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BRAIN RESEARCH

Research Report

Inhibitory mechanisms following electrical stimulation of tendon and cutaneous afferents in the lower limb

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ABSTRACT

Electrical stimulation of the Achilles tendon (TES) produced strong reflex depression (duration > 250 ms) of a small background contraction in both heads of gastrocnemius (GA) via large diameter electrodes localized to the tendon. The inhibitory responses were produced without electrical (M wave) or mechanical (muscle twitch) signs of direct muscle stimulation. In this study, the contribution of presynaptic and postsynaptic mechanisms to the depression was investigated by studying conditioning effects of tendon afferent stimulation on the mechanical tendon reflex (TR) and magnetic motor evoked potential (MEP). TES completely inhibited the TR over an ISI of 300 ms that commenced before and continued during and after the period of voluntary EMG depression. Tendon afferent conditioning stimuli also partially inhibited the MEP, but over a short time course confined to the period of voluntary EMG depression. The strength and extended time course of tendon afferent conditioning of the TR and its failure to produce a similar depression of the MEP are consistent with a mechanism involving presynaptic inhibition of Ia terminals. Cutaneous (sural nerve) afferent conditioning partially inhibited the TR and MEP over a short time course (ISI < 100 ms) resembling the inhibition seen in the voluntary EMG. This was consistent with the postsynaptic origin of cutaneous inhibition of the motoneurons.

1. Introduction

There is currently no satisfactory means to study Ib-mediated effects in man. Nerve stimulation produced weak mixed responses from Ib and Ia afferents (reviewed Pierrot-Deseilligny and Burke, 2005). Thus, the functional significance of the Ib pathway in humans remains unclear. Our knowledge of autogenic Ib inhibition is largely derived from animal experiments, particularly from decerebrate and anesthetized cats (Jami, 1992; Proske, 1981).

Burne and Lippold (1996) showed that tendon electrical stimulation (TES) during an ongoing voluntary contraction produces a strong reflex depression. The electrically induced tendon reflex (TRE), consisting of a series of excitatory and inhibitory components, was shown in several upper limb muscles, particularly extensor digitorum communis and flexor pollicus. Although the contributing afferents were localized to the muscle tendon in these previous studies, their afferent type is still uncertain. Burne and Lippold (1996) suggested that Ib afferents may mediate the response, though

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Abbreviations: CSP, Cutaneous silent period; TR, Tendon reflex; TES, Tendon electrical stimulation; TMS, Transcranial magnetic stimulation; EMG, Electromyogram; MEP, Motor evoked potential; TRE, Electrically induced tendon reflex

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it was subsequently argued that the depression originates from group III tendon afferents (Priori et al., 1998). Recent lower limb experiments further implicated large diameter tendon afferents in mediating the TRE. These studies compared the TRE depression with that following cutaneous afferent (sural nerve) stimulation. Gastrocnemius (GA) depression produced by cutaneous stimulation was of higher threshold, longer latency and persisted after partial tibial nerve block when the TRE was completely depressed (Khan and Burne, 2009). The group mean threshold was 1.7× perceptual threshold for tendon inhibition compared with 3.4× perceptual threshold for cutaneous inhibition (Khan and Burne, 2009). Stimulus strengths of 7.6× the perceptual threshold produced a maximal TES reflex whereas 10–12× perceptual threshold (above pain threshold) was needed to produce a maximal cutaneous response. The onset latency for the first period of inhibition (I_1) was 47–49 ms and 90 ms for tendon and sural nerve stimulation, respectively. Further, the tendon response was lost completely over 10–25 min of ischemic nerve block when the cutaneous response to mild noxious stimulation persisted. It was thus concluded that the blocked tendon fibers were larger than group III, presumably of group I origin. Thus, the above experiments provided further support to the conclusion that large diameter tendon afferents gave rise to the TRE.

Khan and Burne (2007) recently reported that common muscle cramp could be inhibited by the TRE, leading to the conclusion that reduced inhibitory feedback from tendon afferents may have an important role in generating cramp. The TRE depression was found to be weakest under the same conditions that favour cramp. It is strongly dependent on joint angle, being weakest in the fully shortened muscle and negatively correlated with the mean background contraction (Khan and Burne, 2007) as previously reported for Ib inhibition of homonymous and synergistic muscles (Fournier et al., 1983; Pierrot-Deseilligny and Fournier, 1986; Stephens and Yang, 1996).

