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# Neurogenic Shock

*Anna Volski and Daniel J. Ackerman*

## Abstract

Neurogenic shock is a state characterized by hypotension, bradycardia, and other evidence of autonomic dysfunction. The most common cause is acute spinal cord injury (SCI), which will be the subject of our focus. Because the typical autonomic reflexes may be either abolished or dysregulated, appropriate treatment requires an understanding of the neuroanatomic substrate for the change. In this chapter, we will explore the root cause for neurogenic shock, differentiating it from spinal shock, and discuss those patients at risk and generally accepted treatment paradigms. The timeframe for manifestation of neurogenic shock is variable and it can quickly progress to cause secondary injury or death, so appropriate monitoring requires a high level of suspicion and diligence.

**Keywords:** neurogenic, shock, hypotension, bradycardia, hypothermia, autonomic, sympathetic, vasomotor, dysreflexia, spinal cord

## 1. Introduction

Imagine that you are in the trauma bay receiving a patient with a suspected high spinal cord injury due to a motor vehicle crash. Emergency medical responders sign out to you that the blood pressure has been fine on the way in, 110/60 mmHg with a heart rate in the 60s. As you complete your primary survey and get the patient on to your monitors you find the pressure has plummeted to 80/50 but rather than tachycardia the patient's heart rate is only 45. The rhythm is sinus bradycardia, the hemoglobin on your initial lab is 14.4 g/dl and there is no clear source of blood loss.

All too often neurogenic shock is an under-recognized but deadly cause of hypotension, bradycardia, and other complications related to spinal cord injury. In this chapter, we examine the definition, diagnosis, and treatment taking special care to differentiate it from spinal shock. We also briefly discuss autonomic dysreflexia and the role that neurogenic shock and autonomic dysreflexia can play in the rehabilitation setting.

## 2. Methods

Searches were conducted using the PubMed database for “neurogenic shock.” The Lewis Katz School of Medicine online textbook library was also referenced using the same search terms, as were hard copies of reference textbooks 10 and 11.

### **3. Background**

#### **3.1 History**

The contemporary understanding of “neurogenic shock” was born with Alfred Blalock’s “classification of peripheral circulatory failure,” which he described in articles between 1927 and 1942. His “pure” types of shock included cardiogenic, hematogenic (better known as hypovolemic), neurogenic, and vasogenic (anaphylactic and septic) [1]. In descriptions of the neurogenic type, Blalock wrote: “the primary alteration is vasodilatation dependent on diminished constrictor tone as a result of influences acting through the nervous system,” a description that has persisted [2]. At the time, Blalock associated neurogenic shock with spinal cord injury, spinal anesthesia, and vasovagal syncope [2]. Though our definition of neurogenic shock has evolved since Blalock’s time, his classification system remains, and so do the challenges of defining, identifying, and managing neurogenic shock.

#### **3.2 Neurogenic shock vs. spinal shock**

Neurogenic shock is considered distributive in nature and refers to the loss of vasomotor tone and the instability that subsequently follows due to an imbalance in the autonomic nervous system (ANS) [3, 4]. Loss of sympathetic tone leads to unopposed parasympathetic control, manifested by refractory hypotension and bradycardia [3]. Other aspects of neurogenic shock include temperature dysregulation, autonomic dysreflexia, and orthostatic hypotension [5]. Aside from bradycardia and hypotension, many patients develop autonomic dysreflexia defined as a profound autonomic response to what would typically be a mild stimulus such as bladder or bowel distension [6]. The presence of a focal neurological deficit is not required for diagnosis, and although this is most often encountered in the setting of an acute SCI, theoretically any damage resulting in the loss of cerebral control of the autonomic nervous system may place a patient at risk for neurogenic shock. Neurogenic and spinal shocks are distinct consequences of spinal cord injury and the terms should not be used interchangeably.

Neurogenic shock most often occurs after an acute injury above T6, with a possible incidence of 29% in the cervical SCI population and 19% in the thoracic SCI population [4]. The onset may be variable in relation to the timing of the injury, but in SCI patients it most commonly manifests within 2 h of the trauma [7]. In most patients it is transient and may last for 1–6 weeks after injury [5, 8].

