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Mechanical and electromyographic responses to stretch of the human anterior tibial muscle at different levels of contraction

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Summary. The EMG response and the mechanical response to 2 degree stretch of the human anterior tibial muscle was studied during contractions ranging from 0% to 80% of maximal voluntary contraction (MVC). The EMG response showed three distinct peaks M1, M2, and M3 with peak latencies of 59 ms, 86 ms, and 120 ms respectively. At low background torques M1 dominated while M2 and M3 were small or absent. M2 and M3 dominated above 40% of MVC and M2 in particular showed "automatic gain compensation", i.e. it constituted a – more or less – constant proportion of the background EMG for all contraction levels. The ratio between M1 amplitude and background EMG steadily decreased with contraction level. Even though the summed contributions of M1, M2, and M3 to some degree showed automatic gain compensation, this was not the case for the mechanical response to stretch. Between 0% and 30% of MVC the reflex mediated mechanical response increased approximately in proportion to the contraction level, but the reflex mediated mechanical response peaked at 40% of MVC and declined to zero at 80% of MVC. This discrepancy between EMG and mechanical response was explained by a simple model. The regression line between rectified and filtered tibialis anterior EMG and torque was used to predict the mechanical response from the EMG response. At increasing contraction levels the twitch elicited by supramaximal electrical stimulation decreases, and we reduced the predicted mechanical response by the same factor as the twitch. This simple model predicted the mechanical response for all contraction levels, making it possible to assess the "functionality" of reflexes even when accurate measurements of muscle force or intrinsic muscle properties are not possible.

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Introduction

If a muscle is stretched, an EMG response is elicited. This response consists of one or more peaks in the rectified EMG. The amplitude of the peaks varies with stretch amplitude, stretch velocity, and the contraction level of the muscle being stretched (Berardelli et al. 1982). With respect to the origin of the different peaks there is general agreement that the earliest peak is mainly due to excitatory input from primary spindle afferents in the stretched muscles (Marsden et al. 1983) and to a lesser degree due to a removal of inhibition from antagonist muscles (Sinkjær and Hoffer 1987). The origin of the later peaks is a matter of more controversy (Hagbarth et al. 1981; Hultborn and Wigström 1980; Marsden et al. 1973; Matthews 1987; Melvill Jones and Watt 1971). We will label the peaks in the EMG response M1, M2, and M3 (Lee and Tatton 1975). This study had three main purposes:

First, to describe the EMG responses in the human anterior tibial muscle to stretch during voluntary contraction ranging from zero to maximal voluntary contraction (MVC). To our knowledge this has not been done for any human muscle.

Second, to evaluate the "automatic gain hypothesis" (Marsden et al. 1976, 1983; Matthews 1986). According to this hypothesis the reflex gain increases in proportion to the contraction level, thus maintaining reflex responses in the EMG with a constant proportion to the background EMG. For contraction levels well below 50% of MVC, Matthews (1986) provided evidence that the mechanical responses also

increased in proportion to the contraction level. Data from decerebrate cat soleus muscle (Hoffer and Andreassen 1981) and from human anterior tibial muscle (Sinkjær et al. 1988) indicates that the "automatic gain control" does not function for the mechanical response above 30%–40% of MVC. We therefore decided to investigate both EMG and mechanical responses at high contraction levels.

The third purpose of this study was to investigate the relationship between EMG and mechanical responses to stretch. Animal studies (Houk et al. 1970; Hoffer and Andreassen 1981) and human studies (Allum and Mauritz 1984; Sinkjær et al. 1988) indicate that the stretch reflex and the intrinsic muscle properties have roughly equal contributions to the total mechanical response to stretch. We shall try to build a model that permits prediction of the mechanical reflex response from the EMG response. Such a model will make it possible to assess how "functional" the stretch reflex is, even in muscles where the contribution due to intrinsic muscle properties cannot be determined.

