


The Hyperreflexia

The Pathophysiology of 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.

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 (1).***

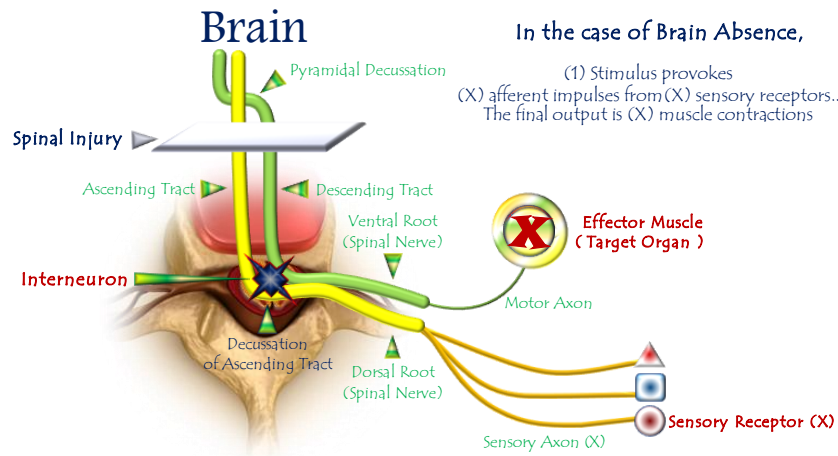



Figure (1)
The Pathophysiology of the Clonus, 1st Hypothesis

[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 hypothesis 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.*

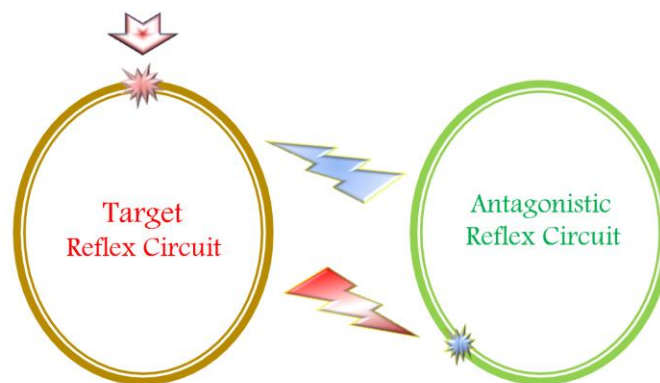
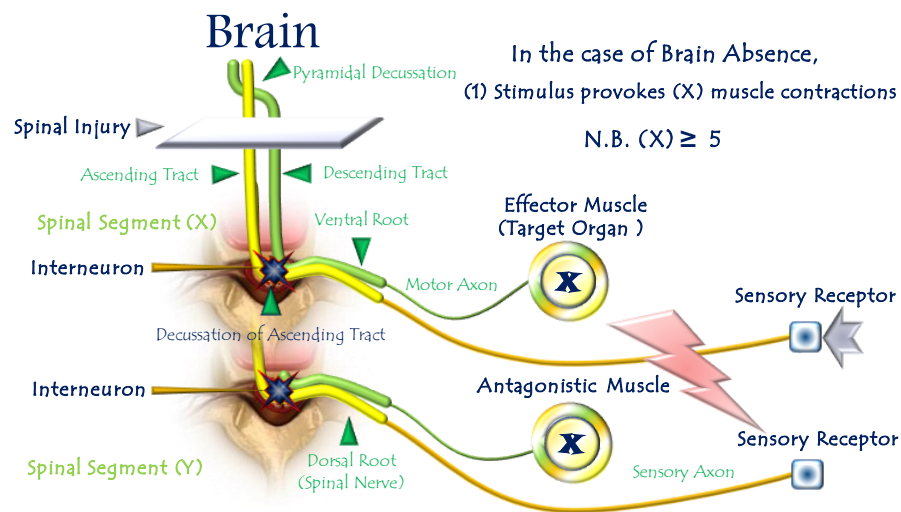
2. The Clonus, 2nd Hypothesis of Pathophysiology

[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 (2)**.



2nd Hypothesis In Other words

Figure (2)
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.*




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*Non- Traumatic Non- Embolic Acute Thrombosis of Radial Artery
(Buerger's Disease)*



Isolated Axillary Tuberculosis Lymphadenitis



Free Para Scapular Flap (FPSF) for Skin Reconstruction



Three Steps of Neural Conduction

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