

available at www.sciencedirect.comwww.elsevier.com/locate/brainres**BRAIN
RESEARCH****Research Report****Afferents contributing to autogenic inhibition of gastrocnemius following electrical stimulation of its tendon****Serajul I. Khan, John A. Burne***

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ABSTRACT

Electrical stimulation of the Achilles tendon produced strong reflex inhibition of the ongoing voluntary EMG activity in the two heads of the gastrocnemius (GA) muscle in all tested subjects. The inhibition was seen clearly in both averaged and single sweep surface EMG records. The inhibitory response was produced without electrical (M wave) or mechanical, (muscle twitch) signs of direct muscle stimulation. The onset latency and duration for the first period of inhibition (I_1) were 47–49 ms and 67 ms, respectively. A second inhibition (I_2) had an onset latency of 187–193 ms and duration under 40 ms. Non-noxious stimuli in the range of 2.6–7.6× mean perceptual threshold, when delivered to four locations over the GA tendon, all produced clear inhibition of the voluntary muscle activity. The inhibition was maximal when the cathode was a large metal plate located near the musculotendinous junction and decreased approximately linearly with distances more distal to that site. The effect of passive muscle stretch on the electrically induced tendon reflex inhibition (TRE) was tested at ankle joint angles incremented in steps of 20°. It was found that TRE is strongly dependent on joint angle, being maximal in the fully stretched muscle. TRE was lost completely after partial tibial nerve block. In comparison, GA inhibition produced by cutaneous (sural) nerve stimulation was of a higher threshold, longer latency and persisted after partial tibial nerve block. We thus demonstrated a powerful autogenic inhibition in the lower limb arising from tendon afferents in conscious subjects that is increased by passive muscle stretch and likely to originate from group I tendon afferents.

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1. Introduction

The autogenic inhibition due to Golgi tendon organs was first studied in human subjects in recordings from gastrocnemius medialis and soleus after stimulation of Ib fibers in the gastrocnemius medialis nerve in the lower and medial parts of the popliteal fossa (Pierrot-Deseilligny et al., 1979). The resulting Ib inhibition is contaminated by Ia excitation and reported as quite weak (Pierrot-Deseilligny et al., 1979;

Yanagawa et al., 1991; Delwaide et al., 1991; Downes et al., 1995; Stephens and Yang, 1996). The weak Ib inhibition was attributed partly to the superimposed monosynaptic Ia excitation which obscures the full extent of the inhibition. More recently, Burne and Lippold (1996a) showed that electrical stimulation over muscle tendons produces a strong reflex inhibition of a small voluntary contraction. The response consisted of a series of inhibitory and excitatory components I_1 , E_1 , I_2 , and E_2 and was described in several upper

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Abbreviations: DF, dorsiflexor; PF, plantarflexor; EMG, electromyogram; MVC, maximum voluntary contraction; TRE, electrically induced tendon reflex; TES, tendon electrical stimulation; GTO, Golgi tendon organ

limb muscles. This inhibition occurred with a relatively long latency (~55 ms in forearm extensors and ~95 ms in the tibialis anterior) leading to the suggestion that the suppression was due to an Ib mediated polysynaptic pathway.

In later studies, the initial inhibition (I_1) was shown to be reduced in some patient groups, especially Parkinson's disease (Burne and Lippold, 1996b), essential tremor (Burne et al., 2002) and dystonia (Priori et al., 2001). I_1 was also recently shown to inhibit common muscle cramp, providing evidence for the reflex origin of the EMG activity associated with cramp (Khan and Burne, 2007). These studies underline the clinical importance of Ib mediated effect in humans.

Since there is currently no satisfactory means to study Ib mediated effects in man independently of evoked Ia fiber activity (reviewed Pierrot-Deseilligny and Burke, 2005), their functional role and clinical importance is still poorly understood. Tendon electrical stimulation (TES) may thus provide a novel method for the further investigation of Ib effects in man if it can be shown to selectively activate tendon afferents.

Although the reflex afferents were localized to the tendon (Burne and Lippold, 1996a), some uncertainty remained concerning the species of tendon afferent mediating the response. While Burne and Lippold (1996a) suggested that Ib afferents may mediate the response to TES, it was later argued that the inhibition originates from group III tendon afferents (Priori et al., 1998).

