

THE GAIN MODULATION OF THE HETERONYMOUS EXCITATION OF QUADRICEPS WITH CHANGES IN POSITION OF THE KNEE AND HIP JOINTS IN HUMANS

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ABSTRACT

Objective: A potent heteronymous excitation of quadriceps motoneurons via common peroneal group I and II afferents has been demonstrated in normal subjects. The aim of this study was to establish the contribution of knee and hip proprioceptors on the modulation of this biphasic facilitation.

Methodology: Single 0.2ms pulses were delivered to common peroneal nerve at the intensity of 2x motor threshold in tibialis anterior. The peak to peak amplitude and area of the excitatory responses were measured in ongoing EMG of voluntarily contracted rectus femoris at different knee and hip joint positions.

Results: The reflex showed a significant inhibition with the knee flexion ($P < 0.0001$) and hip extension ($P < 0.01$).

Conclusion: It was concluded that coordinate activation of ankle flexors and knee extensors could be modulated by the knee and hip joints proprioceptors.

KEY WORDS: Common peroneal nerve, Reflex, Reflex of inhibition, Quadriceps femoris.

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INTRODUCTION

The motor activity in muscles is the result of the interaction between central and peripheral inputs. This can modify the motor activities to meet the demands of the external requirements and can occur at the site of convergence of different pathways on a common interneuron.¹ In this interaction process the peripheral inputs can be modulated that would be mani-

festated as the changes in the reflex gain.² It has been shown that this gain modulation of the reflexes can be produced by the influences of the peripheral inputs as well as the descending inputs. The best known example of this sensori-sensory modulation is the modulation of the H reflex during active and passive movement of the ipsilateral and contralateral leg.³

In man, stimulation of group I & II afferents in the common peroneal nerve (CPN) has been shown to evoke biphasic excitation of quadriceps motoneurons (CPQ reflex),³ with the early phase attributed to non-monosynaptic group I and the late phase to higher threshold group II afferents. It has been suggested that a significant part of the cortical excitation to motoneurons of thigh muscles could be relayed via this lumbar excitatory premotoneurons.^{3,4} The role of this neural pathway in the motor control of quadriceps (Q) would be more prominent when we consider its possible contribution to spasticity and rigidity in hemiplegic⁶ and parkinsonian patients⁷ respectively.

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The concept that the putative CPQ reflex could be modulated by different ascending or descending inputs is important when we realize the potential of trans-joint reflexes to assist in controlling the motor action of the limb as a whole during movement. In this particular case, the functional consequence would be the coordination of knee extensor and ankle flexor contraction required at about the point of heel strike during locomotion.

SUBJECTS AND METHODS

The experiments were carried out on 14 healthy volunteer subjects (mean age 27 ± 4 years, height 177 ± 5 cm and weight 73 ± 9 kg), of whom 5 were female and 9 male. The experiments were approved by the local ethics committee and were undertaken with the understanding and written consent of each subject. The subjects were seated in a chair with the hip at about 100° and the ankle fixed at an angle of approximately 110° . The lower leg was attached to the moving arm, which could be fixed firmly at different angles. Different knee joint positions from 180° to 130° of flexion, every 10 degree, were tested. Two hip joint positions, 110° and 180° (sitting and lying down) were also tested in 10 subjects with the knee and the ankle joints fixed at constant 180° and 110° position respectively.

Surface EMG was recorded continuously for 60 seconds from tibialis anterior (TA) and rectus femoris (RF) of the right leg. The potentials were amplified, band pass filtered between 10 Hz and 3 kHz and digitized at 1 kHz (1401, CED, Cambridge). Thirty pulses of 0.2 ms duration at 0.5 Hz were applied to the CPN at the level of caput fibulae. The threshold current for evoking an M wave in TA was recorded at each knee position and subsequent stimulus intensities were calculated accordingly. The reflex was elicited by the intensity of 2 MT in TA which would activate group I and II afferents.⁸ Subjects were instructed to maintain the level of integrated EMG at 20% of maximum voluntary contractions (MVC) during the test. A monitor was used to provide the subjects with visual feedback.

