In the Name of God

the Compassionate and the Merciful

Cutaneomuscular Reflexes in the Lower Limbs in Man

A Thesis Submitted for

the Degree of Doctor of Philosophy

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by

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Dedicated to
my wife and children

Dedicated to my father who
passed away during my
Ph.D course

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Abbreviations

Abductor hallucis	**AH**
Analogue to Digital	**A/D**
Cambridge Electronic Design	**CED**
Centigrade	**C**
Centimetre	**cm**
Cumulative sum	**cusum**
Cutaneomuscular reflex	**CMR**
Electromyography	**EMG**
First early excitatory component of response	**E1**
First early inhibitory component of response	**I1**
First dorsal interosseous	**FDI**
Gastrocnemius	**GAS**
Hertz	**Hz**
Hamstrings	**HAMS**
Hoffmann reflex	**H-reflex**
Kilogram	**Kg**
Kilo-Hertz	**KHz**
Kilometres per hour	**Km/h**
Maximal Voluntary Contraction	**MVC**
Meters per second	**m/s**
Milliampre	**mA**
Millisecond	**msec**
Microvolt	**µV**
Number	**n**
Perceptual threshold	**PT**
Personal computer	**PC**

Probability	**p**
Proprioceptive Neuromuscular Facilitation	**PNF**
Quadriceps	**QUAD**
Sensory Nerve Action Potential	**SNAP**
Second	**sec**
Second excitatory component of response	**E2**
Second inhibitory component of response	**I2**
Standard deviation	**SD**
Third excitatory component of response	**E3**
Tibialis anterior	**TA**
Volt	**V**

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Declaration and list of publications

The experimental work and other research which make up this thesis was carried out entirely by myself. No part of the material has previously been presented for any other degree.
Part of work contained in this thesis has been, or will be published as follows:

1. Bagheri, H. and Baxendale, R. H. (1993). Non-noxious cutaneomuscular reflexes in the human lower limb. Proceeding XXXIInd Congress. I.U.P.S, Glasgow **170.39/p** (Poster Communication).

2. Bagheri, H. and Baxendale, R. H. (1994). Does posture alter cutaneomuscular reflexes in the human lower limb. Journal of Physiology **479**, 29P, (Oral communication).

3. Bagheri, H. and Baxendale, R. H. (1994). The effects of the increasing of stimuli and intensity of contraction on the cutaneomuscular reflexes in tibialis anterior. International Symposium on Alpha and Gamma motoneurones in London, (poster communication, in Press).

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5. Bagheri, H. and Baxendale, R. H. (1994). The effects of stimulus intensity on the cutaneomuscular reflexes in tibialis anterior in man. Journal of Physiology **483**, 80-81 P, (Oral communication).

Summary

Mild electrical stimulation of the toes can produce cutaneomuscular reflexes in the muscles of the lower limb of normal adults (Gibbs et al, 1993). The general pattern is polyphasic with mixed excitations and inhibitions. In the present thesis an attempt was made to investigate the nature of the cutaneomuscular reflexes in different muscles of the lower limb such as tibialis anterior, gastrocnemius, quadriceps, hamstrings and abductor hallucis. The effects of site of stimulation, stimulus intensity, temporal summation, background force, posture and skin cooling were investigated on the nature and magnitude of the cutaneomuscular reflexes.

Experiments were performed in 62 healthy subjects aged between 20 and 38 years old. The subjects were seated, stood erect, or lay relaxed during experiments. Cutaneomuscular reflexes were elicited by stimulation of the hallux, the heel, the lateral border of the foot, the plantar surface of the foot and the shank at intensities up to three times perceptual threshold. The reflexes were identified as modulations in the averaged rectified surface electromyogram of the muscles under study.

Cutaneomuscular reflexes were recorded in tibialis anterior, gastrocnemius, quadriceps, hamstrings and abductor hallucis. Reflexes were elicited most frequently in tibialis anterior but even in this muscle there was some variability in the pattern of responses. Cutaneomuscular reflexes consist of up to four components designated: E1, I1, E2 and I2. The later excitation (E2) is the most consistent feature of reflexes

elicited by stimulation of hallux, whereas stimulation of skin innervated by the sural elicits early inhibitions (I1) in all cases. Cutaneomuscular reflexes were much less frequently observed in quadriceps and hamstrings.

