Revisit Spinal Shock: Pattern of Reflex Evolution during Spinal Shock

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When the spinal cord is suddenly severed, all the fundamental functions of the spinal cord below the level of injury including the spinal cord reflexes are immediately depressed, which is referred to as spinal shock. The resolution of spinal shock occurs over a period of days to months, and spinal shock slowly transitions to spasticity. The definition of spinal shock and the pattern of reflex recovery or evolution remains as an issue of debate and controversy. The identification of clinical signs that determine the duration of spinal shock is controversial. The underlying mechanisms of spinal shock are also not clearly defined. Various authors have defined the termination of spinal shock as the appearance of the bulbocavernosus reflex, the recovery of deep tendon reflexes, or the return of reflexic detrusor activity. However, many questions remain to be answered, such as: When should we define spinal shock as the end? What types of reflexes appear first among polysynaptic cutaneous reflexes, monosynaptic deep tendon reflexes, and pathological reflexes? Should it include changes in autonomic reflexes such as a detrusor reflex?

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Introduction

After an acute onset of spinal cord injury, there is a sudden loss of reflexes and muscle tone below the level of injury, termed spinal shock. The term "spinal shock" was first introduced in 1840 by Hall[17], which suddenly showed a decrease in muscular irritability and no reflexes in spinal paralysis. Before Hall's description, Whyte in 1750 reported the same motor phenomenon, but there was a relatively clear definition of loss of sensation accompanying motor paralysis with gradual recovery of reflexes. But he did not use the term shock and there was no anatomical basis for reflexes understood at the time.[12,34] Initially, it was defined by Bastian[5] in 1890 as a complete severance of the spinal cord resulting in a total loss of motor and sensory function below the level of the lesion, as well as permanent extinction of tendon reflexes and muscle tone despite the reflex arc remains intact. Flaccid motor paralysis is observed immediately after acute onset of complete spinal cord injury below the level of injury, without motor responses to external stimuli. Sherrington[34] replaced Bastian's use of the term "permanent" with a "temporary" extinction of the reflexes below the level of the lesion.[16] The definition by Sherrington[34] has been used to date as transient extinction of reflexes below the level of spinal cord injury.[7]

Spinal shock is pronounced only in the primates, especially in humans, due to such a dominance of an inhibitory mechanism in the spinal cord.[26,43] In general, the more severe the physiologic or anatomic transection of the spinal cord, the more profound the spinal shock. Generally, spinal shock does not occur with slowly developing spinal cord injury.[2] The pattern of natural course following spinal cord injury distinguishes between sudden onset and slow changes in the spinal cord. Transection of the spinal cord in humans leads to two phenomena, spinal shock below the level of injury and unusual Schiff-Sherrington phenomenon above.

Over the next days and weeks, motor reactions to external stimuli gradually reappear systematically.[10,12,22] We know the spinal shock from old observations as follows: The reflex activity begins with gradual and often plantar response. Cutaneous reflexes can occur before deep tendon reflexes. The recovery of the bladder reflex will follow the recovery of cutaneous and deep tendon reflexes.[1,10,23,30,47]

The definition of spinal shock and the pattern of reflex recovery or evolution and muscle tone recovery remains as issue of debate and controversy.[2,22] The lack of consensus on clinical symptomatology defining the duration of spinal shock continues. Some clinicians interpret spinal shock as ending with the appearance of the bulbocavernosus reflex.[20,39] Others[19,48] state that spinal shock ends with the recovery of deep tendon reflexes and may not reappear for several weeks in complete human spinal cord injury. Still other clinicians define the resolution of spinal shock as the recovery of detrusor reflex after injury.[12] If the duration of spinal shock is defined by the initial recovery of any reflex, then it probably lasts no longer than 20 minutes to 1 hour. However, if spinal shock is defined as an absence of deep tendon reflexes, its duration is several weeks.[19]

