Review Article

The stretch reflex and the contributions of C David Marsden

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Abstract

The stretch reflex or myotatic reflex refers to the contraction of a muscle in response to its passive stretching by increasing its contractility as long as the stretch is within physiological limits. For ages, it was thought that the stretch reflex was of short latency and it was synonymous with the tendon reflex, subserving the same spinal reflex arc. However, disparities in the status of the two reflexes in certain clinical situations led Marsden and his collaborators to carry out a series of experiments that helped to establish that the two reflexes had different pathways. That the two reflexes are dissociated has been proved by the fact that the stretch reflex and the tendon reflex, elicited by stimulation of the same muscle, have different latencies, that of the stretch reflex being considerably longer. They hypothesized that the stretch reflex had a transcortical course before it reached the spinal motor neurons for final firing. Additionally, the phenomenon of stimulus-sensitive cortical myoclonus lent further evidence to the presence of the transcortical loop where the EEG correlate preceded the EMG discharge. This concept has been worked out by later neurologists in great detail , and the general consensus is that indeed, the stretch reflex is endowed with a conspicuous transcortical component.

Key Words

C David Marsden, stretch reflex, tendon reflex, transcortical loop

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Introduction

The stretch reflex or myotatic reflex refers to the contraction of a muscle in response to its passive stretching. When a muscle is stretched, the stretch reflex regulates the length of the muscle automatically by increasing its contractility as long as the stretch is within the physiological limits. When a muscle lengthens, the muscle spindle located inside the muscle is stretched, and the rate of neural firing of muscle spindle afferents increases. This augments alpha motor neuron activity in the anterior horn cell pool, causing the muscle fibers to contract and therefore resist the stretching. Another subset of neurons then direct the antagonistic muscles to relax by the mechanism of reciprocal inhibition and in this way the entire reflex process functions to maintain the muscle at a constant length. Gamma motor neurons regulate how sensitive the stretch reflex is by tightening or relaxing the fibers within the spindle. In the early 1950s, John Eccles, a pupil of Sherrington and the Nobel Laureate in 1963, and his colleagues used the stretch reflex as the model to study the physiology of synaptic transmission in the peripheral nervous system. On passing electrical current into the sensory neuron in the quadriceps femoris, the motor neuron innervating the muscle produced a small excitatory postsynaptic potential (EPSP) and while passing the current through the hamstring muscles, the

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antagonist for the quadriceps, an inhibitory postsynaptic potential (IPSP) was recorded. The sum of several EPSPs from multiple sensory neurons converging onto the motor neurons caused them to fire, thus contracting the quadriceps. On the other hand, IPSPs subtract from this sum of EPSPs, preventing the motor neuron from excessive and uncontrolled firing.[1-4] Eccles and Lundberg and others further showed that Ia or spindle afferents with annulospiral endings subserve both postural reflexes and coordination of movements while Ib afferents from Golgi tendon organs provide protection against excessive tension by autogenic inhibition and initiating the flexion of the antagonists.[4-6]

Figure 1 shall explain the anatomico-physiological basis of the alpha-gamma collaboration in the mediation of the stretch reflex.

The stretch reflex has been for ages, thought to be a spinal reflex of short latency. In 1924, Liddell and Sherrington demonstrated the tonic response to stretch in the decerebrate cats and named it the "*tonic reflex*." In addition, Sherrington wrote that "*there could be little doubt that the knee jerk, a reaction, long familiar to the physician, is a fractional manifestation of it… The physician in testing the knee jerk is in fact, testing the stretch reflex of an anti-gravity muscle*,"[7,8] and Denny-Brown and Liddell adduced enough evidence to suggest that the stretch reflex is a spinal process following their experiments on "*spinal*" dogs.[9] An extensive account of the spinal cord and the mediation of reflex through it can be obtained from the masterly treatise, Reflex Activity of the Spinal Cord, compiled by Creed *et al*. in 1938.[10]

In 1953, Merton, the physiologist from Cambridge, proposed a servo control model for the generation of movement which was based on the stretch reflex. He felt that the muscle spindles and the γ-motor neuron system were an integral part of the servo mechanism controlling the length of the muscle and suggested that the reflex helped control posture by increasing the activation of the muscle in direct proportion to the degree of its stretch.[11,12] Merton proposed that the whole system worked like the servo system on a car steering. If the brain wished to contract a muscle to a certain length, it commanded the small sense organs in the muscles to react as if they had been already stretched to the intended position. The result would be reflex contraction of the muscle in question, and this meant that the nervous system only needed to specify the end position of any movement. This was the first attempt to explain how the nervous system might achieve accurate control of movements. Merton's initial experiments were designed to investigate how the sensory information from muscle spindles was used in normal voluntary movement as opposed to the rather unnatural example of the tendon jerk.[11]