The mean latency of the earliest component of cutaneomuscular reflexes is about 50 msec which suggests oligosynaptic spinal pathways. Later components have latencies of about 80 msec which would allow time for a supraspinal pathway. Pure excitations and inhibitions were seen in some subjects but mixed excitations and inhibitions were more common, particularly with stimulation at higher intensities. Cutaneomuscular reflexes can be elicited by single shocks but the amplitude increased significantly when a second or third stimulus pulse was added. The intensity of the background contraction against which the cutaneomuscular reflexes is elicited had a strong effect on the magnitude of cutaneomuscular reflexes. The magnitude of the reflexes was significantly greater when they were elicited during voluntary contraction in a seated position than when the muscle was posturally active during standing. Significant differences were found between the amplitudes of the different components of the cutaneomuscular reflexes as the standing position was adjusted.

The excitatory and inhibitory components of the cutaneomuscular reflexes were decreased during the cooling of the skin and were restored when the skin was rewarmed. Excitations were easier to abolish than inhibitions. This effect can be attributed to changes in the magnitude of the afferent volleys.

The results show that the E1, I1 and E2 are independent components of the reflex. In addition, it is suggested that there may be at least two populations of cutaneous afferents responsible for eliciting the mixed excitations and inhibitions observed in cutaneomuscular reflexes.

Cutaneomuscular reflexes are likely to be most effective when muscles are operating in a low force range at about 20% MVC or less of their maximum force. The results in this thesis also suggest that E2 component of cutaneomuscular reflexes is modulated in task-dependant manner.

INTRODUCTION

1. Introduction

1.1. General history

Mechanical and thermal cutaneous stimulation is employed during a number of physiotherapy treatment techniques to facilitate weak voluntary contraction of muscle during recovery from lesions of the central or peripheral nervous system. Cutaneous stimulation is an important component in manually resisted exercise techniques such as proprioceptive neuromuscular facilitation (PNF).

Probably the most famous sign in clinical neurology is the plantar response described by Babinski (1896). The Babinski response is a pathological response; it may be taken as an indicator that there is an abnormality of function in the central nervous system. His description was of a reaction to a nociceptive stimulus consisting of flexion at the hip and knee and dorsiflexion of the ankle and great toe (Walshe, 1956). Babinski stated that this sign indicates a disturbance of function of the pyramidal system. Investigation of reflex activity of the spinal cord began in the nineteenth century in experiments on the spinal and decerebrated cats by Charles Sherrington (1910). In parallel with the progress of animal experiments on the spinal organisation and supraspinal control of flexor reflexes, work continued on human flexor reflexes. Research in cutaneomuscular reflexes of the leg originated from studies of the Babinski sign and associated phenomena (Kugelberg, Eklund and Grimby, 1960, Grimby, 1963a, Meinck, Strehlow and Koehler, 1981).

Innocuous electrical stimulation of cutaneous afferents elicits reflex responses in the muscles of the upper and lower extremities of human

subjects (Caccia, McComas, Upton and Blogg, 1973, Jenner & Stephens, 1979, 1982; Gibbs, Shiers, Mallik, Harrison and Stephens, 1993). These responses were identified from averaged rectified electromyograms. The earliest responses have latencies consistent with spinal reflexes whilst the absence of late components in patients with neurological deficits led Jenner & Stephens (1979) to conclude that the cutaneomuscular reflex (CMR) had a supra-spinal component. These polyphasic CMRs elicited by innocuous stimulation are quite distinct from the protective withdrawal responses elicited by noxious stimulation as first described by Sherrington (1910) and then later investigated in the human lower limb by Hagbarth (1960).

1.2. Techniques for identifying spinal reflexes

Monosynaptic testing

Monosynaptic testing of motoneurone excitability was introduced by Renshaw (1940) and has been used by a number of investigators to examine reflex changes (Lloyd, 1943c, 1946b). Lloyd used the monosynaptic test technique to investigate the time course and linkage of the excitatory and inhibitory synaptic actions evoked from different afferent fibre groups. Gassel and Ott (1970b) reported excitability changes in a population of triceps surae motoneurones after cutaneous stimulation of the dorsal and plantar surfaces of the distal foot in humans.

H-reflex

The H-reflex, originally described by Hoffmann (1918, 1922), is the electrical analogue of the ankle jerk reflex. It is usually recorded from the soleus in the adult but may be recorded from the other muscles. The H-reflex is known as a monosynaptic reflex which can be used to assess the motoneurone pool excitability noninvasively. It can be evoked by using near motor threshold current to stimulate the group Ia afferent fibres from the muscle spindle. The H-reflex latency is a measure of the conduction delay along the sensory Ia afferent fibres, a central delay and a conduction delay along the motor axons. If stimulation at near motor threshold is done repeatedly, it excites the same motor neurone pool each time. Hence, the latency of the H-reflex is constant when recorded with surface electrodes (Andrews and Bruyninckx, 1986). In man, as in the cat, the electrical threshold and conduction velocity of group Ib

afferents are very similar to those of Ia afferents (Pierrot-Deselligny, Bergego, Katz and Morin, 1981) so that the H-reflex afferent volley will be heavily contaminated by Ib afferent activity (Burke, Gandevia and McKeon, 1983). They suggested that the H-reflex cannot be considered a purely monosynaptic reflex.