Definition of Spinal Shock

Spinal shock was initially considered for arterial hypotension after spinal cord injury.[18] The definition has evolved into a permanent extinction of tendon reflexes. Further changes to the definition have been revised to include all findings relating to the physiological and anatomical transection of the spinal cord that leads to depressed spinal reflexes for a limited time. Complete or relatively complete spinal cord lesion are followed immediately by complete loss of motor and sensory functions below the level of the lesion, when sudden onset, as well as complete loss of tone with no deep and superficial reflexes. The phenomenon in which tone and reflex activity disappear completely below the level of injury is called spinal shock. That is, spinal shock is defined as a condition of transient physiologic, rather than anatomic, reflex depression of spinal cord function below the level of injury. Spinal shock is usually temporary. Spinal shock should not be confused with neurogenic shock, is characterized by the loss of reflexes, detrusor activity, and muscle tone below the level of injury. Neurogenic shock is hemodynamic changes, one of autonomic components during the acute phase of spinal cord injury, which is caused by loss of sympathetic tone and unopposed parasympathetic function, leading to hypotension and bradycardia.[25,47] It is commonly seen when the level of injury is above T6.

A period of spinal shock can be expected after a significant spinal cord injury, defined as a decrease in excitability of spinal cord segments at and below the level of injury. There is absent somatic reflex activity and flaccid muscle paralysis below the level of injury. There were observations[2,15,43] that the most peripheral somatic reflexes of the sacral cord segments (the anal reflex and bulbocavernosus reflex) may never disappear or may return within minutes or hours of the injury, although classic teaching refer to generalized areflexia below the level of the lesion for days to months.[16] Preserved sacral reflex arcs such as bulbocavernosus and anal reflex during spinal shock due to high level cervical cord injuries should not be confused with sacral sparing. If distal sacral reflex arcs can be attributed to high level cervical spinal cord injury, they may be depressed or they may become areflexic within hours to days after injury.[2] However, functions of proximal segments to the level of injury can also be depressed.[2] Although the course of spinal shock is well known, the actual phenomenon is poorly understood, with few or no recent additional research to underlying studies for decades.

Spinal shock usually lasts for days or weeks after spinal cord injury and the average duration is 4 to 12 weeks. Spinal shock is terminated earlier and the pyramidal tract signs and defense reactions occur sooner in incomplete lesions than with complete transverse lesions. The identification of clinical signs that determine the duration of spinal shock is controversial. There is no uniform consensus on defining the cessation of spinal shock. Most references define the end of spinal shock with a return of specific reflexes. However, not all reflexes are uniformly depressed in each patient. Reflexic changes are individualized. The resolution of spinal shock occurs over a period of days to months, and spinal shock slowly transitions to spasticity. Various authors have defined the termination of spinal shock as the appearance of the bulbocavernosus reflex, the recovery of deep tendon reflexes, or the return of reflexic detrusor activity. Nevertheless, there are many questions to answer, such as: When should we define spinal shock as the end? What types of reflexes appear first among polysynaptic cutaneous reflexes, monosynaptic deep tendon reflexes, and pathological reflexes? Should it include changes in autonomic reflexes such as a detrusor reflex?

Pathophysiology

When the spinal cord is suddenly severed, all the fundamental functions of the spinal cord below the level of inju-

ry, including the cord reflexes, are immediately depressed, which is referred to as spinal shock. The underlying mechanisms of spinal shock are not clearly defined. And there has not been a convincing explanation for the recovery of the reflexes. Many hypothetical mechanisms of spinal shock have been introduced. According to modern concepts, spinal shock can be mediated by synaptic changes in spinal cord segments below the level of injury, such as by enhancement of presynaptic inhibition[8] and high concentration of glycine,[37,38] as a major inhibitory neurotransmitter, as well as by hyperpolarization of spinal motoneurons.[24,29]

