Acute Management of Cervical Spine Trauma and Spinal Cord Injury

ABSTRACT

Cervical spine injury can have life-changing consequences. At every stage of injury, we can intervene to meaningfully change patients' outcomes. On the field, a high index of suspicion is critical. Spinal immobilization prevents secondary injury, but immobilization, particularly use of a hard board, must be kept to a minimum. In the trauma bay, perfusion of the spinal cord is a priority to help prevent secondary spinal cord injury. This means addressing any cause of hypotension and understanding how to manage neurogenic shock. In the spinal-cord injured patient, hemodynamic management is an important adjunct.

KEYWORDS: Spinal Cord Injury; Trauma; Cervical; Ankylosing Spondylitis

CME Pre-test Quiz

Introduction and Clinical Scenario

In this article we will take you step by step from the moment of a cervical spine injury, to the ambulance, to the trauma bay, the CT scanner, all the way to the doorway of the OR. At each stage we will discuss what can you do to optimize this patient's outcome.

Sideline Evaluation of a Cervical Spine Injury

We start at the sidelines of a football game. You are the doctor covering. You watch in horror as the quarterback takes a head-on hit as his blind side is uncovered. His neck snaps back violently, as though he was slammed by a truck. He hits the ground, immobile.

Let's look at two different scenarios. In the first, as you rush onto the field and the crowd of players clears from around him, you see him slowly trying to get up. He

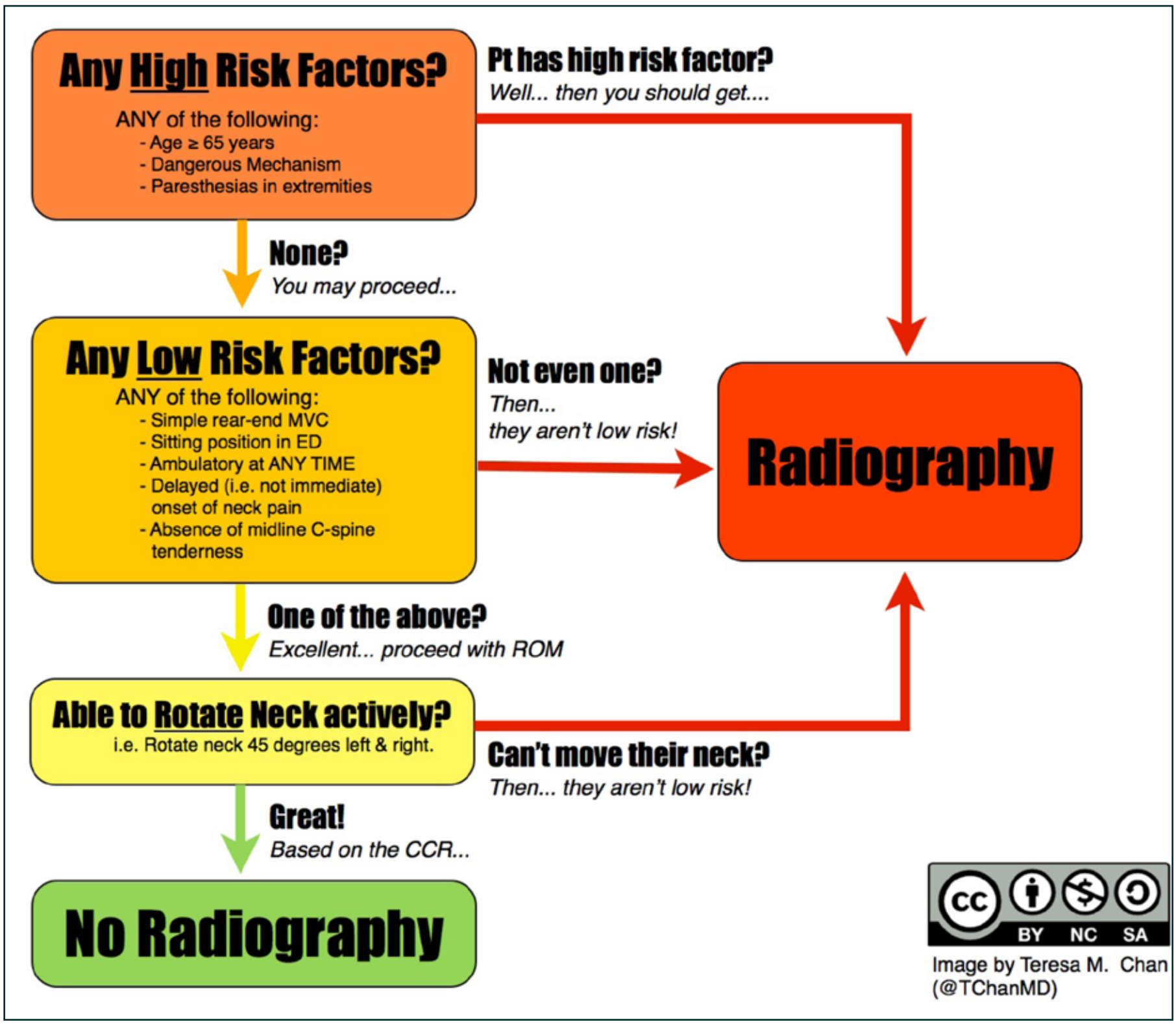
Mohamed Sarraj, MD, *Division of Orthopedic Surgery, McMaster University. Hamilton, Ontario*

