AMMAR YASEEN MANSOUR

The Upper Motor Neuron Injury

The Pathophysiology of the Symptomatology
(Personal View)

N.B.
The Arabic version of this article is the reference,
read it on the following links:

أذيَّاتُ العصبونِ المُحركِ العلويّ
بحثٌ في فيزيولوجيا الأعراضِ والعلاماتِ السَّريريَّة
The Upper Motor Neuron Injuries
The Pathophysiology of Symptomatology

1. The Spinal Reflex, The Traditional Physiology

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

In the traditional concept of the spinal reflex, the circuit of the reflex is entirely present quite below in the spine, and is out of the control of the brain. In such way, the afferent impulse directly passes from the sensory neuron (SN) to the lower motor neuron (LMN) via the intermediate neuron (interneuron). These three elements are the native residents of the same side of one or two adjacent spinal segments. They always cooperate in order to respond to an external stimulus as fast as possible.

However, it is worthwhile to mention the peripheral elements of the spinal reflex as well; i.e., the sensory receptor, the afferent sensory neural fiber, the efferent motor neural fiber, and finally the effector muscle (target organ).

In such concept, the interneuron, which is always present, closes the circuit of the spinal reflex. Moreover, it is considered the corner stone of the spinal reflex.

Consequently, the upper motor neuron (UMN) has nothing to do with the spinal reflex itself. The UMN observes and evaluates without interfering in such process; ***figure (1).***

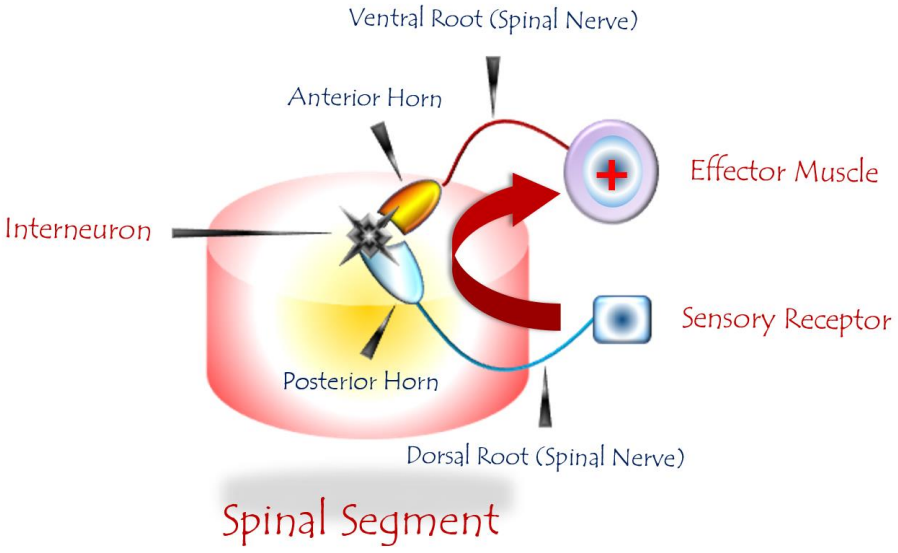

Figure (1)
The Traditional Physiology of Spinal Reflex
(The Lower Motor Neuron Circuit)

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

2. *The Spinal Reflex, The New Physiology*

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

I do believe the spinal reflex to be the function of the upper neuron (i.e., the brain). In such way, the stimulus provokes an afferent impulse that arrives to the brain via the sensory ascending tract. Thereafter, it is up to the brain to process the afferent data, and then to make the adapted reaction vis-à-vis the stimulus. Then, the efferent impulse takes the

descending motor tract in order to reach the lower motor neurons and then to the effector organ (target organ) sequentially.

Thus, the brain plays the essential role in the spinal reflex. Actually, it lays at the summit of the reflex circuit. It processes all the afferent data, and then makes the adapted reaction, which ensuite is sent to the effector organ via the lower motor neuron; ***figure (2).***

