Mechanism of Spasticity

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- Abstract -

Spasticity is one of the most common clinical condition that we encounter in neurological practice. It is characterized by increased deep tendon reflexes, clonus, and other associated features. In this review, the basic anatomy of the spinal cord along with descending pathways subserving the muscle tone and reflexes is presented. Historical animal experiments and clinical studies are discussed to help understand the neurophysiology of spasticity.

Key Words: Spasticity, Gamma motor neuron, Muscle spindle neurophysiology

Spasticity is a clinical term. A lexical definition as “a state of hypertonicity, or increase over the normal tone of a muscle, with heightened deep tendon reflexes” is not only pedantic but also insufficient to cover the wide spectrum of clinical phenomena that we appreciate in practice. We readily recognize spasticity when we see it because of the following characteristics:

1. Deep tendon reflexes have a lowered threshold to tap, increased responses, and spreading.
2. Tonic stretch reflexes (ie resistance to passive movement) are similarly affected.
3. Clonus may appear.
4. Stereotypic and idiosyncratic reflexes may appear.

In this brief review, I will discuss the basic neuroanatomy of spinal cord, descending pathways and pathophysiology of spasticity.

In the spinal cord, there are two motor output neurons, alpha and gamma. So called “upper motor neurons” control the excitability of these “final common pathway”. The gamma-spindle loop plays a major role in maintaining and controlling the muscle tone and reflex sensitivity. The loop is under the control of descending and local neural connection.

Alpha motor neuron innervates the extrafusal muscle fibers, the fibers that generates the power of muscle contraction. Gamma motor neuron innervates the intrafusal muscle fibers, which is in the muscle spindle. The components of muscle spindle are intrafusal fibers, sensory endings, and motor axons. Motor axons are from the gamma motor neurons, and are located in the contractile polar regions. Afferent sensory endings are in the noncontractile central area of the muscle spindle.
the intrafusal fibers, and are sensitive to stretch of these fibers. Contraction of intrafusal fibers by gamma motor neurons pulls on the central regions from both ends, and increase the sensitivity of the sensory endings to stretch.

There are three kinds of intrafusal fibers: dynamic nuclear bag, static nuclear bag, and static nuclear chain fibers. A single group Ia afferent fibers innervate all three types of intrafusal fibers, forming a primary ending. A group II afferent fibers innervate the chain and static bag fibers, forming a secondary endings. Two types of efferent axons innervate different intrafusal fibers. Dynamic gamma motor axons innervate only dynamic bag fibers; static gamma motor axons innervate various combinations of chain and static bag fibers. The primary ending is highly sensitive to the velocity of stretch, the dynamic part; where as the secondary ending is sensitive to the degree of stretch, the static part. By controlling the activity of gamma motor neurons, the sensitivity of the stretch reflexes is regulated.

Sherringtonian transection experiments\(^2\) show that there are facilitatory and inhibitory descending pathways mainly centered in the brainstem. When the intercollicular transection is made, a marked rigidity in the antigravity muscles appear. Hyperactive phasic and tonic stretch reflexes, and clonus appear, which are the features of spasticity. This model is drastically different from the clinical reality, however, provides the basic understanding of spasticity. Complete spinal transection abolishes ipsilateral decerebrate rigidity, showing that the rigidity depends on intact afferent fibers. When cutaneous branches of peripheral nerves were cut, decerebrate rigidity persisted. When afferent fibers from muscle were cut, rigidity was abolished. He concluded that muscle receptors are responsible for decerebrate rigidity, and proposed that muscle spindles are the receptors most likely involved.

Classically descending motor neuron systems are divided into two: the pyramidal and the extrapyramidal system. The pyramidal system crosses the midline at the medullary level distal to the vestibular nucleus. The extrapyramidal system crosses just caudal to the red nucleus. Hemisection of the brain stem at the level of the pons rostral to the decussation of the pyramidal tract abolishes the rigidity on the ipsilateral side, showing that the extrapyramidal system supports decerebrate rigidity. Pyramidal transection or ablation of the motor cortex, the origin of the pyramidal tract produces flaccid paralysis, again showing that the extrapyramidal system, not the pyramidal system is involved in rigidity.

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Ablation of pericruciate cortex causes a transient rigidity with motor signs resembling classical decerebrate rigidity. Electrical stimulation of certain brain areas in this model augmented the rigidity. Such areas were called facilitatory areas. Electrical stimulation of other areas decreased or abolished the decerebrate rigidity. These areas were called suppressor areas.

Facilitatory areas exert their excitatory effects on the spinal cord directly by descending pathways such as the vestibulospinal tract or the rubrospinal tract, or indirectly by way of the lateral reticular formation (ie the lateral reticulo-spinal tract). The lateral reticular formation is a pencil-shaped network of cells extending from the medulla into the thalamus. Input from this formation is derived from most of the sensory system. The cells of this nucleus is inherently active, thereby providing a continual facilitation to the lower motor neurons. Suppressor areas exert inhibitory influence by way of medial reticular formation. The medial reticular neurons are not tonically active. They must be driven by impulses from the suppressor areas if they are to exert their inhibitory effects on the lower
motor neurons. Studies using these models provided insights into the brain organization of spasticity in man. In clinical reality, spasticity has more diverse pictures than what Sherringtonian animals show. The spinal neurons are interconnected in such a way that various levels of reflexes and voluntary movements are possible, both of which are under the descending fractionated control. When the spinal cord is transected, the caudal spinal cord is in an autonomous state and inherent reflex movements appear. The examples of built-in reflex movements are ipsilateral flexion and contralateral extension movements. These stereotypic movements appear with any stimulation. However, when the lesion is incomplete, which is a clinical reality, diverse idiosyncratic movements appear. In spastic paraparesis, stimulation of the affected part or volitional attempts to move results in activation of most of the muscles simultaneously in agonists and antagonists. Reciprocal inhibition is absent in most of the time. The intended movement may result in an opposite movement by disturbed reciprocal inhibition and spreading to other muscles. Electrophysiologic study of phasic stretch reflexes in spastic paraparesis shows that reflex latency time is normal. However, there are larger amplitude spikes, multiple rather than single spikes, after-discharge of motor units, and spreading of the reflex responses.

Passive stretching of muscles in normal person does not activate any muscles. In spasticity, activity occurs in the muscle stretched and spread to other muscles. Noxious cutaneous stimulation produced certain patterns such as cyclic response, flexion reflex, idiosyncratic response, excitatory or inhibitory later responses.

Positioning of limb determined the response, indicating the importance of afferent input. Habituation, dishabituation, and sensitization of the reflexes can be produced by changing multiple stimulation paradigm, again indicating the importance of afferent input and dynamic nature of spinal interconnection.

References: