ISSN : 2350-0743

*International Journal of Recent Advances in Multidisciplinary Research*

*Vol. 12, Issue 02 pp. 10786-10788, February, 2025*

# RESEARCH ARTICLE

## CARDIOVASCULAR MANAGEMENT OF NEUROGENIC SHOCK

***Oscar Daniel Guerrero Ortiz and Alejandro Obregón Corona**

Centro Medico ABC, campus Santa Fe

**ARTICLE INFO**

***Article History***

Received 19th November, 2024
Received in revised form
17th December, 2024
Accepted 26th January, 2025
Published online 28th February, 2025

***Keywords:***

Neurogenic shock, Anesthetic management, Spinal cord injury, Hemodynamic stability, Vasopressor therapy, Perioperative care.

****Corresponding author:***
***Oscar Daniel Guerrero Ortiz***

**ABSTRACT**

Neurogenic shock results from acute spinal cord injury, affecting both sensory and motor conduction at the cervical and upper thoracic levels. This leads to hemodynamic alterations characterized by hypotension, bradycardia, and other autonomic dysreflexias due to the loss of sympathetic tone. This condition is associated with high morbidity and often necessitates surgical intervention, requiring a multidisciplinary approach and posing a challenge for anesthesiologists in perioperative management. The primary treatment goals are neuroprotection, neuroregeneration, and the prevention of secondary injury. Management begins with a thorough preoperative evaluation, including airway management planning, induction strategies that preserve hemodynamic stability, fluid resuscitation, anticipation of potential bleeding risks, and the administration of vasopressor agents and blood products.



***Citation: Oscar Daniel Guerrero Ortiz and Alejandro Obregón Corona. 2025.*** "Cardiovascular Management of Neurogenic Shock*", International Journal of Recent Advances in Multidisciplinary Research,* 12, (02), 10786-10788.

# INTRODUCTION

The term "neurogenic shock" was first introduced by surgeon Alfred Blalock in the late 1920s, describing peripheral circulatory insufficiency associated with spinal cord injury, primarily characterized by significant hypotension and bradycardia. Modern understanding recognizes that these hemodynamic changes result from the loss of sympathetic activity in preganglionic neurons, leading to microvascular hypoperfusion of the spinal cord.

**Epidemiology:** The incidence of acute spinal cord injury varies globally, with traumatic causes being the most frequent. In the United States, the estimated incidence is 40 per million people per year (approximately 12,400 cases annually), compared to 15 per million in Western Europe. In Mexico, a study published by the National Institute of Rehabilitation reported an incidence of 18 per million inhabitants, with a peak age of occurrence in the 30s and a male-to-female ratio of 6:1. The most common cause identified was falls from height.

**Pathophysiology:** The initial injury leads to mechanical disruption of the spinal column, causing compression or transection of the spinal cord. This damages neurons, ligodendrocytes, vasculature, and compromises the blood-brain barrier. A sustained cascade of secondary injuries follows, exacerbating spinal cord damage and neurological dysfunction. Proinflammatory cytokine levels (TNF-α, IL-1β) rise within minutes post-injury. Subsequent alterations in the blood-spinal cord barrier contribute to progressive inflammation, leading to mechanical compression that can extend across multiple spinal segments, worsening the injury.

**Anesthetic Management:** Initial management involves early injury identification, cardiovascular resuscitation, and definitive surgical treatment. The primary goal is to mitigate secondary injury and improve neurological outcomes. Cervical or thoracic spinal cord injury with phrenic or intercostal nerve paralysis can result in hypoventilation and hypercapnic-hypoxemic respiratory failure. Rapid sequence intubation (RSI) with spinal immobilization is the standard approach. Videolaryngoscopy provides superior glottic visualization and reduces intubation time when performed with a neutral neck position. Hemodynamic assessment is crucial since cervical and thoracic injuries disrupt sympathetic-parasympathetic balance, resulting in neurogenic shock. Initial treatment includes fluid resuscitation to restore intravascular volume and transiently increase venous return. Due to sympathetic denervation, cardiac output remains low, with impaired inotropy and chronotropy. A neurological evaluation should determine injury severity. Motor and sensory function

deteriorate below the lesion level. The American Spinal Injury Association (ASIA) Impairment Scale (AIS) classifies neurological impairment through dermatome-based sensory exams, myotome-based motor assessments, and rectal examinations.