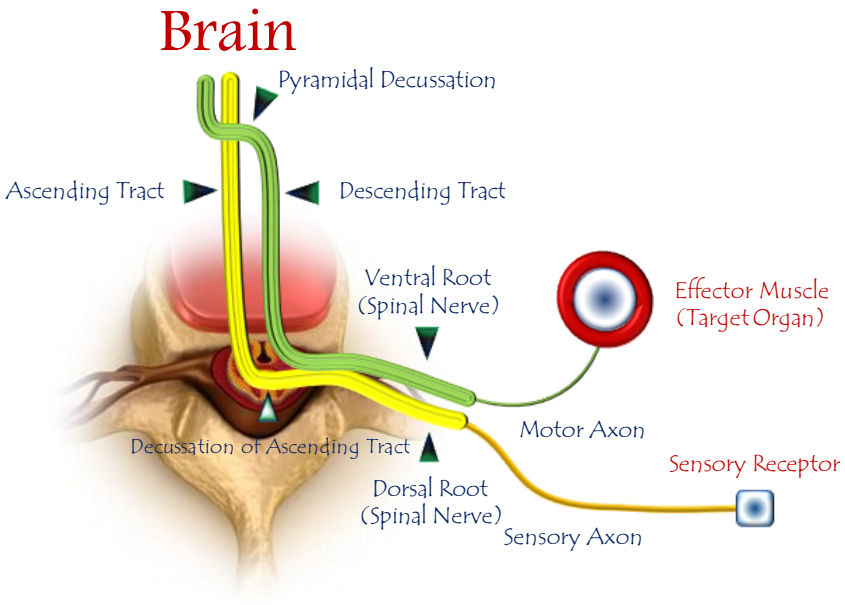

Figure (2)
The New Physiology of Spinal Reflex
(The Upper Motor Neuron Circuit)

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.

3. The Hyperreflexia, The New Pathophysiology

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

The upper motor neuron injuries indicate the blockage of both the ascending and the descending neural tracts. Since, in order to skip the induced blockage, an intermediate bypass is generated in between by the intermediate neurons (the interneurons). Hence after, the afferent impulses will pass directly from the sensory neuron of the ganglion into the lower motor neurons of the anterior horn of the spine, and to the effector organ sequentially.

Indeed, what is for a long time supposed to be the physiology of the spinal reflex becomes for me the actual pathophysiology of the hyperreflexia. Moreover, this pathological lower motor neuron circuit does not exist but in the case of upper motor neuron injuries. However, it will remark all the pathological manifestations of the spastic paralysis (or spastic paresis); ***figure (3).***

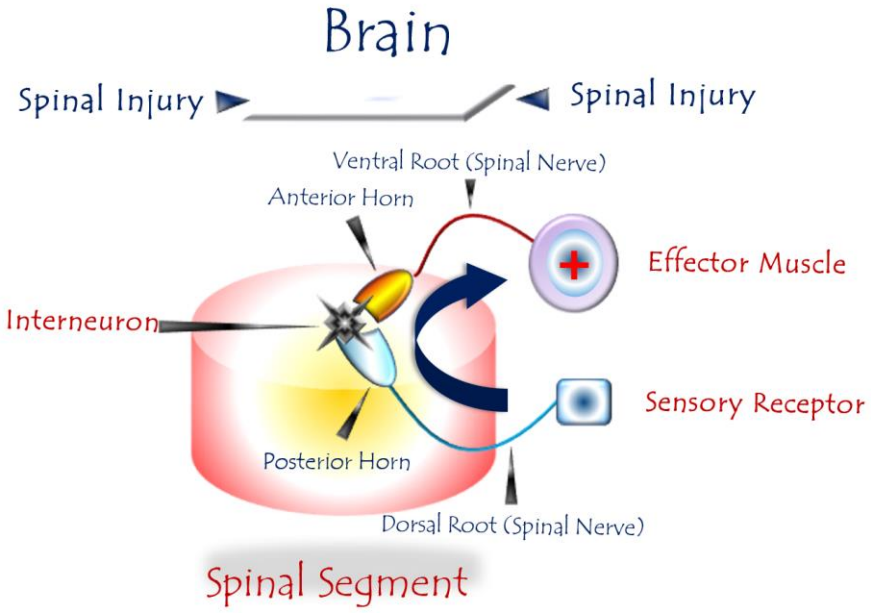

Figure (3)
The New Pathophysiology of Hyperreflexia
(The Lower Motor Neuron Circuit)

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.

3.1. The Overactive Hyperreflex, The Pathophysiology

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

Thanks to the data processing in the brain, the physiological spinal reflex is always unilateral, well measured, and particularly is coherent with the nature of the stimulus. Actually, the afferent impulses get their own senses in the brain, and nowhere else. It is up to the brain to receive all the afferent data, to analyze them, and then to make the appropriate decision vis-à-vis the contingent stimulus.