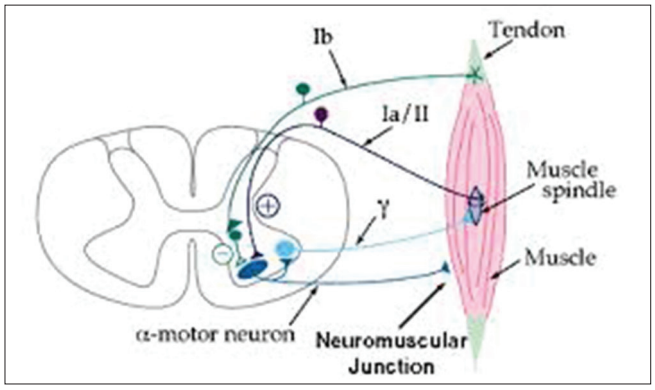

Figure 1: Schematic diagram explaining the anatomico-physiological basis of the alpha-gamma collaboration in the mediation of the stretch reflex. Source: www.skyblue.com

For a long time, it was believed that the stretch reflex evoked by passive stretching of a muscle and the tendon reflex elicited by tapping the identical muscle tendon were identical and that both were spinal in character, subserving the same spinal reflex arc. However, disparities in the status of the two reflexes in certain clinical condition led Marsden, Merton and Morton, a neurologist, a physiologist, and an engineer, respectively, often referred to as the 3Ms from the National Hospital for Nervous Diseases, Queen Square, London, to carry out a series of experiments in the late 1960s and early 1970s, that helped to establish that the two reflexes had different anatomical pathways, and therefore, they were not truly the same.[13] The latency of the tendon jerk is extremely short, and there are reasons to believe that these are mediated at the level of the spinal cord. However, the view harbored over a long period of time was that the stretch reflexes were also mediated by this simple spinal arc. If this hypothesis is treated as acceptable, then it follows that in diseased states, the tendon jerk and the response to passive stretching should reveal identical abnormal response.[13] Marsden *et al*. argued that since the two reflexes are dissociated in certain clinical conditions, there is a strong possibility that they are mediated by two different pathways.[13] Mathews wrote cogently in the regard, "*The essential thing to be kept in mind in thinking about the tonic component of the stretch reflex is that it represents a steady motor output in response to a steady barrage of afferent input. This allows for neural integrative mechanisms of a far higher order of complexity than one can hope to find displayed in the tendon jerk resulting from a single synchronous volley*," and Ragnar Granit, the Nobel Laureate in 1967, fervently held the identical view.[14,15]

To prove their hypothesis, the team of 3Ms devised a simple, yet elegant experiment where Marsden advanced his right flexor pollicis longus muscle for electromyographic studies. The instrument, an electric motor with low inertia, was devised by Morton while the entire experiment was supervised by Merton.[16,17] The proximal interphalangeal joint of the thumb was moved at the physiological rate of 200°/s, and 8–16 trials were taken which were averaged in the computer. The flexed thumb, already contracting under the influence of the flexor pollicis longus, was extended and thus the stretch reflex on extending the thumb was readily studied. The team observed that there were multiple components for the reflex, and the latency for such a stretch reflex, approximately 45 ms, was too long for a pure spinal reflex, and this was nearly double for that of a spinal tendon reflex, which was in the range of 22 ms elicited by tapping the thumb with a tendon hammer and recorded through the same set of electrodes. Importantly, such results were already provided by Hammond in their study on the stretch reflex in the biceps muscle in 1956, where Merton was a coinvestigator.[18,19]

In the 1980s, Deuschl and Lücking proposed that the long-latency reflex (LLR) from hand muscles (known as LLR II)

had two other components, namely LLR I and LLR III, which were elicited by stimulation of the median nerve and recorded from the thenar muscles. They further proposed that LLR II was mediated by Group Ia muscle afferents. LLR I and LLR III are rarely elicited in normal subjects, but they have some significance in disease states. Enhanced spinal tendon reflex and reduced LLR I are frequently observed in Parkinson's disease, essential tremor, and reflex myoclonus whereas LLR II is often absent in Huntington's disease and some focal cerebral lesions. Delayed or absent latencies of the LLR II have been described in multiple sclerosis whereas enhanced LLR III may occur in cerebellar diseases.[20-23]