Surface electrical stimulation of cutaneous afferents also produces a complex reflex response consisting of excitatory and inhibitory components (Caccia et al., 1973; Garnett and Stephens, 1981, 1980) visible against a small voluntary contraction and apparent similarities in the cutaneous and tendon responses complicate the interpretation of earlier studies (reviewed Floeter, 2003). Hence, criteria that could distinguish the TRE from cutaneous reflexes are needed.

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 GA. The optimal stimulation

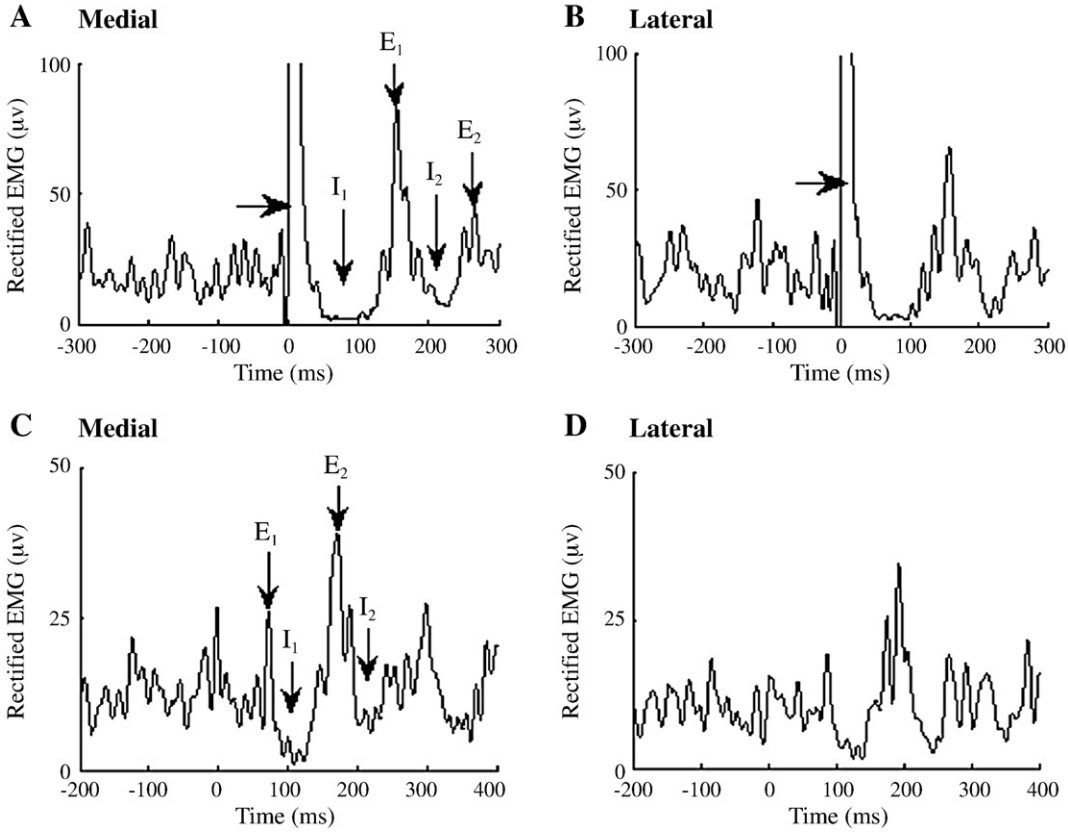

Fig. 1 – Effect of tendon stimulation on a small voluntary contraction of the medial (A) and lateral (B) heads of GA and simultaneous inhibition of the medial (C) and lateral (D) heads of GA following cutaneous sural nerve stimulation. The inhibitory and excitatory components following tendon electrical stimulation are represented by I_1, I_2, E_1 and E_2. The stimulus occurred at 0 ms on the time axis. The arrows represent the stimulus artifact.