Brian Drew, MD FRCSC, *Division of Orthopedic Surgery, McMaster University, Hamilton, Ontario*

quickly shakes his head and says "Doc, I swear I'm fine. I'm good to play. Don't even worry." What do you do?

When thinking about ruling out cervical spine injury in an alert stable patient, two clinical guidelines are front and centre. First, the NEXUS criteria, and second, the Canadian C-Spine Rule. A recent comparison article in the New England Journal of Medicine found that the Canadian C-spine rule had superior sensitivity (99.4% vs 90.7%) and specificity (45.1% vs 36.8%) when compared to the NEXUS criteria.[1] Below is a flowchart from the blog "Canadiem" outlining the use of the Canadian C-spine rule (this patient likely fails to be cleared because of the mechanism described above).[2]

The other thing to keep in mind in a well-appearing person who suffered a trauma to the head and neck is concussion. In a patient with GCS 15 who has had C-spine injury or basilar skull frac-



ture ruled out, concussion must be considered, and you should follow the golden rule; if in doubt, sit them out.

The second scenario is the real focus of this article. After the massive impact on the field, the crowd clears, and the quarterback is laying lifeless in the same position. He can't get up. Perhaps he has some numbness in his right arm. Perhaps he can't feel his legs. What do you do? First, take a deep breath. Next, call for help. The priority is always life over limb so make sure his airway is patent, he's breathing, and he has a pulse. Take off the facemask on the helmet and get him onto a hard board. Next, we think about the C-spine. Should you take off his helmet and shoulder pads? This is a critical question and matters greatly when you consider that underneath all that, there may be an unstable cervical spine fracture. That awareness makes the reasoning clearer. Your number one priority here is to immobilize the spine and prevent secondary injury. Removing the helmet alone extends the neck relative to neutral. Removing the shoulder pads alone drops the shoulders and flexes the neck. What we want is to keep the neck neutral. So, if you have enough hands and can maintain neutral alignment of the neck, remove both together. If not, leave them both on until he gets to the trauma bay.

OK, the helmet and shoulder pads are off, airway is clear, he's on a hard board and being lifted to the ambulance. Wait a minute… we forgot the cervical collar! Although there has been some criticism of the routine use of hard cervical collars, nearly all guidelines recommend their use in patients with potential spinal cord injury (SCI).[3,4] Ahn *et al.* performed a broad systematic review and assembled an expert team to publish guidelines on the prehospital management of possible spinal cord-injured patients. For initial spinal immobilization, they recommended the use of a hard-cervical collar, spinal board, and head immobilization with padding for the occiput and sacrum. Prolonged use of a hard spinal board can be dangerous, with soft tissue pressure ulcers beginning in as little as 30 minutes of immobilization.[5,6] Therefore, transfer off the hard spinal board as soon as possible, preferably after completion of the primary survey.[5]

Neurogenic Shock

You get to the trauma bay and the patient is suddenly hypotensive and bradycardic. There's no obvious bleeding, the primary survey is normal, and the FAST exam (Focused Assessment with Sonography in Trauma) for internal bleeding is negative. As you may have guessed, this patient is in neurogenic shock.

Neurogenic shock usually presents within 2 hours of injury and can occur suddenly in previously stable patients with normal vital signs, so your index of suspicion in the trauma bay must remain high.[7] The incidence of neurogenic shock, defined as an sBP <100 accompanied by a heart rate <80 beats per minute, is reported in between 14 and 44% of spinal cord injuries. The pathophysiology of neurogenic shock is related to a sympathetic blockade at the level of the spinal cord which prevents the compensatory ability to either vasoconstrict or increase cardiac output by increasing heart rate. The classic triad is hypotension, bradycardia, and peripheral vasodilation. Though commonly classified as a different category than hypovolemic shock (which typically presents with tachycardia), hypovolemic shock is still a significant confounder in the management of traumatic neurogenic shock. Patients with neurogenic shock lose their ability to compensate for degrees of hypovolemia that they otherwise would have managed physiologically.[7] Both should be treated concomitantly, and guidelines suggest initial management of neurogenic shock with intravenous fluid resuscitation to compensate for the vasogenic dilation resulting from unopposed parasympathetic tone. Another key point to consider is that vasopressors may in fact worsen perfusion in settings of hypovolemia and exacerbate secondary injury.[8] So, always fluid resuscitate first. If hypotension persists, then management proceeds to vasopressors and inotropes.[9]