Sherrington's hypothesis[34] was one of the most explainable mechanisms of spinal shock, in which sudden withdrawal of facilitatory influences of the descending pathways leads to a disruption of synaptic transmission and interneuronal conduction. The neurophysiological hypotheses are based essentially on the withdrawal of supraspinal facilitation and increased segmental inhibition. If the neurophysiological hypothesis is one aspect that explains the spinal shock mechanism, another aspect depends on the neurotransmitter. The most explainable neurochemical mechanism is three to four fold increase of glycine, an amino acid neurotransmitter, in absence or depression of reflexes during spinal shock.[37,38] High concentration of the inhibitory amino acid neurotransmitter, glycine, is associated with flaccidity following spinal cord injury or spinal shock.[38]

The relative importance of different pathways causing spinal shock is not well understood, but in lower animals, the important descending influences appear to be reticulospinal and vestibulospinal tract, while in higher animals, including man, corticospinal connections are probably more important.[3] Spinal shock occurs due to the loss of normal facilitation and/or inhibition of spinal cord interneurons and motoneurons from corticospinal, rubrospinal, vestibulospinal, and reticulospinal pathway.[4,28] Supraspinal segmental inhibition has been confirmed by several electrophysiological studies during spinal shock, with results of presynaptic inhibition and block monosynaptic and polysynaptic reflex arcs.[9,33]

The loss of tone and depression of the reflexes may be the result of a disturbance of the fusiform, γ-efferent, system that regulates the sensitivity of the muscle stretch receptors.[45] Gamma-motoneurons that regulate muscle spindle tension may potentially be fired to maintain background excitability in muscle spindles. Gamma-motoneurons may lose tonic descending facilitation distal to the level of spinal cord injury, resulting in decreased muscle spindle excitability and decreased segmental input to motoneurons by stretch reflex afferents. The disturbance of fusiform function is

caused by the loss of normal spinal cord activity, which depends on continuous tonic discharges from higher centers, including the tone discharge transmitted through the vestibulospinal and reticulospinal tracts.[45]

There were further observations that an upward spread of reflex depression, the Schiff-Sherrington phenomenon, is not uncommon. After a few hours to a few weeks, the spinal neurons gradually regain their excitability. This phenomenon seems to be a natural feature of neurons in the nervous system. That is, after the source of facilitatory impulses has been lost, it at least partially increases the level of natural excitability to compensate for the loss. In most non-primates, the excitability of the cord centers returns to nearly normal within a few hours to a day. In humans, however, they are often delayed for weeks and sometimes never completely. Conversely, recovery is sometimes excessive and as a result some or all spinal cord functions. Evolutionarily the higher species have greater degrees of spinal shock, suggesting that new descending tracts phylogenetically may be responsible.[34] Although the distal spinal cord below the level of injury has received the most attention, researchers have known for nearly a century that the proximal spinal cord is also undergoing changes, and these cephalic effects are known as the Schiff-Sherrington phenomenon.[2,16,32,34] In early clinical series, such a loss was supposed to be an extension of concussion in the area of injury, but later laboratory experiments suggest the phenomenon.[2] There was a different hypothesis explaining the recovery of reflexes. Nonsynaptic diffusion neurotransmission (volume transmission) and unmasking have been postulated to explain the recovery of reflexes. Reflex recovery may be associated with upregulation of receptors in synapses and on the surface of partially denervated spinal cord cells, resulting in increased sensitivity to neurotransmitters and other neuroactive substances released at the surviving synapses or elsewhere and transported in the extracellular fluid.[3]

Recovery from spinal shock and development of spasticity is caused by synaptic reorganization such as augmentation of latent synapses on spinal motoneurons, which are normally present but ineffective,[40] as well as collateral sprouting of axons from undamaged systems, which in turn may reinnervate partially denervated spinal neurons.[27,29]

