Chapter

Spinal Shock: Differentiation from Neurogenic Shock and Key Management Approaches

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Abstract

The conceptual differentiation of spinal and neurogenic shock tends to be misunderstood among clinicians. In order to better illustrate the differences in definition, presentation, and development of spinal shock (SS) from neurogenic and other forms of shock, we present herein a clinically relevant summary of typical characteristics of SS. First described in the eighteenth century, the continued investigation into the disease process and the response of neural structures to spinal cord trauma have led to a more complete description and understanding. We will begin in the first part of the chapter describing the etiology of SS, including a working definition, as it pertains to complete spinal cord injuries (SCIs). This is followed by the summary of pathophysiology and clinical presentations associated with each clinical phase of SS. Finally, we explore treatment options and considerations as they relate to incomplete SCI. We hope that by presenting a clear and well-delineated overview of SS, we will allow the clinician to better understand and more accurately predict the evolution of this process. This, in turn, should facilitate the ability to deliver better care for the patient.

Keywords: areflexia, clinical management, hyperreflexia, spinal injury, spinal shock, shock, trauma

1. Introduction and epidemiology

The specific definition of spinal shock (SS) has evolved over the past two centuries. Nonetheless, a significant level of ambiguity, controversy, and confusion still exists when differentiating between neurogenic shock (NS) and SS. Whytt first described this clinical entity in the 1750s without using the term "shock" and without the understanding of the underlying basic science and anatomy to accurately inform the definition. Rather, he focused on the observation that SS was associated with a loss of sensation accompanied by motor paralysis with initial loss but gradual recovery of reflexes [1]. The definition was then expanded over by Hall in the early 1840s, officially utilizing the terms "spinal shock" and "reflex arc" [2]. Another contributing factor to the previously elusive definition is the lack of uniform clinical presentation, manifestation, and duration of SS. Due to the substantial clinical variability and heterogeneity of presentations, we must first discuss the definitional aspect of SS so that the reader may have a clear idea and a

Phase	Timing	Neurological changes
1	0–1 day	1. Decreased spinal and supraspinal excitation 2. Loss of 5-HT production leading to loss of plateau potentials 3. Reduction of available synapses and dendrites
2	1–3 days	1. Increased postsynaptic sensitivity 2. Receptor upregulation due to decreased neurotransmitter activity
3	1–4 weeks	1. Increased neurotrophin activity allows for increased synaptic growth 2. Increased interneuron growth 3. Plateau potentials recovered in spinal neurons
4	1–12 months	1. Synapse growth in long axons

Table 1.
Four phases of spinal shock by Ditunno et al.

working model for our subsequent discussions of diagnosis, patient presentation, and treatment approaches.

Ditunno made a subtle point that the controversy surrounding the definition of SS could be attributed to observations made clinically. More specifically, he noted that not all reflexes are eradicated in a strictly binary on/off fashion. Some reflexes may only be depressed and yet still can be technically elicited. Finally, he noted that the resolution of SS does not occur in a binary fashion and often follows a prolonged course of weeks to months [3]. Similar observations by Illis suggested that the definition of SS cannot be comprehensive without including subcomponent definitions of clinical phases [4]. For the purposes of this chapter, we will utilize Ditunno's four-phase model of spinal shock, building upon the groundwork described by various pioneers such as Whytt and Hall [1, 3]. This model allows for clarification of the ambiguity surrounding the disease process while still retaining flexibility to appreciate the variability among clinical presentations. The details of Ditunno's four-phase model can be seen in **Table 1** [3].

The phases are organized according to post-injury time and the nervous system's response to insult. Of note, we will hold off on the discussion of each phase until the *Etiology and Pathophysiology* section as the separation of phases requires delving into how the neurons are responding to their environment as time progresses.

In 2007, there was an estimated global spinal cord injury (SCI) incidence of 2.3 cases/100,000 inhabitants [5]. It has been estimated 45% of SS cases are associated with motor vehicle collisions (MVC), 34% with domestic accidents, 15% with sporting accidents, and 6% with self-harm [6]. The incidence of SCI can vary across geographic, socioeconomic, and cultural factors, including the prevalence of contact sports and differences in primary transportation modality. All of the above factors are important determinants of the incidence of SCI. Finally, no discussion of the topic of SCI is complete without mentioning the tremendous human and economic cost associated with these injuries worldwide [7–9].

2. Spinal shock: etiology and pathophysiology

Spinal cord injuries (SCI) are typically divided into two subtypes, complete and incomplete. An SCI is considered incomplete if there is some degree of residual motor and/or sensory function below the neurologic level of injury that includes the lowest sacral segments, where the neurologic level of injury is defined as the most caudal level at which both motor and sensory modalities remain preserved [10]. It follows that patients affected by a complete SCI will not retain sensory or motor function in the lowest sacral segments.

Before directing our discussion to the management in incomplete SCI, additional information will be provided regarding complete SCIs, specifically in the context of SS. The understanding of key processes surrounding complete SCI is conceptually easier, especially when compared to the understanding of incomplete SCI. It is important to note that although severed neurons are separated from descending input—both excitatory and inhibitory—there remains synaptic contact with associated interneurons and reflex afferents, and even new synaptic connections can be established with sprouting neurons [11, 12]. Our subsequent discussion will describe the previously outlined "four phases" of spinal shock.