Though there is no single preferred vasopressor, some authors recommend the use of Norepinephrine as a combined alpha and beta agonist. Use of an alpha selective agonist alone such as phenylephrine may precipitate reflex bradycardia in an already bradycardic patient.[7]

Back to our clinical scenario, our primary survey has been cleared, 4L of crystalloid did nothing to help the hypotension, but a round of Norepinephrine has finally brought the sBP over 100. Now what? This is only the first step. We will discuss mean arterial pressure (MAP) targets as they relate to spinal cord injury later, but neurogenic shock alone is an indicator that you need close hemodynamic management of this patient after they leave the trauma bay. They will need invasive blood pressure monitoring and a setting that can maintain MAP targets. Most authors suggest hemodynamic monitoring and management for 5-7 days after initial presentation for this population.[9]

Spinal Shock

'Spinal shock' and 'neurogenic shock' are commonly confused terms and we need to distinguish

between the two. Neurogenic shock is a term used to describe the autonomic consequences of spinal cord injury. In other words, the systemic, cardiovascular effects we just described. Spinal shock refers to finding directly related to the spine. Spinal shock is characterized by a lack of reflexes and loss of muscle tone below the level of injury. Neurogenic and spinal shock can occur separately or together. Spinal shock is temporary and resolves spontaneously days or weeks after the injury. The greater the injury to the spinal cord, the longer spinal shock will take to resolve.[10] In addition to flaccid paralysis and loss of the common reflexes, the bulbocavernosus reflex is perhaps the most important. The bulbocavernosus reflex is frequently used as the lighthouse of spinal shock, most sensitive to both its onset and resolution. The reflex entails anal contraction in response to squeezing the glans penis or clitoris, or pulling on a foley catheter and is mediated through the pudendal nerve.[11] Spinal shock can indicate a worse prognosis, but critically, its presence means that you cannot give a prognosis based on the current examination. If a patient appears fully paralyzed but is really in spinal shock, they cannot be assigned a spinal cord injury grade.

Spinal Cord Injury and ASIA scale

The American Spinal Injury Association (ASIA) published the International Standards for Neurological Classification of Spinal Injury to standardize the assessment and reporting of spinal cord injury.12 The ASIA Impairment Scale (AIS) is colloquially referred to as "the ASIA scale" and grades spinal cord injury from E (normal) to A (complete). The "Neurologic Level of Injury" refers to the most caudal spinal segment with normal sensation and antigravity strength. When we refer to a "C7 ASIA B" patient, this says the patient has antigravity function in the triceps but has no motor function from C8 down (sensory incomplete). While using this grading system is beyond the scope of primary care management, understanding the terminology is helpful.

Steroids

Now the primary survey is complete, you determine that the patient's legs are definitely weak. The question arises, should you administer steroids? In the world of spinal cord injury, the mere whisper of the 'S' word can get tempers flaring. The argument from biology is that steroids can reduce inflammation and decrease the consequences of secondary injury. That seemed confirmed in early clinical trials. NASCIS II was a landmark study that used a protocol of methylprednisolone as an initial bolus of 30 mg/kg followed by infusion of 5.4 mg/kg/hour for 23 hours given within eight hours of injury. This was a multicentre double-blinded randomized con-

trolled trial that enrolled nearly 500 patients.[13] The study found a statistically significant improvement in motor scores in a subset of patients. This study was met with wide enthusiasm when it was published in 1990 and administration of methylprednisolone for SCI quickly became standard of care across North America. Over the next few decades, the methodology of the study was brought into question, with authors pointing out that the subgroup analyses included 78 potential subgroups, and a P-value of 0.05 would yield a positive result 1 in 20 times by chance alone.[14] Subsequent studies found that administration of high dose methylprednisolone increased rates of gastro-intestinal bleeding and pneumonia.[15,16] In fact, 2013 guidelines from the American Academy of Neurosurgeons no longer recommended the administration of methylprednisolone in any case.[17,18] To add to the confusion, subsequent expert recommendations DID recommend methylprednisolone as an option within 8 hours of SCI, particularly in young healthy patients with incomplete spinal cord injuries. In summary, steroids in SCI are an option but not routine.[19]