The averaged EMG of 50 ms pre and 100 ms post-stimulus period was used for extracting the data. The mean potential and 2 standard deviation (SD) of it was calculated for 50 ms pre-stimulus period from the averaged EMG. Only the responses that were beyond the 2SD were considered statistically significant. The size of the responses was measured by both the global p-p amplitude and the area of the twin peak reflexes in non-rectified and rectified averaged EMG respectively. The maximum magnitude of the response recorded was used as a reference for normalization of all the other joint positions.

REPEATED MEASURE ANOVA test was used to test the significance of differences between responses at different knee positions using a SPSS program version 10 for windows. Pairwise comparisons (Bonferoni test) were used to reveal the most effective joint positions. Differences between the means were considered statistically significant at a level of $p < 0.05$.

RESULTS

The effect of the knee joint position: Stimulation of CPN resulted in consistent biphasic excitatory responses in RF with average latency of 29.4 ± 2 ms for the earliest phase and total duration of 15.7 ± 2 ms. The p-p amplitude and area of the CPQ reflex was reduced significantly ($P < 0.0001$, $P < 0.01$) by knee flexion (Fig-1). The pooled data for all subjects (Fig-2) shows an abrupt decrease in the first 10° of

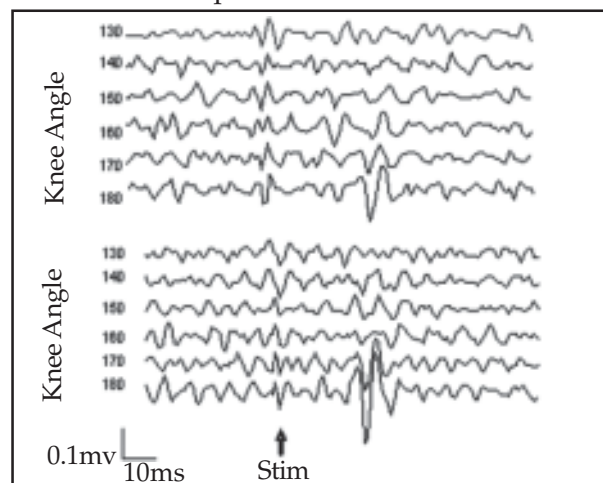


Fig-1: The averaged non-rectified EMG of RF at different knee angles in two volunteers.

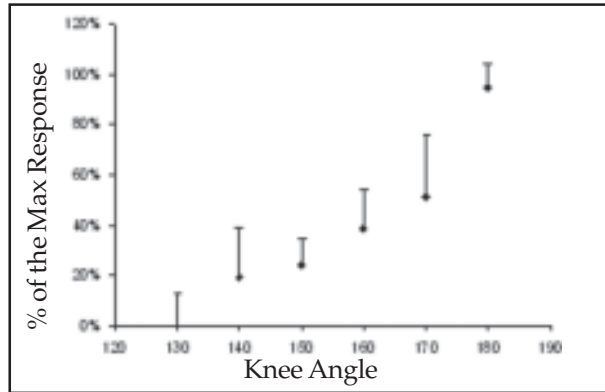


Fig-2: The mean area of CPQ reflex at different knee angles flexion. The differences in the reflex magnitude between 180° and other knee positions were highly significant ($p \leq 0.005-0.0002$). However, the attenuation of the reflex magnitude at other two positions elsewhere in the range was smaller and often not statistically significant. This became significant statistically, if the mean values of every 30° of knee flexed positions were compared.

The pattern of these changes was different among the subjects. Nine out of 14 subjects showed a very abrupt decrease in the size of the response in the first 10-20° of flexion. In these individuals no significant response could be seen in non-rectified EMG after 30° of flexion (Fig-3b). The other five subjects showed gradual attenuation of the reflex towards more flexed positions (Fig-3a). This was mainly started after 10° of flexion.

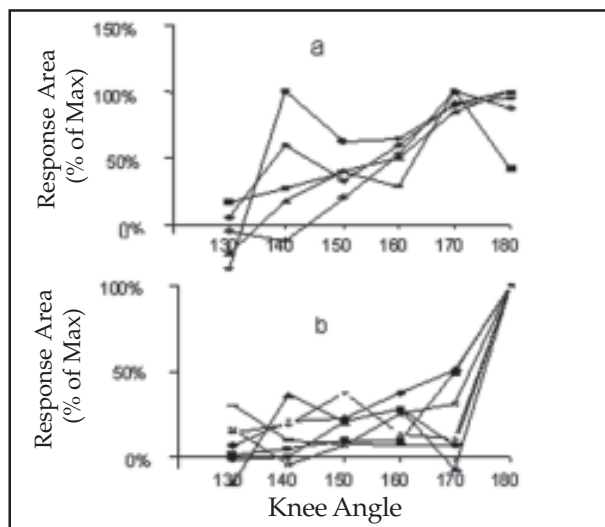


Fig-3: Two different pattern of modulation (a & b) of the CPQ reflex at different knee positions.