Spinal proprioceptive reflexes

Proprioceptive reflexes such as stretch reflexes evoked in man by abrupt stretch of a contracting muscle or by muscle vibration. The former produces a complex response which contains reflex activity of spinal origin, reflex activity of long latency, possibly of long loop origin (Burke et al, 1983). The tendon jerk reflex is a good example of a spinal proprioceptive monosynaptic reflex though it also contains polysynaptic components. The tendon jerk reflex can be elicited by a tendon tap direct on the tendon of the muscle under question such as quadriceps or triceps surae. Burke et al, (1983) believed that the afferent volley responsible for the ankle jerk and H-reflex is not a homogeneous volley in group Ia afferents from primary spindle endings in triceps surae. Both volleys are contaminated by activity in a wide variety of afferents, not only from other mechanoreceptors in triceps surae but also from mechanoreceptors in skin and in other muscles.

Single motor unit EMG recording

Single motor unit recordings have furnished insight into the intrinsic organisation of the reflex pathways. Garnett and Stephens (1980) studied the changes in the probability of firing of motor units active during voluntary muscle contraction in human first dorsal interosseous muscle in response to cutaneous afferent stimulation. The threshold of motor

unit recruitment was taken as that level of voluntary contraction strength at which the motor unit under study first began to fire steadily as the subject slowly increased his force of contraction over several seconds. The subject receives continuous visual feedback of his force output from an auditory monitor and frequency meter display. Meanwhile stimuli were delivered to the digital nerves and a peristimulus time histogram of the occurrence of motor unit spikes following each stimulus accumulated over a number of sweeps. They described a reflex response consisting of 3 phases, early excitation followed by inhibition followed by late excitation.

Peristimulus time histogram (PSTH)

During normal voluntary contractions, the discharge pattern of single motoneurones is determined by the summed effects of a very large number of different synaptic inputs. The effect of a particular input can be determined by constructing presentation of a suitable stimulus. This procedure extracts from the naturally occurring spike train only those changes in firing which are time-locked to the stimulus. The effect of a given input is revealed in terms of the specific contribution it has made to the total firing pattern of the cell. The detection of change in the discharge pattern of a neurone in response to a stimulus is commonly achieved by logging the times of occurrence of spikes relative to the stimulus. A histogram is constructed from the spikes occurring during the presentation of a number of stimuli. The result is referred to as the peristimulus time histogram (PSTH). In fact, the PSTH is a correlogram between a stimulus and the discharge train of impulses of a neurone and is formed from the sum of a number of trials (Ellaway, 1978).

The peristimulus time histogram (PSTH) provides a means of correlating the discharge of neurones with other events. Since Ellaway (1978), the cusum technique has been applied to PSTH (Buller, Garnett and Stephens, 1980, Garnett and Stephens 1980, Baxendale, Davy, Ellaway and Ferrell, 1991). The cumulative sum (cusum) derived from the PSTH facilitates the detection of small changes in the PSTH that may be obscured by random fluctuations in counts. The cusum integrates differences from the mean control level of counts in the PSTH. Any signal in the data that is related to the stimulus appears as a change in the slope of the cusum.

EMG modulation curve technique (surface EMG recording)

This technique is based upon the observation that during tonic activity, motor unit discharge increased with facilitation of the reflex and decreased with inhibition of the reflex (Gassel and Ott, 1969, 1970 a, b). Systematic application of the EMG modulation curve technique revealed oscillating sequences of excitation and inhibition with onset latencies often under 50 msec (Meinck et al, 1981).

Triphasic responses to cutaneous stimulation have been recorded by surface EMG from the first dorsal interosseous muscle by Jenner and Stephens (1979, 1982). Choa and Stephens (1982) extended this work to the lower limb examining the tibialis anterior. Gibbs et al (1993) investigated the reflex activity of the different muscles of the lower limb and trunk with surface electromyography.

