

The Spinal Shock

The Pathophysiology of the Spinal shock

"Innovated Conception"

How could a spinal section at the level of the cervical spine (CS) block a spinal reflex (SR) that is governed by the first sacral spinal nerve, for example? Obviously, the distance between the site of the spinal injury and the conventional neural center governing the reflex is so far.

Moreover, in spite of a non-recovered complete high cervical spine section, how could the blocked spinal reflexes come back later? In addition, why the returned reflexes are of different characters from the original ones?

These major questions will be at the core of the following article. Herein, you will find out my innovative conception about the actual circuit of the spinal reflexes.

By adopting this innovative conception, one can clarify all the mysteries contouring the spinal shock, and can identify the actual pathophysiology hiding behind the offensive characters of the returned spinal reflexes as well. Moreover, we might finally arrive to a certain therapeutic conclusion that may help changing the dark destiny of such lesions.

1. The Spinal Reflex

1.1. The Conventional Spinal Reflex Circuit

The conventional circuit of the spinal reflex consists of the lower sensory neuron, the lower motor neuron (LMN), and the intermediate neuron. These three major components of the circuit reside at the same and/or quite adjacent spinal segments. Conventionally, this circuit is named the lower motor neuron circuit (LMNC).

Directly, the afferent impulse is conducted from the lower sensory neuron to the lower motor neuron via the intermediate neuron. Then, the efferent impulse leaves the lower motor neuron toward the effector muscle(s); figure (1).

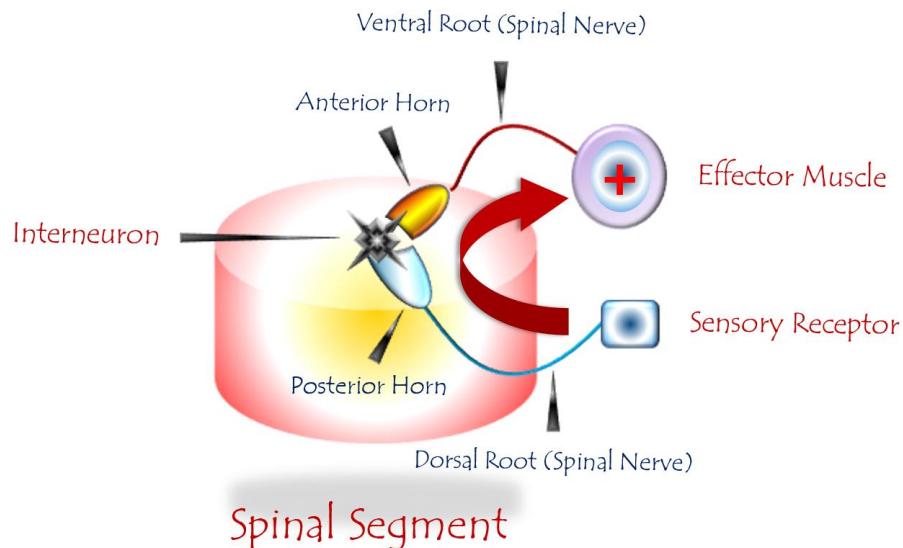


Figure (1)
The Traditional Physiology of Spinal Reflex
(The Lower Motor Neuron Circuit)

For more details concerning the Traditional Physiology of the Spinal Reflex,
see the linked video:

The spinal reflex contains three partners;
the sensory neuron (SN), the intermediate neuron, and the lower motor neuron (LMN).
The three elements reside the ganglion of the dorsal root of the spinal nerve,
the posterior horn, and the anterior horn of the spine, respectively.

They can be in one or in two adjacent spinal segments.
The afferent impulse arrives to the sensory neuron firstly. Then, via the intermediate
neuron it reaches the LMN. The LMN reacts and hence sends the motor orders to the
target organ (effector muscle).

The Upper Motor Neuron has nothing to do with the spinal reflex itself.
The UMN observes and evaluates without interfering in such process

1.2. The Innovated Spinal Reflex Circuit

I do believe the intermediate neuron of the spinal reflex circuit actually is the upper motor neuron (UMN) itself. In such a way, the intermediate neuron (i.e., the upper motor neuron) becomes the absolute governor of the spinal reflex.

The afferent sensory impulses end in the lower sensory neurons. The ascending neural tracts conduct the afferent impulses toward the upper sensory centers in the brain. The upper neural centers treat the afferent information, and then arrive to a certain conclusion. The UMs receive this conclusion, and in turn decide the appropriate motor reflex(s). The

efferent motor orders descend to the lower motor neurons (LMNs). Finally, the LMNs execute the motor orders.

Accordingly, the circuit of the spinal reflex should be consisted of the lower sensory neuron, the lower motor neuron, and at the middle of the circuit, and at its top hierarchy as well, is the upper motor neuron. This newly suggested circuit of the spinal reflex is named as the upper motor neuron circuit (UMNC).