2.1 Phase I

As outlined in **Table 1**, **Phase I** of SS is marked by areflexia/hyporeflexia, a consequence of the loss of descending mediation. This phase occurs from 0 to 24 hours from time of injury. Under normal circumstances, both spinal motor neurons and interneurons receive certain baseline levels of background excitatory input from supraspinal axons. When an individual wishes to initiate voluntary movement, additional stimulus is superimposed above this "background activity." Supraspinal inputs mediating the background excitation of spinal motor neurons and interneurons are numerous and include vestibulospinal and reticulospinal pathways [13]. These two pathways will now be discussed in more detail.

The vestibulospinal pathway arises from first-order neurons located in Scarpa's ganglion which is situated in the distal part of the internal auditory meatus [14]. Afferents are sent from the ganglion through the vestibular part of the eighth cranial nerve before entering the brainstem at the pontomedullary junction. Upon entry, there are four second-order vestibular nuclei; however, we shall focus on the medial and lateral vestibulospinal tracts for the purposes of our current discussion. The medial and lateral vestibulospinal tracts arise from the medial and lateral vestibular nuclei, respectively [15, 16]. The latter descends the entire length of the spinal cord ipsilaterally and plays a crucial role in walking upright, while the former descends bilaterally in the medial longitudinal fasciculus and terminates at the mid-thoracic level, facilitating the integration of head and eye movements [17, 18].

The reticulospinal pathway arises from the brainstem, the pontine reticular formation, and the medullary reticular formation [19, 20]. Pontine reticular fibers traveling in the pontine reticulospinal tract remain uncrossed as they descend in the medial longitudinal fasciculus, terminating in axial and limb muscles involved in posture and gait stability [21]. At the level of the muscle, their effects are at least threefold: (a) facilitation of movement, (b) regulation of reflexes, and (c) contribution to muscle tone. The medullary reticular fibers traveling in the medullary reticulospinal tract serve a slightly different role [22–24]. First, the fibers originating from the medullary reticulospinal formation are located bilaterally in the reticulospinal tracts as they descend; however, most of the fibers remain uncrossed. As they terminate on axial and limb muscles, they serve an inhibitory role during the modulation of voluntary movement and reflexes.

In addition to supraspinal inputs, serotonergic (5-HT) neurons and noradrenergic (NE) neurons originating from the raphe nucleus and locus coeruleus, respectively, may also play a role in the background excitatory input as they influence spinal cord motor systems [25]. Mechanistically, this may involve the production of plateau potentials [26, 27]. The plateau potentials originate on dendrites, believed to be mediated through sustained activation of $Ca2^{+}$ channels, and provide amplification of excitatory inputs, with approximately sixfold "gain," thus allowing for prolonged neuronal firing with minimal excitatory input, as well as contributing to the background basal excitatory stimulation [28–30].

Baseline excitability in muscle spindles may also be handled in part by gamma-motor neurons [31, 32]. Upon SCI, gamma-motor neurons caudal to the injury may lose their ability to influence motor neurons via stretch reflex afferents as they lose their tonic descending facilitation. The loss of descending inhibition of inhibitory pathways within the spinal cord must also be considered, primarily because it likely contributes to decreased spinal reflexes [33, 34].

Finally, some of the more delayed developments involving the injured cord, both metabolic and structural, could contribute to the observed areflexia/hyporeflexia characteristics of SS. At the same time, the observed areflexia/hyporeflexia usually occurs immediately post-SCI, making any other pathophysiologic considerations secondary—rather than primary—factors [35, 36]. This "secondary factor" list includes (a) dendritic retraction and synaptic degeneration seen within 1–3 days post-SCI; (b) impaired delivery of metabolites and secretion of neurotrophins; and (c) the impact of growth factors caudal to the neurologic level of injury [36–38].

Upon traumatic injury resulting in complete SCI, the baseline excitation from supraspinal inputs will be lost, leading to hyperpolarization of the neurons [39]. This hyperpolarization leads to the neurons becoming less excitable and yields the clinical picture in **Phase I**.

2.2 Phase II

Appearing 1–3 days following the SCI, the return of cutaneous reflexes is observed [3]. It is still unknown whether this is due to replacement of synapses or to denervation supersensitivity. Morphological changes in the synapses have been documented within hours to days of SCI; however, these synapses may not become functional until weeks—or even months—have passed, making this an unlikely contributor to **Phase II** developments [40–45].