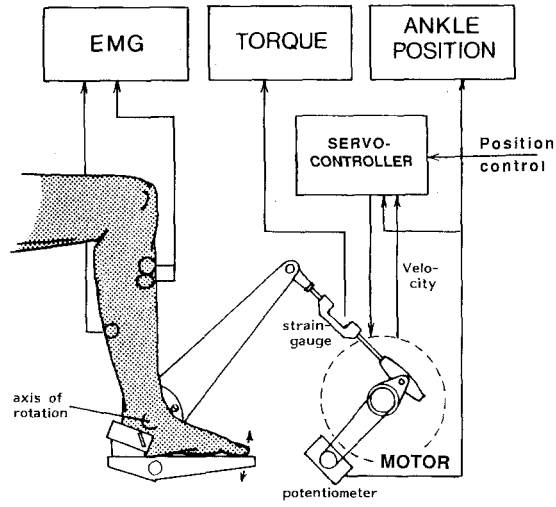

Fig. 1. Experimental set up. The subject was seated with the left foot strapped to a pedal, which can be moved by the motor. The motors axis of rotation is aligned with the ankle joints axis of rotation. Torque on the platform was measured by the strain gauge and ankle position by the potentiometer. Torque, ankle position, and EMG from the anterior tibial, the medial, and lateral gastrocnemius muscles were recorded

Methods

Experimental protocol

Eleven normal male subjects age 20–37 years were examined. During an experiment the subject sat in a chair with the left foot strapped to a platform. Knee and ankle angles were approximately 100 degrees. The ankle angle was defined as the angle between the sole of the foot and the shaft of tibia. Skin temperature was 35° C, maintained by a lamp and a sensor on the skin over the anterior tibial muscle (AT). The subjects were asked to produce the maximal possible contraction of the ankle dorsiflexors for 1 s. This was repeated three times and the highest of these values was chosen as the subjects MVC. Afterwards the subjects were required to maintain a given percentage of their MVC. During the contraction the dorsiflexors were stretched by rotating the platform. Two degrees stretches and releases with 35 ms rise time and a duration of 500 ms were applied with a frequency of 1.1 Hz. The level of contraction and the requested background torque were shown simultaneously on an oscilloscope to give the subject a visual feedback of the contraction level. During the perturbations the subjects were asked not to intervene (Crago et al. 1976).

To determine the intrinsic response, i.e. the mechanical response without reflex contributions, similar perturbations were also tested on contractions of the anterior tibial muscle obtained by electrical stimulation applied to the deep peroneal nerve. By changing the stimulation frequency from 7 to 50 Hz and the stimulation current from 4 to 20 mA the contraction level was varied from a few to approximately 70% of MVC. During stimulation the EMG showed no reflex response. For details see Sinkjær et al. 1988).

Mechanical set up

The axis of rotation of the platform was aligned with the axis of rotation of the ankle joint (Fig. 1). The platform was rotated by a motor (CEM, model 26).

Data collection

Bipolar EMG surface electrodes were placed parallel to the tibia over the anterior tibial muscle (AT). The distance between the centers of the electrodes was 2 cm. Another pair of electrodes was placed with one electrode over the lateral and one over the medial head of the gastrocnemius muscle (LG/MG). EMG signals were band-pass filtered (20 Hz to 2 kHz). The AT-EMG signal was full wave rectified and first order lowpass filtered (20 Hz). The AT-EMG, the rectified AT-EMG, the LG/MG-EMG, the platform torque, the position of the platform, and the velocity of the platform were displayed on a Siemens Elema 803 Mingograph. The rectified AT-EMG and the platform torque signals were averaged over 40 to 50 stretches by a Nicolet Viking and stored for later analysis.

Data analysis

The torque increment relative to the background torque was measured from the averaged signals 200 ms after stretch onset (Fig. 2). The contribution from the stretch reflex to the total torque increment was calculated by subtracting the intrinsic torque increment from the total torque increment (Sinkjær et al. 1988).

The EMG response consisted of from one to three peaks designated M1, M2, and M3. The onset latency, the peak latency, the duration, and the amplitude of M1, M2, and M3 were determined. The amplitude was calculated as the EMG level at the peak minus the EMG level before the stretch (Fig. 2).

In most recordings M1, M2, and M3 were roughly triangular. The mean EMG increment from the onset of M1 to the end of M3 was therefore approximated by one sixth of the sum of the M1, M2, and M3 amplitudes.

In a few persons a fourth peak was seen at high contraction levels. This peak was not analysed since the onset latency (150 ms) was so long that voluntary responses could have interfered.

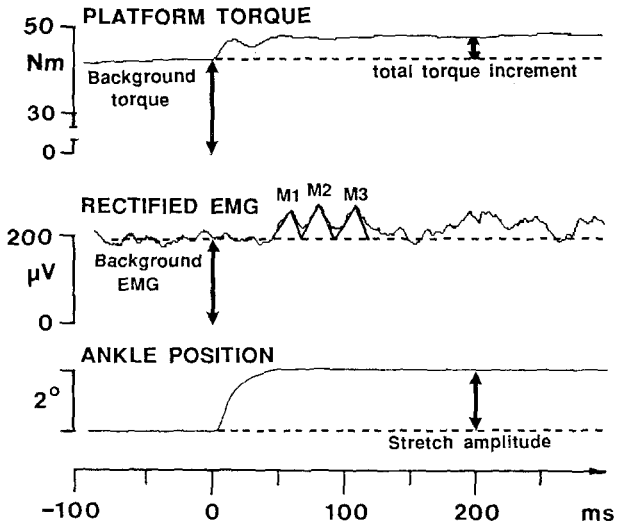

Fig. 2. From top to bottom, the torque on the platform, the rectified AT-EMG, and the ankle position. Torque and EMG signals were usually averaged 40 times. Three peaks labelled M1, M2, and M3 were seen in the EMG. The onset latency, peak latency, amplitude, and duration of M1, M2, and M3 was measured in the rectified and averaged AT-EMG. The mean EMG increment was calculated as one sixth of the total sum of the amplitudes of M1, M2, and M3, assuming triangular shapes. Torque increments were measured 200 ms after onset of stretch

Trials were rejected if cocontraction was observed in the LG/MG EMG recordings. Cocontraction was occasionally present at very high contraction levels but could usually be eliminated by careful instruction of the subject. Possible effects of fatigue were reduced by allowing subjects to rest between contractions. At higher contraction levels the duration of the contractions were limited to approximately 6 s.

Results

EMG responses at different background torques

EMG responses to 2 degree plantar stretches of the ankle with a rise time of 35 ms were recorded.

M1, M2, and M3 amplitudes. M1 had a large amplitude even at low background torques. In most subjects M1 increased by a factor of two with increasing background torque. M2 and M3 had amplitudes around zero at 10% of MVC, but increased to a plateau of about 100 μV for background torques from 40–80% of MVC. Each of the subjects showed a linear relationship between background torque and background EMG. The slopes of the regression lines for torque and EMG differed between subjects due to differences in electrode positions, thickness of subcutaneous fat etc. To compensate for this the M1, M2, and M3 amplitudes were divided by the slope of the regression line connecting EMG background and background torque in % of MVC for that subject. Figure 3A–C shows mean and standard error of the normalized M1, M2, and M3 amplitudes versus background torque for all subjects. M2 and M3 amplitudes rose rapidly with torque, and exceeded M1 amplitudes for background torques greater than 40% of MVC. The mean EMG increment (Fig. 3D), calculated as one sixth of M1 plus M2, plus M3 (see Methods), increased with contraction levels up to 50% of MVC, where it reached a plateau.