**Anesthetic Goals**

- Minimize secondary injury.
- Optimize cardiovascular function for adequate spinal cord perfusion.
- Implement intraoperative monitoring, including continuous pulse oximetry, ECG, blood pressure monitoring, capnography, and temperature regulation.

**Neuromonitoring**

- Level I evidence supports intraoperative somatosensory evoked potentials (SSEP) and transcranial motor evoked potentials (TcMEP).
- Electromyography aids in detecting and preventing nerve root injuries during decompression or pedicle screw fixation.
- Volatile anesthetics decrease SSEP amplitude and prolong cortical response latency. Motor evoked potentials are more sensitive to inhaled agents. Total intravenous anesthesia (TIVA) is recommended when motor responses are monitored intraoperatively.

**Pharmacologic Considerations**

- Pre-induction anticholinergic agents (glycopyrrolate, atropine, ephedrine) prevent bradycardia.
- Transcutaneous pacing may be necessary in high spinal cord injuries.
- Induction agents should be selected cautiously. Propofol, while commonly used, can exacerbate hypotension due to sympathetic denervation and reduced venous return.
- Mean arterial pressure (MAP) should be maintained between 85–90 mmHg intraoperatively. MAP >90 mmHg increases bleeding risk and reduces surgical field visibility, while MAP <85 mmHg worsens secondary spinal cord injury.

**Hemodynamic Management**

- Blood loss increases with each additional instrumented spinal level. Prophylactic placement of two large-bore IV catheters is recommended.
- Central venous catheterization may be indicated for vasoactive medication administration or if peripheral access is limited.
- The spinal cord has minimal anaerobic reserve, and injured tissue is highly susceptible to hypoxia and ischemia. A liberal transfusion strategy is often preferred over a restrictive approach in neurogenic shock.

**Vasopressor Therapy**

- **Lesions at T1-T4:** Sympathetic denervation of the heart leads to predominant vagal activity, causing hypotension and bradycardia. **First-line therapy: norepinephrine (α1 and β1 agonist).** Dopamine and epinephrine are alternative options.
- **Lesions below T6:** Hypotension results mainly from peripheral vasodilation, while cardiac innervation remains intact. **First-line therapy: phenylephrine (α1 agonist).**
- Dopamine is associated with higher cardiogenic complications (e.g., ventricular tachycardia, atrial fibrillation, troponin elevation) and should not be first-line therapy.

**Emerging Technologies**

- **Intrathecal lumbar catheters:** Directly measure spinal cord pressure, aiding in hypotension prevention and secondary injury mitigation.
- **Near-infrared spectroscopy (NIRS):** Transdural monitoring of oxygenated and deoxygenated hemoglobin concentrations provides real-time tissue oxygenation assessment.

**Table 1. Vasopressor Agents for Neurogenic Shock Management**

| Agent | Receptors | Dose | Recommendations |
|---|---|---|---|
| Norepinephrine | α1 (+++++), β1 (+++) | 0.05-0.5 µg/kg/min | First-line for ≥T6 lesions; recommended for <T6 with tachyarrhythmia history. |
| Phenylephrine | α1 (+++++) | 0.5-2.0 µg/kg/min | First-line for <T6 lesions without bradycardia; avoid in ≥T6 lesions. |
| Dopamine | α1 (+++), β1 (++++) | 4-10 µg/kg/min | Avoid as first-line due to cardiovascular complications. |
| Epinephrine | α1 (+++++), β1 (++++) | 0.05-0.5 µg/kg/min | Consider for refractory hypotension and/or bradycardia. |