Denervation supersensitivity is defined as increased neuronal firing in response to a neurotransmitter [46]. This phenomenon has been shown to occur in both the peripheral (PNS) and central (CNS) nervous systems, including the brain and the spinal cord [47–51]. The proposed mechanisms involves upregulation of mRNA transcription and protein translation that begins within hours and peaks within days post-SCI, which is within the time scale of empirically observed changes [52]. More specifically, the overall process leads to increased synthesis and insertion of receptors into the postsynaptic membrane, altered synthesis and assembly of receptor subunits, decreased removal and/or degradation of receptor(s), and reduced excitatory neurotransmitter reuptake [52–55]. Mechanistically, NMDA glutamate receptors, serotonin 2A, and vanilloid VR1 receptors have been shown to increase either in association with mRNA synthesis or the observed density at the synapse [54, 56–58]. Hypoactivity of neurons has been shown to constitute a sufficient stimulus to increase production of the NMDA glutamate receptors [55]. Although the exact details are yet to be elucidated, neurotrophins, growth factors, and their respective receptors have been shown to stimulate an increase in transcription and translation [59–64]. Postulated downstream effects involve the modulation of NMDA receptors, resulting in increased excitability and/or decreasing GABA synaptic inhibition [65]. These effects seem to play a role in the development of SS during the initial period of 1–3 days post-SCI [3].

2.3 Phases III and IV

Stages III (1–4 weeks) and **IV (1–12 months)** of SS are often linked together and are best described through the lens of the human tibial H-reflex. The H-reflex has been used to model the recovery of reflexes caudal to SCI over time [66, 67].

In this context, an interesting phenomenon is observed beyond post-injury "day 3" temporal marker. More specifically, there is an increased reflex excitability observed at 2–4 weeks post-SCI with an increase in latency and another increase in reflex excitability at approximately 3–4 months post-SCI [3].

Overall, it has been shown that the 2–4-week mark increase in excitability can be attributed to axon-supplied synapse growth and/or disynaptic interneurons, while the increase in excitability at 3–4 months is mediated by primary afferents and/or soma-supplied synapse growth [3]. The timing of the observed changes in excitability suggests that there is an axon-length-dependent rate of synapse growth. Two mechanisms have been proposed to explain this phenomenon: (a) two periods of synaptic growth—early findings dependent on axonal synthesis and the later growth period dependent on somal synthesis and (b) disynaptic stretch reflex pathways, such as the Golgi tendon organ reflex, are preferentially hyperexcitable relative to the monosynaptic Ia afferents to motoneurons [3].

3. Diagnosis and clinical presentation

3.1 Phase I

Caudal to complete SCI within the first 24 hours, Phase I will present with flaccid, paralyzed muscles and deep tendon reflexes (DTRs) being initially absent. While the DTRs such as the ankle jerk (AJ) and knee jerk (KJ) are absent, a pathologic reflex, delayed plantar response (DPR), is often the first to return and should be observed within hours post-SCI [68]. Other cutaneous and polysynaptic reflexes such as the bulbocavernosus (BC), cremasteric (CM), and anal wink (AW) can also be seen to return during Phase I. Location of the lesion can be determined based on presenting symptoms. Lesions above the mid-pons will cause decerebrate rigidity, while those located below the mid-pons cause hyporeflexia [69]. In addition to skeletal motor and reflex findings during this time, there are autonomic findings that may be relevant if the lesion is in the cervical area. Findings include hypotension, atrioventricular conduction block, and bradyarrhythmia, and these can be continued through Phases II and III [3]. These findings are consistent with neurogenic shock, detailed in a separate chapter.

3.2 Phase II

One to 3 days post-SCI, the clinician should expect to see continued reflex return. Building upon Phase I, the cutaneous reflexes, BC, AW, and CM, become stronger [3]. Except for two patient populations, namely, the elderly and children, DTRs are still absent; however, the tibial H-reflex returns around the 24-hour marker [70, 71]. In the elderly, DTRs and the Babinski sign can occur during this phase [68]. Although not known for certain, the presence of pre-existing subclinical myelopathy might contribute to this early recovery as some animal studies have exhibited quicker recovery of DTRs in the setting of prior upper motor neuron lesions [68, 72, 73]. Children exhibit similar recovery, showing DTRs sometimes 3 days post-SCI, which might be attributable to their still developing descending supraspinal tracts, predisposing them to spinal hyperreflexia. The recovery of cutaneous reflexes during phase II is likely due to receptor plasticity [3].

3.3 Phase III

The third phase (days 4–30) is marked by early hyperreflexia. Excluding the two patient populations discussed in Phase II, almost all patients will regain DTRs

during this period [3, 68]. The return of these reflexes is as follows: Babinski sign recovery will follow AJ recovery closely, with the AJ preceding the return of the KJ [3, 68, 74]. The clinician should expect to see most DTRs resolve during this phase with only 10% persisting beyond **Phase III** [3]. Ditunno discussed the variability of reflex return regarding the timing trend. There have been studies showing reduced tendon reflex excitability in certain trained populations, such as ballet dancers and power-trained athletes, relative to untrained or even endurance athletes [75–77]. There has also been evidence that pre-SCI experiences could influence the reflex excitability below an SCI [78, 79]. During this time the clinician will have to be aware of the developing autonomic functions. There is expected improvement in the bradyarrhythmia and hypotension described before; however, around this time autonomic dysreflexia can arise and is most commonly due to a distended bladder or bowel causing a massive sympathetic outflow below the neurologic level of injury [3]. Autonomic dysreflexia can lead to difficult-to-control hypertension and bradycardia and is most commonly seen in patients with SCI at or above T6 but has been seen as low as T10 [80].