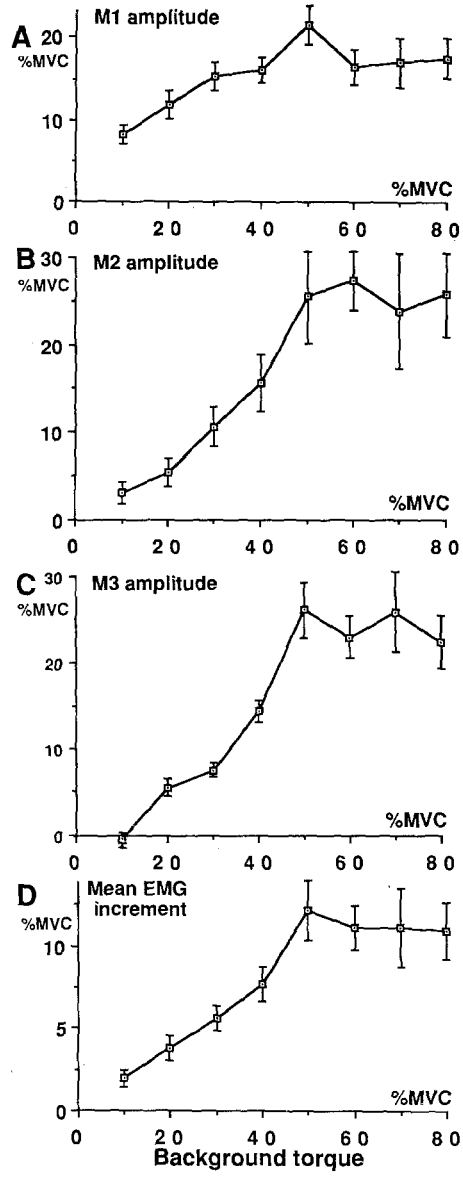

Fig. 3A–D. Mean values of normalized M1, M2, and M3 amplitudes and mean EMG increment for all subjects. Horizontal bars indicate ± 1 standard error (N = 11)

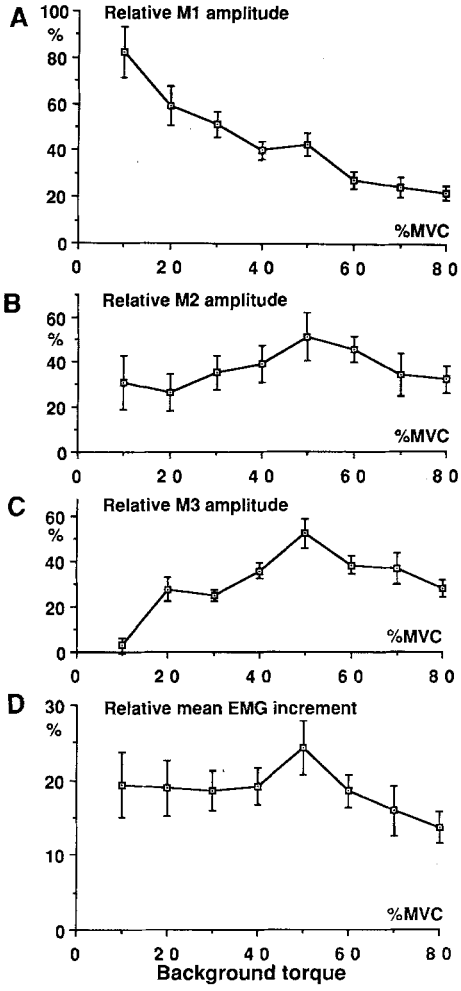

Fig. 4A–D. Relative amplitudes (amplitude/background EMG) of M1, M2, and M3 and relative mean EMG increment. Mean values of all subjects. Horizontal bars indicate ± 1 standard error ($N = 11$)

The relative importance of the M1, M2, and M3 peaks was assessed by plotting the amplitudes in percent of the background EMG for all subjects (Fig. 4). At low background torques the M1 amplitude was approximately the same size as the background EMG, but declined with background torque and was only about 20% of the background EMG at 80% of MVC.

M2 ranged from 27–51% of the background EMG, peaking at 50% of MVC. M3 ranged from 13–52% of the background EMG, also peaking at 50% of MVC. The mean EMG increment in percent of the background EMG was relatively constant from 10% to 60% of MVC, and declined for higher levels of contraction (Fig. 4D).

M1, M2, and M3 latencies and durations. Onset and peak latencies and durations of M1, M2, and M3 are shown for one subject in Fig. 5.