Conversely, spinal shock is the transient loss of reflexes and sensorimotor function that manifests acutely after injury to the spinal cord. It is a symptom of underlying spinal cord injury and the term “shock” in this situation does not refer to cardiovascular instability. Spinal shock is characterized by flaccid paralysis, anesthesia, and areflexia or hyporeflexia [3, 7]. Note that often enough the two may be present in the same patient but their natural course and treatment are distinct; furthermore there are often other potential causes for shock in the trauma patient (e.g. hypovolemic secondary to acute blood loss) clouding the diagnosis. It has been proposed that there are four phases of complete spinal shock resolution: hyporeflexia or areflexia (0–24 h), initial return of reflexes (1–3), early hyperreflexia (day 4 – 1 month) and spasticity (1–12 months) [7]. The total duration of spinal shock depends on the definition of its resolution. Resolution has been defined as the appearance of any reflex, the appearance of the bulbocavernosus reflex, return of reflex detrusor functions, or the return of deep tendon reflexes [7, 9]. Depending on which definition you use, spinal shock can last anywhere from days to months.

	Spinal shock	Neurogenic shock
Definition	Transient loss of reflexes and sensorimotor function below level of injury	Loss of sympathetic tone with unopposed parasympathetic control, leading to cardiovascular instability
Blood pressure	Hypotension	Hypotension
Heart rate	Bradycardia	Bradycardia
Associated autonomic symptoms	Difficulty breathing, bowel and bladder dysfunction, priapism	Autonomic dysreflexia, orthostatic hypotension, temperature dysregulation
Motor	Flaccid paralysis	Varies with injury
Reflexes	Areflexia or hyporeflexia in early stage, hyperreflexia in later stage of resolution	Varies with injury
Duration	Days to months	Most often 1–6 weeks
Treatment	Stabilization and treatment of underlying injury	Fluids and vasopressors with appropriate temperature monitoring

**Table 1.**  
*Comparison between spinal shock and neurogenic shock [3–5, 7–9].*

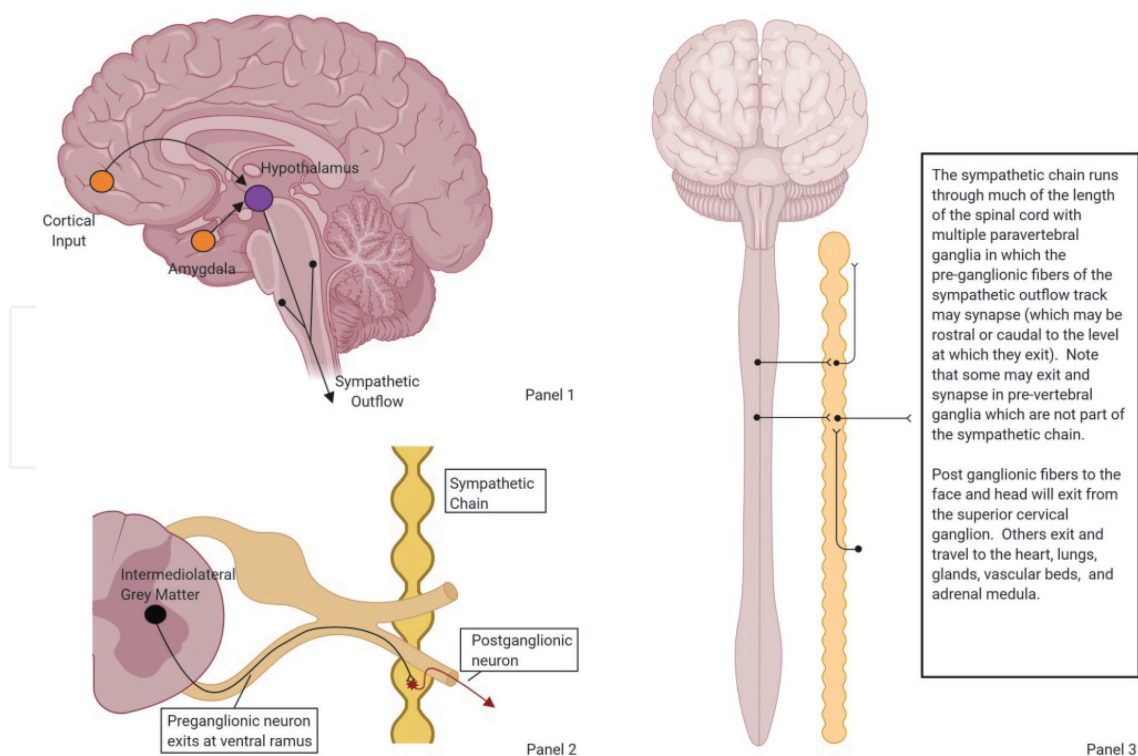
Spinal shock and neurogenic shock may occur at the same time in a patient, complicating management, but they are not synonymous (see **Table 1**). Perhaps the most significant distinction is the difference in their management. The mainstay of treatment of neurogenic shock is fluids and vasopressors. As a transient symptom of spinal cord injury, spinal shock is expected to resolve on its own, in a predictable manner. Once spinal shock resolves, the underlying injury may be more accurately assessed.