3.4 Phase IV

One to 12 months post-injury, spasticity and hyperreflexia usually set in, characteristic of **Phase IV**. The remaining DTRs not extinguished during **Phase III** of SS should become absent during this period [3]. Minimal stimuli will elicit cutaneous reflexes, Babinski sign, and DTRs. It has been estimated that there will be detrusor paralysis recovery by 4–6 weeks [3]. The autonomic dysreflexia described in Phase III can also develop during Phase IV, including malignant hypertension, and following its emergence can become chronic/protracted.

4. Treatment

A detailed history is imperative for accurate diagnosis and treatment of spinal shock. As mentioned previously, prior patient life experiences (i.e., athletes, ballerinas, etc.) may play a role in the rate of hyperreflexia appearance [3]. Thus, a thorough history will help guide appropriate expectations of the clinical evolution of reflexes. The history will also help direct the clinician to what developments could be expected as these can depend on the type, severity, and timing of the incident. Certain substances and chemical mediators for reducing inflammatory processes, protecting neurons, and regenerating neural capacities have been investigated for efficacy in the management of SCI [6, 81–88]. Corticosteroids, specifically methylprednisolone, have been postulated to be part of a generalized recommendation to help alleviate inflammatory processes mediated by neutrophils and macrophages; however, clinical trials and non-randomized studies point to not having this as a general recommendation [81–85]. It has been recommended that a young patient, free of any underlying disease which could be influenced by corticosteroids, could be started on a short trial of methylprednisolone with a loading dose of 30 mg/kg with a maintenance dose of 5/mg/kg/h for the next 24 hours [6]. Symptomatic medications for autonomic dysfunction can include treatments for headaches, flushing, elevated blood pressure, orthostasis, and bladder and abdominal distension. Prompt attention to bowel and bladder hygiene, bladder catheterization, cautious use of bowel preparations, and anticholinergic medications may help with any associated hemodynamic instability. There are ongoing investigations into G-CSF and FGF-2, among others, as possessing neuroprotective qualities as well as stem cells of varying stages, olfactory ensheathing cells, and mesenchymal

stromal cells that are possible candidates for regenerating neural capacities [86–88]. Consequently, the clinician should remain up to date on the current literature for therapeutic developments. Providers should also keep in mind that lesions above T6 can be accompanied by neurogenic shock, and we refer you to the neurogenic shock chapter for the diagnosis and management of that phenomenon.

Current guidelines and recommendation can be split up based on the location of SCI.

Cervical SCI:

1. Immediate immobilization through traction and alignment.

2. Identify if injury is above c5.

    a. Above C3: Immediate mechanical ventilation.

    b. C3–C5: Monitor closely for respiratory decompensation and ventilate if necessary.

    c. Maintain supportive care and ensure SBP > 90.

3. Neurosurgery consult to determine if neurosurgery is necessary [89].

Thoracolumbar SCI:

1. Stable fractures: Stabilization with brace from 6 to 12 weeks.

2. Unstable fractures: Surgical decompression [90].

Sacral SCI:

1. Unstable sacral fractures:

    a. Identify any active bleeds.

    b. Immediate reduction.

2. Stable sacral fracture:

    a. Reduction with brace for up to 4 months.

    b. Limit activity.

While it has been a standard practice to give high-dose methylprednisolone after spinal cord injury, recent studies have found that there is no advantage of steroids when considering neurological recovery [91, 92]. Given that SCI can result in long periods of immobility, it is important to consider antithrombotic prophylactic treatment. If patient is on bed rest, gastric and skin ulcer precautions must also be in place.

5. Considerations for incomplete SCI

Incomplete SCI can be classified using the American Spinal Injury Association (ASIA) into three broad categories. Grade A, B, and C injury designations are based

on functions that are preserved. **Table 2** describes the preserved functions in all grades. Incomplete spinal cord injuries can be categorized into four types: central cord syndrome, anterior cord syndrome, posterior cord syndrome, and Brown-Sequard syndrome.

The incidence of incomplete SCI has reported range from 40 to 50% of all spinal injuries [93–95]. Central cord syndrome tends to be the most common injury with posterior cord being the rarest of the incomplete spinal injuries. We will start by exploring the central cord syndrome. Most cases of incomplete SCI are caused by motor vehicle accidents (MVA), falls, and swimming injuries [96, 97].

5.1 Central cord syndrome

Central cord syndrome (CCS) is seen primarily in patients in the fifth decade of life and beyond and is usually a result of hyperextension injury [95, 98]. In younger patients, CCS is usually due to high-velocity trauma. CCS in older patients tends to occur in the setting of pre-existing degenerative narrowing of the spinal canal; this narrowing combined with hyperextension can cause an expanding hematoma that exerts pressure on the spinal cord [99]. Depending on the location and severity, we see a different range of symptoms. Milder injuries can result in burning sensation of the upper extremities. Most presentations consist of weakness in all limbs, with upper extremities more affected than the lower extremities. Majority of central cord injuries are due to a lesion at the levels of C4–C6. Patients with the following history and signs should be evaluated for CCS [100].

