Manual Therapy 15 (2010) 388–393

Contents lists available at ScienceDirect

Manual Therapy

journal homepage: www.elsevier.com/math

Original article

Challenging presumptions: Is reciprocal inhibition truly reciprocal? A study of reciprocal inhibition between knee extensors and flexors in humans

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ARTICLE INFO

Article history:
Received 3 March 2009
Received in revised form
14 December 2009
Accepted 6 March 2010

Keywords:
Reciprocal inhibition
Quadriceps
Hamstrings
H reflex

ABSTRACT

Reciprocal inhibition (RI) between different muscles has been used as an explanation for the effect of some treatments. Consequently, there may be a presumption that RI is bi-directional and equal between every agonist antagonist muscle pair. That is, the strength of RI from agonist to antagonist is equal to that from antagonist to agonist. With this in mind we investigated RI between quadriceps and hamstrings using 2 techniques to explore if a) it is evoked between this agonist antagonist pair and b) if it is equal and opposite in strength. Firstly, electromygraphic (EMG) activity of one muscle was recorded whilst stimulating group Ia afferents from the other. The second approach involved conditioning a reflex evoked in one muscle by stimulating Ia afferents from the other. Using the first approach, short-latency inhibition thought to be RI, was observed more frequently ($p < 0.000$) and was larger ($p < 0.05$) from femoral nerve stimulation to hamstrings than the inhibition evoked in quadriceps by sciatic nerve stimulation. The second approach revealed a similar pattern. RI between quadriceps and hamstrings is not actually reciprocal i.e. not equal in both directions. Our presumptions about the frequency and strength of other pathways between different agonist antagonist pairs need to be assessed.

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1. Introduction

Proprioceptive neuromuscular facilitation (PNF) and muscle energy techniques (MET) are widely used amongst physiotherapists in the management of neuromusculoskeletal disorders. They are generally thought to be a fast and effective way to increase the flexibility of muscles and may work by improving muscular relaxation, thus allowing a greater magnitude of movement during the stretch (Chaitow, 2001). One mechanism, thought to underlie some PNF and MET techniques, is known as reciprocal inhibition (RI) (Chaitow, 2001). RI occurs when agonist Ia afferents are activated; they inhibit the antagonist motoneurone pool using the Ia inhibitory interneurone, thus creating a disynaptic Ia inhibitory pathway.

RI has been extensively studied in animals (Sherrington, 1893, 1897, 1913; Lloyd, 1941; Eccles et al., 1956; Hultborn, 1976; Baldissera et al., 1981) and is particularly potent between the knee extensors and knee flexors in the cat (Eccles and Lundberg, 1958; Lundberg, 1970; Hultborn, 1972). RI has also been studied in humans for example between ankle dorsiflexors and plantarflexors (Kots and Zhukov, 1971; Mizuno et al., 1971; Simoyama and Tanaka, 1974; Tanaka, 1974; Ashby and LaBelle, 1977; Kudina, 1980; Pierrot-Deseilligny et al., 1981; Iles, 1983, 1986; Shindo et al., 1984; Crone et al., 1985, 1987; Crone and Nielsen, 1989; Nielsen and Kagamihara, 1992; Nielsen et al., 1992; Shindo et al., 1995), wrist extensors and flexors (Day et al., 1981, 1983, 1984; Cavallari et al., 1984) and between the elbow extensors and flexors (Katz et al., 1991). Surprisingly however, only one study has investigated RI between knee extensors and flexors in humans (Bayoumi and Ashby, 1989).

Despite the lack of evidence, some clinicians may presume that RI occurs between every agonist/antagonist muscle pair. In addition the term reciprocal implies that a given Ia inhibitory pathway will be matched by similar inhibition in the reverse direction. Indeed, the Oxford English Dictionary defines the term reciprocal as "affecting two parties equally" (Soanes, 2002). Hence, to be truly reciprocal one might assume that the inhibition should not only be bi-directional but also occur with equal frequency and amplitude. Consequently, clinicians might presume that a given strength of inhibition occurs from an agonist to its antagonist to the same degree as from the antagonist to the agonist, irrespective of each muscle's size or functional relationship.

Whilst Bayoumi and Ashby (1989) demonstrated that RI was bi-directional but not equal between the quadriceps and hamstrings (stronger from extensors to flexors than the reverse), other studies exploring RI have not questioned whether RI is bi-directional and

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doi:10.1016/j.math.2010.03.004

equal (Kudina, 1980; Pierrot-Deseilligny et al., 1981; Day et al., 1984; Crone et al., 1987) and hence this is relatively unexplored. Thus the purpose of this investigation is to extend the studies of Bayoumi and Ashby (1989) by using different techniques to explore RI between quadriceps and hamstrings in humans, in order to assess a clinician's presumption that RI is thought to be bi-directional and equal in strength.

2. Methods

With ethical approval from UCL research ethics committee and informed consent, recordings were taken from a group of 14 healthy subjects aged between 21 and 48 years. To examine RI, two experimental paradigms were used.

Eight subjects were comfortably seated in an armchair. Using the difference in amplitude of the inhibition to generate the power and a two tailed test with a confidence level of 5%, the power of using eight subjects is 97.2%. The left leg was positioned upon a stool with the hip in approximately 90° flexion and the knee resting at approximately 10° from full extension. Surface electromyography (EMG) was recorded using adhesive electrodes (Medicotest, Blue sensor electrodes) placed edge to edge with the recording area 3 cm apart and positioned over the distal muscle bellies of the vastus medialis oblique (VMO) and vastus lateralis (VL), positioned 10 cm above the patella medially and laterally, and over the medial and lateral hamstrings (15 cm above the centre of the popliteal fossa medially and laterally). The EMG was amplified (Digitimer NL824) and filtered (Neurolog NL125) with a bandwidth of 30–3 kHz. The data was converted from an analogue to a digital signal at a sampling frequency of 4 kHz (CED 1401) and stored for later analysis by CED Signal software.