#### 4. Anatomic and epidemiologic considerations

The autonomic nervous system constitutes the involuntary control of many crucial systems of the body. Described as a system of visceral sensory inputs and motor responses, it maintains homeostasis and responds to both internal and external stimuli by manipulating the balance between its main divisions, the sympathetic and parasympathetic systems [10]. Although much of the autonomic system includes spinal reflex arcs with visceral motor neurons originating in ganglia lying peripheral to the spinal cord, there is overarching control exerted by multiple systems in the brain (**Figure 1**) [5]. It is the loss of this input from above that produces the dysfunction of the system, leading to shock [3]. The ANS maintains control of vital functions in the heart, vasculature, lungs, liver, digestive and para-digestive organs, glands, and reproductive organs. Although there are many involved neuropeptides, norepinephrine is the most common effector molecule in the sympathetic division and exerts its influence on both alpha and beta receptors, as well as the adrenal gland [10].

Estimating the true incidence of neurogenic shock is difficult for multiple reasons. The overall definition is reasonably broad, and a patient may experience multiple subtypes of shock at the same time. Furthermore, there may not be a simple direct imaging correlate that is easily elucidated (for example, one can see a significant anterior or lateral cord injury and correlate the physical symptoms to the level of the lesion, but isolating the level of injury in the intermediolateral





**Figure 1.**

*Overview of sympathetic outflow. Panel 1 (top left): CNS control of the sympathetic nervous system originates in multiple brainstem areas and nuclei as well as the hypothalamus, which also receives input from the cortex and amygdala. The combined input creates a sympathetic outflow tract which descends the spinal cord to the intermediolateral gray matter. Panel 2 (bottom left): At multiple levels from T1 through the rostral lumbar spine the preganglionic neurons will exit through the ventral rami and then jump to the sympathetic chain where they may ascend, descend, or synapse at that level before exiting again as part of the spinal nerves. Panel 3 (right): Zoomed out view of the sympathetic chain with multiple Para-vertebral ganglia in which the preganglionic neurons may synapse. The exiting post ganglionic noradrenergic neurons provide direct sympathetic input to the heart, lungs, glands, vascular beds, and adrenal medulla. Note that some sympathetic neurons may exit and not synapse in the sympathetic chain but travel to pre-ganglionic neurons to synapse (such as the celiac and mesenteric ganglia) [10, 11]. Created with Biorender.com.*