1. Patients over 50 years of age: Hyperextension with a previous history of degenerative changes in the spinal canal.

2. Patients under 40 years of age: High velocity trauma (MVA, skiing, etc.).

3. Sensory Loss: Cape-like distribution (upper extremities and thorax with sacrum spared).

4. Motor loss: Weakness that is more prominent in the upper extremities than lower extremities.

5. Autonomic regulation: Loss of bowel and bladder. Orthostatic hypotension may also be seen [101].

Any patient that is being evaluated for incomplete SCI should have a high-resolution computed tomography (CT) to identify spinal fractures, dislocations,

Grade	Description
A	Complete spinal cord injury. No motor or sensory function
B	Motor function is lost, while sensory function is preserved
C	Sensory function is lost, with motor function spared at the sacral level
D	Sensory functions intact, and all motor functions are at least grade 3/5 (able to move against gravity, but not against active resistance)
E	No loss of function noted

Table 2.
American Spinal Injury Association (ASIA) classification for incomplete spinal cord injuries at level of injury.

and potential hematomas [100]. A magnetic resonance imaging (MRI) should be considered when CT is normal, but CCS is still suspected. In roughly 4–6% of individuals with CCS, it is possible that all imaging, with the exception of MRI, can show no abnormalities. Once the severity on the CCS is identified and classified using the American Spinal Injury Association (ASIA) scale (**Table 2**), management pathway can be selected [102]. The Congress of Neurological Surgeons recommends that patients receive immediate surgery in cases of fractures or dislocations [103, 104]. However, decompressive surgery in CCS is controversial as many studies looking at outcomes comparing surgical and nonsurgical management have been inconclusive. The use of steroids is not recommended as it has been shown no benefit when compared to observation [105–109].

It has been noted that 75–80% of patients can regain full neurological recovery [96, 110, 111]. Depending on the ASIA score that was determined during admission, one can begin determining prognostic considerations [112]. Usually younger patients with CCS from traumatic injuries tend to have the best prognosis [113]. The timeline for recovery can be up to 1 year after injury. Patients will usually regain functions in an ascending manner [99].

5.2 Brown-Sequard syndrome

Unlike CCS, the Brown-Sequard syndrome (BSS) is a rare type of incomplete SCI [114]. It is usually seen in penetrating trauma, including knife and gunshot wounds. It can also occur with the loss of vascular supply due to a herniation or edema to a hemisection [115–117]. BSS presents with ipsilateral loss of motor function, ipsilateral loss of sensation, and proprioception and contralateral loss of pain and temperature [114]. These symptoms are due to a lesion involving the corticospinal, dorsal column, and spinothalamic tracts, respectively. In some cases, there is loss of bowel and bladder function. BSS has the best prognosis of all the incomplete spinal cord injuries. Roughly 90–99% of patients gain back full function [99]. Diagnosis should be suspected based on a combination of physical examination/presenting signs and confirmed with an MRI. Management is similar to CCS, consisting of conservative approach with a strong focus on early rehabilitation. Surgery is indicated in the following scenarios [118–121]:

1. Lesion requiring decompression.

2. Presence of a tumor.

3. An abscess compressing the spinal cord.

Complete recovery following BSS can take up to 2 years. However, most patients regain full motor skills within the first 6 months. Pain and temperature sensations tend to recover before full motor function is regained [122, 123]. It is vital that patients receive immediate physical therapy following the acute treatment phase to maximize recovery. During the treatment and management phase of BSS, providers must be careful in completely addressing the underlying condition that lead to BSS, such as spinal cord herniation or a CSF leak through a dural tear, as these could lead to permanent loss of neurologic function [124, 125].

5.3 Anterior cord syndrome

Anterior cord syndrome (ACS) is a rare incomplete SCI that accounts for approximately 1–3% of spinal injuries [95]. It also has the worst prognosis of all the

incomplete SCI, with only 10–20% of patients achieving some level of functional recovery [126]. ACS has two primary pathogenetic mechanisms. In about 90% of cases, it is caused by decreased vascular perfusion to the anterior spinal artery which supplies the anterior 2/3 of the spinal cord [95, 126]. Another possible cause is from increased direct pressure on the spinal cord caused by compression trauma or "over-flexion" [127]. The first signs of ACS include bilateral loss of motor function, pain, and temperature sensation. These findings are more dominant in the lower extremities. Patients also tend to present with loss of bladder and bowel function [126]. Presentation of ACS is usually acute with severe back pain and loss of neurologic function mentioned. The best confirmatory test is a spinal MRI; however, computed tomography angiography (CTA) may be used for faster diagnosis. Emergent surgical management may be required depending on the underlying pathology responsible for the ACS (e.g., aortic aneurysm). Once the underlying condition is treated, management of ACS is similar to other SCIs and consists of physical and occupational therapy. While the patient may never regain the lost motor and sensory function, it is vital that physical therapy is provided on a regular basis to prevent contractions and spastic paralysis [128].

5.4 Posterior cord syndrome

Posterior cord syndrome (PCS) has an incidence of roughly <1% [95, 99]. Like ACS it carries a very poor prognosis. The causes of PCS include vascular compromise to the posterior spinal artery, trauma, multiple sclerosis (MS), vitamin B12 deficiency, and syphilis. Since PCS affects the posterior aspect of the spinal cord containing dorsal column fibers, one typically sees presentations that involve loss of proprioception and vibratory sensation with motor function being preserved. Patients occasionally will have sensation of "electric shocks" running down their spine, which is known as Lhermitte's sign and can indicate MS or a metabolic deficiency [121, 128]. CTA might allow for rapid diagnosis of vascular comprise/threat and allow for emergent treatment. However, MRI imaging showing infarctions is the most reliable method of confirming the diagnosis [99]. Once the underlying pathology is treated, PCS management will require rigorous physical and occupational rehabilitation course [121].