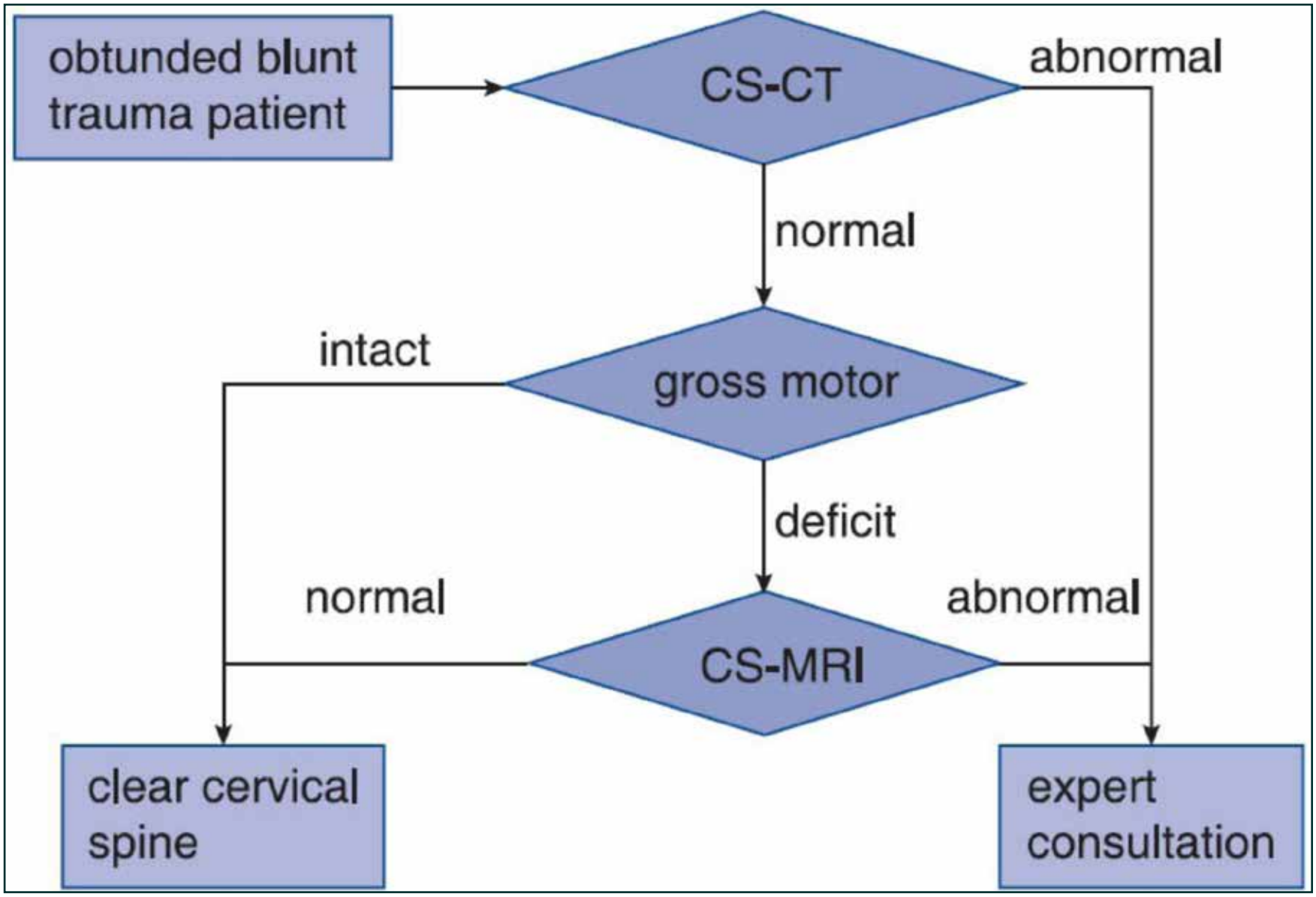
Spinal Cord Perfusion

What else can you do medically to help improve this patient's outcome? Consider the phases of spinal cord injury. This is an extremely well researched topic and the focus of cutting-edge basic science research. The initial phase is related to immediate hemorrhage and cell death from direct compression at the impact site. Secondary mechanisms are many, but one of the key things to understand is that cellular repair mechanisms following initial injury are impaired.[20] The edematous and damaged spinal cord now has higher metabolic demands and is simultaneously less able to tolerate stress.[21] This makes the spinal cord susceptible to second-hit ischemia from hypoxia or hypoperfusion. We can modify this risk by ensuring good ventilation and oxygenation, and by maintaining recommended MAP targets over 85 mmHg for 5-7 days.[22]