Electrical, 1 millisecond (ms) square-wave pulses were delivered ipsilaterally and percutaneously at three second intervals (Digitimer DS7A stimulator) to either the femoral or sciatic nerve. When stimulating the femoral nerve the anode (a gauze-covered metal plate) was positioned over the proximal lateral aspect of the thigh. The cathode (a roving and gauze-covered probe) was used to locate the femoral nerve superficially in the femoral triangle, medial to the femoral artery. The sciatic nerve was stimulated at mid-thigh level between the bellies of biceps femoris and semitendinosus and the anode was positioned just above the gluteal fold. A recording of surface EMG activity of the muscle being stimulated was monitored throughout the recording session to visualise evoked responses and to monitor the degree of background activity. The surface EMG of the antagonist was also monitored using a light biofeedback device. The device was set so that 100% of maximum voluntary contraction (MVC) lit 10 lights. This was established by the researcher applying a manual resistance in the appropriate direction against the leg, whilst the knee was positioned at 90° of knee flexion. The subject was then asked to maintain a stable, weak (30% of MVC, i.e. to light up 3 of the lights of the biofeedback device) contraction throughout the recording period.

The motor threshold (MT) was established by varying the stimulus intensity until a motor response was evoked in 5 out of 10 stimuli. The stimulation intensity was then varied and ranged from 0.8 up to 2 times MT. Following 100 stimuli the EMG from the antagonist was rectified and averaged. The mean activity was established during a 50 ms pre-stimulus time frame. A cursor was placed horizontally in order to mark this mean activity. Inhibitory reflexes were considered to be present if activity dropped below the lower 95% confidence interval for the pre-stimulus mean and lasted for a minimum of 8 ms (Wohlert, 1996). The latency of any reflex response was taken from the first clear deflection from the mean activity (see Fig. 1).

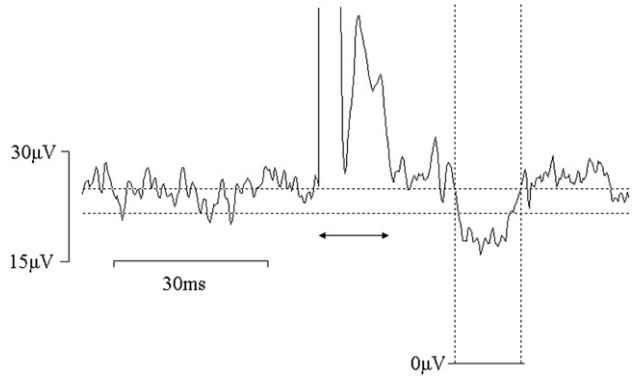

Fig. 1. Rectified and averaged EMG of hamstrings with stimulation of the femoral nerve at 1.5 × motor threshold (MT) constructed from 100 stimuli. The horizontal arrow marks the stimulus artefact. The mean activity was established during a 50 ms pre-stimulus time frame. A horizontal cursor was positioned on this pre-stimulus mean (top horizontal cursor). To establish if a reflex was present, a second horizontal cursor was positioned at the lower 95% confidence interval. If the response dropped below this cursor for 8 ms the response was said to be present. To establish the latency of the reflex, a vertical cursor was then carefully positioned at the point of the first clear deflection from the mean activity. To establish the amplitude of the reflex a second vertical cursor was positioned and marked the return to the level of the mean activity. A total area was defined by the area between the first and second vertical cursors, the top horizontal cursor and a line marking 0 μV. The amplitude of the reflex was then calculated as a proportion of this total area.

The amplitude of the reflex was determined in the following manner. Cursors marked the onset and offset of the rectified reflex. The amplitude of the reflex was calculated as the area of the reflex below the pre-stimulus mean level, as a proportion of the total area of the reflex (see Fig. 1). As the method was repeated on the subjects, the fastest latency of any response from each muscle group, from each individual, was then noted and the mean and standard deviation calculated for these group results. The fastest latency, rather than the average latency for each individual was chosen in order to best reflect the fastest possible latency for any response. The frequency of occurrence and size of any inhibition for each muscle group was also determined and was compared using a Mann–Whitney *U* test and *t*-tests respectively. The level of significance was set where $p = 0.05$.

To help assess whether any evoked response is due to disynaptic inhibition, the latency of the actual response can be compared to an estimation of the latency of disynaptic inhibition between these two muscles. An estimate of the fastest theoretical time for a disynaptic Ia inhibitory pathway can be deduced from the sum of the conduction time of Ia afferents from the site of stimulation to the spinal cord, the central delay and the conduction time from the spinal cord to the thigh muscle. The conduction time of the peripheral pathways can be calculated using knowledge of the conduction distance of each pathway. The appropriate conduction distances were measured from a combination of fourteen cadavers and skeletons. An estimate was made of the position of the site of the stimulation and the distance from this location to the spinal canal was measured. An average was then calculated.

In order to investigate the inhibition that occurred using the first experimental paradigm in greater detail, additional experiments were carried out using eight subjects. This second paradigm is well established within the electrophysiological literature and it is called conditioning a test reflex (Stephens and Yang, 1996; Leonard et al., 2006). As described previously, a low intensity stimulus is delivered to the antagonist's nerve – this is called a conditioning stimulus. This low intensity stimulus

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