6. Conclusions

It is important to distinguish the differences between spinal shock and neurogenic shock, both in terms of definitions and clinical manifestations. Spinal shock encompasses a diverse set of injuries involving various parts of the spinal

	Spinal shock	Neurogenic shock
Damage location	Different areas of the spinal cord	Sympathetic pathways—above T6 vertebral level
Systemic hypotension	Possible, depending on the location and severity of injury	Always
Onset time	Sudden to days	Sudden
Time to resolution	Weeks to months	Hours to days

Table 3.
Spinal shock versus neurogenic shock.

cord, whereas neurogenic shock tends to be a result of spinal injuries above the level of T6. Spinal shock occurs in phases (I–IV) that are temporally distributed over a period of weeks to months, whereas neurogenic shock tends to have sudden onset that requires more urgent management. **Table 3** outlines the key differences between spinal and neurogenic shock. Patients with SS and injuries above the level of T6 should always be evaluated for neurogenic shock symptoms, such as

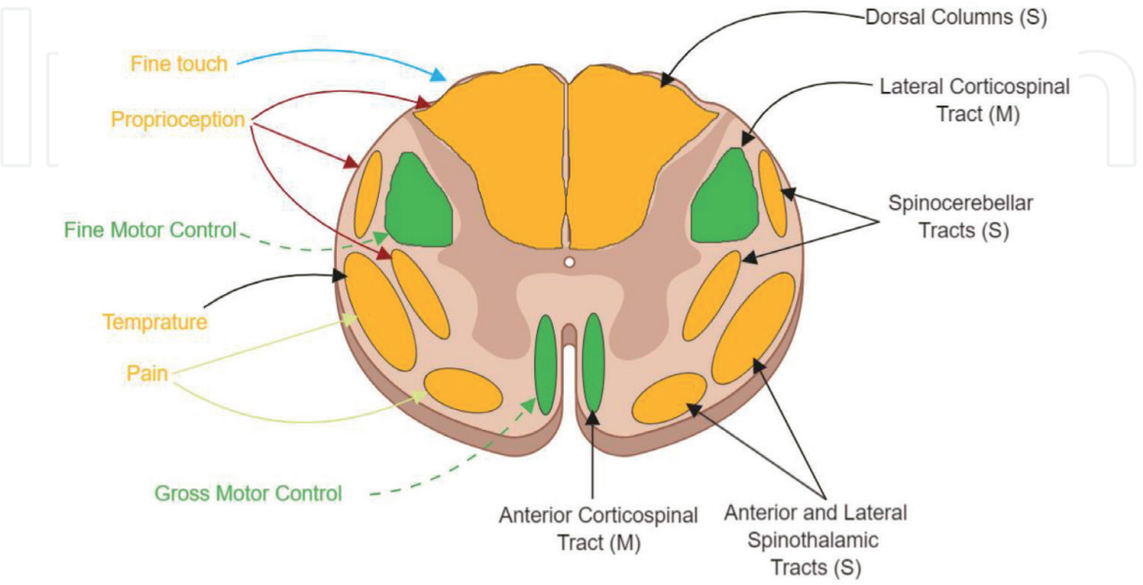

Figure 1.
This represents the different tracts on a T8 spinal cross section. The sensory pathways (S) and motor pathways (M) are identified with specific characteristics depicted on the right. This image was created using Biorender and is used here based on the terms and conditions of Biorender®.