Clinical Implications of Spinal Shock

Clinical implications of spinal shock can be summarized as follows: the higher species, the greater degree of spinal shock; the more severe anatomical transection, the more profound spinal shock; the more distal segment from the level of

injury, the later depression of reflexes; the more abrupt injury, the more prominent spinal shock; the more profound spinal shock, the worse prognosis. The presence of spinal shock appears to be prognostic only for the temporal profile of the injury mechanism. Spinal cord injury with concomitant spinal shock usually has a worse prognosis than the same degree of spinal cord injury without spinal shock because the injury is occurred over a shorter period of time.[10,16] In addition, patients with equivalent degree of spinal cord injury and spinal shock may do somewhat better if they resume reflex early.[2,16]

Spinal shock occurs mainly in sudden onset of spinal cord lesion as in the traumatic, infectious, or vascular varieties of transverse myelopathy, and it is only rarely seen in slowly progressive lesions such as tumors of the spinal cord, spondylotic myelopathy, or multiple sclerosis.[31] After a while, the cutaneous reflexes and the muscle stretch reflexes appear again, but the muscle stretch reflexes appear in an exaggerated form and a pathologic response occurs when the spinal shock subsides. When the reflex automatism of the isolated spinal cord is established, the result is always spasticity or hyperactive reflexes with abnormally spreading to adjacent isolated spinal cord segment.[2,16] This usually occurs after an interval of 3 weeks to a month. Clinically, an infection such as severe urinary tract infection or infected pressure injury will prolong the period of spinal shock.[16] If spinal shock is not physiologically identical, the later development of an infectious process, particularly severe sepsis, can be the cause.

Spinal shock is a commonly used term that represents a lack of descending facilitation after upper motor neuron lesions. It is sometimes difficult to clinically distinguish between upper and lower motor neuron lesions after spinal cord injury due to spinal shock. Spinal shock is more pronounced in severe spinal cord injury and at higher neurological levels of injury. The somatic component of spinal shock are flaccid motor paralysis, loss of sensory function, and loss of deep tendon and cutaneous reflexes. Autonomic reflexes are variably influenced depending on the level of injury. The autonomic component is the loss of sympathetic tone and unopposed parasympathetic function, resulting in hypotension, bradycardia, and skin hyperemia.[2] If the distal segments of the spinal cord are not damaged, but simply isolated from higher centers, there is usually a return of reflex detrusor contractility. Initially, such reflex activity is not maintained properly and only low pressure changes occur, but the strength and duration of such involuntary contractions typically increase, producing involuntary voiding, often resulting in incomplete bladder emptying. The return of reflex bladder activity typically occurs as an involuntary voiding between catheterizations and occurs with the recovery of deep tendon reflexes in the lower extremities.

It is important to differentiate decreasing blood pressure between from circulatory shock and neurogenic shock during spinal shock. Neurogenic shock is a type of distributive shock that consists of the hemodynamic triad of hypotension, bradycardia, and peripheral vasodilatation, resulting in loss of sympathetic stimulation to the blood vessels and unopposed vagal activity.[25] When spinal shock begins, the arterial blood pressure drops almost immediately, sometimes down to about 40 mmHg, indicating that the activity of the sympathetic nervous system is almost blocked. The pressure is normalized within a few days, even in humans. Circulatory shock requires volume replacement, but neurogenic shock requires vasopressors.[25] Although hypovolemic circulatory shock is associated with tachycardia, loss of thoracic sympathetic innervation (T1-T5) can inhibit tachycardia and vasoconstriction as signs of hypovolemia in patients with both conditions coexisting.[25,47]

Spinal shock is characterized by complete loss of autonomic nervous function below the level of injury resulting in loss of bladder tone and paralytic ileus as well as flaccid, areflexic paralysis of skeletal and smooth muscles. As the vasomotor tone is lost, the dependent lower extremities become edematous and patient may be particularly vulnerable to deep vein thrombosis and pulmonary embolism.