However, in case of brain absence, the afferent impulses are directly deviated toward the lower motor neuron, and immediately to the effector muscle ensuite. The effector muscle will then get all the power burden of the afferent impulse as it is, and then accordingly contracts.

Without the brain filter and treatment, and despite the insignificancy of the stimulus itself, such a raw weak afferent impulse might be of a great impact on the effector muscle. In such way, the contraction of the effector muscle might be exaggerated, over measured, unadjusted, and is especially incoherent with the stimulus energy; ***figure (4).***

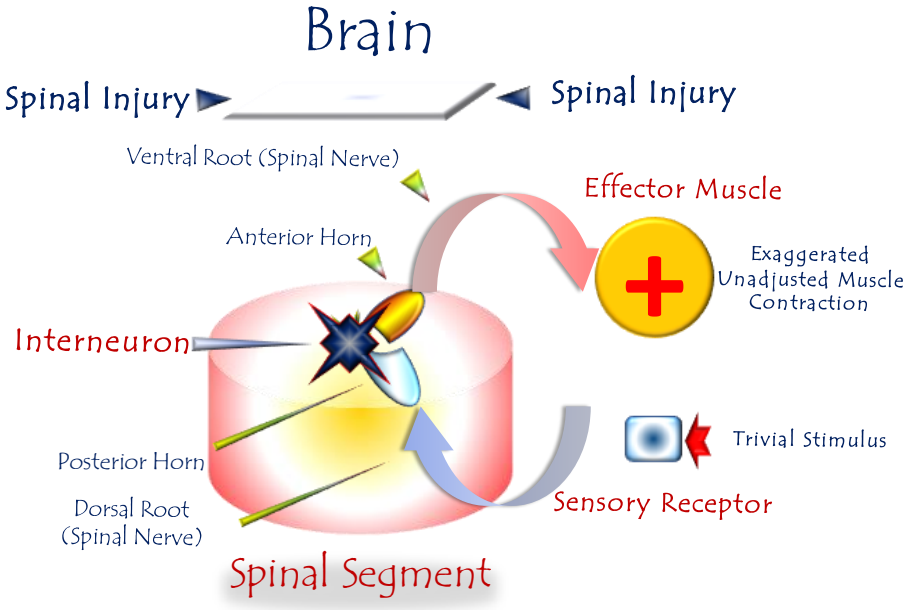

Figure (4)
The Pathophysiology of Overactive Hyperreflexia

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

Without the brain filter and treatment,
and despite the insignificancy of the stimulus itself,
such a raw weak afferent impulse might be of a great impact on the effector muscle.
In such way, the contraction of the effector muscle might be over measured, unadjusted,
and is especially incoherent with the stimulus energy.

3.2. The Bilateral- Response Hyperreflex, The Pathophysiology

For more details concerning the Pathophysiology of the Bilateral- Response Hyperreflex, see the linked video:

Thanks to the data processing in the brain, the spinal reflex is always unilateral. However, in the upper motor injuries, the hyperreflex might manifest bilaterally. Since the interneurons of the two halves of the spinal segment do interlink between each other, the hyperreflex of both sides unite in one neural network. Hence, the two emerged pathological circuits of the hyperreflexia become one. Therefore, the activation of one circuit will unduly activate the contralateral one, and vice versa; ***figure (5).***