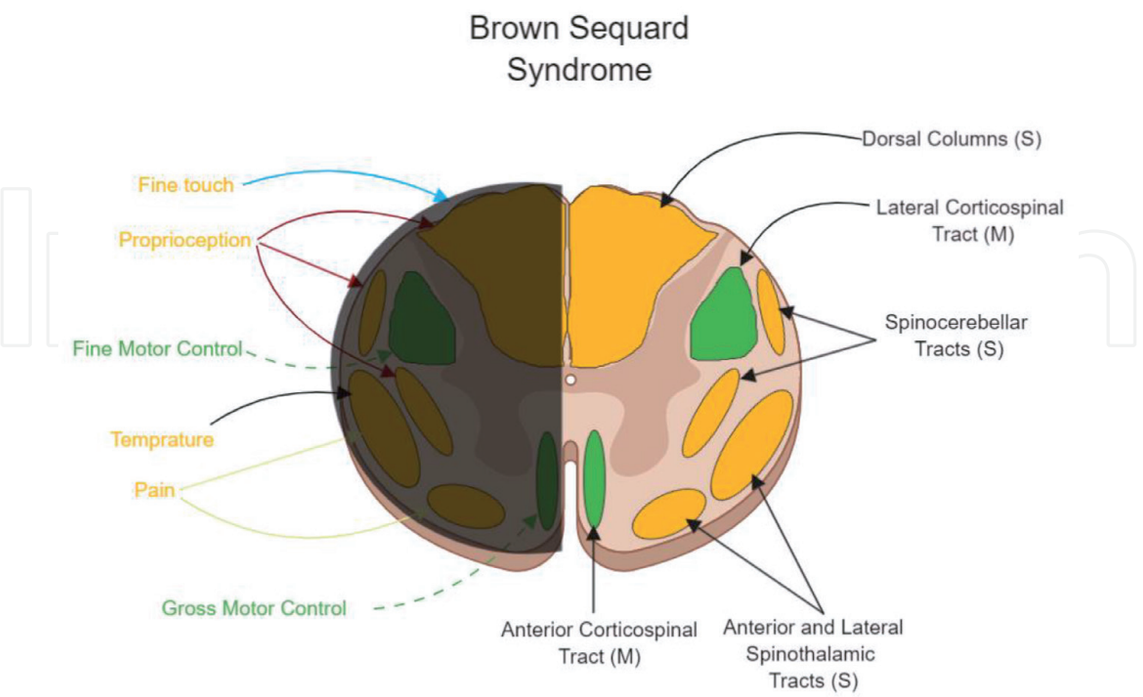

Figure 2.
Image representing lesion that would be considered Brown-Sequard and the pathways involved in the hemisection injury. This image was created using Biorender and is used here based on the terms and conditions of Biorender®.

hypotension, hypothermia, and bradycardia. Both complete and incomplete SS injuries can develop hypotension but will not develop systemic vasodilation (as would be seen in the event of neurogenic shock). Accurately differentiating neurogenic and spinal shock is important because it will help clinicians in determining important management decisions in patients with SCI (**Figures 1–5**).

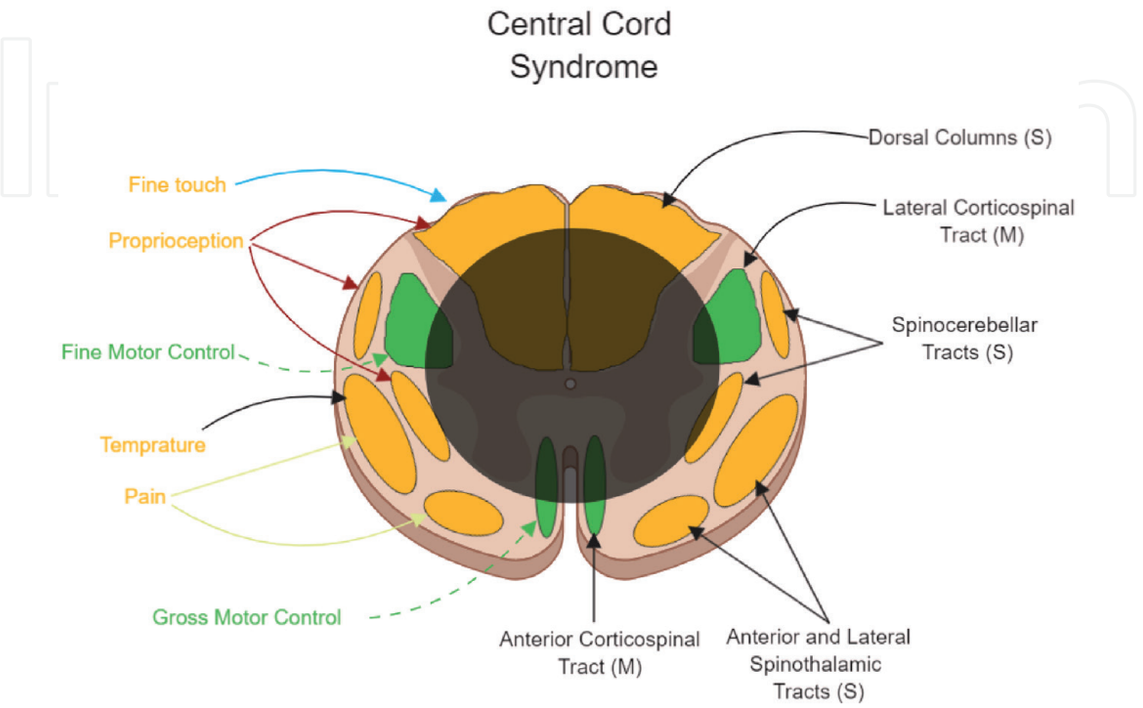

Figure 3.
Image represents central cord injury and the pathways involved. This image was created using Biorender and is used here based on the terms and conditions of Biorender®.

