead (2 wks) | Dead | No | No | No | C45 Sub |
| Early | 82 yr | M | MVA | C4 ASIA D | <1 hr | C4 ASIA A | Dead (7 wks) | Dead | No | No | No | No |
| Early | 54 yr | F | Fall | C8 ASIA A | <12 hrs | C5 ASIA A | 34 (5 mo) | (−)10 | ACDF C56 C67 | Day 3 | No | No |
| Early | 28 yr | M | MVA Vert. Artery Injury | C4 ASIA A | <24 hrs | Brain Dead | Dead | Dead | No | No | No | C56 Bil. jump facet |
| Delay | 19 yr | M | Sport -ski | C5 ASIA A | 3–10 day | C2 ASIA A | 0 (2 yrs) | (−)12 | A/P Cerv | <24 hrs | 102.4 2 day | C56 Bil. Jump facet |
| Delay | 19 yr | M | MVA | C5 ASIA A | 5–8 day | C3 ASIA A | 10 (2 yrs) | (+)4 | A/P Cerv | Day 2 | No | C45 Bil. Jump facet |
| Delay | 27 yr | M | Sport softball | C5 ASIA A | 4–8 day | C2 ASIA A | 2 (1 yr) | (−)6 | PCF C4–6 | <9 hrs | 102.8 8 day | C56 Uni. Jump facet |
| Delay | 16 yr | M | Sport wrestle | C4 ASIA A | 4–9 day | C1+ ASIA A | 0 (2 yrs) | (−)4 | A/P Cerv | <8 hrs | 105.2 6 day | C34 Bil. Jump facet |
| Delay | 45 yr | M | Sport dive | C5 ASIA A | 3–9 day | C4 ASIA A | 3 (8 mo) | (−)5 | A/P Cerv | Day 2 | 103.1 8 day | C56 Uni. Jump facet |
| Delay | 25 yr | M | MVA | C6 ASIA A | 5–11 day | C5 ASIA A | 16 (8 mo) | (−)4 | PCF C5–7 | Day 13 | No | C56 Uni. Jump facet |
| Late | 28 yr | M | MVA | C5 ASIA A | 7 days | Brain Dead | Dead | Dead | No | <24 hrs | No | HCD C56 FT Fracture |

MVA = motor vehicle accident; ASIA = American spinal injury association level of injury; ASC. Time = time of initial spinal cord ascension; Ascend = motor level of neurologic deterioration; M. Score = motor score by ASIA criteria at follow-up and time in parenthesis; Change = motor score change by ASIA criteria from initial to follow-up examination; OR = operative procedure; Time OR = time of operative procedure; Fever = highest temperature and onset of febrile episode; HCD = herniated cervical disc; Bil. = bilateral; Uni = unilateral; FT = foramina transversaria; A/P = anteroposterior; Cerv = cervical; ACDF = anterior cervical decompression and fusion; Vert = vertebral.

in all patients with spinal cord injuries. The presence and extent of cervical cord edema and/or hemorrhage, intravertebral disc disruption or herniation, and evidence of extracranial vertebral artery injury were determined. Other parameters analyzed included demographics, mechanism of injury, Allen and Ferguson fracture classification,[2] and ASIA grade and affected vertebral level on admission and during hospital stay. The timing of skeletal traction, the timing and type of operative treatment, episodes of hypotension, and hemoglobin level on admission and during deterioration, body temperature fluctuations, and the administration of heparin were also noted.

The mechanisms of injury were categorized as motor vehicle accident, fall, sport-related (diving, wrestling and softball), and miscellaneous. The occurrence and timing of neurologic deterioration were recorded. Early deterioration was defined as new neurologic loss within 24 hours of injury, delayed deterioration as new neurologic loss beginning 24 hours to 7 days after injury, and late deterioration as new neurologic loss beyond 7 days after injury. Comparisons among categories were evaluated using Fisher exact, $\chi^2$, and unpaired *t* tests with level of significance at $P < 0.05$.

## ■ Results

Twelve of 186 patients (6.0%) with acute traumatic CSCIs showed neurologic deterioration or ascension within 30 days after injury (Table 1). The mean age of patients was 38 years (range, 16–82) with a male predominance of 11 to 1. All but two patients had complete injuries at admission. One patient with incomplete injury and another that was neurologically intact had early complete cervical cord injuries after cervical immobilization. No patient with a penetrating injury had subsequent neurologic deterioration. In addition, no patient experienced neurologic worsening within 48 hours of surgical treatment. No differences in gender, age, mechanism of injury, operative treatment or level of injury were identified among these patients. However, there was an association between peritrauma death and secondary neurologic deterioration ($P = 0.033$).

