Reflex inhibition following electrical stimulation over muscle tendons in man

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Summary

Electrical stimulation over selected muscle tendons in alert human subjects produced, in each muscle, a reflex inhibition of muscle activity. This inhibition, when maximal, was seen in the surface EMG as an interval of complete electrical silence during a sustained voluntary contraction. The inhibition was clearly visible in single sweeps and in averaged records. Its onset latency and duration were respectively, 56±4.9 and 46±11.8 ms in extensor digitorum communis, 71±6.1 and 46±10.5 ms in extensor pollicis brevis, 77±11.2 and 47±10.5 ms in extensor pollicis longus, 72±7.3 and 43±8.6 ms in abductor digiti minimi, and 97±3.5 and 43±2.8 ms in tibialis anterior. The inhibitory response was produced at low stimulus intensities (<10 mA) without electrical (M wave) or mechanical (muscle twitch) signs of direct muscle stimulation. It therefore did not arise from stimulation of la afferents (muscle spindles). The response arose from tendons since it occurred at lowest threshold when stimulation was applied directly over the tendons of the five different muscles studied. At low stimulus intensities, the response declined sharply when the stimulating electrodes were moved to the skin immediately adjacent to the tendons. The response did not arise from skin afferents since it was also present when stimuli were delivered to the tendon by subcutaneous needle electrodes and it was not reproduced by stimulation of cutaneous nerves in the region of the tendon. In another series of experiments on extensor pollicis brevis, five skin locations were stimulated while overlying the tendon and again while the skin was stretched so that they were lying 0.6-0.8 cm dorsal to the tendon. In these experiments the response was again greatly attenuated when the stimulation was not directly over the tendon, although the same cutaneous sites were stimulated. The inhibition was followed by a pronounced excitatory component (El) of peak latency 120-140 ms. The results of the study provide evidence for a powerful autogenic inhibitory reflex in man. The evidence is consistent with the possibility that the response arises from Golgi tendon organ afferents.

Keywords: tendon reflex; Golgi tendon organ; autogenic inhibition; man

Abbreviations: ADM = abductor digiti minimi muscle; EDC = extensor digitorum communis muscle; EPB = extensor pollicis brevis muscle; EPL = extensor pollicis longus muscle; TA = tibialis anterior muscle

Introduction

Relatively little is known of the characteristics of tendon organ reflexes in man. Human reflex studies have most commonly employed transcutaneous nerve stimulation and recording techniques, the interpretation of which is complicated by the activation of several fibre types including muscle spindle afferents of various kinds, skin afferents, subcutaneous tissues, joint receptor afferents, tendon organ afferents and pain fibres. In addition there can be problems from antidromic activation of motor fibres.

Identification of different afferents has been generally dependent upon differences in fibre diameter and the associated differences in electrical threshold and conduction velocity. This method has only limited usefulness since the fibres which subserve different sensory modalities tend to show substantial overlap in the distribution of their fibre diameters. This is particularly true of the la fibres subserving muscle spindles and the lb fibres innervating Golgi tendon organs.

Because of the limited data on man (see Cavallari et al., 1985), our knowledge of autogenic inhibitory effects is largely derived from animal experiments, specifically from decerebrate and anaesthetized cats [reviewed by Prosk (1981,
Electrical recording

Stick-on, chlorided silver electrodes were attached to the skin overlying the belly of the muscles, extensor digitorum communis (EDC), extensor pollicis brevis (EPB), tibialis anterior (TA), extensor pollicis longus (EPL) and abductor digiti minimi (ADM). Careful attention to skin degreasing and the use of electrode jelly enabled inter-electrode impedance as measured with a Wien bridge to be kept below 5 kΩ. Amplifiers were conventional and had filters with their 3 dB cut-off points set to 1.6 Hz and 3 kHz for low frequency and high frequency signal conditioning, respectively. Amplifier gains were controlled by software via a digital interface.

