

The sensitized motoneuron as a possible missing link in the development of myofascial trigger points ?

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Myofascial trigger points (mTp) are confined zones in the musculature of hyperirritable permanently contracted fibers. They are very common in the skeletal musculature, and each shows typical pain patterns.

The mechanisms of the development of mTp are mostly not known in spite of accepted theories and hypotheses.

Most of the theories deal with the vicious circle on the muscular level due to accumulated substances in that region which maintain this contracture .

Another hypothesis as to the origin of mTps could be to consider the motoneuron, which innervates the zone and which through a failure of inhibition has become sensitized, resulting in a spontaneous release of Acetylcholine (Ach) in the synaptic cleft.

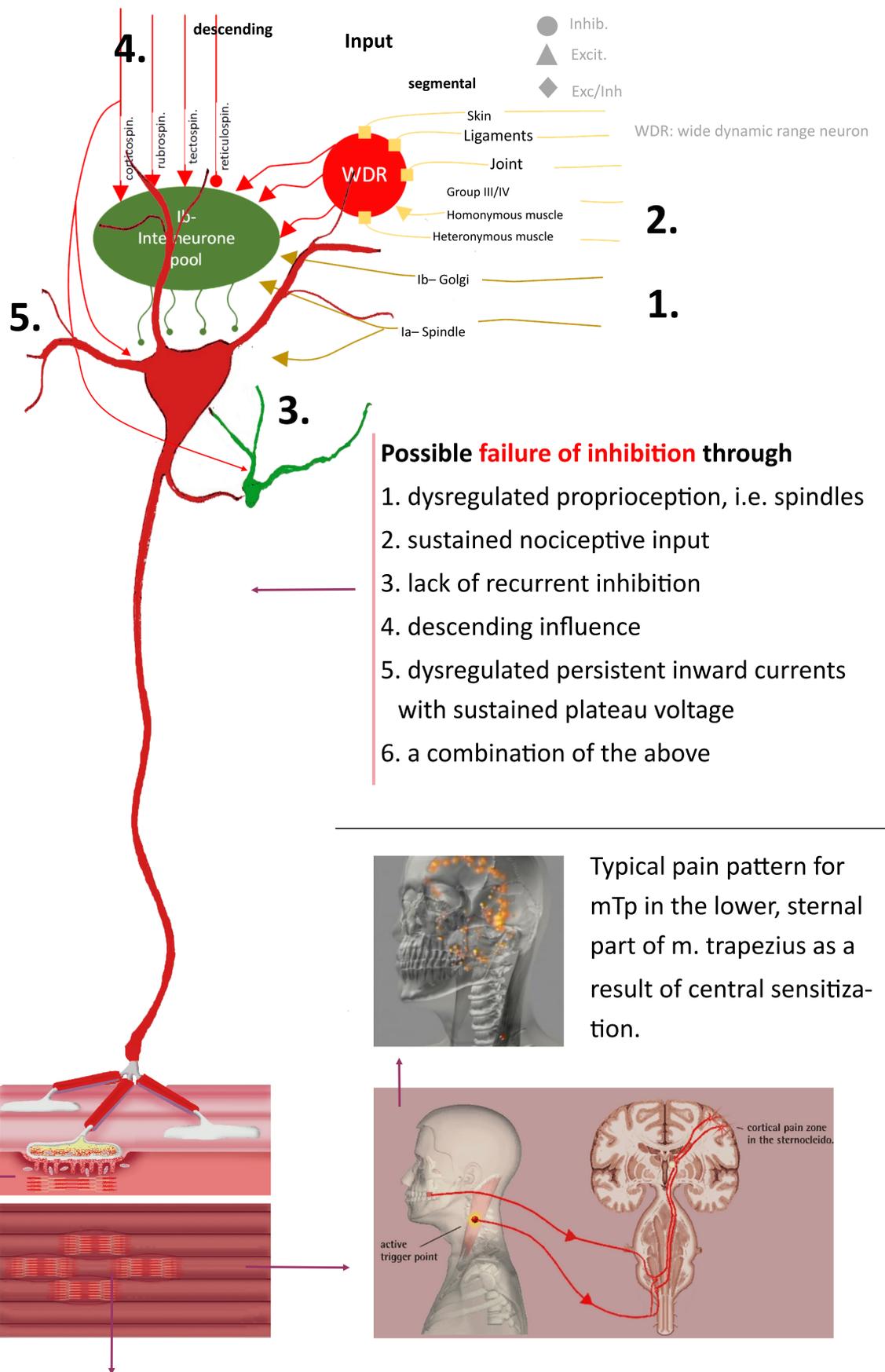
Online research in this direction delivered no results.

The observed sustained release of Ach, typical for motor endplates over mTp, has lead to the commonly used description:

dysfunctional motor endplate

Sustained contractions of single muscle fibers lead to a zone which lacks sufficient oxygen and shows lowered ph and ATP. This in turn causes a malfunction of the Ca^{2+} metabolism and a release of nociceptive substances such as substance P, bradykinin etc., which sensitize the first and eventually the second nociceptive afferent by evoking dormant nociceptive collaterals.

The release of reactive oxygen species (ROS) in the muscle fiber is also blamed for the vicious circle of contractures without the primary influence of the motoneuron in question.



Amongst other effects, **oral splints** alter the output of periodontal sensors and spindles in the involved musculature. Although their efficacy has been proven very well in the treatment of myofascial pain syndrome in dentistry, the neuronal pathways involved are as yet unknown.

The question to be answered is:

Are oral splints effective partly due to inhibitory reflexes on sensitized motoneurons?