Four of the five patients in the early group (mean age 56 years) developed neurologic worsening during application of cervical immobilization less than 24 hours after

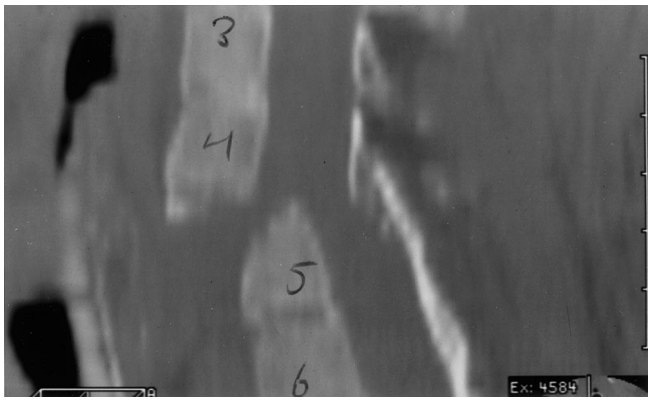


Figure 1. A 38-year-old man with ankylosing spondylitis had development of a C5 ASIA A complete spinal cord injury after a motor vehicle accident. Computer tomography (CT) sagittal reformat showed severe subluxation at C4–C5. During halo vest placement, the injury ascended to C3.

injury. The fifth patient initially had paraplegia but died of bilateral vertebral artery injuries. Two of the four patients whose condition worsened with cervical immobilization had ankylosing spondylitis that resulted in three-column shear injuries after minor trauma. One was initially neurologically intact before progressing to a C6 ASIA A CSCI during halo vest immobilization (Figure 1). In the other patient with ankylosing spondylitis the injury progressed from C5 to C3 ASIA A CSCI during traction and immobilization. The third patient had an incomplete central cord injury (C4 ASIA D) after a fall. In the emergency department, the patient was extremely agitated and would not remain recumbent while immobilized in a rigid cervical collar. The injury quickly ascended to a C4 complete CSCI with the patient's self-manipulation of his neck. The fourth patient was an obese woman with a C8 ASIA A severe central cord injury after a fall whose injury ascended to a C5 ASIA A level after halo vest placement because her body habitus precluded adequate immobilization.

All six patients in the delayed deterioration group sustained flexion–distraction injuries with facet dislocations and had neurologic deterioration beginning between 24 hours and 7 days after injury. Their mean age was 25 years (range, 16–45); four patients had a C5 ASIA A injury, and one each had a C4 ASIA A and C6 ASIA A injury. Three patients had bilateral facet dislocations (Stage 3) and the others had unilateral facet dislocations (Stage 2). Initial MRI examination performed less than 8 hours after injury showed hyperintense T2 signal in the spinal cord at the level of injury in all, intraparenchymal hemorrhage in five, and vertebral artery occlusions in two (Figure 2, A–C). All were initially managed with skeletal traction for reduction on the day of admission, and then underwent surgical stabilization (four anteroposteriorly and two posteriorly). Their neurologic conditions remained unchanged for at least 48 hours after surgery. Three patients had delayed neurologic deterioration of three levels: one of two levels, and two deteriorated at a single level. Deterioration included decreased tidal volume and decline in motor function evident in examination. Subsequent MRI during deterioration showed increased white matter signal when compared with initial T2-weighted MRI sequences.

In five of six patients, a sustained period of hypotension (systolic blood pressure below 90 mm Hg) was observed preceding neurologic deterioration. Although four episodes occurred during operative treatment, none worsened during the first two postoperative days. In addition, four had elevated body temperature (>101.5 F), occurring 2 days after ascension of neurologic injury in three and 1 day earlier in the other. None had identifiable infection around the time of hyperthermia. Patients in this delayed deterioration group were somewhat younger ($P = 0.079$) and typically had sports-related injuries ($P = 0.0052$).

Two patients, both 28 years old, had symptomatic vertebral artery dissections causing cervicomedullary ischemia and subsequent brain death. One patient was categorized in the late deterioration group (neurologic deterioration occurring more than 7 days after injury), whereas the second had bilateral vertebral artery occlusions immediately after the accident. The severe vascular ischemia probably resulted in the atypical acute deterioration observed in this second patient. Both died of cerebral ischemia. There was a significant correlation between ascending lesions from symptomatic vertebral injuries and death ($P = 0.0010$).

