

# *Upper Motor Neuron Lesions*

## *The Pathophysiology of Symptomatology*

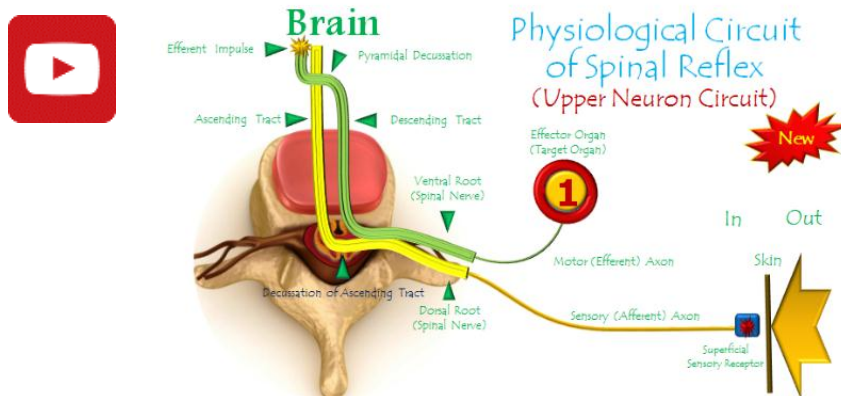
### *(Innovated)*

#### **1. Spinal Reflex, New Hypothesis of Physiology**

*I do believe the spinal reflex to be the function of the upper neuron (i.e. the brain). In such a 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 (s), which ensuite is (are) sent to the effector organ (s) via the lower motor neurons.*

*For more details, watch the linked video:*

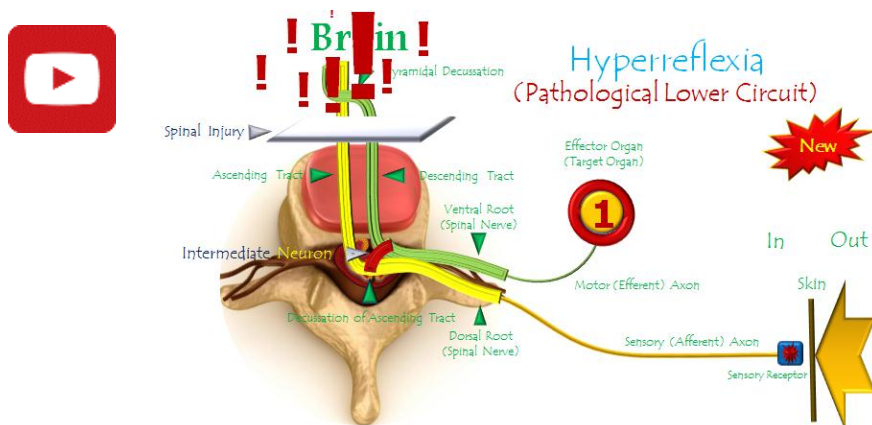


#### **2. Hyperreflexia, New Hypothesis of Pathophysiology**

*The upper motor injuries indicate the blockage of both ascending and descending neural tracts. Since, in order to skip the induced blockage, an intermediate bypass is generated in between by the intermediate neurons. 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 spine, and to the effector organ (s) subsequently.*

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

For more details, watch the linked video:



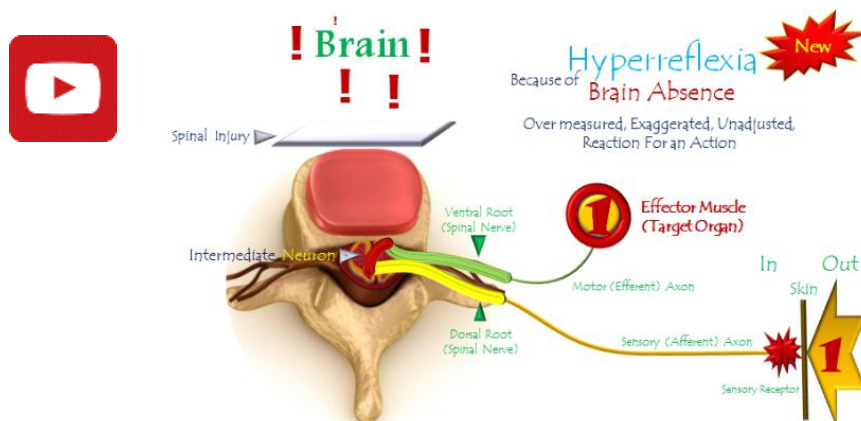
## 2.1. Hyperreflexia (1), Pathophysiology of Hyperactive Hyperreflex

Thanks to data processing in brain, the physiological spinal reflex is always unilateral, well measured, and particularly is coherent with the nature of 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 neurons, and immediately to the effector muscle(s) ensuite. The effector muscle(s) will then get all the power burden of the afferent impulse(s) as it is, and then accordingly contract(s).