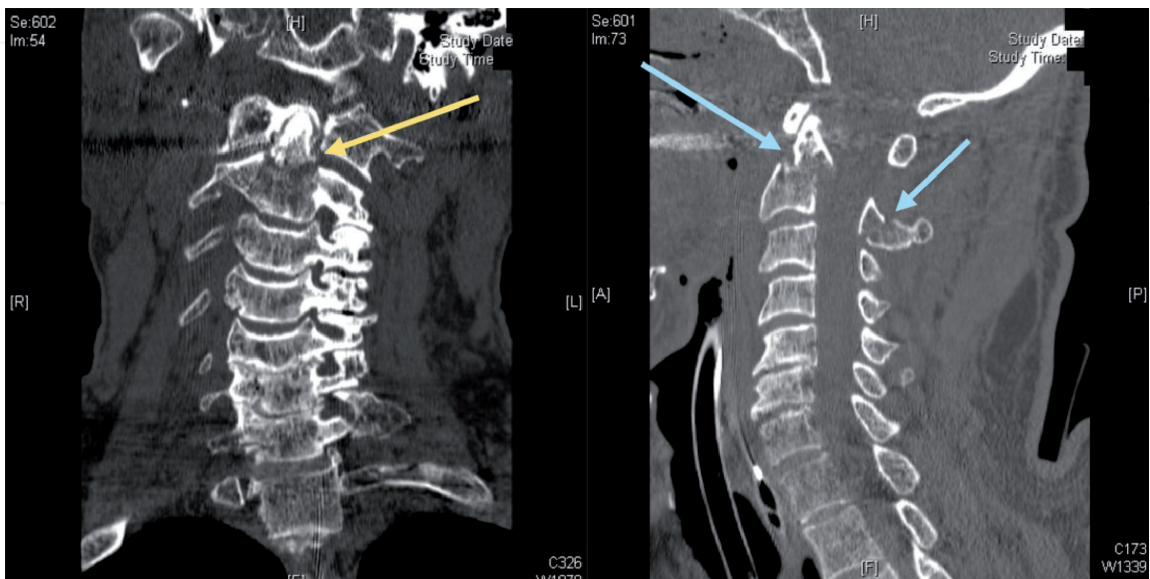
gray matter and ruling out other causes of shock is more challenging). Studies of incidence after SCI have been widely variable, ranging from 14 to 44% depending on the criteria used [4]. Extrapolating from a range of papers, criteria have ranged from systolic blood pressures (SBP) <70 mmHg up to <100 mmHg, and heart rates (HR) <50 beats per minute (BPM) up to  $\leq 90$  BPM in various combinations [12]. Examples include SBP <90 mmHG or HR <50 BPM as a cutoff, more recently others have used SBP <100 mmHG and HR <80 BPM, and still others simultaneous SBP <90 mmHg and HR <80 BPM with some variability in terms of whether or not lab values accounting for hypovolemia were examined as a confounder [4]. In one cohort of patients with isolated spinal cord injury (the majority of which were related to blunt trauma), defining neurogenic shock as SBP <100 mmHg, HR <80 BPM or both, out of 490 cases the incidence of hypotension was 25.8% and of classic neurogenic shock (hypotension and bradycardia) was only 19.3% [13]. What is suggested but not thoroughly quantified in the literature is that the incidence is highest in cervical spine injury and somewhat less for upper thoracic injury (above T6) while SCI lower than T6 would be considered rarely associated with neurogenic shock [3]. There is also not a single consensus in regard to penetrating vs. blunt trauma as to which is more likely to lead to neurogenic shock. Considering that SCI accounts for only about 5% of major trauma cases, a lack of experience may play a role in limiting identification and definitive management even at centers of reasonably high volume [13].