## ■ Discussion

The reported incidence of neurologic deterioration after spinal cord injury ranges from 1.8% to 10%.[8,12,15,16,25,36] Although a prospective analysis by Marshall et al[25] identified specific causes of deterioration in 12 of 14 patients, only 69 patients with complete CSCI were identified among the 283 patients studied. Frankel[16] reported eight cases of progressive neurologic deterioration among 800 patients with spinal cord injury treated with postural reduction, except 1 who underwent open reduction. Others have observed rapid subacute neurologic deterioration involving all regions of the spinal cord.[1,4,8,11,36] Belanger et al[4] described three patients with severe delayed neurologic loss after spinal injury, including one with a cervical flexion distraction injury in whom deterioration ascended on day 7.

Three temporal subgroups were identified in this series with similarities in pathogenesis of ascension. The early group consisted of patients with new neurologic deterioration less than 24 hours after injury and was typically related to halo vest or traction immobilization. Another patient with paraplegia showed acute worsening and died of bilateral vertebral artery occlusion. Because displaced cervical fractures and/or dislocations can cause spinal cord compression and neurologic injury, cervical manipulation and traction are used to quickly restore anatomic spinal alignment and maintain immobilization. Although risks of injury are considered small and may be minimized by small incremental increases in

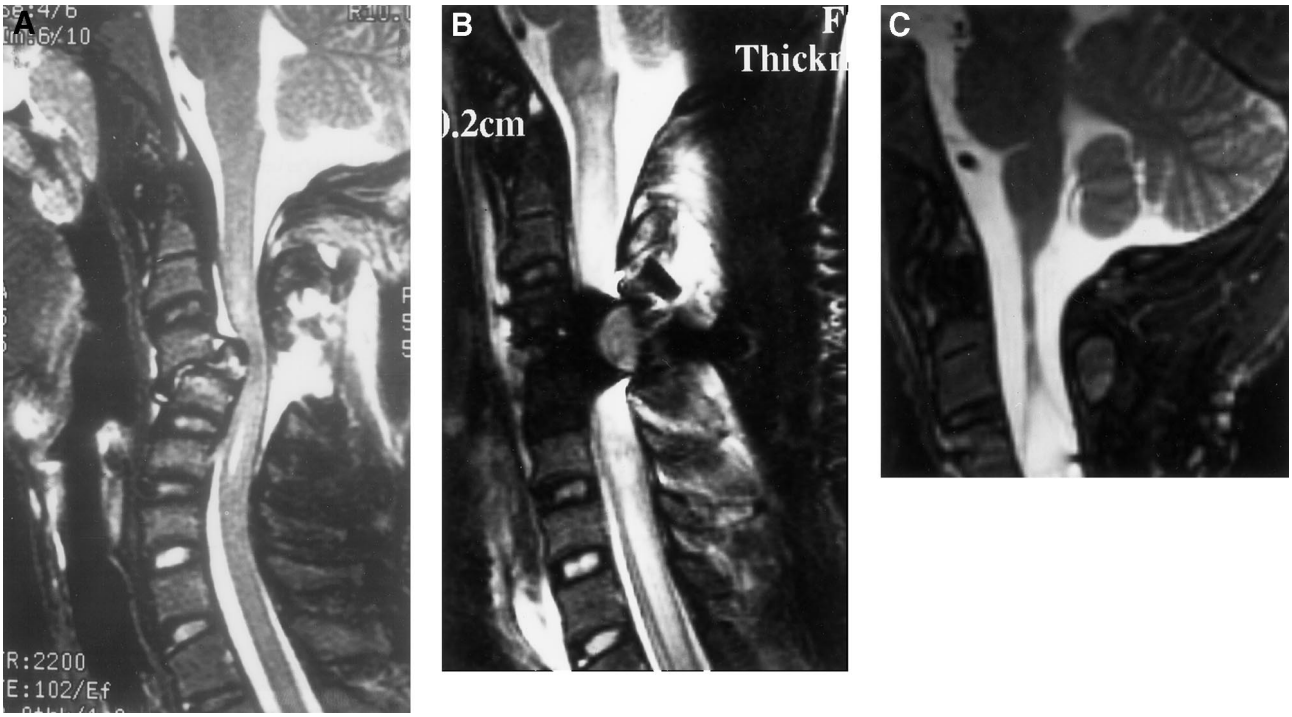


Figure 2. A 16-year-old boy sustained a C4 ASIA A injury while wrestling that resulted in quadriplegia. A magnetic resonance (MRI) scan confirmed a flexion–distraction injury with C3–C4 bilateral facet dislocation (**A**). The initial MRI illustrated focal edema and intraparenchymal hemorrhage. The patient underwent an acute (<8 hours) C3–C4 anterior cervical decompression and fusion followed by posterior cervical wiring to restore anatomic alignment. A postoperative sagittal MRI within 24 hours after injury showed the spinal canal decompressed and no significant change in the parenchymal edema or hematoma. Five days after the injury progressive loss of bicep and deltoid function occurred, along with decreased vital capacity. Another MRI showed much more extensive white matter signal and normal vertebral arteries (**B**). The injury ascended to C1, despite high-dose steroids. At 2 years, imaging showed an atrophied spinal cord in the same distribution of the prior white matter changes. The patient never regained the motor function displayed at admission (**C**).

traction with sequential neurologic examinations,[33] these techniques can result in loss of neurologic function due to spinal cord distraction,[8,18,23] ligamentous rupture,[20,27] or disc protrusion.[14,28]

Two of the four patients in the early group with neurologic worsening occurring after immobilization had ankylosing spondylitis. Ankylosing spondylitis sustained in spinal trauma can be difficult to manage.[5,12,20,25,29] Multisegmental ossification across interspaces results in a stiff but brittle column that can fracture from shear forces, resulting in two highly mobile segments. Consequently, small changes in alignment with skeletal traction are extremely difficult to achieve. The conditions of both patients with ankylosing spondylitis acutely worsened during either initial halo vest placement or during traction. One patient was neurologically intact before sustaining complete tetraplegia after placement of the halo vest, whereas the second lost further function of his upper limbs after traction and halo vest placement (Figure 1). Imaging studies were performed and excluded compressive hematomas as the underlying cause in either patient. Inadequate immobilization in central cord injuries of a combative patient and an obese patient were followed by neurologic deterioration.

