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Surface electromyography reveals males have a slower patellar reflex than females

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ABSTRACT

In humans the cross sectional area of spinal motor neurons at L3 is larger in males than in females. Since these contribute to the control of the quadriceps femoris muscle group and are involved in the patellar reflex (PR), gender differences in the PR are expected. We have investigated this possibility using a group of 28 young subjects (14 male and 14 female) aged 20–22 years. The PR was quantified by the muscle compound action potential (MCAP) from the surface electromyogram (sEMG) of the vastus lateralis muscle. We found that the PR latency in females (17 ± 0.19 ms), was significantly ($p < 0.001$) faster than in males (21 ± 0.37 ms). This 4 ms difference in latency could not be ascribed to differences in stature or thigh length. In conclusion, for the age range tested females posses a significantly faster patellar reflex than males. We suggest that the slower PR latency of male subjects may arise in part from their larger α-motorneurons: such that longer integration times are required for the summation of postsynaptic excitation to be sufficient to excite α-motorneurons.

1. Introduction

In humans, it is well established that sexual dimorphism exists within the neuroanatomy of the central and peripheral nervous systems (Morris et al., 2004). Although the best characterised neuroanatomical dimorphisms are those associated with reproduction (Forger and Breedlove, 1986), sexual dimorphism of other motor neuron pools exists. For example, motorneurons in the anterior horn of the spinal cord at levels C5 and L3 from humans have the largest area in males (Yuan et al., 2000). L3 nerves contribute to the femoral nerve, and are involved in the control of the vastus lateralis (VL) and the patellar stretch reflex (PR) (Schubert and Keil, 1968). Consequently, the larger size of these motorneurons in males (Yuan et al., 2000) suggests that their L3 α-motor efferents should possess faster conduction velocities, and endow males with a monosynaptic PR that has a shorter latency than females.

To date, quantification of the PR by surface electromyography (sEMG) has predominantly used populations of mixed gender (e.g. Karst and Willett, 1995; Frijns et al., 1997; Uysal et al., 1999). Few studies have examined sEMG solely within populations of the same sex, and none with the specific objective of exploring gender differences in the PR: the primary aim of this study.

2. Methods

2.1. Subjects

Twenty eight subjects, 14 male and 14 female, took part in the study. All were 3rd year students at the University of Nottingham with an age range between 20 and 22 years old; Table 1 shows their anthropometric information. The recruitment of participants was by opportunity sampling and all were näive to the experimental method. Each subject filled in a questionnaire to provide background health information, to check for previous or known existent neurological conditions; all subjects were unaware of any prevalent neurological or other health problem/s. Studies conformed to the standards set by the *Declaration of Helsinki*, and the ethical committee of the Faculty of Medicine, University of Nottingham, granted ethical approval. Subjects gave written informed consent and were free to withdraw from the study at any point.

2.2. Electrode placement

We specifically chose to study the PR in the VL due to the sexual dimorphism of L3 α-motorneurons. We followed the European recommendations for surface sensor placement and signal processing according to 'SENIAM' (Hermens et al., 1999). After palpation of the VL, circular ECG conductive adhesive electrodes (diameter 22 mm; Medi-Trace Mini 100 Snap Electrode) were placed 2/3rds of the measured difference from the anterior superior iliac spine (ASIS) to the lateral side of the patellar, with an inter electrode distance of 30 mm (centre to centre), parallel to the fibre direction. To

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Table 1
Summary of the anthropometric details for 14 male and 14 female subjects. Data are presented as mean ± SEM (upper line) with their range (lower line). Significant difference is determined by Student's *t*-test.

Gender	Height (m)	Weight (kg)	Thigh length (cm)
Male	1.83 ± 0.02	80.3 ± 1.7	48.6 ± 1
	1.71–1.95	70–90	41–54
Female	1.67 ± 0.02	57.3 ± 1.3	44.8 ± 1
	1.58–1.79	47–64.7	39–53
Mean difference	0.16 ± 0.02***	23.0 ± 2.2***	3.8 ± 1.4*

* $p < 0.05$.
*** $p < 0.001$.

reduce skin impedance, its surface was abraded. A reference strap electrode was fitted around the ankle.