## 5. Initial evaluation and stabilization

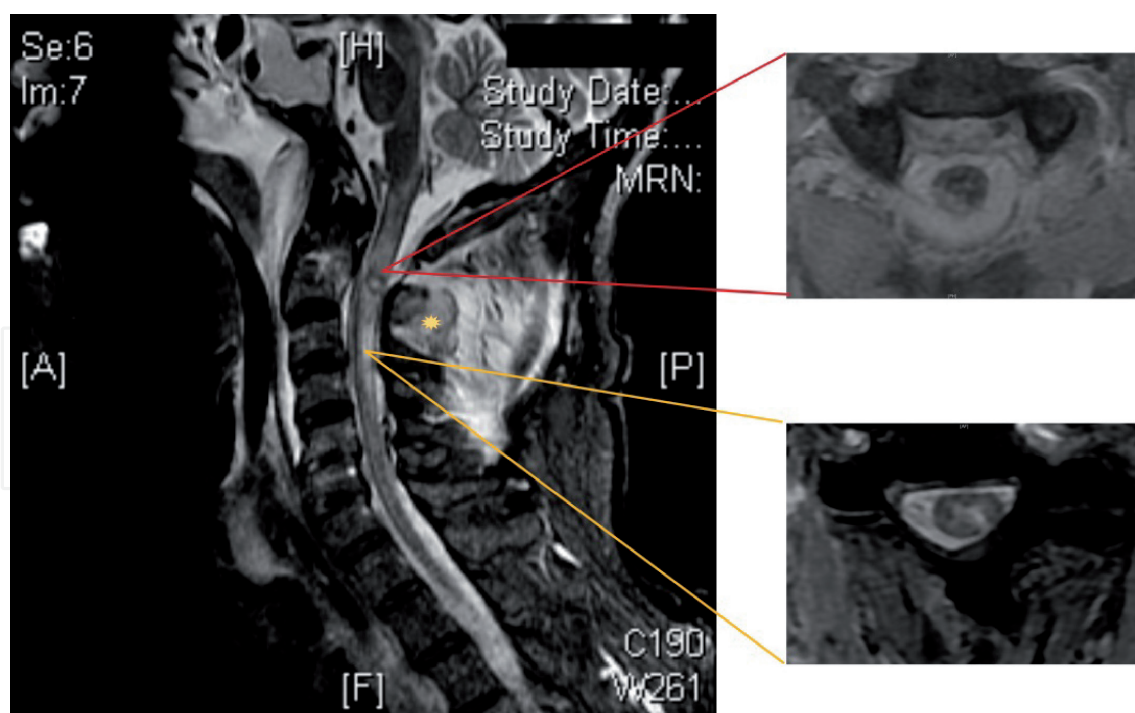
### 5.1 Identification of patients

Case Presentation: 70 y/o male presents by air to a level I trauma center following a bike accident wherein he was thrown down a hill. It is unclear if he was wearing a helmet. He was initially resuscitated by bystanders as he was in cardiac arrest, and then intubated in the field prior to arrival (GCS 3-T). At the time of arrival he is found to have bradycardia with HR 53 BPM and initial BP was 112/74 with mildly low body temp 96.3 F (35.7 C). He became more bradycardia and did not respond to atropine requiring another brief round of cardiopulmonary resuscitation. Although the initial hemoglobin on his arterial blood gas (ABG) was 13.3 g/dl, he was also transfused four units of packed red blood cells (PRBC's). Computed Tomography (CT) of the head and cervical spine showed an occipital condyle fracture as well as a type III (low) dens fracture with 6 mm distraction and a c2 spinous process fracture (**Figure 2**). Pressor support with norepinephrine was initiated and preparations were made for trans-venous pacing in the event of refractory bradycardia. Magnetic resonance imaging (MRI) the following day confirmed a likely distraction injury with cord edema and hemorrhage (**Figure 3**). Interestingly, his hemoglobin by hospital day 1 had increased to 17.5 g/dl suggesting that his perceived response to transfusion may have been related to volume resuscitation and pressers rather than the PRBC's. He continued to have issues with bradycardia but did not require trans-venous pacing. Considering his severe high cervical spine injury with resultant tetraparesis and complications he was transitioned to comfort directed care on hospital day 3.

Case Discussion: The case above illustrates the complexities in early identification of neurogenic shock as a distinct entity. Because the signs of neurogenic shock are somewhat variable in terms of timeframe from injury to onset, and in light of differences between individual patients and systems in regard to fluid resuscitation in the field, a high index of suspicion is necessary from the time of initial evaluation through the early hours and days of intensive care. Any patient presenting with a spinal cord injury should be considered to be at risk with those having higher level



**Figure 2.** Coronal (left pane) and sagittal (right pane) CT scan views of the cervical spine showing a type III odontoid fracture (yellow arrow), C1/C2 fracture distraction (long blue arrow) and C2 spinous process fracture (short blue arrow). Created with Biorender.com.



**Figure 3.**

*Sagittal STIR c-spine MRI (left pane) with noted intra-axial edema and hemorrhage at the base of the odontoid (red triangle and top axial slice), more inferior cord edema (yellow triangle and bottom axial slice), and significant posterior compartment paravertebral edema (yellow star). Created with Biorender.com.*

injuries at higher risk (**Figure 1**). The authors suggest that the American Spinal Injury Association Autonomic Standards Assessment Form [14] is a reasonable place to start and takes into account blood pressure, heart rate, sweating, temperature regulation, the bronchopulmonary system, and the lower urinary tract and bowel. Even with this tool, however, no specific definition of bradycardia/hypotension is forthcoming; thus it will need to be set by individuals and institutions. As there is no single accepted treatment cutoff for the bradycardia and hypotension, it may be important for systems to consider their patient population in relation to prior studies and establish parameters for automatic physician notification during hemodynamic monitoring with reasonable case reviews to establish the best local standard.