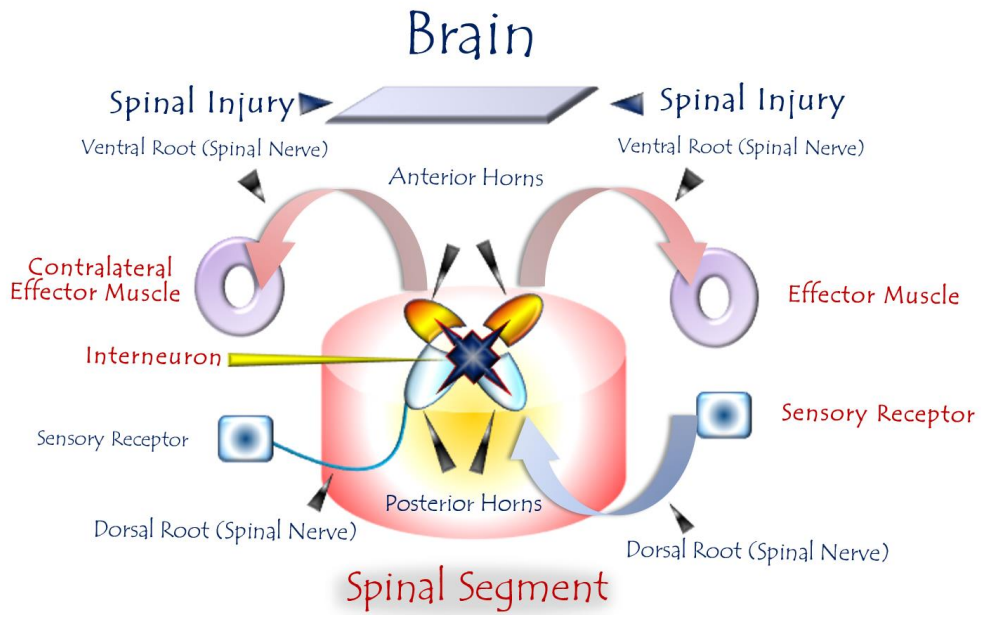

Figure (5)
The Pathophysiology of Bilateral- Response Hyperreflexia

For more details concerning the Pathophysiology of the Bilateral- Response Hyperreflex, see the linked video:

Upon their efforts to discharge their burdens of raw data,
the sensory neurons (SNs) sometimes arrive to communicate with the lower motor neurons (LMNs) of both sides; the ipsilateral and the contralateral sides, of the same spinal

segment. Therefore, the two emerging hyperreflexes circuits unite in one pathological circuit, which is the bilateral- response spinal hyperreflex.

An example of the prementioned hyperreflex, a tapping on the right patellar tendon can provoke the extension of both knees simultaneously.

3.3. The Extended Hyperreflex, The Pathophysiology

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

Thanks to the data processing in the brain, each spinal reflex has its own predesignated trigger points. Actually, these trigger points are the only keys to launch the reflex circuit.

In the upper motor neuron injuries, and because of the brain absence, the interneurons start activating the ancient deserted neural connections and/or creating new neural connections. Therefore, the hyperreflex recruits new trigger points for its pathological circuit. In such way, the hyperreflex extends its sector of work; ***figure (6).***

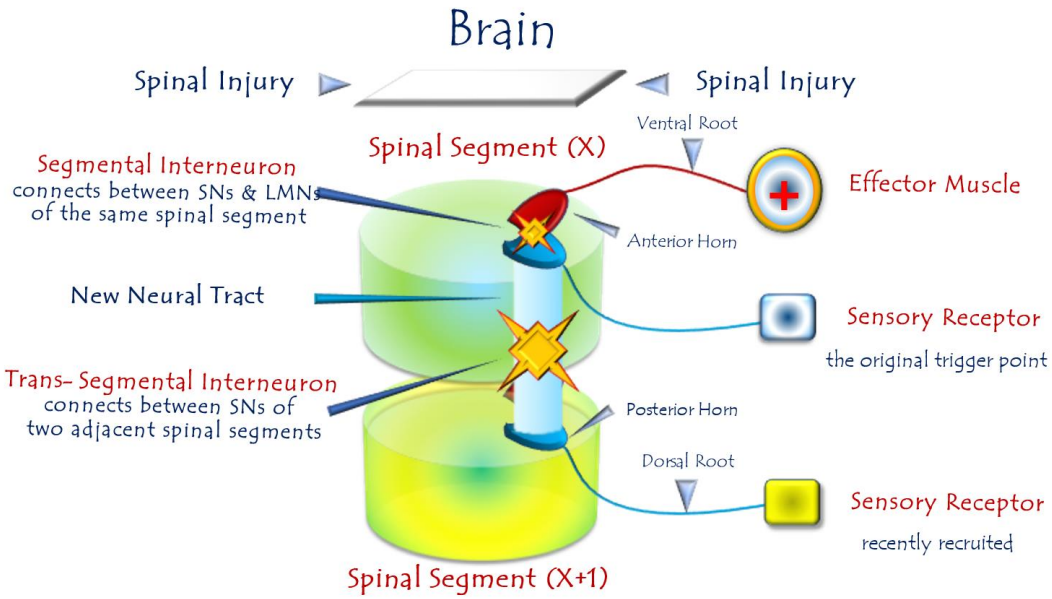

Figure (6)
The Pathophysiology of Extended Hyperreflexia

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

Upon their efforts to discharge their burdens of raw data,
the SNs arrive to communicate with the other SNs of the adjacent spinal segments,
as well as with the LMNs of the same spinal segment.
Therefore, the SNs of many spinal segments work together
to serve one spinal hyperreflex circuit.
Consequently, in addition to the muscle's tendon, the stimulation of the muscle body itself
and/or the cutaneous coverage van provoke the contraction of the effector muscle.