Imaging of Suspected Cervical Injury

Imaging is somewhat less contentious. Plain X-rays have a sensitivity as low as 52% at detecting cervical spine injury and should never be relied on when a CT scanner is available.[23] If there is any clinical suspicion for cervical spine injury (either by mechanism or clinical examination) a CT scan should be obtained. With the increasing availability of MRI and the unavoidable medicolegal concerns, there is now the question of the necessity of magnetic resonance imaging. In a patient with neurologic deficit, MRI can indicate ligamentous injury, determine the extent of cord edema, and define the zone of injury. A

more contentious point is whether an MRI is indicated as part of cervical spine clearance in an obtunded patient where the physical examination is unclear. Is a normal CT scan enough? Presently, the literature recommends CT scan alone as being sufficient to rule out unstable cervical spine injury with a reported 100% negative predictive value in identifying unstable cervical spine injury.[24,25] An interesting prospective study done at the Foothills Medical Centre in Calgary put this claim to the test. It included 402 intubated blunt trauma patients in the ICU with normal CT scans and asked; how many of these patients with normal CTs had abnormal dynamic imaging, i.e. flexion/extension X-rays? They found only one, and on further review that CT was actually mis-reported as normal.[26] What about MRI? A large systematic review using MRI on 1535 obtunded blunt trauma patients with normal CT scans found eleven (0.7%) with unstable injuries requiring surgery. The authors of that study still recommend clearance of the cervical spine in obtunded patients with normal CT scans in the absence of gross motor dysfunction.[27] If there is a clinical concern and MRI is unavailable, flexion-extension plain radio-



Borrowed from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4231259/

graphs can be taken with the patient in a well-fitted cervical collar. The authors recommend this algorithm which we use in our practice.

Finally, what about CT angiography (CTA) and vertebral artery injury? Recent studies have shown that when ordered outside of strict criteria, CT angiography had little utility and rarely changed management.[28] Table 1 outlines the modified Denver criteria for screening Blunt Cerebrovascular Injury (BCVI) where any one sign/symptom or risk factor should prompt a cervical CTA.[29]

Management of the Patient with an Ankylosed Spine

The ankylosed spine presents unique challenges. At our institution, we have unfortunately seen several patients with ankylosed spines sustain spinal cord injuries in the hands of well-meaning health care providers. The spines of patients with Ankylosing Spondylitis (AS) or DISH (Diffuse Idiopathic Skeletal Hyperostosis) behave more like long bones than normal spines. Osteoporosis is common in AS, and spinal fractures are reported at four times the incidence of the general population with an eleven-fold higher risk of spinal cord injury.[30] A fixed kyphotic deformity must be recognized in the ER since if these patients have their heads placed flat on a bed, or placed in a cervical collar, they can easily fracture and sustain a spinal cord injury.[31] Recognition of pre-existing deformity is of paramount importance.

Table 1: Modified Denver Criteria for BCVI Screening

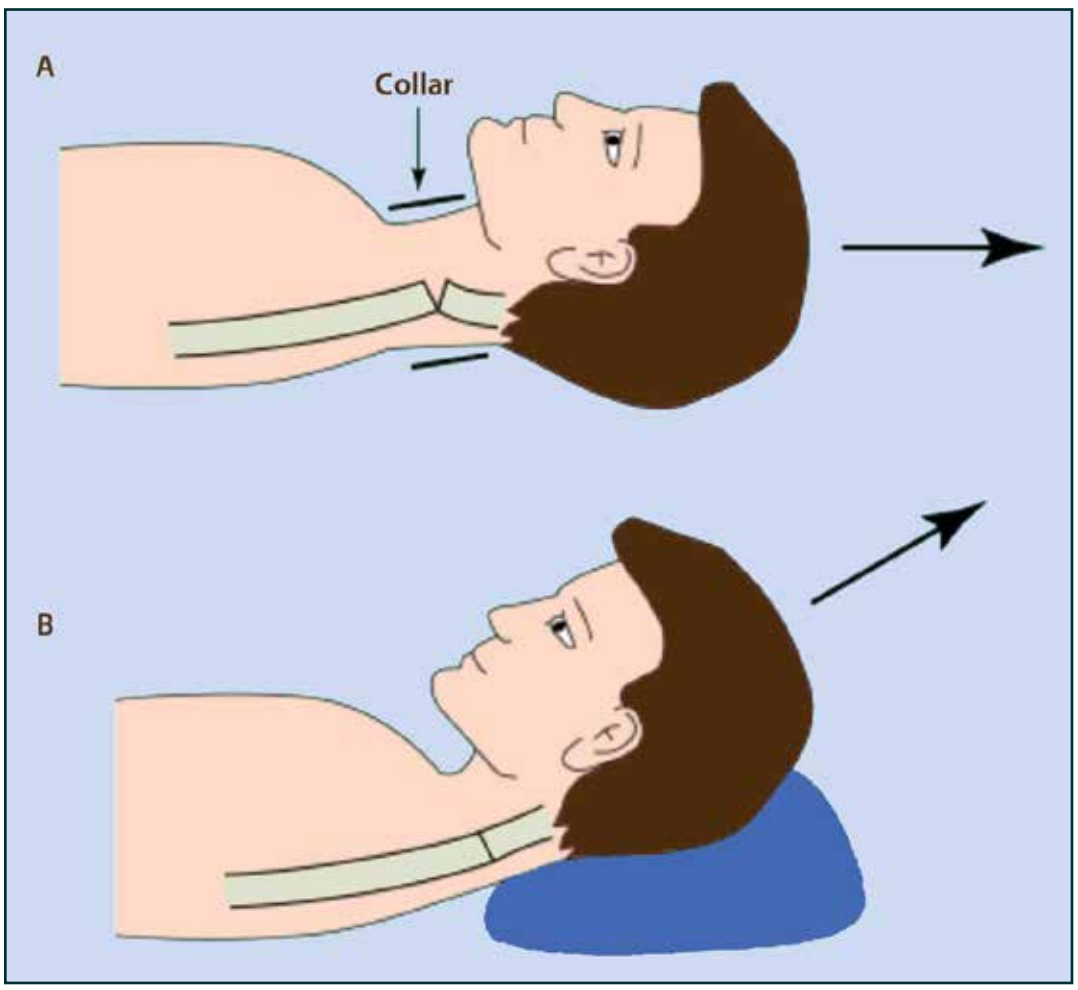
Signs/Symptoms	Risk Factors
• Arterial hemorrhage • Cervical bruit • Expanding neck hematoma • Focal neurologic deficit • Neuro exam inconsistent with head CT • Stroke on CT or MRI • Unexplained neurologic deficit	• Midface Fractures (Le Fort II or III) • Basilar Skull Fracture with carotid canal involvement • Diffuse axonal injury with GCS<6 • Cervical vertebral body or transverse foramen fracture, subluxation, or ligamentous injury • Any fracture at C1-3 • Hanging with anoxic brain injury • Seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling, pain, or altered mental status.