Beyond the early infancy, the LMNC does not exist in a healthy individual. Gradually after birth, the UMN possess the decision-making authority. In such a way, the LMNs satisfy executing the descending motor orders only; figure (2).

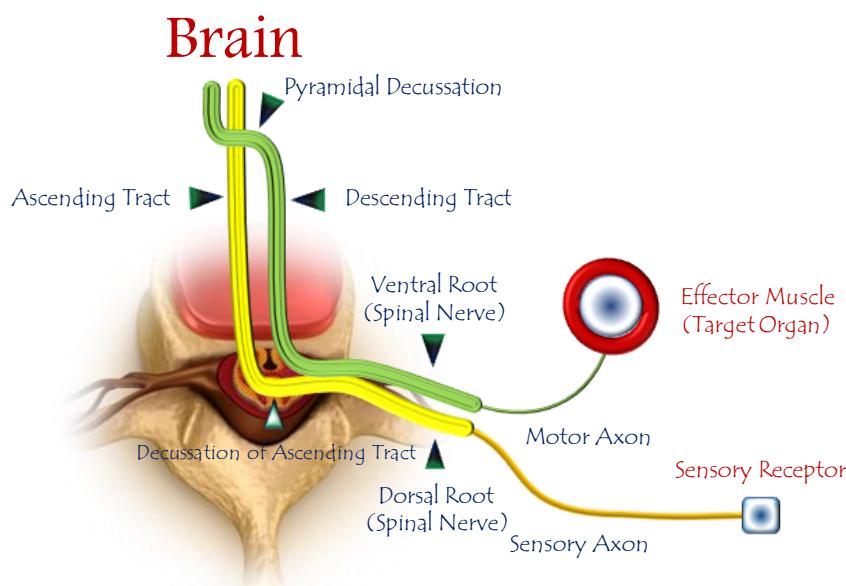


Figure (2)
The New Physiology of Spinal Reflex
(The Upper Motor Neuron Circuit)

For more details concerning the New Physiology of the Spinal Reflex,
see the linked video:

*I do believe the spinal reflex runs in this manner:
The afferent sensory impulses end in the sensory neuron.
The ascending neural tracts conduct the afferent impulses toward the brain.
The Brain treats the afferent data, and then arrives to a certain conclusion.
The upper motor neuron (UMN) receives this conclusion, and in turn decides the appropriate motor reflex.
The efferent motor order descends to the lower motor neurons (LMN).
Finally, the LMN executes the motor order.*

1.3. The Lower Motor Neuron Circuit (LMNC)

The LMNC is the innovated term that I frequently use to refer to the pathological situation where the LMN takes the decision-making authority instead of the UMN.

Normally, the LMNC does not exist. However, when it exists, it indicates the total blockage of the descending and/or the ascending neural tracts and/or the lesion of the UMN in the brain; figure (3).

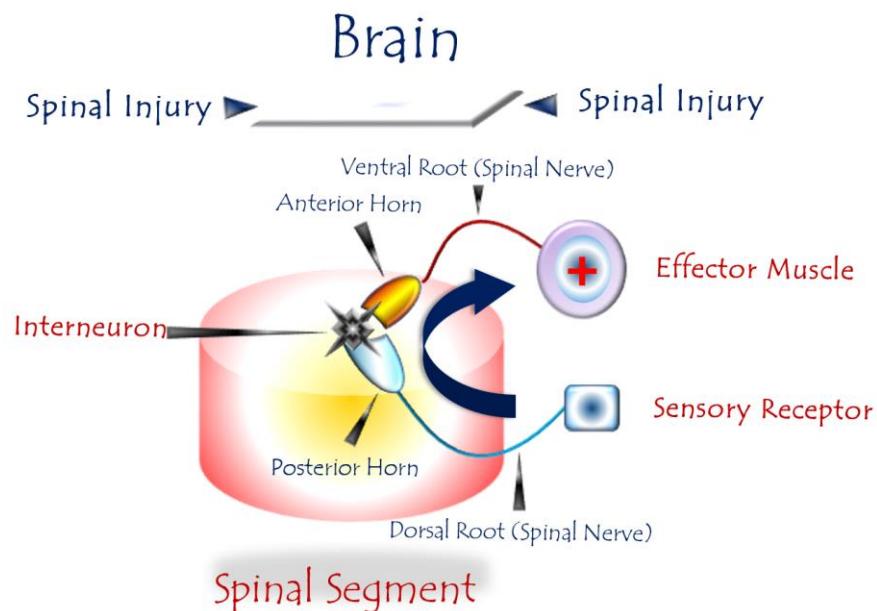


Figure (3)
The New Pathophysiology of Hyperreflexia
(The Lower Motor Neuron Circuit)

For more details concerning the Pathophysiology of the Hyperreflexia,
see the linked video:

I do believe:

In healthy persons, the lower motor neuron circuit (LMNC) does not exist. However, when it does exist, it indicates the blockage of the descending neural tracts and/or the lesion of the upper motor neurons in the brain. The LMNC is a pathological anatomical and functional circuit. Unfortunately, when it exists it worsens the outcome of the neural injury.

