

Spasticity: Pathophysiology and Neural Control

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Abstract

In discussing spasticity and its pathophysiology we presented evidences for cellular alteration and their neurocontrol. We outline clinical practice of recognizing and describing spasms and spasticity in order to emphasize residual descending influence to the spinal cord. Finally, we provide published evidences that such residual descending influence can be exerted under an attempt of an altered motor task and exert facilitation of excitation and suppression of the central state of the spinal cord reflex activity.

Keywords: spasticity, pathophysiology, neurocontrol

1 Introduction

For a description of spasticity, clinicians are using velocity-dependent increase in resistance of a manually stretched muscle group and increase in tendon jerk reflex irradiation. These two findings are common to different forms of spasticity, i.e. in cerebral hemiplegia, cerebral palsy, brain stem disorders, chronic spinal cord injury. A large number of electrophysiological studies have been performed to examine changes in inhibitory and excitatory spinal cord functions in humans with upper motor neuron dysfunctions and spasticity, i.e. on alpha and gamma motoneuron excitability, presynaptic inhibition acting on Ia afferents, recurrent inhibition, short-latency Ia reciprocal inhibition, autogenic inhibition, postspike afterhyperpolarization, plateau potentials etc. All of these numerous neurophysiological studies in humans demonstrated that there is no single pathophysiological factor responsible for spasticity. Thus the statement of Denny-Brown in 1980 [1] that "Spasticity is a complex disability not identifiable with any single reflex or synaptic transmitter" is still applicable when we are discussing the pathophysiology of spasticity.

Studies of spasticity in experiments with mammals (e.g. mice, rats, cats, monkeys) by mechanically inducing selective lesions of motor cortical structures and descending pathways from brain stem lead to the finding that spasticity arises when the parapyramidal fibres of the inhibitory system are interrupted, either to the cortico-reticular fibres above the level of the medulla (cortex, corona radiata, internal capsule) or to dorsal reticular tract in the spinal cord. These experimental studies on animals lead to the assumption that, also in humans, muscle tone is controlled by the inhibitory dorsal reticular tract and the facilitatory medial reticulospinal and vestibulospinal tracts and when misbalanced, e.g. due to spinal cord, brain stem, brain hemisphere lesion, can cause muscle hypertonia.

2 Spasticity and Spasms

When the spinal cord is deprived of supraspinal control, there is a correspondingly large increase in activity of the spinal motor neurons. This is manifested as spasms in the form of prolonged, uncontrolled, excessive contractions of skeletal muscles and as pathological responses to all forms of stimulation: tactile, noxious and proprioceptive. This excessive activity interferes with basic reflexes organized at segmental levels of the spinal cord. As this activity continues, it takes an abnormally long time for one movement to be replaced by another. Motor responses become limited in their variety of stereotyped movements and local signs of responses are lost.

Spasms are described in clinical practice as involuntary movements. However clinical neurophysiological evaluation with surface polyelectromyography (sPEMG) in people suffering from those involuntary movements reveals two important characteristics: i) spasms have rather constant features of motor unit activity and ii) those specific distribution of motor unit activity of limb and trunk

muscles usually can be initiated and stopped by a volitional attempt, a motor task, that involves activity of the muscles involved in the spasms. However, volitional effort for initiation of spasms or suppression of it is different when compared with the volitional effort with intact nervous system. It is necessary to be aware of those differences and to spend some time to learn how to learn the appropriate task.

Figure 1 and 2 illustrate above description that spasms can be volitionally initiated and suppressed. In figure 1 we are showing sPEMG recordings in three subjects with three different severities of spinal cord injury and resulting spasticity. Figure 1A shows motor unit activity of ankle dorsal and plantar flexors during volitional motor task with some co-activation in the ipsilateral quadriceps and adductor muscle groups. There is no activity in the contralateral lower limb muscle groups. The volitional effort of the Jendrassik manoeuvre did not have any effect on the activity of motor units. Motor unit activity during selective volitional motor tasks and absence of activity during Jendrassik maneuver is characteristic for neurological conditions when spinal cord after injury recovered all descending functions to the lumbar cord.