Methods

Subjects

We performed 46 experiments on a total of 29 subjects. Their age range was 23–72 years. All subjects gave informed consent for electrical recording and stimulation using methods that had been accorded prior approval by the local Ethics Committee.

Electrical stimulation

Silver/silver chloride electrodes filled with conducting jelly were placed on the skin for transcutaneous stimulation of structures beneath. Unless otherwise stated, the stimuli were rectangular pulses of 0.05 ms duration and voltages up to 400 V. In some experiments constant current stimulation was used (Digitimer D57, 5–45 mA).

In experiments on EDC, the cathode was placed directly over the tendon, usually <1 cm proximal to the retinaculum, and the anode 1.5 cm lateral to it. A monopolar arrangement was used in experiments on EPB, where the anode was a large metal plate which was taped to the dorsum of the hand. The cathode was a small gold-plated disc electrode which was taped to the skin directly over the tendon.

The stimuli were timed with respect to the averaging program (and sometimes other external events) using a crystal controlled oscillator and frequency divider (Digitimer D100). Stimulation and recording sites were carefully chosen with regard to the surface anatomy. Reference was made to cadavers and the dissection photographs of McMinn and Hutchings (1988).

Skin stretch experiments on EPB

Because tendon stimulation was generally transcutaneous, it was necessary to assess the possible contribution of skin afferent stimulation to the inhibitory response. The EPB was selected for further experiments on this question because of its long and prominent tendon. In each subject, three to five sites 1 cm apart were selected and marked on the skin along the line of the EPB tendon. They fell 1–5 cm proximal to the metacarpo-phalangeal joint. A 5 cm strip of Micropore tape was then secured to the skin so that it ran immediately dorsal to and parallel to the tendon line of stimulation sites (see Fig. 4 below). Recordings were then made in response to stimulation at each site as it lay directly over the tendon.
Table 1 Summary of I responses

<table>
<thead>
<tr>
<th>Muscle</th>
<th>EPB</th>
<th>EPL</th>
<th>EDC</th>
<th>TA</th>
<th>ADM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latency (ms)</td>
<td>71.3±6.1</td>
<td>77.5±11.2</td>
<td>56.1±4.9</td>
<td>97.5±3.5</td>
<td>75.4±3.2</td>
</tr>
<tr>
<td>Duration (ms)</td>
<td>46.0±10.5</td>
<td>47.5±10.6</td>
<td>46.5±11.8</td>
<td>43.5±2.8</td>
<td>48.2±8.3</td>
</tr>
<tr>
<td>Inhibition (%)</td>
<td>64.3±14.9</td>
<td>50.6±28.2</td>
<td>82.2±16.5</td>
<td>94.1±10.2</td>
<td>78.3±16.2</td>
</tr>
<tr>
<td>Max stimulus</td>
<td>30 mA</td>
<td>30 mA</td>
<td>214 V</td>
<td>300 V</td>
<td>30 mA</td>
</tr>
<tr>
<td>No. of responses</td>
<td>90</td>
<td>14</td>
<td>235</td>
<td>48</td>
<td>34</td>
</tr>
</tbody>
</table>

A total of 392 inhibitory responses were recorded from 29 normal subjects. The response was clearly identified in all experiments, though the threshold and maximal stimulus intensities showed some inter-subject variability. Population means and their standard errors are shown.

Fig. 2 Data pooled from all subjects to show the mean relation between strength of stimulus given to Golgi tendon organ (GTO) afferents in EDC and the amplitude of the resulting I wave. The error bars show standard deviations. Recordings were made from the skin over the belly of EDC and stimulation was given directly over the central tendon of EDC just proximal to the extensor retinaculum.

Fig. 3 Four sweeps of raw EMG, each showing the response to a single stimulus (at the marker) to EDC (rectangular pulse of 300 V, 50 μs). In the bottom trace the time calibrations are 50 ms and the marker amplitude is 100 μV. Note a period of inhibition of firing, beginning 55 ms after the stimulus that is followed by a rebound excitation.
The data from these muscles are summarized in Table 1. The obtained without contamination by muscle action potentials EDC and EPB. In these muscles the responses could be tendons and most of the data reported here were taken from their respective tendons: EDC, EPB, EPL, TA and ADM. Experiments from the following muscles after stimulation of different muscles in 46 experiments conducted on 29 subjects.