The delayed group was the largest subset with deterioration occurring between 1 and 7 days after CSCI. All patients had flexion–distraction injuries with facet dislocations. Frankel[16] described six of eight patients with delayed progressive loss of thoracic neurologic function who also had fracture dislocations. Similarly, seven of the eight patients had deterioration between 1 and 8 days after injury. Comparatively frequent neurologic deterioration after fracture dislocation has been reported by others.[1,8,11,12] Frankel[16] proposed that thrombosis, hematoma, or inflammatory necrosis caused the neurologic deterioration. Moreover, he suggested that an inflammatory response may occur, given febrile episodes in seven of eight patients. Ascension ceased in two of his patients after systemic anticoagulation, one of whom first underwent operative exploration for unexplained neurologic deterioration and had a swollen, pink spinal cord. However, ascension in the other six patients spontaneously ceased without anticoagulation. For example, one patient's neurologic deterioration from a T10 to a T3 complete injury ceased after infusion of high-dose steroids.[16]

All six patients in the delayed group had flexion–distraction injuries with facet dislocations and were treated with high-weight closed reduction and subsequent operative therapy. None had neurologic worsening in the first two postoperative days. Although two had vertebral artery injuries, the frequency of asymptomatic vertebral artery injury was similar to that in previous reports (range, 24–80%).[19,22,35]

All six patients were treated with high-dose steroids during neurologic deterioration without reversal of neurologic loss. One also had mannitol therapy, as suggested in Frankel,[16] without success. Another was treated with intravenous heparin, limiting ascension to only one additional level. An MRI performed in each patient with neurologic deterioration showed increased white matter signal on T2-weighted images without compressive hematoma or malalignment. It seems unlikely that this signal represented reversible vasogenic edema, given the persistent neurologic dysfunction despite steroid therapy. In fact, whereas Bracken et al[7] reported an 11.2 motor score improvement after 6 months in the control group and a 16.0-point improvement with the use of intravenous steroids, the follow-up motor scores of the delayed group were 4.5 points worse after a mean of 1.4 years (Table 1).

Because these patients not only lost function acutely but were also unable to regain their initial motor level despite methylprednisolone and aggressive rehabilitation, it seems that an irreversible ischemic process had occurred. Therefore, the MRI findings may represent cytotoxic edema from vascular insufficiency that is followed by subsequent cord infarction. For example, one patient whose injury ascended from a C4 to a C1 ASIA A injury had a late MRI showing prominent loss of neurologic tissue in the same distribution as the previous white matter signal change (Figure 2, A–C).