2.3. Experimental procedure

Subjects were seated such that the legs dangled freely with the knees slightly flexed. Tendon taps were applied manually with a hammer, such that its head was perpendicularly orientated to the tissue. To maximise the amplitude and success rate of a PR, the most sensitive region to elicit a PR was located and marked. This spot was repetitively tapped to produce at least ten positive PRs (a trial), one trial with the subject seated in a relaxed position with eyes closed and then another trial with the subject performing the Jendrassik manoeuvre. For the latter, subjects were instructed to interlock their left and right hands and pull to create maintained tension across the shoulders in response to an aural prompt issued 2 s before tapping the tendon, a second aural command instructed them to terminate this manoeuvre. A positive reflex was taken as the production of a clear lower leg jerk and a compound muscle action potential (CMAP) on the sEMG trace in response to a tap. Relaxation of the VL was determined by the lack of CMAP activity in the sEMG record immediately prior to stimulus application. To avoid artefacts that may arise though fatigue or subject anticipation, the force of the stimulus was randomly varied from subthreshold upto a value that either elicited maximal PR magnitude, or was painful. Stimuli were applied only when the lower leg was stationary and with a minimum rest period of at least 5 s between taps. This procedure was repeated in the same manner for each subject on both legs and was performed exclusively by author HV. We tested 56 limbs with and without the Jendrassik manoeuvre which equates to a total of 112 trials.

2.4. Data collection and analyses

The sEMG was recorded in bipolar mode, collected and stored using a Powerlab 26T with a dual BioAmp and Lab Chart software (ADInstruments Oxfordshire, UK). The sEMG was filtered with a 0.5–2 kHz bandpass filter, sampled at 10 kHz and stored for subsequent analyses. The acquisition software was configured such that 1 s of the sEMG, 0.5 s pre- and 0.5 s post-trigger, was captured. This arrangement allowed for the detection of background EMG activity prior to the stimulus.

For stimulation, a tendon hammer (MLA93; ADInstruments, Oxfordshire, UK) with a built in peizo electric sensor was used to both quantify the applied force and trigger the recording. The voltage output of the tendon hammer is proportional to the force applied; this was sampled at 10 kHz and smoothed with a three point median filter.

Given our specific aim, we studied the pre-mechanical, electrophysiological latency of the VL PR. Only the shortest and fastest latency component of the CMAP reflex response was measured: the delay from the stimulus trigger (time = 0) to the onset of the CMAP (see Fig. 1). The magnitude of the PR was quantified as the integral of the CMAP to the end of sampling (50 ms): the area under the curve (AUC; mV ms). Although, the peak to peak value of the CMAP was highly and linearly correlated ($p < 0.0001$) to its AUC, the AUC has the advantage of ease of measurement and indifference to CMAP waveform.

2.5. Statistical analysis

The dominant leg was defined by preference in sport. All group data, unless stated otherwise, are given as the mean ± standard error of the mean (SEM) with *n*, the number of limbs used for calculation. For statistical analyses PRISM version 5 software (GraphPad Software, San Diego California, USA) and StatsDirect (StatsDirect Ltd, Altrincham, Cheshire, UK) were used. Data distributions were deemed normal with the D'Agostino & Pearson omnibus normality test. Statistical tests used are given in the text. Statistical significance was taken at the level of $p < 0.05$.

3. Results

3.1. Description of raw data

Fig. 1 illustrates a typical time course for the applied force and sEMG recorded from the VL during a PR. The stimulus is not instantaneous but has a defined rising phase, which peaks between 2.5 and 3.5 ms after onset; a timing independent of force magnitude and consistent between trials. The sEMG shows a clearly defined biphasic compound muscle motor action potential (CMAP) whose latency is measured as the time between the two points indicated by the dashed lines. The CMAP appears to have a blunted shape compared to previous published results (e.g. Safronov, 2006; but see Uysal et al., 1999). Since neither the high sampling frequency (10 kHz) or filtering employed can readily account for this shape, its shape may relate to the expanded time base employed or impedance an changes that arise during limb movement. Because electrodes were positioned away from the IZ, the CMAP shape is unlikely to be affected by changes in waveform superimposition that may have arisen during movement (Rainoldi et al., 2000).

3.2. Stimulus–latency response relationship

Only 24 out of 112 trials demonstrated reflexes that possessed a significant increase in latency with stimulus magnitude (e.g. Fig. 2a); Spearman rank correlation coefficients, *r*, for these associations ranged from 0.43 to 0.73 (median 0.54, 13 limbs) with, and from 0.44 to 0.84 (median 0.7, 11 limbs) without, Jendrassik conditions, respectively. Of these, seven subjects maintained positive correlations between latency and stimulus magnitude in the same limb both with and without Jendrassik conditions. Correlation between latency and stimulus strength occurred independently of any correlation between stimulus strength and the AUC. Non-parametric linear regression of the correlated latency data yielded a median slope of 0.06 ms N^{-1} (range 0.02–0.43) and an intercept of 16 ms (range 13.5–24). All other trials failed to demonstrate a correlation between latency and stimulus strength (e.g. Fig. 2b).

3.3. AUC–latency response relationship

In 16 trials, latency was negatively correlated with AUC magnitude (Fig. 3a and b); Spearman rank correlation coefficients, *r*, for these associations ranged from −0.61 to −0.51 (median −0.55). Correlation was independent of Jendrassik conditions and gender (Fisher's exact test).