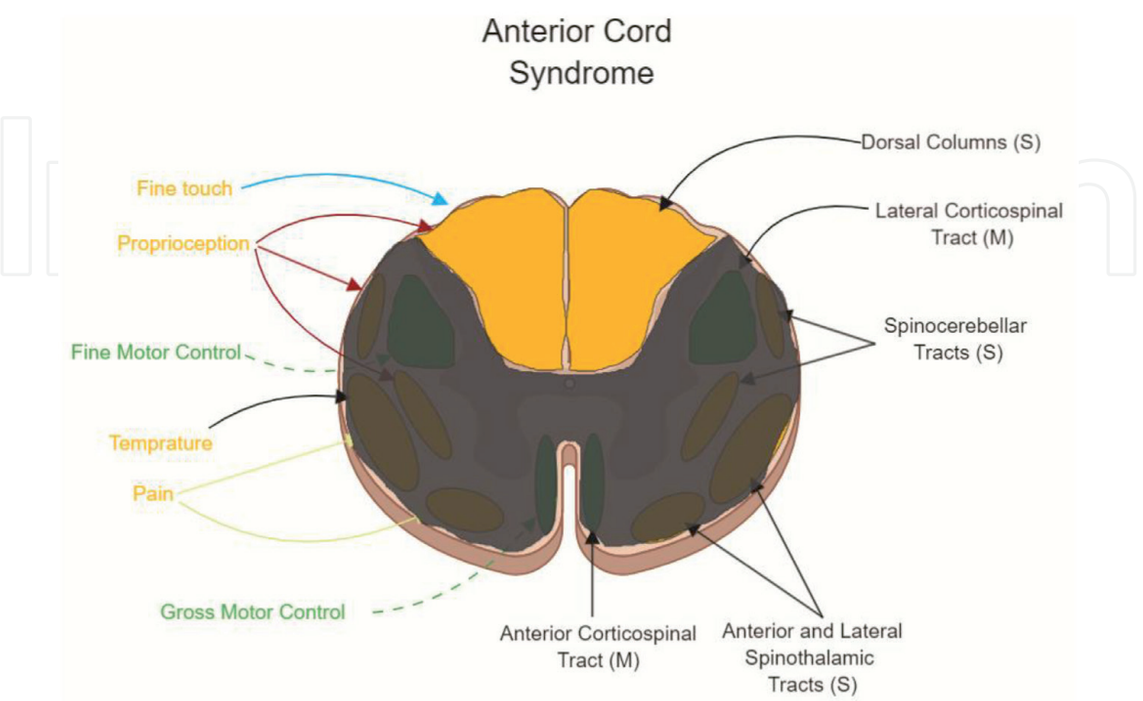

Figure 4.
Image represents anterior cord injury and the pathways involved. This image was created using Biorender and is used here based on the terms and conditions of Biorender®.

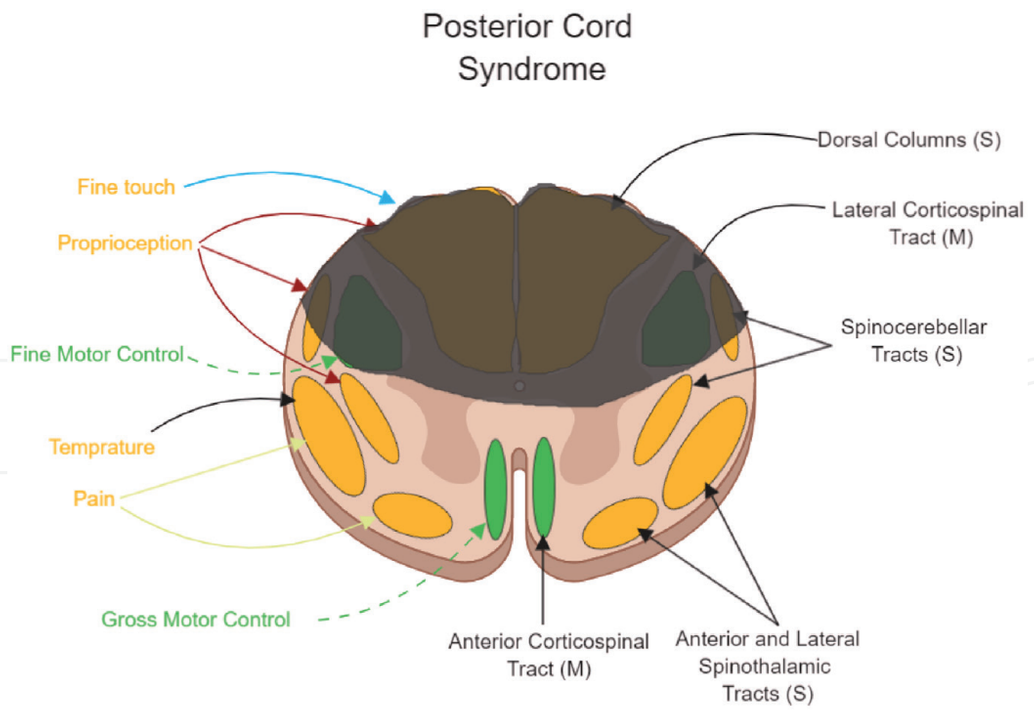

Figure 5.
Image represents posterior cord injury and the pathways involved. This image was created using Biorender and is used here based on the terms and conditions of Biorender®.

Glossary

SS	spinal shock
SCI	spinal cord injury
DTR	deep tendon reflex
DPR	deep plantar reflex
CM	cremasteric
KJ	knee jerk
BC	bulbocavernosus
AJ	ankle jerk
AW	anal wink
CCS	central cord syndrome
BSS	Brown-Sequard syndrome
ACS	anterior cord syndrome
PCS	posterior cord syndrome
CSF	cerebrospinal fluid

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