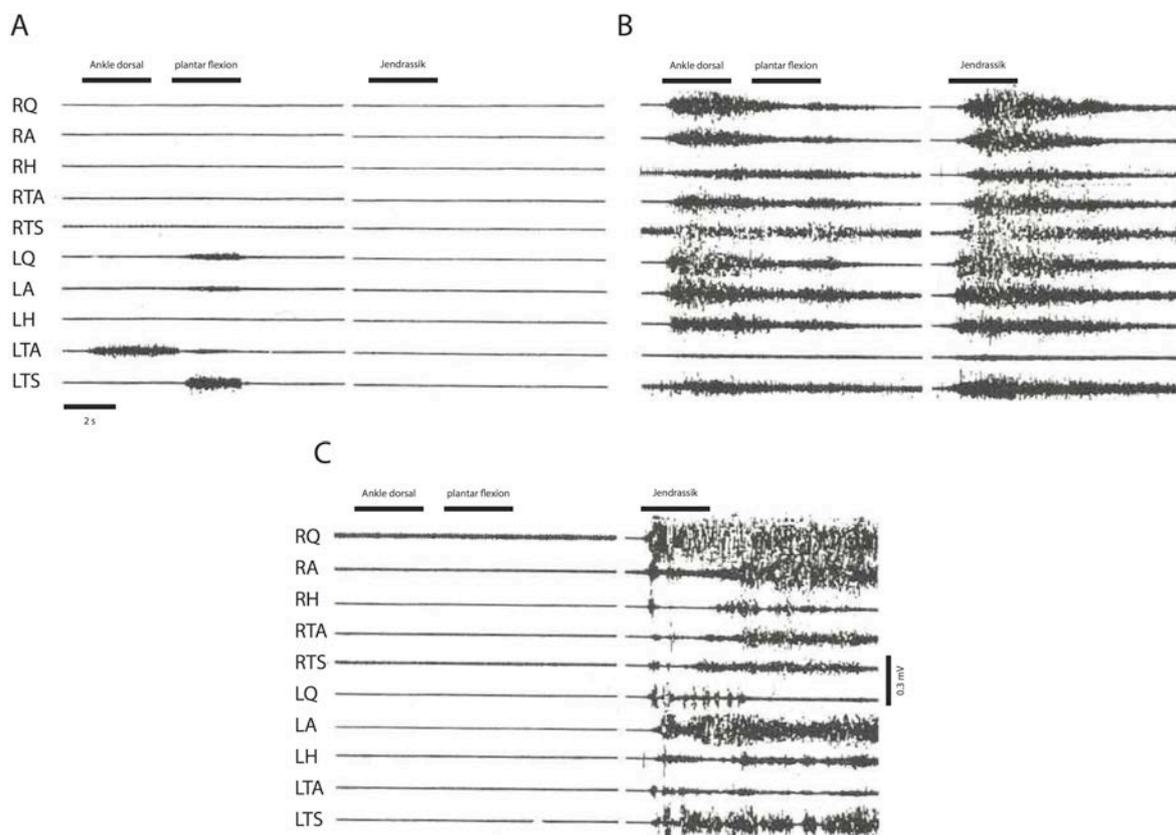


Figure 1 Surface polyelectromyographic recording of the response to ankle dorsal and plantar flexion and Jendrassik manoeuvre in three spinal cord injury subjects with different degrees of preserved brain control through the lesion (increasing severity of the injury from A to C). R: right, L: left, Q: quadriceps, A: adductor, H: hamstring, TA: triceps surae, TS: tibialis anterior (adapted from Dimitrijevic et al. [2]).

Figure 1B illustrates a characteristic finding in a person after chronic thoracic spinal cord injury with preservation of some residual but non-selective, diffuse motor unit activity during volitional motor task of ankle plantar and dorsal flexion of right limb. Excessive activity also on the ipsi- and contralateral lower limb can be seen. Moreover, Jendrassik manoeuvre in the contrast to the previous subject is effectively activating all muscle groups of both limbs. This means that volitional motor task and volitional reinforcement manoeuvre are actually capable to elicit spasms.