It is agreed, however, that the profound systemic hypotension that characterizes neurogenic shock may lead to hypoperfusion of the spinal cord with subsequent ischemia and secondary injury [15]. To improve outcomes, prompt and aggressive treatment of hypotension should be undertaken in a monitored intensive care unit, with adequate cardiopulmonary and ventilatory support [8]. Medical treatment consists of sufficient fluid administration as well as vasopressor therapy for sustaining blood pressure and maintaining perfusion [4]. That being said, it should be noted that the data regarding pressor use in SCI may be conflicting in this regard, as a distinction needs to be made between pressor use in an attempt to stabilize or improve the motor and sensory loss related to SCI, and that to preclude hypotension and bradycardia related to neurogenic shock from causing complications such as systemic hypoperfusion and cardiac arrest among others. According to one author, up to 100% of patients suffering from neurogenic shock may also have bradycardia, with 71% reported as having severe bradycardia (HR < 46 BPM) and 16% progressing to cardiac arrest [16].

## 5.2 Patient management

Fluid resuscitation is the first line therapy for hypotension in the setting of neurogenic shock [17]. Maintenance of blood volume influences both blood pressure



and blood flow around the site of injury [8]. If there is an inadequate response to fluid resuscitation, agents with  $\alpha_1$  and  $\beta_1$  adrenergic receptor activity should ideally be used to increase sympathetic activation [15]. What is otherwise considered routine care such as suctioning, as well as abdominal changes such as elevated bladder and bowel pressures, are known to produce wide swings in heart rate and blood pressure that may be refractory to treatment [18]. These changes should be anticipated and prevented as much as possible.

Blood pressure can be further augmented through the administration of intravenous vasopressor agents. These include norepinephrine, epinephrine, dopamine, phenylephrine, as well as concurrent atropine in patients with significant bradycardia [15]. There are some prior reports of transitioning individuals that need extended treatment with a non-intravenous agent to propantheline, aminophylline, theophylline, and ephedrine although the evidence is extremely limited [16]. Enteral pseudoephedrine has also been used successfully as an adjunctive therapy [16].

Current management guidelines dictate that mean arterial pressure (MAP) should be maintained above 85–90 mmHg for the first 5–7 days of therapy [19]. This resuscitation target has been questioned due to the lack of quality evidence showing a positive effect on outcomes [8]. Additionally, maintenance for 5–7 days may be insufficient because certain individuals benefit from longer management [4]. One study has shown that vasopressor therapy achieving the MAP goal is more likely to cause complications than to improve neurological outcomes, with dopamine leading in complications [20]. As such, the risk of vasopressors should be balanced against their benefits in each individual patient, and there should be clear goals for use in regard to improvement of the sensory and motor deficits vs. cardiovascular stabilization.

A recent study suggests that maintenance of a spinal cord perfusion pressure (mean arterial pressure – cerebral spinal fluid pressure) above 50 mmHg is a stronger predictor of neurologic recovery than systemic MAP and may also be useful in guiding management [21]. More studies with high quality evidence are needed to establish reasonable treatment goals that are linked to improved patient outcome.

## **6. Rehabilitation and recovery**

Rehabilitation in patients with spinal cord injuries should be comprehensive, interdisciplinary, and patient-centered, with goals that are individualized and realistic. Interventions should not be delayed and complications need to be anticipated and promptly identified.

Neurogenic shock can persist for 1–6 weeks after the initial injury, certainly long enough to interfere with rehab in some cases [8, 22]. In addition to that, patients with spinal cord injuries are vulnerable to a number of cardiovascular complications which should be anticipated in the course of rehabilitation, and some with prolonged or severe bradycardia may require permanent pacemakers [16]. Cardiovascular complications are the leading causes of morbidity and mortality in patients in both the acute and chronic stages of spinal cord injury [17]. Common complications include autonomic dysreflexia, orthostatic hypotension, reduced cardiovascular reflexes and absence of cardiac pain during ischemia [18].

Independent of neurogenic shock, autonomic dysreflexia (AD) is a potentially fatal complication that occurs in 48–90% of patients with injuries above T6 [17]. It is caused by the loss of supraspinal sympathetic modulation and is characterized by sudden episodes of hypertension, headache, and tachycardia with prevailing reflex bradycardia [23, 24]. Additional sympathetic features include piloerection and cool extremities



due to vasoconstriction below the level of injury [23]. In contrast to the sympathetic response below the level of injury, a parasympathetic response may predominate above the level of injury. A compensatory baroreceptor response leads to reflex bradycardia. Other features include flushing, sweating, and nasal congestion [24].