# CONCLUSION

Neurogenic shock results from acute spinal cord injury, leading to severe hemodynamic alterations such as hypotension and bradycardia. This medical emergency often necessitates surgical intervention and presents significant challenges for anesthesiologists. Initial treatment focuses on fluid resuscitation to restore intravascular volume and cardiac output, followed by vasopressor administration tailored to injury level and patient-specific risks. Norepinephrine is recommended for high thoracic (≥T6) and cervical lesions due to sympathetic denervation and unopposed vagal activity, which cause hypotension and bradycardia. α-agonists should be avoided at these levels to prevent exacerbation of bradycardia. Dopamine is associated with adverse cardiovascular effects and should be avoided when possible. Future advancements in cardiovascular management for neurogenic shock focus on improved spinal cord blood flow monitoring through invasive and non-invasive techniques, optimizing perfusion, and preventing secondary damage.

# REFERENCES

1. Volski, A., Ackerman, D. J. (2019). Neurogenic Shock. In: Stawicki, S. P., Swaroop, M. (Eds.). *Clinical Management of Shock - The Science and Art of Physiological*

*Restoration*. Intech Open. https://doi.org/10.5772/intechopen.89915
2. Mathews L., Kla K. (2020). Anesthesia for adults with acute spinal cord injury. In: Pasternak J. (Ed.), *Wolters Kluwer: Uptodate*.
3. Ahuja, C., Wilson, J., Nori, S., et al. (2017). Traumatic spinal cord injury. *Nat Rev Dis Primers*, 3, 17018. https://doi.org/10.1038/nrdp.2017.18.
4. Perez R., Martin del Campo S., Renan S. (2008). Aspectos epidemiológicos de la lesión medular de la población del Centro Nacional de Rehabilitación. *Revista Mexicana de Medicina Física y Rehabilitación*, 20, 74–84. https://www.medigraphic.com/pdfs/fisica/mf-2008/mf083-4d.pdf.
5. Stephen Bonner, MRCP FRCA FFICM, Caroline Smith, FRCA. (2013). Initial management of acute spinal cord injury. *Continuing Education in Anaesthesia Critical Care & Pain*, 13(6), 224–231. https://doi.org/ 10.1093/ bjaceaccp/mkt021.
6. Sacino A., Rosenblatt K. (2019). Early Management of Acute Spinal Cord Injury-Part I: Initial Injury to Surgery. *J Neuroanaesth Crit Care*, 6(3), 213-221. https://doi.org/ 10.1055/ s-0039-1694688.
7. Lee YS., Kim KT., Kwon BK. (2021). Hemodynamic Management of Acute Spinal Cord Injury: A Literature Review. *Neurospine*, 18(1), 7-14. https://doi.org/ 10.14245 /ns.2040144.072.
8. Menacho ST., Floyd C. (2021). Current practices and goals for mean arterial pressure and spinal cord perfusion pressure in acute traumatic spinal cord injury: Defining the gaps in knowledge. *J Spinal Cord Med*, 44(3), 350-356. https://doi.org/10.1080/10790268.2019.1660840.
9. Casha S., Christie S. (2011). A systematic review of intensive cardiopulmonary management after spinal cord injury. *J Neurotrauma*, 28(8), 1479-95. https://doi.org/10.1089/neu.2009.1156.
10. Hawryluk G., Whetstone W., Saigal R., et al. (2015). Mean Arterial Blood Pressure Correlates with Neurological Recovery after Human Spinal Cord Injury: Analysis of High Frequency Physiologic Data. *J Neurotrauma*, 32(24), 1958-67. https://doi.org/10.1089/neu.2014.3778.
11. Iovine JA., Villanueva RD., Werth CM., et al. (2022). Contemporary hemodynamic management of acute spinal cord injuries with intravenous and enteral vasoactive agents: A narrative review. *Am J Health Syst Pharm*. https://doi.org/10.1093/ajhp/zxac164.
12. Manolopoulos, P., Boutsikos, I., Boutsikos, P., et al. (2020). Current use and advances in vasopressors and inotropes support in shock. *Journal Of Emergency And Critical Care Medicine*, 4. https://doi.org/10. 21037/jeccm. 2019.12.03.

*******