Figure 1C illustrates the other extreme of the elicited motor activity in response to the Jendrassik manoeuvre. Actually the Jendrassik manoeuvre is another kind of volitional motor task. Thus, in this subject, spasms can be activated by effort of motor task of the upper limbs and resulting in motor unit

activity of lower limbs. In the same time the selective volitional motor task of the paralyzed lower limbs has no effect.

Neurophysiological evidences for partial preservation of excitatory inflow to the lumbar spinal cord are asking for exploration of residual suppression capabilities of the brain to the reflex activity below the level of spinal cord injury. This aspect has been examined in 50 chronic spinal cord injury patients by testing amount of EMG activity of muscles involved in plantar withdrawal reflex [3].

Figure 2 shows an example of a sPEMG recording in one clinically complete subject and shows a significant suppression of muscle group activity of quadriceps, hamstring, tibialis anterior and triceps surae when they have been asked to suppress withdrawal plantar stimulation response. This finding was present in half of the studied subjects. We shall keep in mind that all of these subjects did not have any ability for volitional movement as well as for sensation. Therefore these measurements are showing that withdrawal reflex movement, which resembles flexor spasms of the lower limb, can be suppressed with volitional effort even in subjects with clinically complete spinal cord injury. Naturally again it is important to be aware that the quality of perception of motor task for the subject who is performing suppression is very different from one where the central nervous system is intact and has to be learned.

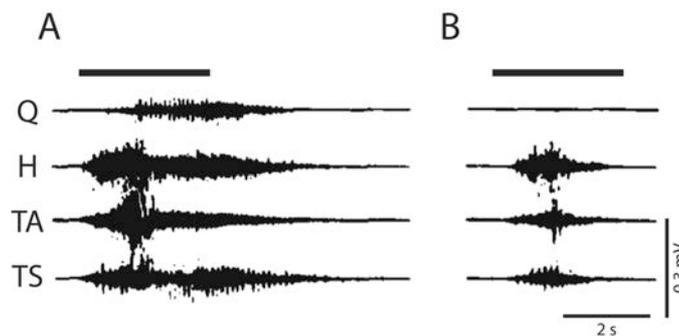


Figure 2 Plantar reflex response of a clinically complete spinal cord injury patient. A: plantar stimulation with no instruction to the patient. B: plantar stimulation with asking the patient to suppress the response. Q: quadriceps, H: hamstring, TA: tibialis anterior, TS: triceps surae (adapted from Cioni et al. [3]).

3 Spinal Interneuronal System

The interneuronal network of mammals has a greater degree of flexibility than often assumed although many textbooks continue to describe spinal reflexes as being highly stereotyped [4]. Cortico-bulbospinal pathways are ending within spinal cord gray matter, where their axons connect with dendrites and axons of interneurons in somatotopic organization together with afferents from peripheral neurons and their primary, secondary and tertiary afferents. Individual spinal cord interneurons can maintain widespread connections with a variety of motoneurons across a large portion of the spinal cord [5,6].

The distribution pattern of descending system terminals within the dorsolateral and ventromedial parts of the intermediate zone of spinal interneurons has been extensively studied [7]. Different sets of interneurons in distinct parts of the intermediate zone possess the same characteristics as the descending pathways, which terminate on them. There is definite evidence that the spinal interneuronal population is not separated in two subgroups, one involved in spinal reflex activity and another in supraspinal-spinal interaction, but rather is a dynamic entity that integrates many functions in order to produce harmonious movements. The premotor neural circuitry forms an "integration center" that is the last link of numerous peripheral and central nervous system pathways [8]. Cortical, subcortical, brain stem, medullar and spinal cord lesion of descending tracts will induce degeneration and corresponding interneurons will be released from brain control. In a case where all descending axons are injured (as it is the condition with accidental traumatic spinal cord injury with complete separation between rostral and caudal portion of the spinal cord) the spinal interneurons will maintain integrity with peripheral afferents. Under such a circumstance, the spinal interneuronal network can become "generator of spasticity". Is it necessary for the development of the "spinal network spasticity generator" to have structural changes by sprouting of primary sensory peripheral fibres to the sites of

degenerated descending axons [9] or is the spinal interneuronal system sufficiently multifunctional and able to reconfigure? We don't know for sure the answer to this question, but so far, neurophysiological research in spastic spinal cord injury people supports the idea that the spinal network has a dominant role in generating features of spasticity [10].