3.4. The Multi- Motor- Response Hyperreflex, The Pathophysiology

For more details concerning the Pathophysiology of the Multi- Motor- Response Hyperreflex, see the linked video:

Thanks to data processing in the brain, every spinal reflex has its own independent predesignated circuit. So, the concept of "One Reaction for One Action" rests valid.

However, in upper neuron injuries, and because of the brain absence, the interneurons start activating the old unused (abandoned) neural roads and/or creating new neural connections. In such way, many reflex circuits would be interlinked. So that, one stimulus can activate all of them simultaneously; ***figure (7).***

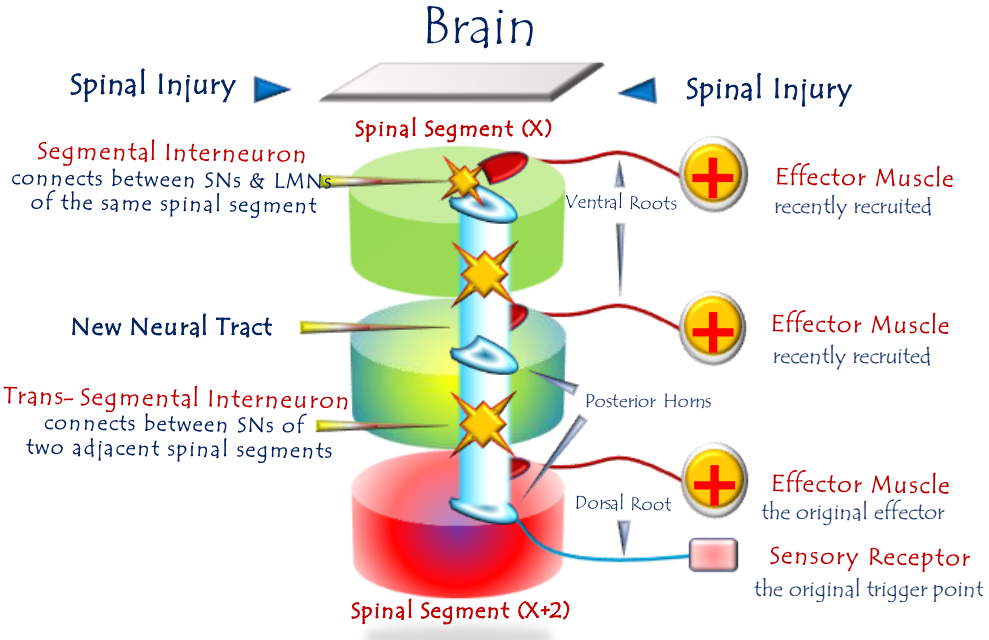

Figure (7)
The Pathophysiology of Multi- Motor- Response Hyperreflexia

For more details concerning the Pathophysiology of the Multi- Motor- Response Hyperreflex, see the linked video:

In the upper motor injury,
the SNs of one segment arrive to communicate with many LMNs of the adjacent spinal segments. Thus, the SNs of a specific spinal segment (X+ II) could supply the LMNs of the adjacent segments (X) & (X+I). Therefore, as an example, the stimulation of the right patellar tendon could simultaneously provoke the extension of the right knee,
the flexion of the right hip joint as well.

4. The Clonus

Clonus is a rhythmic, oscillating, stretch reflex, the cause of which is not totally known; however, it relates to the lesions of the upper motor neurons and therefore is generally accompanied by the hyperreflexia. It can be evaluated in many joints but is most commonly seen in the ankle joint by briskly dorsiflexing the foot.

Sometimes, we obtain a similar movement in normal individuals, however it should be less than five oscillations in such cases. Provided, it is accompanied by other signs and/or symptoms of hyperreflexia, one should think of its morbidity even in a low rhythm.

4.1. The Clonus, 1st Hypothesis of Pathophysiology

For more details concerning the 1st Hypothesis of Pathophysiology of the Clonus, see the linked video:

Actually, one stimulus can activate a group of different receptors that cohabit in the same zone of contact. These different receptors are supplied by different types of sensory axons. Since each axon has its own velocity of neural conduction, the related afferent impulses will reach the target subsequently, in different time, as well.