The Transcortical Hypothesis

From the experiments conducted and gathering useful information from previous works, Marsden *et al*. questioned whether the stretch reflex had a cortical component where on passively stretching a muscle, the afferent impulse travelled up the spinal cord to the motor cortex and returned to the spinal efferent neurons to result in a separate reflex of long latency. This possibility was first mooted by CG Philips in 1969 from his extensive physiological works on the baboon's hand, and he suggested that the spinal stretch reflex "*had been overlaid in the course of evolution by some transcortical circuit*," and he coined the term "*transcortical stretch reflex*."[24] In actual practice, such an afferent pathway had been traced in the same animal travelling to the depths of the central sulcus in proximity to the motor area.[25] Therefore, the efferent limb for the stretch reflex is formed by the fast corticospinal fibers which end monosynaptically in the spinal motor neuron pool, and Mathews stated emphatically that new experiments have eliminated the alternatives, leaving the transcortical hypothesis in command of the field.[26,27] Similar results, indicating the operation of the long-latency transcortical pathway for the human thumb reflexes, have also been reproduced by Capaday *et al*., by the study of transcranial magnetic stimulation and others as well.[27,28] It appeals to reason that the disparity between the stretch reflex and the tendon reflex would be more pronounced for muscles whose motor neurons are far away from motor cortex in the brain, and in their subsequent experiments, Marsden *et al*. lent his long flexor of the great toe and the masseter muscle for studies. The latency for the great toe turned out to be 75 ms and that for the masseter, 13 ms. However, the latency of the ankle tendon jerk was about 37 ms and that of the jaw jerk, 8 ms only.[13] This study almost incontrovertibly suggested that two different pathways existed for the stretch reflex and the tendon reflex and that in the former, almost certainly a transcortical contribution operates in its genesis. In recognition of the works of the 3Ms, this pathway has been named M1, M2, and M3 long-latency loop by Tatton and Lee in their subsequent works. M1 represents the brief spinal reflex arc, and M2 and M3 are the long ones with a transcortical component, M3 being mediated by the cerebellum.[29,30]

The issue whether stretch reflexes were abolished in lesions of the dorsal column, the afferent pathway for the reflex, was resolved when in one subject with a demyelinating plaque at the level of the C2 spinal segment, well above the segment subserving the reflex for the flexor pollicis longus, the stretch reflex was absent though all the upper limb jerks were within normal limits. The contralateral upper limb was normal in every aspect.[13]

Stimulus Sensitive Myoclonus and Further Vindication of Transcortical Loop

One important evidence that the transcortical loop operates in the genesis of the stretch reflex is the phenomenon of stimulus-sensitive myoclonus or cortical loop reflex myoclonus.[13,31] In 1975, Shibasaki and Kuroiwa observed that spontaneous myoclonus had a cortical component, and Sutton and Mayer and Rosen *et al*. studied focal stimulus-sensitive myoclonus in the early 1970s.[32-34] At around the same time, Marsden *et al*. investigated one subject by extending the flexed thumb and observed myoclonic jerk involving the upper extremity and the shoulder, and the latency was in the range of 50 ms which was equal to the normal stretch reflex for the said muscle. It was felt that the cerebral cortex was involved in its genesis since the latency of the jerk was the same as that of the normal stretch reflex.[13,16,17] In a seminal paper in 1979, Hallett *et al*. classified cortical reflex myoclonus into cortical reflex loop myoclonus where the electroencephalogram correlates are time locked to the (Electromyography) EMG bursts and reticular reflex myoclonus where the cortical spikes are not so.[35-37] In summary, stimulus-sensitive myoclonus can be conceived as a gross overreaction of the motor cortex to an incoming volley of sensory input and as had been suggested by Greenfield; it could be due to the lack of inhibition on the motor cortex from cerebellar Purkinje cells, and postmortem examination revealed substantial loss of these cells.[13] That the myoclonic jerks are of cortical original is also evident from the work of Kugelberg and Widen on a patient who presented with myoclonus in the lower limb. During operation, abnormal spikes were recorded from the contralateral cortical leg area, and focal cortical excision led the amelioration of the symptom. Similar experiments were also carried out earlier by Adrian and Moruzzi in anesthetized animals.[38,39] Later works have observed that apart from stretching of the muscles, there is evidence of transcortical pathways with cutaneous and vibratory stimulation as well.[40,41]

Concluding Remarks

Thus, Marsden *et al*. had the suspicion that the tendon reflex and the stretch reflex travelled through different pathways. Their works paved the way for the understanding of conditions such as stimulus-sensitive myoclonus and the absence of the long-latency loop in some cortical lesions. John Rothwell, a long-time associate of Marsden and currently, Professor of Human Physiology, Institute of Neurology, Queen Square, London, feels that many pathways link the afferent input from muscle spindles with motor neurons, and many of these pathways are within the local spinal segments, with the monosynaptic one being the simplest. Of course, there are multiple other possible connections through interneurons. What was special about the suggestion of the 3Ms was the pathways linking muscle spindle and the motor neuron that traversed the cortex also could participate in the stretch reflex. The 3Ms put spinal and cortical circuits in the same continuum and put forward the concept that the cerebral cortex also participates in the genesis of the stretch reflex (personal communication). This significant addition to the

classical Sherringtonian concept that stretch reflexes were purely spinal in nature is a benchmark in the subsequent study of cortical functions and human neurophysiology.

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