Clinical Observations of Reflex Evolution during Spinal Shock

There is a clinical phenomenon of spinal shock with sequential rostrocaudal depression of reflex activities after spinal cord transection and recovery of reflexes in caudorostral pattern.[15,23,31] If the duration of spinal shock is defined by the initial recovery of a reflex, it will not take more than 1 hour. When spinal shock is defined as absence of deep tendon reflexes or autonomic reflexes, its duration lasts several weeks or months.[12,22] We should also pay attention to the Schiff-Sherrington phenomenon. Proximally propagated depression or loss of reflex activity in the proximal segments to the level of injury is by the Schiff-Sherrington phenomenon which is affected by proximal interneuronal inhibition during spinal shock.

There are several characteristics of spinal shock. The severity of injury is related to the severity of spinal shock. Spinal cord injuries first change the reflexes that occur in the nearest segment of the injury, then change the reflexes more distal away from the injured segment. Thus, high-level cer-

vical injuries may have a longer preservation of sacral reflexes such as preserved bulbocavernosus and anal reflex. The observation that reflex depression or extinguishment occurs in a proximal to distal pattern suggests a physiological explanation for this change. However, spinal shock occurs immediately after spinal cord injury, but reflexes do not decrease or disappear in some segments for some periods. The segment of the spinal cord most distal to the transection may be more likely to retain some reflex activities. In clinical series, patients with high level cervical cord injuries are likely to retain distal sacral reflex such as bulbocavernosus and anal reflex despite loss of all other reflexes.[2] Guttmann[15] has found that the ankle jerk, plantar response, anal sphincter and bulbocavernosus reflex are still present immediately after spinal cord transection and may disappear only after a certain latent period. During this time, there may be present some reflex activity in the sacral segments, but reflex activity in the detrusor muscle of the bladder may be absent.

Spinal shock includes a suppression of autonomic activity as well as somatic activity, and the bladder is acontractile and areflexic. Radiologically, the bladder shows smooth contour with no trabeculation. The bladder neck is usually closed and competent unless previously undergone surgery or if the patient has no thoracolumbar spinal cord and sympathetic injury. Some electromyographic activity can be recorded in the striated sphincter and the maximum urethral closure pressure is lower than normal but still maintained at the level of the external sphincter. However, there is no normal guarding reflex that the striated sphincter contracts during filling and no voluntary control. The bladder storage pressure is low. Catheterization is necessary to solve urinary retention.

The autonomic component of spinal shock after spinal cord injury may last from days to weeks, but if a somatic component is present, it usually lasts for hours after injury. In fact, one-third of patients with spinal cord injuries can not have significant loss of reflexes without somatic spinal shock after injury. An earlier observation by Riddoch[31] showed that the sacral or caudal segment of the spinal cord after complete transection has less reflex depression than the rostral segments. The reflex depression is usually more severe and lasts longer in the segments of the isolated cord that is closer to the transection than the distal segment.[15] Other observation showed greater depression of reflexes in the rostral segments due to loss of a greater number of descending propriospinal and encephalospinal pathways.[23] Dimitrijević and Nathan[11] have suggested a very important postulation that cutaneous reflexes are the least depressed and recover

sooner because of less obvious long descending fibers contributing to the central excitatory state.

Reflexes return sequentially rather than simultaneously. During the early return of reflexes, the stimulus should be strong or summated and the response is easily fatigued.[3,44] According to Guttmann's classic spinal cord injury study[14-16], the resolution of somatic component of spinal shock was traditionally signaled by the return of the bulbocavernosus reflex and the anal cutaneous reflex, a polysynaptic spinal reflex mediated by the S2–4 via the conus medullaris. Observations before Guttmann show other phenomenon of reflex activity during spinal shock. It is not consistent with the caudorostral recovery of reflexes, for example, cremasteric reflex occurs as early as the bulbocavernosus reflex and delayed plantar response.[13,31,43] The cremasteric reflex comes from L1–2, bulbocavernosus reflex from S3–4, and delayed plantar reflex from S1. In recent decades, at least since Guttmann, no detailed observation of the reflex behavior during spinal shock have been performed.[2] It is understood that the clinical observation of reflex change since spinal cord injury in humans can not be an easy task. However, as much clinical observations as possible should be required to more clearly define spinal shock. The questions remain: How to define the spine shock? How to define when spinal shock stops? What is the first reflex after spinal cord injury and during spinal shock? Is there difference in the reflex recovery pattern depending on the reflex types?