SUMMARY OF KEY POINTS

1. Minimize time in rigid immobilization as much as feasible.
2. Spinal shock is temporary flaccid paralysis and loss of reflexes. You cannot give a prognosis for a spinal cord-injured patient in spinal shock.
3. Recognize patients with stiff spines (such as in ankylosing spondylitis) and immobilize them in their natural position of comfort to avoid secondary injury.
4. What we can do to improve neurologic outcomes in spinal cord injury: Maintain spinal cord perfusion through oxygenation and blood pressure management, avoid secondary injury through immobilization, and facilitate early surgical decompression (<24hr)

Patients should be immobilized in their natural position with as many pillows, blankets, or sandbags as necessary.

Spine fractures in AS can be devastating and a high index of suspicion in these patients is recommended. There is a significantly higher incidence of both non-contiguous injury and epidural hematoma. We recommend a whole spine CT scan followed by an MRI if abnormalities are detected.[32]



Surgical Timing

If you diagnose a cervical spine injury that requires surgery, how quickly should the operation be scheduled? The purpose of surgery is to first stabilize and then decompress the spinal cord. It would be logical to think emergent decompression is preferred, and that the faster we can get to the OR, the better. Evidence is beginning to support this intuition. A large prospective cohort study in 2012 found that surgical decompression within 24 hours had improved neurologic outcome compared to decompression after 24 hours.[33] This finding has been confirmed in further rigorous analyses.[34] Current research is beginning to show that there may be benefit to 'ultra-early' decompression within 8-12 hours after injury, although this has yet to be confirmed in large prospective controlled studies.[35–37] What we

CLINICAL PEARLS

1. The Canadian C-spine Rule is the preferred algorithm to clear the cervical spine after trauma
2. X-rays are not sufficient to rule out cervical spine injury. CT scan is the gold standard.
3. Neurogenic shock is a distributive syndrome characterized by the triad of hypotension, bradycardia, and peripheral vasodilation. First line treatment is fluid resuscitation, then vasopressors.
4. Patients with stiff spines (ankylosing spondylitis or DISH) have high rates of spine fractures and non-contiguous injuries. Full spine CT scans should be obtained.

do know is that "Time is Spine" and the patient should be decompressed as soon as possible; definitely within 24 hours.[38]

Conclusion

A cervical spine injury is intimidating to the clinician and potentially devastating to the patient, but the principles of management are simple and straightforward. Immobilize the patient in their own neutral position to avoid secondary injury, maintain perfusion of the cord (with oxygen and adequate blood pressure) and facilitate speedy decompression and stabilization.