4 Spasticity and Spinal Cord Injury

When we studied spasticity in the complete spinal cord injury people, findings were the following:

1) Single stimulus, manually induced by "natural" proprioceptive or exteroceptive stimulus elicit responses of continued motor unit activity throughout the duration of movement usually showing increases and decreases of motor unit activity simultaneously with that of the antagonist [11].

2) Single tendon taps can cause considerable activity in muscles other than the one, which the tap has been stretched. Silent periods occur in muscles other than the one tapped. The muscles activated during the first 100 ms following the tap on a tendon are activated according to the normal pattern of reciprocal innervation. The spread of activity after the first 100 ms is due to afterdischarge of the motor units of the tapped muscle and of many other muscles [12].

3) Repetitive stimuli of lower frequency and repetitive responses: 1-6 Hz of nociceptive skin or stretch stimulus can induce a sustain feature after an initial short period of progressive increase of the responses followed by period of progressive decrease of the response until full habituation of cutaneo-muscular or proprioceptive reflexes is established. At that stage repetitive and regular input can reconfigure the interneuronal network and set up gate control of the input by presynaptic inhibition [13,14].

4) Repetitive posterior root stimulation of "higher" frequencies (up to 120 Hz): We have found that in complete and incomplete spastic spinal cord spasticity can be suppressed or increased depending on frequency, amplitude and site of stimulation [15]. Furthermore, we have shown how a particular window of frequency of dorsal roots of upper lumbar cord segments can elicit extension or stepping-like movements in otherwise complete and spastic spinal cord injury people [16,17].

5 Remarks

We learned from the short review that in the absence of supra-segmental axons within spinal interneuronal network it is possible to demonstrate alternative reflex pathways, interaction between various spinal neuron populations, reconfiguration and adjustment of the operation of the interneuronal network. Moreover, the feature of spasticity in people with absent cerebral control of the spinal cord network depends also on the profile of the segmental, peripheral input: i) single stimulus, ii) repetitive stimuli of low frequency and iii) higher frequencies. Described results of our studies of spasticity by application of external control of the afferent inputs and their content and profile highly suggest that spasticity is a result of the interaction between peripheral afferent input, and reduced, altered, central input to the spinal interneuronal systems. Thus our hypothesis is that different types and patterns of peripheral and/or central afferent inputs cause altered functional configuration of the spinal interneuronal network, which become generators of spasticity.

Here the presentation of studies on spasticity in the incomplete spinal cord injury people and others spastic conditions, like hemiplegia, cerebral palsy, have been omitted. However, basic functional properties of spinal interneuronal system there are not changed and motor output expression is a result of altered interaction between suprasegmental and segmental interneuronal activity.

6 Definition of Spasticity

Spasticity is neurological condition manifested by impaired control of muscle tone and movement due to altered descending brain input to the spinal cord gray matter, spinal interneuronal system. The reduced brain control capacity of the cord by neurological disorders and intact segmental peripheral input transforms the spinal interneuronal network activity to the generator of exaggerated muscle tone, lower threshold of stretch reflexes, their increased gain, widening trigger area for local cutaneo-muscular withdrawal reflex responses and from reduced to absent voluntary movement, up to involuntary movements, spasms.

7 Conclusion

Spasticity and spasms in the neurological conditions of spinal cord or head injury, multiple sclerosis, stroke, cerebral palsy or other conditions with upper motor neuron dysfunctions are a cause of disability and in severe forms cause in addition to impairment of motor control also altered sensation and pain. There are quite a few modalities for control of spasticity, from physical, neurophysiological, pharmacological, chemical, functional neurosurgical to neurobiological under development. However, their effectiveness for modification of muscle hypertonia and involuntary movement have often side effects that diminish the desire for their application. A side effect free alternative, which can be applied, is the utilization of brain modification of residual motor function to convert involuntary spasms to functional movement under volitional control.

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