Normally, the afferent impulses will reach the brain consequently. Thereafter, it is up to the brain to treat the afferent data, and then to make the adapted reaction vis- a- vis the stimulus. In such way, one stimulus can have just one single adjusted response.

However, in the upper neuron injuries, the things are no longer the same. The brain function of processing the afferent data is no more functional. Moreover, the consecutive afferent impulses will be directly deviated toward the effector muscle (Target Organ) via the intermediate neurons and the lower motor neurons sequentially. So that, each afferent impulse will obtain its own response; (X) afferent impulses can then provoke (X) reflexive responses; ***figure (8).***

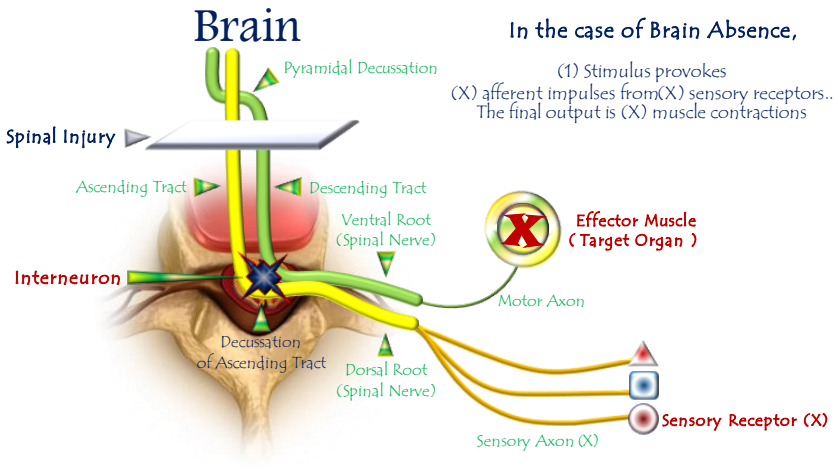

Figure (8)
The Pathophysiology of the Clonus, 1st Hypothesis

For more details concerning the 1st Hypothesis of Pathophysiology of the Clonus, see the linked video:

The different velocity of neural conduction in the different sensory axons will be the base of my first hypothesisi of clonus.
In the case of brain absence, all the afferent impulses will directly arrive to the effector muscle. Each afferent impulse will provoke its own muscle contraction.
Consequently, (X) afferent impulses will provoke (X) muscle contractions..
Which is the Clonus.

4.2. The Clonus, 2nd Hypothesis of Pathophysiology

For more details concerning the 2nd Hypothesis of the Pathophysiology of Clonus, see the linked video:

Normally, one stimulus launches its related reflex circuit only. For an example, tapping on Achilles tendon launches its own reflex circuit (i.e., the ankle jerk reflex). However, in the upper motor injuries and because of the induced overactive hyperreflex, the contraction of the related muscles (i.e., the Gastrocnemius and Soles muscles in our example) can be brisk and too severe. The severe muscle contraction could activate another group of receptors that belong to the antagonistic muscle(s) (i.e., the Tibial muscle in the same example).

In turn, the severe tibial muscle contraction will activate the receptors of the antagonist muscles (i.e., the Gastrocnemius & the Sole muscles). Hence, a vicious circle of two opposite overactive hyperreflexes arises. In such a way, one overactive hyperreflex activates the antagonistic overactive hyperreflex circuit, and vice versa.

However, every coming hyperreflex circuit will be of less energy than the precedent. At times, the muscle contraction ends to be too weak to launch a new circle of the vicious circle. The clonus then stops. Moreover, the abused muscles of both sides of the vicious circle will eventually exsanguinate the stock of energy. So finally, they give up the game. And the clonus also stops; ***figure (9).***