Electrical stimulation of cutaneous afferents has also been reported to produce a complex reflex response consisting of excitatory and inhibitory components in man (Caccia et al., 1973; Garnett and Stephens, 1980, 1981) that are visible against a small voluntary contraction. It has therefore been suggested that the effects of TES may be partly or wholly due to cutaneous afferent stimulation (Floeter, 2003). Thus, further investigation is needed to identify features that distinguish between cutaneous and tendon afferent responses.

TES has not been investigated in lower limb muscles. The large muscle–tendon anatomy of the triceps surae group should be suitable to investigate the most effective sites of stimulation in relation to tendon anatomy. Described here is a study of the reflex response to electrical stimulation of the Achilles tendon in the two heads of the GA muscle. Stimulation at different locations on the tendon confirmed the responses' close relationship to tendon anatomy. The species of afferent mediating the reflex was investigated by comparing it over a range of stimulus intensities with the response to stimulation of the purely cutaneous superficial branch of the sural nerve. We then compared the timecourse of reflex depression in tendon and cutaneous afferents during progressive tibial nerve block.

As noted above, TES was shown to effectively inhibit common muscle cramp (Khan and Burne, 2007). Thus, a common pathway originating in tendon afferents modulates cramp-generated EMG and voluntary EMG activity. The cramp experiments confirmed the common observation that cramp is enhanced in the shortened muscle and relieved by passive muscle stretch, an effect that has been anecdotally attributed to the Golgi tendon organ (GTO) reflex though no solid evidence for this has been cited. We therefore investigated the relationship between the TES reflex and ankle joint angle in order to further implicate it in the mechanisms of cramp and the GTO reflex. If the magnitude of the TES reflex is strongly related to muscle length, then modulation of tendon afferent input may provide a mechanism for the effects of stretch on cramp.

We report here that electrical stimulation over the GA tendon produces a powerful reflex inhibition of the ongoing voluntary contraction in both heads of the GA muscle and we provide evidence that the origin is in large diameter tendon afferents. This result is consistent with the view that the inhibition may arise from Ib afferents, providing an effective means to further investigate the role of these afferents in conscious human subjects. This conclusion is further

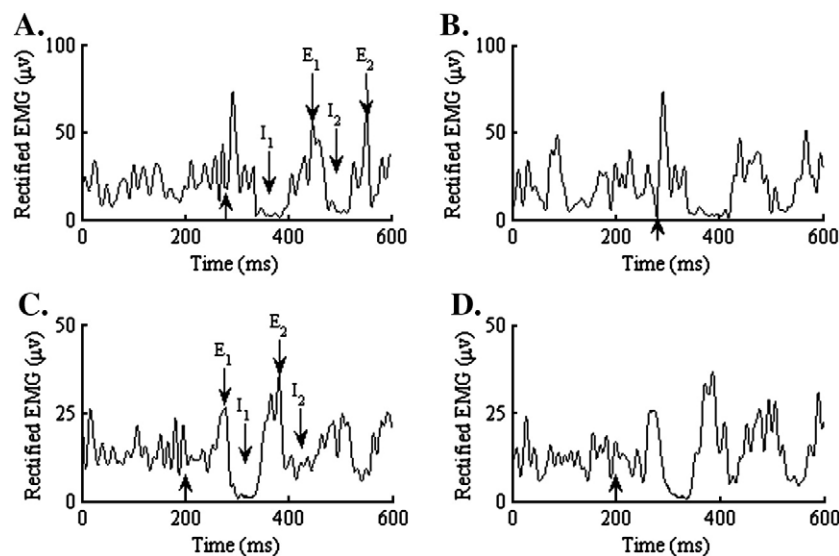


Fig. 1 – Tendon reflex response during a normal voluntary contraction of 20% of MVC in the medial (A) and lateral (B) heads of the GA muscle following proximal (90% of tendon length) electrical stimulation. Simultaneous inhibition in the medial (C) and lateral (D) heads of the GA muscle following cutaneous stimulation of the sural nerve. The ankle joint angle was 70°. The arrow on the time scale indicates the time of stimulation.

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