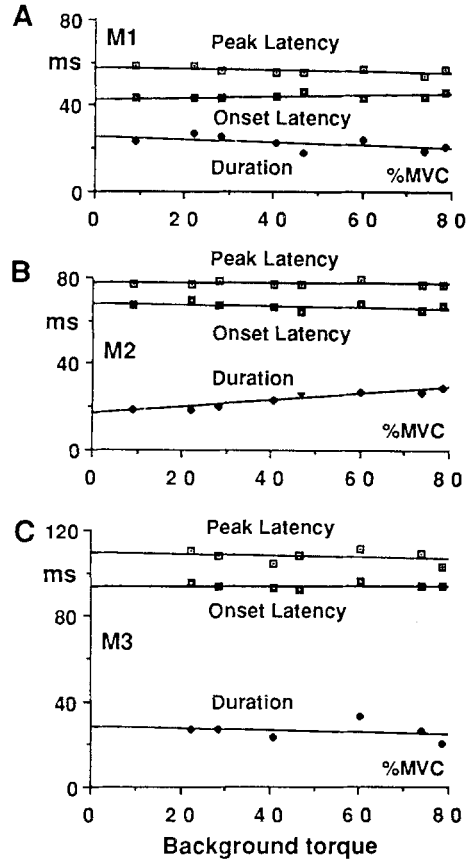

Fig. 5A–C. Peak latency, onset latency, and duration of the M1, M2, and M3 responses in the rectified AT-EMG as function of background torque in one subject

Table 1. Range of mean latencies and durations for 11 subjects and decline of latencies from 10–80% MVC (mean ± SE.)

	Peak latency ms	Onset latency ms	Duration ms	Latency decline ms
M1	57– 62	44– 51	22–27	1.3 ± 0.6
M2	79– 93	69– 80	18–25	1.3 ± 0.7
M3	110–130	97–113	25–33	8.5 ± 1.9

For each subject mean latencies and durations were calculated by averaging over all background torques. Table 1 gives the range of these mean values for all subjects. Onset of M3 occurred 5 to 10 ms after M2 ended for low and intermediate levels of contraction.

For most of the subjects the peak latencies decreased with background torque ($P < 0.05$, sign test). From 10% to 80% of MVC the peak latencies of M1 and M2 declined by about 1.3 ms while the peak latency of M3 declined by about 8.5 ms. M3

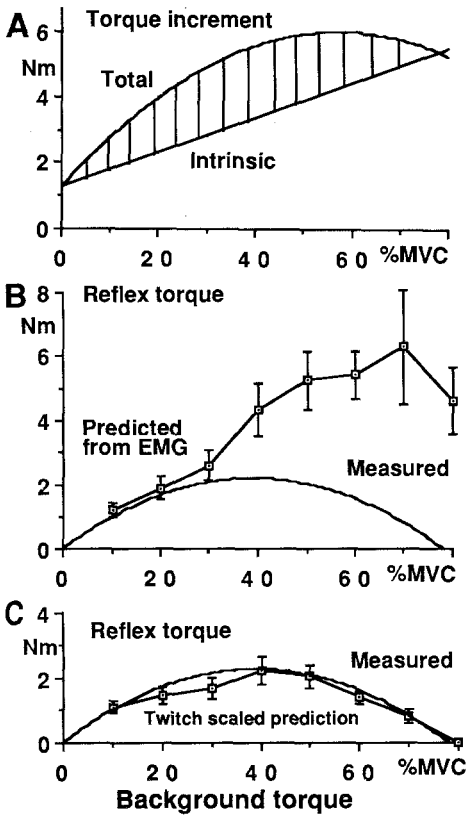

Fig. 6A–C. EMG response compared to reflex mediated mechanical response as function of the level of contraction in 6 subjects. **A** Mechanical measurements. Total torque increment described by a second order polynomium and intrinsic torque increment by a first order polynomium fitted to the mean values of all subjects. The shaded area indicates the reflex mediated torque increment. **B** Reflex mediated torque predicted from EMG (averaged values of mean EMG increment divided by the slope of the regression line for background EMG and background torque) and the measured reflex mediated torque increment (shaded area from **A**). **C** Reflex torque predicted from EMG (from **B**) multiplied by the relative twitch amplitude (Twitch scaled prediction) and measured reflex torque (shaded area from **A**). The predicted twitch scaled reflex torque corresponds well with the measured reflex torque for all levels of contraction

declined significantly more than M1 and M2 ($P \leq 0.02$, Wilcoxon's signed ranks test).