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

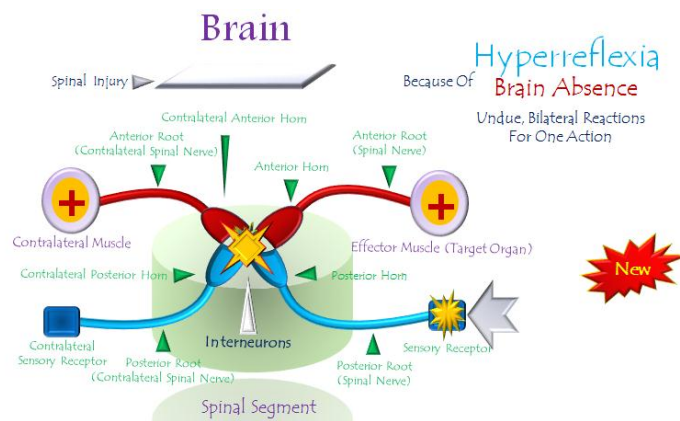
For more details, see the linked video:



## 2.2. Hyperreflexia (2), Pathophysiology of Bilateral- Response Hyperreflex

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

For more details, see the linked video:

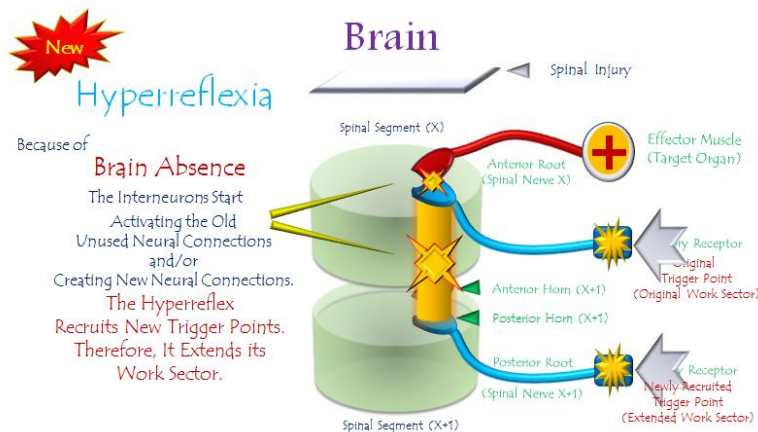


## 2.3. Hyperreflexia (3), Pathophysiology of Extended Hyperreflex

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

In upper motor neuron injuries, and because of 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 a way, the hyperreflex extends its sector of work.

For more details, see the linked video:

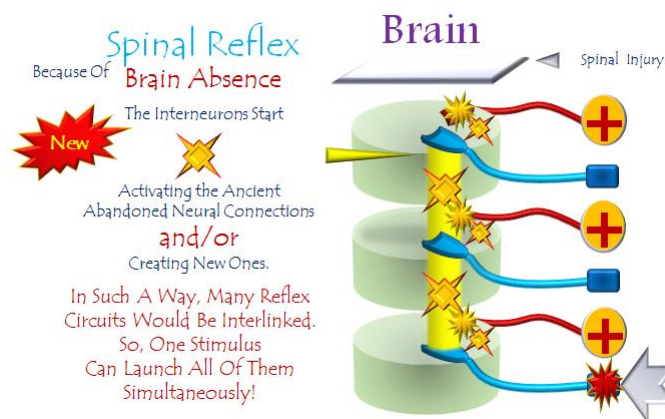


## 2.4. Hyperreflexia (4), Pathophysiology of Multi- Response Hyperreflex

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

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

For more details, see the linked video:



## 3. The Clonus

Clonus is a rhythmic, oscillating, stretch reflex, the cause of which is not totally known; however, it relates to lesions in upper motor neurons and therefore is generally accompanied by 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 such 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 such rhythm.

### 3.1. The Clonus, 1<sup>st</sup> Hypothesis of Pathophysiology

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.

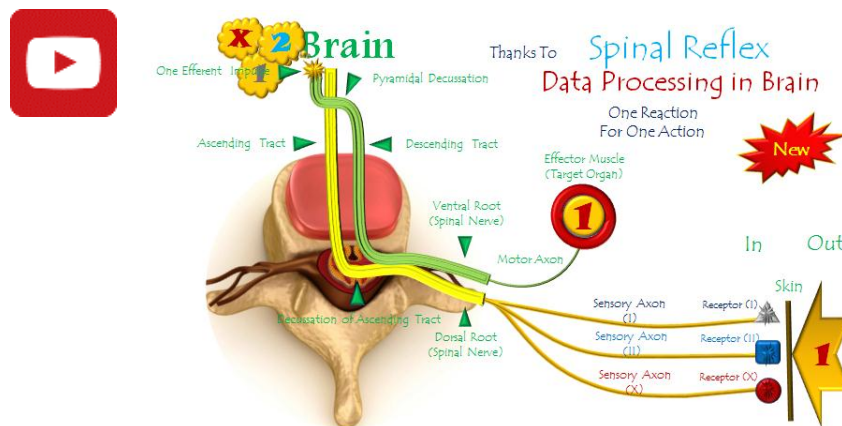
The velocity of neural conduction in some axons will be fast (Sensory Axon I in my video), moderate in others (Sensory Axon II in my video), and slow in the rest (Sensory Axon X in my video).