References

1. Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-Spine Rule versus the NEXUS Low-Risk Criteria in Patients with Trauma. New England Journal of Medicine. 2003;349(26):2510-2518. doi:10.1056/NEJMoa031375
2. BoringEM TC. Tiny Tip | Canadian C-spine Rule Mnemonic. CanadiEM. Published December 29, 2014. Accessed March 18, 2023. https://canadiem.org/tiny-tip-canadian-c-spine-rule/
3. Sundstrøm T, Asbjørnsen H, Habiba S, Sunde GA, Wester K. Prehospital Use of Cervical Collars in Trauma Patients: A Critical Review. Journal of Neurotrauma. 2014;31(6):531-540. doi:10.1089/neu.2013.3094
4. Ahn H, Singh J, Nathens A, et al. Pre-Hospital Care Management of a Potential Spinal Cord Injured Patient: A Systematic Review of the Literature and Evidence-Based Guidelines. Journal of Neurotrauma. 2011;28(8):1341-1361. doi:10.1089/neu.2009.1168
5. Milland K, Al-Dhahir MA. EMS Long Spine Board Immobilization. In: StatPearls. StatPearls Publishing; 2022. Accessed March 17, 2023. http://www.ncbi.nlm.nih.gov/books/NBK567763/
6. Vickery D. The use of the spinal board after the pre-hospital phase of trauma management. Emergency Medicine Journal. 2001;18(1):51-54. doi:10.1136/emj.18.1.51
7. Taylor MP, Wrenn P, O'Donnell AD. Presentation of neurogenic shock within the emergency department. Emerg Med J. 2017;34(3):157-162. doi:10.1136/emermed-2016-205780
8. Cardiovascular changes following spinal cord injury - Western University. Accessed March 17, 2023. https://ocul-uwo.primo.exlibrisgroup.com
9. Ruiz IA, Squair JW, Phillips AA, et al. Incidence and Natural Progression of Neurogenic Shock after Traumatic Spinal Cord Injury. Journal of Neurotrauma. 2018;35(3):461-466. doi:10.1089/neu.2016.4947
10. Ko HY. Revisit Spinal Shock: Pattern of Reflex Evolution during Spinal Shock. Korean J Neurotrauma. 2018;14(2):47-54. doi:10.13004/kjnt.2018.14.2.47
11. Previnaire JG. The importance of the bulbocavernosus reflex. Spinal Cord Ser Cases. 2018;4(1):1-2. doi:10.1038/s41394-017-0012-0
12. Kirshblum SC, Burns SP, Biering-Sorensen F, et al. International standards for neurological classification of spinal cord injury (Revised 2011). J Spinal Cord Med. 2011;34(6):535-546. doi:10.1179/204577211X13207446293695
13. Bracken MB, Shepard MJ, Collins WF, et al. A Randomized, Controlled Trial of Methylprednisolone or Naloxone in the Treatment of Acute Spinal-Cord Injury. New England Journal of Medicine. 1990;322(20):1405-1411. doi:10.1056/NEJM199005173222001