Stimuli that may induce an AD response include bladder distension, detrusor sphincter dyssynergia, kidney or bladder stones, or other painful stimuli such as ingrown toenails, pressure ulcers, infections, fecal impaction, musculoskeletal pain, and menstrual cramps [24]. Sequelae of untreated hypertension in the setting of autonomic dysreflexia include stroke, intracranial hemorrhage, seizures, cardiac arrest, hypertensive encephalopathy, and death [25]. An increase of 20–40 mmHg in systolic blood pressure in people with spinal cord injury should raise suspicion for AD, though the exact definition is not consistent across studies [25].

Primary treatment of AD includes sitting patients upright and lowering their legs, as well as removing or loosening tight clothing or accessories [26]. After that it becomes necessary to identify triggering noxious stimuli and address them. A distended bladder should be emptied with a catheter, a rectal exam may identify impaction, skin should be examined for pressure ulcers and more serious causes need to be suspected because they may not be obvious [26]. Medications that can be administered to help stabilize AD include the calcium channel blocker nifedipine, nitrates, and vasodilatory agents such as hydralazine [18, 23], although hypotension needs to be anticipated and patients will require appropriate hemodynamic monitoring.

Another cardiovascular consequence of spinal cord injury related both to neurogenic shock and autonomic dysreflexia is orthostatic hypotension (OH). It is defined by the American Autonomic Society as a reduction in systolic or diastolic blood pressure of  $\geq 20/10$  mmHg, within 3 min of standing upright [27]. Symptoms occur as a result of reduced cerebral perfusion pressure and include light-headedness, dizziness, syncope, pallor, nausea, fatigue, and sweating. Nevertheless, many patients do not report symptoms despite meeting the definition of OH, and some report symptoms in spite of not fully meeting that definition. Pharmacologic therapy may be used to treat OH but should be done carefully because of the already labile blood pressure in patients with spinal cord injury. The most common treatments are compression stockings, abdominal binders, midodrine, or fludrocortisone [23, 27].

Additional autonomic complications that occur after injury are the reduction of cardiovascular reflexes and the absence of cardiac pain. Cardiovascular reflexes regulate blood pressure, intravascular volume, and temperature [18]. The sensation of pain related to cardiac ischemia may be altered because cardiac pain fibers that travel with sympathetic afferent fibers (visceral sensory fibers) are disrupted in cervical or thoracic injuries above T4 [28]. As a consequence, spinal cord injury patients may have atypical presentations of cardiac ischemia including referred pain.

Other major components that are critical in spinal cord injury rehabilitation are bowel and bladder training, respiratory care, mobilization, as well as physical and occupational therapy. Not only should rehabilitation address the medical aspects of patient care, but the psychological impacts of spinal cord injury as well. A comprehensive approach treating the whole individual gives patients a better chance at achieving optimal functional recovery.

## **7. Conclusion**

Neurogenic shock is a feared and difficult to treat complication of disruption of the sympathetic nervous system which most often occurs in the setting of a spinal cord injury. The refractory hypotension and bradycardia may be extremely

dangerous for the patient, and may lead to cerebral anoxia, cardiac arrest, and more. Although there is no single accepted blood pressure and heart rate cutoff to define neurogenic shock, the signs are reasonably well established and definitive treatment is in line with current critical care management standards. Beyond the short term, neurogenic shock as well as autonomic dysreflexia, which may commonly accompany spinal injuries at the same level, can complicate the rehabilitation process. Hopefully future prospective studies will adopt standard ways of isolating and confirming neurogenic shock and establish treatment paradigms that improve patient outcomes.

### **Conflict of interest**

The authors report no conflict of interest.

### **Author details**

Anna Volski<sup>1</sup> and Daniel J. Ackerman<sup>2\*</sup>

1 Lewis Katz School of Medicine at Temple University, St. Luke's University Health Network, Bethlehem, PA, USA

2 Department of Neurology, St. Luke's University Health Network, Bethlehem, PA, USA

\*Address all correspondence to: [daniel.ackerman@sluhn.org](mailto:daniel.ackerman@sluhn.org)

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