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 a way, one stimulus could have just one single adjusted response.

However, in upper neuron injuries, things are no longer the same. The brain function of processing the afferent data is no more functional. Moreover, the subsequent afferent impulses will be directly deviated toward the effector muscle(s) (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 could then provoke (X) reflexive responses.

For more details, see the linked video:



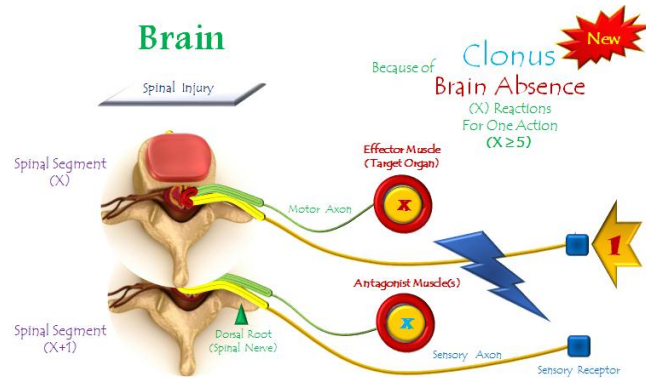
### 3.2. The Clonus, 2<sup>nd</sup> Hypothesis of Pathophysiology

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

In turn, the hyperactive Tibial contraction will activate the receptors of its antagonist muscles (i.e. the Gastrocnemius & Sole muscles). 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.

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

For more details, see the linked video:



.....  
**One can also read:**

- [Neural Conduction, Personal View vs. International View \(Innovated\)](#)
-  [Neural Conduction, Action Pressure Waves \(Innovated\)](#)
-  [Neural Conduction, Action Potentials \(Innovated\)](#)
-  [Neural Conduction, Action Electrical Currents \(Innovated\)](#)
-  [The Function of Action Potentials \(Innovated\)](#)
-  [The Three Phases of Neural Conduction](#)
-  [Neural Conduction in the Synapse \(Innovated\)](#)
-  [Sensory Receptors](#)
- [Nodes of Ranvier, the Equalizers \(Innovated\)](#)
-  [Nodes of Ranvier, the Functions \(Innovated\)](#)
-  [Nodes of Ranvier, Function N1 \(Innovated\)](#)
-  [Nodes of Ranvier, Function N2 \(Innovated\)](#)
-  [Nodes of Ranvier, Function N3 \(Innovated\)](#)
- [The Philosophy of Pain, Pain Comes First! \(Innovated\)](#)
- [The Philosophy of Form \(Innovated\)](#)
- [Spinal Injury, Pathophysiology of Spinal Shock, Pathophysiology of Hyperreflexia](#)
-  [Spinal Shock \(Innovated\)](#)
-  [The Clonus \(Innovated\)](#)
-  [Hyperactivity Hyperreflexia \(Innovated\)](#)
-  [Hyperreflexia, Extended Sector of Reflex](#)
-  [Hyperreflexia, Bilateral Responses](#)
-  [Hyperreflexia, Multiple Responses](#)

- *Nerve Conduction Study, Wrong Hypothesis is the Origin of Misinterpretation (Innovated)*
-  *Wallerian Degeneration (Innovated)*
-  *Neural Regeneration (Innovated)*
- *Wallerian Degeneration Attacks Motor Axons, While Avoids Sensory Axons*
-  *Barr Body, the Whole Story (Innovated)*
-  *Boy or Girl, Mother Decides!*
-  *Adam's Rib and Adam's Apple, Two Faces of one Sin*
-  *The Black Hole is a (the) Falling Star?*
-  *Adam's Rib, could be the Original Sin?*
-  *Pronator Teres Syndrome, Struthers Like Ligament (Innovated)*
-  *Function of Standard Action Potentials & Currents*
-  *Posterior Interosseous Nerve Syndrome*
-  *Spinal Reflex, New Hypothesis of Physiology*
-  *Hyperreflexia, Innovated Pathophysiology*
-  *Clonus, 1<sup>st</sup> Hypothesis of Pathophysiology*
-  *Clonus, 2<sup>nd</sup> 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*

19/6/2020