14. Bydon M, Lin J, Macki M, Gokaslan ZL, Bydon A. The Current Role of Steroids in Acute Spinal Cord Injury. World Neurosurgery. 2014;82(5):848-854. doi:10.1016/j.wneu.2013.02.062
15. Chikuda H, Yasunaga H, Takeshita K, et al. Mortality and morbidity after high-dose methylprednisolone treatment in patients with acute cervical spinal cord injury: a propensity-matched analysis using a nationwide administrative database. Emerg Med J. 2014;31(3):201-206. doi:10.1136/emermed-2012-202058
16. Sultan I, Lamba N, Liew A, et al. The safety and efficacy of steroid treatment for acute spinal cord injury: A Systematic Review and meta-analysis. Heliyon. 2020;6(2):e03414. doi:10.1016/j.heliyon.2020.e03414
17. Resnick DK. Updated Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injury. Neurosurgery. 2013;72 Suppl 2:1. doi:10.1227/NEU.0b013e318276ee7e
18. Walters BC, Hadley MN, Hurlbert RJ, et al. Guidelines for the management of acute cervical spine and spinal cord injuries: 2013 update. Neurosurgery. 2013;60(CN_suppl_1):82-91. doi:10.1227/01.neu.0000430319.32247.7f
19. Fehlings MG, Tetreault LA, Wilson JR, et al. A Clinical Practice Guideline for the Management of Acute Spinal Cord Injury: Introduction, Rationale, and Scope. Global Spine J. 2017;7(3 Suppl):84S-94S. doi:10.1177/2192568217703387
20. Oyinbo C. Secondary injury mechanisms in traumatic spinal cord injury: a nugget of this multiply cascade. Acta Neurobiologiae Experimentalis. 2011;71(2):281-299.
21. Ambrozaitis KV, Kontautas E, Spakauskas B, Vaitkaitis D. [Pathophysiology of acute spinal cord injury]. Medicina (Kaunas). 2006;42(3):255-261.
22. Rabinstein AA. Traumatic Spinal Cord Injury. In: Rabinstein AA, ed. Neurological Emergencies: A Practical Approach. Springer International Publishing; 2020:271-280. doi:10.1007/978-3-030-28072-7_14
23. Holmes JF, Akkinepalli R. Computed tomography versus plain radiography to screen for cervical spine injury: a meta-analysis. J Trauma. 2005;58(5):902-905. doi:10.1097/01.ta.0000162138.36519.2a
24. Tins BJ. Imaging investigations in Spine Trauma: The value of commonly used imaging modalities and emerging imaging modalities. J Clin Orthop Trauma. 2017;8(2):107-115. doi:10.1016/j.jcot.2017.06.012
25. Patel MB, Humble SS, Cullinane DC, et al. Cervical spine collar clearance in the obtunded adult blunt trauma patient: A systematic review and practice management guideline from the Eastern Association for the Surgery of Trauma. J Trauma Acute Care Surg. 2015;78(2):430-441. doi:10.1097/TA.0000000000000503
26. Hennessy D, Widder S, Zygun D, Hurlbert RJ, Burrowes P, Kortbeek JB. Cervical Spine Clearance in Obtunded Blunt Trauma Patients: A Prospective Study. Journal of Trauma and Acute Care Surgery. 2010;68(3):576. doi:10.1097/TA.0b013e3181cf7e55
27. James IA, Moukalled A, Yu E, et al. A systematic review of the need for MRI for the clearance of cervical spine injury in obtunded blunt trauma patients after normal cervical spine CT. J Emerg Trauma Shock. 2014;7(4):251-255. doi:10.4103/0974-2700.142611
28. Hagedorn JCI, Emery SE, France JC, Daffner SD. Does CT Angiography Matter for Patients with Cervical Spine Injuries? JBJS. 2014;96(11):951. doi:10.2106/JBJS.M.00036
29. Biffl WL, Cothren CC, Moore EE, et al. Western Trauma Association Critical Decisions in Trauma: Screening for and Treatment of Blunt Cerebrovascular Injuries. Journal of Trauma and Acute Care Surgery. 2009;67(6):1150. doi:10.1097/TA.0b013e3181c1c1d6
30. Maarouf A, McQuown CM, Frey JA, Ahmed RA, Derrick L. Iatrogenic Spinal Cord Injury in a Trauma Patient with Ankylosing Spondylitis. Prehosp Emerg Care. 2017;21(3):390-394. doi:10.1080/10903127.2016.1263369
31. Papadopoulos MC, Chakraborty A, Waldron G, Bell BA. Exacerbating cervical spine injury by applying a hard collar. BMJ. 1999;319(7203):171-172.
32. Rustagi T, Drazin D, Oner C, et al. Fractures in Spinal Ankylosing Disorders: A Narrative Review of Disease and Injury Types, Treatment Techniques, and Outcomes. Journal of Orthopaedic Trauma. 2017;31:S57. doi:10.1097/BOT.0000000000000953
33. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). PLoS One. 2012;7(2):e32037. doi:10.1371/journal.pone.0032037
34. Badhiwala JH, Wilson JR, Witiw CD, et al. The influence of timing of surgical decompression for acute spinal cord injury: a pooled analysis of individual patient data. The Lancet Neurology. 2021;20(2):117-126. doi:10.1016/S1474-4422(20)30406-3
35. Lee DY, Park YJ, Kim HJ, Ahn HS, Hwang SC, Kim DH. Early surgical decompression within 8 hours for traumatic spinal cord injury: Is it beneficial? A meta-analysis. Acta Orthopaedica et Traumatologica Turcica. 2018;52(2):101. doi:10.1016/j.aott.2017.12.001
36. Eraifej J, Nnadi C, Ganau M. Early and ultra-early surgical decompression for acute spinal cord injury: bracing for the winds of change. Eur Spine J. 2022;31(7):1691-1692. doi:10.1007/s00586-021-07030-9
37. Yousefifard M, Hashemi B, Forouzanfar MM, Khatamian Oskooi R, Madani Neishaboori A, Jalili Khoshnoud R. Ultra-early Spinal Decompression Surgery Can Improve Neurological Outcome of Complete Cervical Spinal Cord Injury; a Systematic Review and Meta-analysis. Arch Acad Emerg Med. 2022;10(1):e11. doi:10.22037/aaem.v10i1.1471
38. Ramakonar H, Fehlings MG. 'Time is Spine': new evidence supports decompression within 24 h for acute spinal cord injury. Spinal Cord. 2021;59(8):933-934. doi:10.1038/s41393-021-00654-0