A consistent inhibitory response could be identified in all experiments, as judged by visual inspection of raw records. In EPB the latency of the I wave was estimated to be 71±6.1 ms and its duration 46±10.5 ms. The actual latency of I was difficult to determine in this muscle and may fall within the range 50–71 ms, since it was masked by the presence of a prominent excitatory component (T) which had a latency of ~50 ms.

The inhibition was also visible in single sweeps displayed on an oscilloscope. Here it appeared as an interruption to the background discharge, though its exact latency and duration varied between sweeps, presumably due to fluctuations in the background activity. At high stimulus intensities, the timing of the response in single sweeps appeared to become more stable, as the background discharge was abolished (Fig. 3).

**Excitatory components E and T**

An excitatory component (E1) usually followed the I wave with a peak latency of 120–140 ms. Under suitable circumstances, a second excitatory wave (E2) was also seen 100–120 ms later than E1 (Fig. 1). The amplitude of E1 was generally related to that of I. Another excitatory component (T) was also seen in some experiments; it was brief and preceded the I wave (latency in EPB, 50 ms). The I response was not dependent on T since the latter was not present in most records.

**The origin of component I**

To test the possibility that the autogenic inhibition following tendon stimulation arises from Golgi tendon organ afferents,
Fig. 4 Records from EPB. The approximate stimulation sites are shown with respect to the tendons of EPB (lower) and EPL (upper). To control for the effects of skin stimulation, four skin locations (a–d), which were 2–5 cm proximal to the metacarpophalangeal joint, were stimulated (20 mA, 50 μs) while overlying the tendon of EPB and again after the skin had been pulled and fixed with tape so that the same skin locations were displaced 0.6–0.8 cm dorsal to the tendon (e–h). This stimulus was just above threshold in order to minimize current spread. The anode was located on the dorsum of the hand so that current flow across the tendon was reduced by dorsal displacement of the skin. The inhibitory response was always larger when the stimulation was centred over the tendon. This effect was not due to cutaneous afferents since the same sites were stimulated before and after skin displacement. With respect to its distribution along the tendon, the response tended to decline as the cathode was moved toward the metacarpophalangeal joint.

Further experiments were conducted to control for the contribution of other afferents, particularly those from skin.

**Skin stretching experiments**

Records from EPB are shown in Fig. 4. This muscle has a long tendon which was clearly defined by thumb extension, enabling accurate placement of the stimulating electrode. An identifiable response was obtained from EPB in all of the 23 experiments carried out in 10 subjects. The threshold of the Golgi tendon organ response from this muscle was under 40 V (or 10 mA) in most subjects. Figure 4 shows a comparison of the effects of low intensity stimulation at five skin locations while overlying the tendon of EPB (records A–D) with similar further stimulation after the skin was pulled and fixed with tape so that the same sites lay 0.6–0.8 cm dorsal to the tendon (records E–H). A sharp decline in response amplitude could be demonstrated with small displacements of the stimulus away from the tendon. These data are summarized in Table 2. The skin stretching procedure excluded the possibility that local variations in skin sensitivity to the stimulus produced the observed distribution of the response. Thus the stimulus had to be

**EDC response distribution**

The topographic distribution of the tendon response was systematically studied by stimulation of the skin in the vicinity of the EDC tendon. When traverses of the active stimulating electrode were carried out, the size of I declined with distance on the skin from this optimal position, either laterally or distally (Fig. 6B).