Comparison of EMG responses and reflex mediated mechanical responses to stretch

The reflex contribution to the torque increment was calculated 200 ms after the onset of a 2 degree plantar rotation. 200 ms after the stretch of the dorsiflexors, the torque recruited by the M1, M2, and M3 peaks has been fully developed.

The reflex contribution was calculated by subtracting the intrinsic contribution to the torque increment, measured during electrical stimulation of the deep peroneal nerve, from the total torque increment (for details see Sinkjær et al. 1988). Figure 6A shows total and intrinsic torque increment averaged over six subjects.

For each subject, a reflex mediated torque increment due to the mean EMG increment was calculated by dividing the mean EMG increment with the slope of the regression line between background EMG and background torque for that subject. Averaged over six subjects this predicted torque increment coincided with the measured reflex mediated torque increment for background torques up to 30% of MVC (Fig. 6B). For higher background torques the predicted torque increment exceeded the measured reflex mediated torque increment.

The amplitude of a twitch elicited by supramaximal nerve stimulation during voluntary contraction decreases with increasing contraction level. The amplitude of the muscle twitch as function of background torque (T_B) is given by the relationship (Woods et al. 1987):

$$A(T_B) = A(0)*[T_{max}-T_B]/T_{max} \quad \text{(eq. 1)}$$

where $A(T_B)$ is the amplitude of the twitch at the background torque T_B and $A(0)$ is the maximal amplitude of the twitch (elicited in a relaxed muscle). T_{max} is the maximal voluntary contraction a subject can keep for 10 s (equals approximately 80% of MVC). The predicted torque increment scaled down according to eq. 1 was in good agreement with measured reflex mediated torque increment for all background torques (Fig. 6C).

Discussion

M1, M2, and M3 responses

The most striking feature of the M1, M2, and M3 responses is their reproducibility. Under our experimental conditions M1, M2, and M3 responses were found in all subjects with surprisingly small variations in latency between subjects (Table 1). Bedingham and Tatton (1984) found a similar reliability in EMG responses from a wrist flexor. Other authors have reported that in the triceps surae muscles responses other than M1 were only present in 40% of the subjects (Berardelli et al. 1982) and only when the duration of the stretch exceeded 20 ms (Allum and Mauritz 1984). Those experiments were made at contraction levels at or below 20% of MVC. In the tibialis anterior muscle we also found that the long latency responses were small or absent at contraction levels below 20% of MVC.

A consistent finding was a small decline in peak latencies (Fig. 5) with contraction level. We attribute the decline of M1 and M2 latencies to the organization of the motor neuron pool (Henneman et al. 1974). According to the size principle only small motor units with slowly conducting axons are recruited by the reflex at low contraction levels. At higher contraction levels larger motor units with faster conducting axons are recruited. One will therefore expect that the average nerve conduction time for the motor units is smaller at high than at low contraction levels in agreement with our observations.

Interestingly, the peak latency of M3 fell by about 8.5 ms, significantly more than for M1 and M2 and more than can conveniently be explained by differences in axonal conduction velocities. This suggests either that M3 is mediated through a longer pathway, which is also organized according to the size principle or that the synaptic delays in the M3 pathway are shortened during strong contractions.

Automatic gain compensation

Within the statistical uncertainty the mean EMG increment after stretch was a constant proportion of background EMG up to 60% of MVC (Fig. 4D). The decline in the EMG increment at 70% and 80% of MVC was marginally significant. The EMG increment thus seems to support the hypothesis of "automatic gain compensation" (Marsden et al. 1976; Matthews 1986), at least for low and intermediate contraction levels. However, the behavior of the individual components of the EMG increment was markedly different. The amplitude of M1, relative to background EMG, declined monotonically with contraction level (Fig. 4A), while M2 and M3 had a flat maximum around 50% of MVC (Fig. 4B, C). If we accept the automatic gain compensation hypothesis as a first order approximation of the behaviour of the motor neuron pool, then the implication is that only the input to the motor neuron pool giving rise to M2 is almost independent of contraction level. The input giving rise to M1 is steadily declining with contraction level while the input to M3 is nearly absent at 10% of MVC, whereafter it behaves somewhat like the input giving rise to M2.

