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  Are changes in spinal excitability related to functional impairment post stroke?

  Spinal inhibition is an important mechanism to modulate the constant afferent activity. Modulation of the afferent activity is a way to control the spinal reflexes. There are different kinds of inhibition based on the site and pathways involved. Presynaptic inhibition (PSI) is one kind of spinal inhibitory mechanism. It involves an interneuron that produces inhibition of the afferent nerve at its presynaptic terminal. Inputs originating from a variety of sources converge on the interneuron responsible for PSI. As a result, a balance of excitatory and inhibitory inputs originating from afferent and descending pathways is responsible for maintaining an appropriate level of inhibition. Loss of this balance of competing inputs leads to impaired PSI post-stroke. Weakness, which can be characterized using grip force, is one among several manifestations of motor impairment post-stroke. However, the correlation between impaired PSI and grip force is not known. In a recent study we found that in persons post-stroke, PSI was 8% on the paretic (significantly less compared to 21% PSI in controls; $p<0.05$) and 10% on the non-paretic side. Grip force on the paretic side was 60% weaker post-stroke compared to control. A significant positive correlation was revealed between grip force and PSI on the paretic side ($r=0.68$). A significant negative correlation between grip force on the paretic side and PSI on the non-paretic side ($r=-0.61$). It is conceivable that as persons post-stroke undergo rehabilitation, recovery of grip strength may occur concomitantly with recovery of PSI.

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