The effect of hip joint positions: Analysis of the pooled data showed that when the knee was maintained at full extension, hip extension from 110° to 180° degrees resulted in a significant decrease in the p-p amplitude ($P \leq 0.02$) and the area of the responses ($P \leq 0.008$). Unlike the knee flexion, the hip extension produced different effects among the subjects. Whilst in four subjects a dramatic collapse of the reflex was recorded ($>80\%$), in two subjects no changes were detected and in the rest only a moderate reduction (less than 50%) of the reflex magnitude was detected.

DISCUSSION

The characteristics of the reflex investigated in the present experiments are similar to those described elsewhere.⁹ The Q excitation is significantly modulated by changes in the knee and hip position. The reflex inhibits at flexed positions of the knee and extended position of hip. The position-dependent modulation of the CPQ reflex has not been observed by previous authors.¹⁰ This could be as a result of different method (cycling) that was employed to produce the changes in the knee position. Considering the usual setting for cycling it would be unlikely that the knee and hip were at fully extended position at any point. This is significant given that these results reported here show the steepest modulation over the last few degrees of the range of knee extension.

The gain of a reflex can be altered strongly by the convergence onto its pathway.¹¹ In present experiments, it appears that the inhibition of the CPQ reflex was mainly from some component of the somatosensory receptor array activated as a consequence of changes in the knee position and / or the hip position. Position and movement of the joints can be signaled by the discharges from receptors within the joint, skin or muscle spindles.¹² Any of these could be responsible for the inhibition of the reflex.

The present experiment does not provide any direct evidence about the origin of the afferents responsible for the observed inhibition of the reflex. Nevertheless, the positive information in the literature mainly supports the

stretch receptors as the main source of the position sense.¹² Therefore, the muscle spindles in Q muscles could be considered as the main source for the observed gain modulation in the CPQ reflex during knee flexion and hip extension.

On the basis of this assumption, different patterns of modulation of the CPQ reflex observed following the changes in the position of knee and hip could be attributed to the variation in the mechanical or neurological properties of the Q muscles among the subjects. Monoarticular and biarticular parts of Q stretch at different magnitude during knee flexion and hip extension. Obviously in the monoarticular parts the internal tension will be developed faster and higher during knee flexion and they remain unchanged during hip extension. The biarticular parts however will not develop comparable tension in single joint movements. The different extent of stretch in the mono and bi-articular parts of Q can be the reason for the weaker reactions produced by hip extension.

It is also possible that different strength of the convergence on the reflex pathway could be responsible for these discrepancies.³ It is plausible that strong inhibitory convergence of afferents from monoarticular parts of Q may produce the sudden drop of reflex amplitude at the first degree of knee flexion in the first group. This projection from the monoarticular muscles are probably very weak in the second group and the mild inhibitory convergence from the biarticular part of Q could be responsible for the gradual and milder inhibition of the reflex as the knee is flexed.

The modulation of this excitatory pathway can be of a functional significance when we bear in mind that these two muscles have a synchronized activity during stance phase of gait. Both have their peak of activity shortly after heel strike where the knee joint is extended and the hip is at flexion, the positions that CPQ reflex would be at its maximum excitability and could have a major role to reinforce the Q activity at this period. Marchand-Pauvert and Nielsen¹³ have shown that at 30-60ms after heel strike the CPQ

reflex was at its highest magnitude and led them to the same conclusion. The increased activity of the reflex during standing straight while leaning backward⁹ also indicates a postural task for this reflex pathway to provide coordinate activation of the pretibial and quadriceps muscles. At the end of stance phase however, Q should remain silent to let the knee flexion. At the end of stance phase of gait hip is at extended position and knee flexion would help the limb to unload. The inhibitory effect on the CPQ reflex at this period could facilitate the limb unloading and start of swing phase.

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