2. The Spinal Shock

For more simplicity, we will imagine a clinical case of a complete traumatic spinal section at the level of the sixth cervical segment (C6).

Clinically, the patient will suffer from two subsequent phases. The first phase is the flaccid paralysis phase, and the second one is the spastic paralysis phase.

In the flaccid paralysis phase, all the spinal reflexes under the level of spinal injury (C6) are blocked completely. Moreover, all sorts of sensation and voluntary motion in the concerned part of the body disappear.

Afterward, the spastic paralysis gradually dominates the clinical picture. The spinal reflexes begin to reappear but in a new character. They become of increased activity and of increased tonicity.

2.1. The Pathophysiology of Flaccid Paralysis

The spinal injury disrupts all the descending and/or ascending neural tracts. Consequently, the LMNs below the site of injury are found deprived of any contact with the UMNs. Since there are no more descending orders, the LMNs keep silent and the flaccid paralysis dominates. For the same cause, the spinal reflexes are completely blocked below the site of cervical injury.

Similarly, the ascending afferent sensory impulses are also blocked at the level of the cervical injury. Therefore, the brain loses the sensory information issued from this part of body under the level of injury.

2.2. The Pathophysiology of Spastic Paralysis

The LMNs and the sensory neurons (SNs) as well, try to find out some alternative pathways in order to overcome this absolute terrible functional silence. Finally, they arrive to reactivate the ancient neural pathways that used to connect between them in the early infancy. Furthermore, for the same purpose, I do not exclude that some new lateral pathways could also be constructed.

In such a way, the SNs arrive to discharge their burdens of raw data directly into the LMNs' field. The LMNs receive these untreated neural impulses as an order to act. Subsequently, the LMNs do act but in a pathological spastic way.

*Herein, in the absence of the UMNC, the LMNC reappears and dominates. This is a pathological circuit that does not exist but in the case of UMNs injuries. Therefore, I call it the **pathological lower motor neuron circuit (LMNC); figure (3).***

The LMNC is an anatomical and a functional circuit. Unfortunately, its presence worsens the outcome of the spinal injury. Actually, when the LMNC installs it precludes the normal function of any recovered UMNC. It is stronger and more functional than the recently recovered UMNC. In somehow, it forms a functional blockage against the spontaneous and/or surgical recovery of the spinal injury.

3. Recommendations

3.1. In Flaccid Phase

For any penetrative injuries to the vertebral column, I recommend an early surgical intervention. It is explorative and reparative surgery at the same time.

It is rare that a penetrative injury to the vertebral column would pass without a serious harm to the spinal cord. It is up to the surgery to find out the eventual spinal injury, to evacuate all the debris and all the bony sequestrations out of the spinal canal, to improve the conditions of the surrounding tissues in order to optimize the process of spinal recovery, and finally to ensure the stability of the vertebral column. Moreover, I recommend suturing the spinal cord and the spinal nerves whenever it seems possible to do so.

3.2. In Spastic Phase

Since the LMNC is quite present, any attempt to improve the outcome of the spinal injuries will be hindered by the well-established strong LMNC. Therefore, it is obligatory to break the LMNC in order to give a real chance to the upper motor neuron circuit to regain the terrain.

I do believe the lower sensory neuron to be the weakest link of the LMNC. Therefore, all the therapeutical efforts should be concentrated in eliminating this element.

By the radiofrequency therapy, one can temporarily or permanently paralyze the lower sensory neuron. Then, the lower motor neuron will do its best to reactivate the ancient neural pathways. Henceforth, one can really test the efficiency of the recovery process at the site of spinal injury. It will be a new chance to come back to the normality; i.e., the upper motor neuron illuminates the lower motor neuron.

Herein, the surgical intervention can also be of great importance. The surgery removes all the constricting fibrous bands, as well as all the bony sequestrations, out of the spinal canal. Thus, some neural pathways could surpass the zone of spinal injury and assure the communication between the UMN and the LMN.

4. Conclusion

As mentioned above, shortly after the forced absence of the influence of the upper motor neuron, the lower motor neuron tries to build its independent circuit. The newly born circuit of the LMN is very active and very sensitive. It stands behind all the pathological symptomatology of the spastic phase.

Moreover, the LMNC is quite strong. It could preclude the spontaneous natural process of recovery as well as those induced by the surgical intervention. In somehow, it is a real obstacle to the ultimate outcome of the spinal cord injuries.

Therefore, in order to improve the outcome of the spinal cord injuries, it is vital to prevent the birth of such pathological lower motor neuron circuit, or to break that circuit in the other late cases.

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2018/6/4