Five of the six patients had significant sustained hypotension (systolic blood pressure <90 mm Hg) before the ascension of the injury. These episodes typically occurred during surgery, 2 days before ascension of the injury. Two patients with intraoperative hypotension had very early surgery (<9hours after injury). Others researchers have associated very early surgery in patients with spinal cord injury with worse clinical outcomes, perhaps secondary to hypotension with induction of anesthesia.[15,21,25] Additional factors may precipitate sustained hypoperfusion. For example, Aito et al[1] described a period of hypotension after the patient sat upright followed by pain and neurologic deterioration. Even mild hypotension has been reported to cause spinal cord infarction due to insufficient collateral circulation.[31] Farmer et al[15] identified four patients with sepsis and delayed neurologic deterioration. The shunting of arterial blood to the venous system during septicemia may result in hypoperfusion of the spinal cord. Marshall et al[25] also noted two patients with neurologic deterioration from unidentified causes, one of whom had "severe metabolic abnormalities." In discussing the results in Frankel,[16] Hardy also described six similar patients with neurologic deterioration: In one who died, spinal arterial occlusion accompanied by cylindrical columns of edematous tissue seven levels above the initial injury site was identified during autopsy. All these cases implicate vascular insufficiency in the pathogenesis of neurologic deterioration. Because impaired vasoreactivity of the injured spinal cord increases susceptibility to vascular insufficiency, either reduced arterial blood flow or impaired venous outflow could increase ischemia in the penumbra of tissue near the injury site, resulting in cytotoxic edema and infarction.

An additional cause of neurologic progression may be propagation of venous thrombosis, resulting in further infarction. After complete spinal cord injury, 80% of patients have altered venous drainage of the spinal cord.[10] The tortuous anatomy of the veins may accentuate the effects of venous stasis and subsequent thrombosis. Frankel[16] illustrated a beneficial effect of anticoagulation in two patients. Anticoagulation may be beneficial in both arterial hypotension and venous stasis, because further propagation of venous thrombosis can be limited and collateral circulation to the ischemic spinal cord improved. Similarly, aggressive hyperdynamic medical therapy in spinal cord injury should maximize perfusion of the ischemic region and may improve outcome. Results in one prospective clinical study indicate beneficial effects of hyperdynamic therapy in the management of spinal cord injury.[34]

Finally, late deterioration may be associated with vertebral artery injury. Patients with cervical fracture through the transverse foramens or dislocation often sustain vertebral artery injuries. Louw et al[22] showed vertebral artery injuries in 10 of 12 patients with facet joint dislocations. Parent reported that 5 of 12 patients with lateral cervical spine dislocation had development of vertebral dissection, whereas Willis et al[35] showed frequent vertebral artery injury with bone fragments penetrating the transverse foramens.[26] Patients with complete spinal cord injuries have a 24–62% frequency of vertebral dissection.[19,35]

However, comparatively fewer (0–11%) become symptomatic from these lesions, despite the prevalence

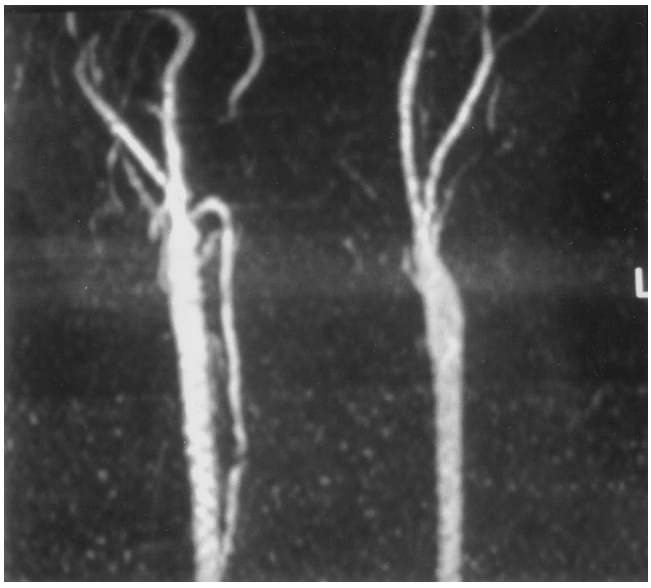

Figure 3. A 28-year-old man had only minimal movement of his upper limbs after motor vehicle accident. On admission, he was unresponsive to stimulation and without movement. Cervical magnetic resonance angiogram showed absence of flow through the left vertebral artery and a focal dissection of the right vertebral artery. The patient's condition deteriorated to brain death because of bilateral vertebral artery occlusion.