While the automatic gain compensation hypothesis describes the EMG increment reasonably well, it fails to describe the mechanical response. The mechanical response increases approximately proportional to background torque up to 30% of MVC, peaks at 50%–60% of MVC and then decreases.

Our findings therefore only support the automatic gain compensation hypothesis for contraction levels up to about 30% of MVC. Rather, for a wide range of background forces, the combined effects of intrinsic muscle properties and reflex action tends to make the torque increment and thereby muscle stiffnes relatively independent of contraction level (Hoffer and Andreassen 1981; Sinkjær et al. 1988). Although our conclusion does not support the automatic gain compensation hypothesis, our data are in agreement with the mechanical responses measured by Matthews (1986) and Hunter and Kearney (1982) who only measured mechanical responses below 30% of MVC.

Is the intrinsic stiffness measured in this study identical to the intrinsic stiffness of a voluntarily activated muscle? Rack and Westbury (1969) showed that sinusoidally stretches to partially tetanized muscles at low stimulation rates reduce the force and that movement in general reduces both force and stiffness, in particular at low stimulus rates (Rack and Westbury 1974). Furthermore, the yielding that occurs when a muscle is stretched in excess of the short range stiffness is much more prominent at low stimulus rates than at higher rates (Joyce et al. 1969; Nichols and Houk 1976). Despite this, when a muscle is stretched rapidly, in excess of the short range stiffness, experimental evidence supports a linear relation between force and intrinsic stiffness, irrespective of stimulus rate and mode of recruitment. As in this study, Hoffer and Andreassen (1981) found a linear relation between force and intrinsic stiffness in the cat soleus muscle during electrical stimulation. Allum and Mauritz (1984) found a linear relation between force and intrinsic stiffness in a voluntary recruited muscle in the first 40 ms following a stretch before reflexes could interfere. Apparently, the rapid stretch resets the status of the cross-bridges, and makes previous history of recruitment irrelevant.

Direct comparison of intrinsic stiffness of the ankle plantarflexors during supramaximal nerve stimulation at different rates, and total stiffness during voluntary contraction was made in a subject with almost absent reflexes. The stiffness measured during electrical stimulation and during voluntary contraction was identical within the measurement accuracy.

Prediction of reflex mediated torque increment from the EMG increment

Previous studies in man (Allum and Mauritz 1984; Sinkjær et al. 1988; Sinkjær 1988), and in animals (Houk et al. 1970; Nichols and Houk 1976; Hoffer and Andreassen 1981) have shown that reflexes make a significant contribution to muscle stiffness, and that muscle stiffness is an important factor in force

generation (Sinkjær et al. 1988). In these studies the reflex stiffness was determined as the difference between the stiffness of a muscle with intact reflexes and the intrinsic muscle stiffness. In particular in man the accurate determination of intrinsic stiffness is technically difficult. A reliable prediction of the reflex mediated stiffness (or torque increment) from the EMG response would therefore serve two purposes: 1) It would make it possible to assess how "functional" stretch reflexes are in a wide range of muscles, and even in patients with motor disorders where measurement of intrinsic muscle stiffness is likely to be difficult or impossible. 2) A model could also serve to explain the above mentioned discrepancy between the EMG increment and the mechanical response: The EMG increment increased roughly in proportion to background EMG, while the reflex mediated torque increment peaked at 40% of MVC.

Two simple models were developed. In the first model the relationship between background torque and background EMG was used to predict the torque increment due to the EMG increment. This model predicted torque increments in agreement with the experimental values for background torques up to 30% of MVC (Fig. 6B). At higher background torques the predicted torque increments were too high. The second model was a modified version of the first. At high contraction levels most of the motor units will be firing close to their fusion frequency. Additional EMG activity in the form of modulation of firing rate will therefore be ineffective in generating additional torque. A simple relation between the size of the twitch elicited by supramaximal stimulation of the muscle nerve and the contraction level (Woods et al. 1987) was used to account for this effect and to modify predicted torque increment. With this modification of the model good agreement between measured torque increments and predicted torque increment was observed for all background torques (Fig. 6C).

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