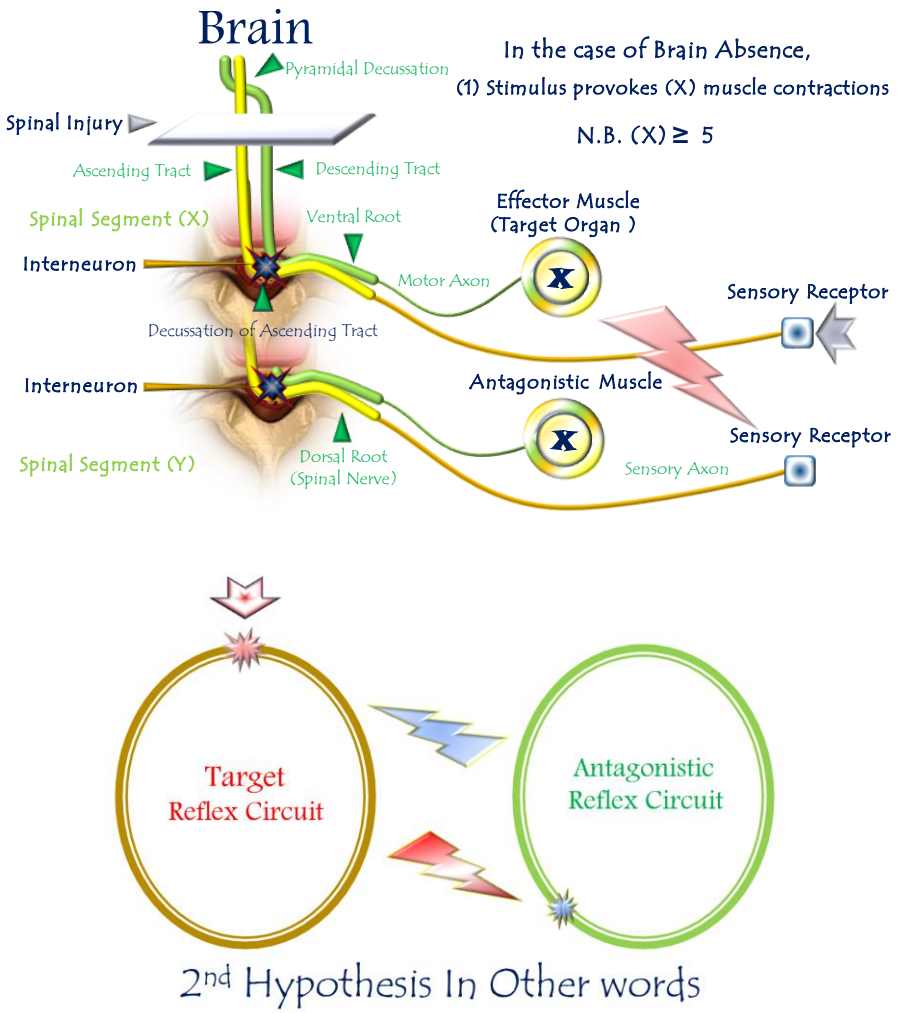

Figure (9)
The Pathophysiology of the Clonus, 2nd Hypothesis

For more details concerning the 2nd Hypothesis of Pathophysiology of the Clonus, see the linked video:

In upper motor injuries,
and because of the induced hyperactive hyperreflex, the contraction of the effector muscles (i.e., the Gastrocnemius and the Soles muscles in the case of ankle jerk reflex) can be brisk and too severe.
However, the severe muscle contraction could activate another group of receptors that

belong to the antagonistic muscle(s)
(i.e., the Tibial muscle in the same example).

Hence, a vicious circle of two opposite hyperactive hyperreflexes arises.
In such a way, one hyperactive hyperreflex activates the antagonistic hyperactive hyperreflex circuit, and vice versa.

5. *The Pathophysiology of the Muscle Spasms and Spasticity*

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

In the spastic phase, why the patient suffers a continuous muscular contraction that can by time induce the rigidity to the affected part of body?

The lower part of the body below the level of the spinal cord injury is subjected to the influence of the LMNC. The LMNC is very active and very sensitive. In such a way, even the trivial cutaneous stimulations (i.e., a sample touch, a blow of air, etc.) as well as the ordinary internal ones (i.e., intestinal movements, intestinal gas, urine and feces retention, etc.) can illuminate the reflexive circuits.

The permanently activated LMNCs impose the contraction of both the flexor and extensor muscles at the same time. In another sense, the simultaneous contraction of the antagonist and agonist muscles is responsible of the spastic situation in the UMNIs; ***figure (10).***