**Stimulation with concentric needle electrodes**

To investigate the contribution of skin afferents to the I wave further, the tip of a concentric needle stimulating electrode was inserted subcutaneously to lie immediately superficial to the tendon of EDC (middle tendon). The central core of the electrode served as the cathode and a large (12×5 cm²) aluminium foil was placed on the palmar aspect of the forearm as the anode. The lack of any skin sensation confirmed that current flow across the skin was removed or greatly reduced. As can be seen from Fig. 6C, the...
Table 2 Skin stretching experiment on EDC (n = 10)

<table>
<thead>
<tr>
<th>Distance of stimulating site from the metacarpo-phalangeal joint (cm)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Area of I wave (mV ms⁻¹)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>On tendon stimulation</td>
<td>3.1</td>
<td>4.55</td>
<td>4.0</td>
<td>5.05</td>
<td>1.9</td>
</tr>
<tr>
<td>Off tendon stimulation</td>
<td>0</td>
<td>0.75</td>
<td>0.8</td>
<td>2.6</td>
<td>1.1</td>
</tr>
<tr>
<td><em>P</em></td>
<td><em>P</em> &lt; 0.01</td>
<td><em>P</em> &lt; 0.01</td>
<td><em>P</em> &lt; 0.01</td>
<td><em>P</em> &lt; 0.05</td>
<td><em>P</em> &lt; 0.05</td>
</tr>
</tbody>
</table>

The areas are the population means for each stimulation site. It can be seen from the statistical comparisons that the ‘on tendon’ stimulation produced significantly greater inhibition at every skin location.

Fig. 6 (A) Axes used to plot the distribution of the EDC Golgi tendon organ response on the skin. The zero point was the point of lowest threshold and was centred on the central tendon. (B) Graphs of I wave amplitude plotted against anatomical location of active electrode. In each case the stimulus was 350 V, 50 s pulse duration. Due to the relatively high stimulus intensity and the multiple tendons of this muscle, the gradient was not as steep as observed for EPB. (C) Use of a concentric needle electrode for subcutaneous stimulation of Golgi tendon organ afferents at the musculotendinous junction of EDC (350 V, 50 s pulses, average of 50 rectified sweeps). The maximum amplitude of the I wave was ~ 0.2 mV.

Configuration and amplitude of the I and EI response to Golgi tendon organ stimulation was not altered by the procedure, confirming that skin afferents were not involved.

Discussion

This study describes a powerful autogenic reflex inhibition which follows electrical stimulation over the tendons of conscious human subjects. The effect may arise from stimulation of Golgi tendon organ afferents since it has been localized to the tendons of five different muscles and control experiments have eliminated the possibility of a significant contribution from skin afferents. Selective stimulation was achieved by placing the stimulating electrodes over the tendons of muscles such as TA, EDC and EPB, all of which have long tendons.

The complex response to tendon stimulation (of EDC) is shown in Fig. 1 and consists of a sudden decline in excitation, the I wave commencing 56±4.9 ms after the stimulus and persisting for 46±11.8 ms. The inhibition was of maximal strength 76±15 ms after the stimulus. Following the I wave was a reflex excitatory component EI lasting 50–100 ms and peaking 120–145 ms after the stimulus to the tendon afferents. Sometimes additional excitatory responses followed EI with a periodicity of between 90 and 120 ms, E2, E3, etc. It seemed likely that the later responses represented the action potential bursts of physiological tremor although no experiments were undertaken to confirm this.

Origin of the inhibitory reflex

The evidence that the inhibitory response was derived from muscle tendons may be summarized as follows. The response could be elicited at low threshold (well below noxious levels) and in the absence of an M wave (of latency under 10 ms) or a visible twitch, indicating that it was not the result of direct motor stimulation. The inhibitory response is not due to stimulation of spindle afferents since this would have produced an excitatory reflex response at <30 ms in the arm (H wave) (40–45 ms in the leg). In fact the earliest excitatory response to follow the tendon stimulus occurred at ~50 ms (the T wave).