A recent study which examined in detail the temporal return of reflexes after spinal cord injury, has challenged above traditional view.[9,22] The study was performed sequential evaluation of the reflexes on arrival at emergency room after spinal cord injury. The evaluated reflexes include delayed plantar response (reflex), bulbocavernosus reflex, cremasteric reflex, Babinski sign, ankle jerk, and knee jerk. The study has shown that the bulbocavernosus reflex may not be the first reflex to recover after spinal cord injury, but pathological reflex, known as the delayed plantar response, precedes or occurs simultaneously with the return of the bulbocavernosus reflex in most acute complete injuries (ASIA Impairment Scale, AIS, A). The delayed plantar response requires an unusually strong stimulus, unlike the Babinski sign or normal plantar response.[46] The delayed plantar response lasts hours to a few days until the evolution of the extensor plantar reflex or Babinski sign, usually within 14 days in subjects with complete injuries.[9,22] It shows a reciprocal relationship with the Babinski sign and the delayed plantar response is gradually replaced by the Babinski sign as observed by Riddoch.[13,22,31,41-43] If deep tendon reflexes are chosen as a cessation criterion for spinal shock, the dura-

tion of spinal shock is longer and will take several weeks or months. Clinical observation suggests that other reflexes after delayed plantar reflex tend to appear in the following order: bulbocavernosus reflex, cremasteric reflex, ankle jerk, Babinski sign, and knee jerk.

The pattern of reflex recovery appears to be cutaneous polysynaptic reflexes before monosynaptic reflexes.[12,22] There is no significant time difference in the recovery of deep tendon reflexes of ankle jerk and knee jerk and evolvement to Babinski sign in complete injuries. Although Guttmann[14-16] showed that the bulbocavernosus reflex recovered first, followed by deep tendon reflexes in a caudal to rostral direction, the study was unable to confirm this recovery pattern. The cremasteric reflex (L2) often precedes the ankle jerk (S1) and the delayed plantar response (S1) frequently precedes the bulbocavernosus reflex (S3−5), which is not compatible with the caudorostral recovery pattern of the reflexes.[22] Differences of reflex recovery in complete injuries according to age were significant. The younger, the more severe spinal shock with delayed development to Babinski sign, the longer duration of the presence of delayed plantar response, and the delayed recovery of deep tendon reflexes.[22] The earlier recovery of deep tendon reflexes in the elderly suggests that spinal stenosis with preexisting subclinical myelopathy can contribute to the rapid recovery of reflexes.[6,22,31] Conclusions of the study were as follows: 1) A delayed plantar response could be the first reflex; 2) The reflex recovery did not follow a caudorostral pattern. The absence of reflexes and the recovery of reflexes in a caudal to rostral sequence are of limited clinical utility; 3) Polysynaptic cutaneous reflexes may be less depressed compared to monosynaptic deep tendon reflexes; and 4) The clinical presentation of reflex activities during or after spinal shock should be reconsidered and the definition of spinal shock including autonomic reflex activities should be reestablished.[22] In addition, a patient with spinal cord injury who has delayed plantar response and/or bulbocavernosus reflexes is not suspected of damaging the conus medullaris and sacral nerve roots.