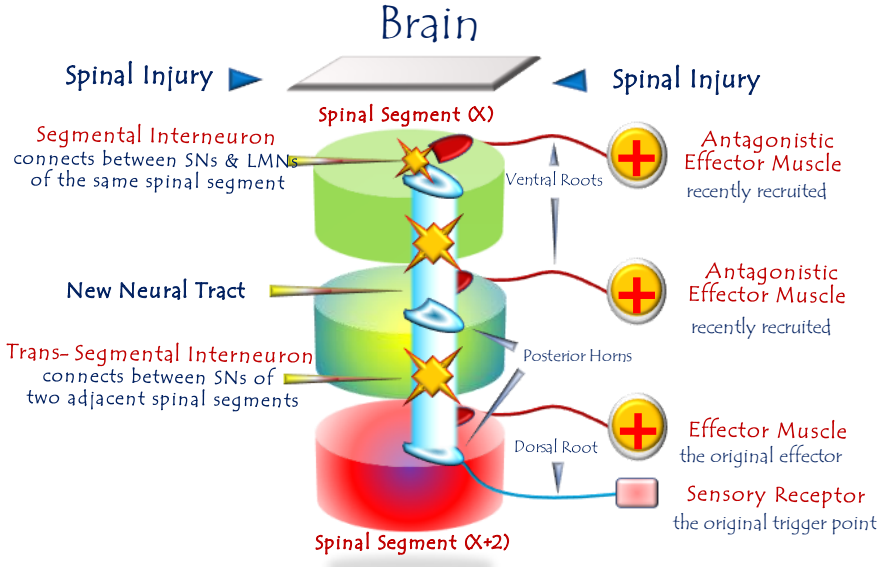

Figure (9)
The Pathophysiology of the Muscle Spasm

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

The lower part of the body below the level of the spinal cord injury is subjected to the influence of the LMNC. The LMNC is very active and very sensitive. In such a way, even the trivial cutaneous stimulations (i.e., a sample touch, a blow of air, etc.) as well as the ordinary internal ones (i.e., intestinal movements, intestinal gas, urine and feces retention, etc.) can illuminate the reflexive circuits.

The permanently activated LMNCs impose the contraction of both the flexor and extensor muscles at the same time. In another sense, the simultaneous contraction of the antagonist and agonist muscles is responsible of the spastic situation in the UMNIs

6. The Pathophysiology of the Triple Flex Reflex

In the spastic phase, why the normal defense reflexes such as the withdrawal reflex do change their characters?

Normally, a painful stimulation of the grand toe of foot provokes the dorsal flexion of the ankle joint, the knee flexion, and the hip joint flexion simultaneously. These triple reactions persist as long as the painful stimulation of the grand toe persists. The purpose of the defense reflex is to protect the individual and to keep him (her) away off the pain source.

In the UMNIs, the withdrawal defense reflex changes its characters. In such a way, even a trivial unpainful stimulation might provoke the same triple reaction. Moreover, the triple response disappears soon regardless of the continuity of the act of stimulation. In this case, it is justifiable to change the name of the withdrawal defense reflex, and adopt the new term "the triple flex reflex" to show the difference.

In the normal defense reflex, the upper neural centers in the brain guide all the process of the withdrawal defense reflex. They accumulate and treat all the afferent raw data. Then, when they suspect such stimulant to be dangerous to the individual, they command the effector muscles to contract and keep contracting as long as the painful stimulant is in direct contact with the organ.

Whereas, in the UMNIs, the LMNC is quite present. Hence, all the afferent sensory impulses from the relevant cutaneous sector will directly be deviated towards the LMNs. However, the LMNs have not the aptitude nor the experience to analyze such unprocessed raw data. The LMNs receive the sum of the afferent as an order to a simple act. Herein, the sensory raw

afferent has no sense but the sense of an order to act. Therefore, the relevant effector muscles do contract, but they do not keep contracting.

In the UMNIs, the decision-making authority is referred to the unskillful LMNs. Since there is no afferent data, the brain centers do not feel threatened by the stimulant what so ever is the stimulant. Unpurposely, the foot is withdrawn for a while, then it is pushed again toward the stimulant, which could be very dangerous. From now on, the defense reflexes lose the justification of existence, and are hence deprived from the instinctive purpose of protection.

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- *Spinal Reflex, New Hypothesis of Physiology*
- *Hyperreflexia, Innovated Pathophysiology*
- *Clonus, 1st Hypothesis of Pathophysiology*
- *Clonus, 2nd Hypothesis of Pathophysiology*
- *Clonus, Two Hypotheses of Pathophysiology*
- *Hyperreflexia (1), Pathophysiology of Hyperactivity*
- *Hyperreflexia (2), Pathophysiology of bilateral Responses*
- *Hyperreflexia (3), Pathophysiology of Extended Hyperreflex*
- *Hyperreflexia (4), Pathophysiology of Multi-Response Hyperreflex*
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- *This Woman Can Give Birth to Female Children More Than to Male Children*

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