It was possible to demonstrate good localization of the stimulus to the tendons of five different muscles; EPB was particularly suited to the localization experiments as its tendon was raised and therefore clearly outlined by extension of the thumb. In 10 subjects, it was shown that low intensity shocks delivered at five different points along the tendon were more effective as measured by the strength of I (mean percentage inhibition) than shocks delivered to five points just 0.5–0.8 mm dorsal to the tendon. In these experiments, the stimulating electrode was moved off the tendon by stretching and taping of the skin, so that test and control shocks were delivered through the same skin locations. By this means, the possibility that local variations in skin sensitivity contributed to the result was excluded.
In the case of EDC, a 'receptive field' could be plotted in respect of the location of the stimulating cathode on the skin area over the tendon. The gradient of this field was not as steep as that observed for EPB. However, this muscle is served by four parallel tendons which would be likely to contribute to the response. Nevertheless, the optimal sites for stimulation of this tendon were the skin areas immediately superficial to the tendon. Lateral deviations from these areas attenuated the I and E1 responses and larger shifts in the electrode position resulted in the abolition of both components. It was therefore clear that skin afferents were not responsible for the inhibitory I wave and that the response was localized to the muscle tendon. The stimulus parameters (brief, low intensity shocks) were clearly appropriate for the Ib afferents and less appropriate for smaller diameter afferents, thus tending to exclude contributions from tendon nociceptive endings that have been reported to produce inhibitory effects (reviewed by Mense, 1993). Also, as previously stated, powerful inhibitory effects were obtained in the absence of noxious sensations. In fact, most subjects reported very little sensation of any type at stimulus levels close to threshold. Thus the evidence strongly favours the Ib tendon afferents, though a contribution from other afferents within the tendon cannot be definitely excluded.

**Timing of the inhibitory reflex**

It has been known for many years that tendon organ afferents give rise to autogenic inhibition. The latency of this response is reported to be only slightly longer than that required for an excitatory reflex response from muscle spindles. Although group Ib and Ia afferents have a similar distribution of fibre diameters and conduction velocities, the later onset of inhibition is generally attributed to the presence of an additional interneuron in the inhibitory pathway. We have argued that the effectiveness of very low tendon stimuli (<40 V) and the mild, non-noxious sensation which accompanied the stimulus would infer involvement of large diameter afferents. However, the responses reported here, in view of their latencies, could be of polysynaptic origin. The significance of a possible polysynaptic Ib reflex is discussed below.

**Role of the polysynaptic Ib reflex**

Fukami and Wilkinson (1977) (see also Fukami, 1981; Wilkinson and Fukami, 1983) found that the response in impulses per second of isolated Golgi tendon organs was fairly linearly related to the applied force (though this was not confirmed in later studies on decerebrate preparations). The isolated Golgi tendon organs behave similarly to muscle spindles with respect to their great sensitivity to small stretches as they also have a low threshold to in-series forces. The work reported here, through the demonstration of strong inhibitory tendon reflexes in alert subjects, adds further support to these observations for an important role of Golgi tendon organs in reflex regulation of force of contraction, as proposed by Houk (1979).

The natural frequency of oscillation of the wrist joint while muscles are contracted, due to the viscoelastic properties of muscles and tendons acting across it and the inertial mass of the hand, is ~8 Hz in large hands and up to 12 Hz in small ones. A single muscle twitch, in the absence of any negative (inhibitory) feedback would be expected to set the joint oscillating in a die-away fashion at this frequency. A period of relative or absolute inhibition, timed to occur 50–100 ms after a twitch would prevent this undesirable oscillation. The timing of the reflex we have studied is appropriate to provide effective damping of joint oscillation during muscle contraction. Stretch reflex activity alone may tend to assist joint oscillation since it has a similar resonant frequency (Lippold, 1973). There appears to be ample substrate for close coupling of Ia and Ib reflex activity through convergence of these afferents onto spinal interneurons (see Jankowska and McCrea, 1983; Harrison and Jankowska, 1985).

This work may thus carry implications for the ways in which various tremors such as physiological, parkinsonian and essential tremor are generated. Indeed, using the Golgi tendon organ stimulation technique reported here, a subsequent study on Parkinson’s disease has suggested a loss of tendon organ inhibition in these patients (Burne and Lippold, 1996).

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