Phases of Reflex Recovery after Spinal Cord Injury

Reflex changes during spinal shock are individualized. The resolution of spinal shock occurs over a period of days to months, and spinal shock slowly transitions to spasticity. It has been suggested that this transition consist of four phases: areflexia or hyporeflexia (0−24 hours), initial reflex return (1−3 days), early hyperreflexia (4 days-1 month),

and spasticity/hyperreflexia (1−12 months).[12] Four phases of spinal shock have been postulated in view of the above clinical presentation of Ko and colleagues[22], presumably paralleled by different pathophysiological processes, but the postulation may not be as well associated with human pathophysiology. In this observational postulation, delayed plantar response and evolution to Babinski sign, recovery of deep tendon reflexes and autonomic reflexes were used for the milestones of transmission to each phase.

The first phase occurs between 0 to 24 hours after injury and is characterized by areflexia or hyporeflexia. There is no deep tendon reflex. The first pathological reflex during this period is the delayed plantar reflex followed by a series of cutaneous reflexes such as the bulbocavernosus, abdominal wall, and cremasteric reflex. Sympathetic dysfunction may cause bradyarrhythmias, atrioventricular conduction block, and hypotension. Motor neuron hyperpolarization explains the changes.[8] Phase 2 occurs between day 1 and day 3 after injury. During this phase, polysynaptic cutaneous reflexes are more prominent, while deep tendon reflexes still do not exist. It is not unusual for elderly individuals and children to experience recovery of deep tendon reflexes during this time. The Babinski sign can also be evident in the elderly. Denervation supersensitivity and receptor upregulation explain these changes in the second phase. The next phase (phase 3) occurs between 4 days and 1 month after injury. Deep tendon reflexes may be returned in the majority of patients and the Babinski sign may appear. It differs from the fourth phase (1−12 months) in which hyperactivity occurs in cutaneous and deep tendon reflexes in response to minimal stimuli. Deep tendon reflexes usually recover by 3-days after injury. There is a big discrepancy in the appearance of this reflex. The recovery of the Babinski sign is almost similar to the return of the ankle jerk. There is also a decrease in delayed plantar reflex. Autonomic changes such as bradyarrhythmias and hypotension begin to subside. This period is reflected by axon-supported synapse growth. The fourth phase is dominated by hyperactive reflexes and occurs from 1 to 12 months after injury. Vasovagal hypotension and bradycardia generally improve within 3 to 6 weeks, but orthostatic hypotension may take 10 to 12 weeks to disappear. During this period, episodes of malignant hypertension or autonomic dysreflexia begin to appear. Soma-supported synapse growth accounts for these findings.

The physiology of the latter two phases is driven by synapse growth and short (phase 3) and long axon growth (phase 4) from intraspinal and segmental afferent sources replacing empty synaptic endings in axotomized supraspinal neurons. This hypothesis suggests that post-injury synaptic

formation is axon-length dependent, activity dependent, and competitive, leading to a gradual termination of spinal shock.[12,30,36] In the clinical presentation, the transition from spinal shock to spasticity is a continuum that gradually increases motor excitability with characteristic changes in muscle tone, spasm, and short- and long-latency reflex excitability.[19,35]

Conclusion

Initially observable reflexes are the polysynaptic cutaneous reflexes such as delayed plantar response, bulbocavernosus reflex, and cremasteric reflex, rather than the monosynaptic deep tendon reflexes, and these distinctions are more evident than caudorostral distinction.[12,22] It seems that the polysynaptic cutaneous reflexes receive less supraspinal facilitation and/or that synaptic areas are less disturbed because descending pathways provide less contributive.[1,21] If spinal shock is defined as the absence of all reflexes, the definition of spinal shock may be reestablished since all reflexes are rarely absent, even in cases of complete injuries. The view of spinal shock that reflex return occurs in a caudal to rostral sequence may also be reconsidered. A more accurate description of spinal shock should be characterized by a period of altered appearance of cutaneous and deep tendon reflexes and the emergence and at times disappearance of pathologic reflexes over days and weeks.[22]

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