of such lesions in cervical spine trauma.[19,23,26,35] Symptomatic vertebral artery injuries may occur rapidly when bilateral or unilateral vertebral artery occlusion prevents cervicomedullary blood flow.[9,24,26,30] One of the patients was unconscious initially with only minimal upper limb movement and had acute progression to brain death caused by bilateral vertebral occlusions (Figure 3). Although this patient had an acute presentation, most symptomatic vertebral artery injuries typically become symptomatic in a delayed manner and are classified as late deterioration.[6,13,30,32] Patients show posterior circulation symptoms including visual field loss, swallowing abnormalities, cranial palsy, hoarseness, or paraesthesias. The delayed onset probably results from thromboemboli after recanalization of the vertebral artery. The late group patient in this series showed increased upper extremity weakness and cranial neuropathy 7 days after CSCI. Despite intravenous heparin, the injury progressed to subsequent brain death (Figure 3).

Neurologic deterioration in complete CSCI (ASIA A) is not uncommon. Specific causes have been identified in temporal subgroups. Inadequate immobilization or halo vest placement in ankylosing spondylitis, early reduction and surgery in young patients with facet dislocations, and symptomatic vertebral artery injury has been associated with secondary deterioration. Management of CSCI may be improved with recognition of these temporal patterns and appropriate intervention. Because the mortality rate in CSCI may be as high as 26%,[15] and in the current study death was associated with neurologic deterioration ($P = 0.033$), it is important to identify the cause of secondary worsening based on the early onset of symptoms to reduce subsequent injury. Adequate early immobilization, particularly in patients with ankylosing spondylosis; maintenance of sufficient perfusion, particularly in patients with facet dislocations; and rapid anticoagulation after symptoms of vertebral artery injury may prevent or at least reduce the extent of secondary neurologic injury after CSCI.

### ■ Key Points

- The temporal onset of secondary neurologic deterioration can be used to categorize complete CSCI as follows:
- Early deterioration
- Onset less than 24 hours
- Typically caused by traction or inadequate immobilization
- Delayed deterioration
- Onset between 24 hours and 7 days
- Ischemia possibly related to hypotension and/or venous obstruction
- Late deterioration
- Onset after 7 days
- Vertebral artery injury (may occur acutely, however, if severe)

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# Point of View

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In the present paper by Harrop et al the authors have examined the incidence and factors associated with neurologic deterioration following cervical spinal cord injury in a cohort of 182 patients evaluated at the Delaware Valley Regional Spinal Cord Injury Center between 1993 and 1999. It was found that 12 of 186 ASIA A patients with complete cervical cord injury sustained neurologic deterioration during the first 3 days posttrauma. The incidence of neurologic deterioration of 6% in this series is consistent with the reports in the literature which vary from 1.8% to 10%.[1,2] Early deterioration (less than 24 hours) occurred in four patients who had either ankylosing spondylitis or a severe central cord injury. Neurologic deterioration in these patients was related to attempts to institute traction/immobilization. Delayed deterioration (between 24 hours and 7 days) occurred most commonly in patients with flexion distraction injuries of the cervical spine. An association between episodes of hypotension and neurologic deterioration is reported by the authors, although precise details regarding this association are lacking. Late deterioration (greater than 7 days) occurred in a single patient with bilateral vertebral artery injuries.

This paper is an important one in that it highlights the incidence of neurologic deterioration following complete cervical spinal cord injury and the potential associated etiologic factors. Clinicians must exert a high degree of caution in treating patients with ankylosing spondylitis, cervical stenosis with associated spinal cord injury, distraction injuries of the spine, and facet dislocations that may be associated with vertebral artery injury.

Of further importance, the clinical observations reported in this paper emphasize the validity of the secondary injury concept of acute spinal cord injury.[3] In particular, persisting compression of the cord, instability of the vertebral column, and vascular disruption with ischemia represent important potentially treatable causes of secondary injury. Other potential mechanisms of delayed deterioration not mentioned by the authors include glutamatergic exitotoxicity, inflammation, and delayed programmed cell death or apoptosis.[4,5] Further work in disease-specific animal models and translational studies in the clinical setting are required to define the importance and potential relevance of these latter mechanisms.

## References

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