Withdrawal flexion reflexes

The flexion reflex is a polysynaptic defence response to noxious stimulation of the skin. Sherrington (1910) examined the reflex responses to nociceptive stimuli in a series of classic experiments on the spinal and decereberate cat. He stated that even though the reflex is most readily elicited from the foot, it has a receptive field which includes the skin of the whole limb as far up as the groin in front, the perineum medially and the ischial region behind. He suggested that noxious stimulus within this field causes not only a contraction of the flexors of the hip, knee and the dorsiflexors of the foot and digits, but also a relaxation of antagonistic extensors and plantarflexors. He also concluded that the reflex exists as a protective mechanism to ensure appropriate withdrawal from potential harm and that the exact nature of this withdrawal, in terms of the muscles activated, varies according to the site and characteristics of the stimulus. This variation he referred to as local sign. He also showed examples of ipsilateral extensor reflexes. Ipsilateral extension reflexes could also be elicited from the skin or the cutaneous nerves. These extension reflexes were further investigated by Hagbarth (1952) who defined receptive areas for both flexor and extensor reflexes in the cat hind limb. He showed that extensor reactions were most readily elicited from localised regions over the extensor muscles themselves.

The pattern of the muscles activated in the reflex is in accordance with its biological function, which is withdrawal of the stimulated limb away from the offending stimulus.(Hagbarth, 1960; Kugelberg et al, 1960; Grimby, 1963a). Meinck et al (1981) elicited the withdrawal reflexes in the muscles of the lower limb by stimulation of the peroneal and tibial nerves, and named these "macroreflexes".

Non-noxious cutaneomuscular reflexes

Electronic averaging instruments are extensively used for investigating cortical evoked potentials and also muscle reflex potentials after sensory stimulation. To distinguish these modulations of averaged EMG from the gross limb movements in classical reflexes, innocuous CMRs have been called "microreflexes" (Meier, Schmidt, Nordmann, Humme and Dahm, 1973).These small amplitude responses evoked by sensory stimulation are not easily seen in the interference EMG record and 50 or more responses must be averaged to disclose the reflex (Meier et al, 1973).

Non-noxious electrical stimulation of cutaneous afferents from the fingers and toes is known to produce polyphasic reflex responses in the upper and lower limbs of the human subjects (Jenner and Stephens, 1979, 1982; Gibbs et al, 1993). These polyphasic responses have been classified as early components, E1 and I1 and late components as E2 and I2 following the convention introduced by Caccia et al (1973) and Stephens and Usherwood (1976) and Choa and Stephens (1982).

Parallel changes in motoneurone pool excitability in response to mild cutaneous stimulation have also been investigated using monosynaptic testing (Gassel and Ott, 1970 a). In addition, Garnett and Stephens (1980) demonstrated changes in the probability of firing of first dorsal interosseous motor units following skin stimulation.

Comparison of withdrawal reflexes and innocuous CMRs

CMRs elicited by innocuous stimulation are clearly distinct from the classical flexion reflex. These differences are in the levels of stimulus intensity which is painful if associated with flexion withdrawal reflexes and innocuous if associated with CMRs. This would seem to imply

involvement of different populations of afferent fibres and a different central pathways. It has been shown that Group A delta and C fibres are responsible for flexion reflexes (Kugelberg, 1948) whereas group II (A beta) fibres are responsible for innocuous CMRs (Chen and Ashby, 1993, Cody, Plant and Richardson, 1988, Garnett and Stephens, 1980). Another difference between flexion reflexes and CMRs is the pattern of muscle activation. Flexion withdrawal reflexes can be elicited without any activity in the muscles at rest whereas innocuous skin stimulation is never powerful enough to recruit any EMG in an initially inactive muscle. CMRs can be elicited in extensor muscles whereas flexion reflexes are associated with extensor inhibition and flexor excitation. Finally, flexion withdrawal reflexes are movements but CMRs modulate EMG without necessarily producing any movement.

1.3. Literature review

Latency of responses

The latency of the response and the nerve conduction velocity of the afferent and efferent fibres together can yield information about the central delay and the origin of the different components of the CMRs.

Techniques for identifying the latency of the CMRs

Evans, Harrison and Stephens (1990) recorded CMRs from FDI muscle following electrical stimulation of the digital nerves of the index finger in man. They measured the latency of E1 component of cutaneomuscular reflexes from stimulus artefact to the first sign of inflexion from the mean background EMG level. The I1 and E2 latencies were taken where the trace departed from the mean EMG level.

Chen and Ashby (1993) elicited reflex responses in upper limb muscles to cutaneous stimuli. They calculated the mean and 95 % confidence intervals of the 50 msec prestimulus EMG. The latencies of onset and termination of periods of excitation or inhibition in the poststimulus period were determined from the points where the post stimulus EMG went outside the confidence interval.

Garnett and Stephens (1980) elicited reflex responses in single motor units in human FDI muscle by cutaneous afferent stimulation. They recorded PSTHs and average rectified surface EMG simultaneously. They measured the latency of the cutaneous responses to points of inflexion in the cumulative sum.

Short latency reflex response (spinal pathway)

In man, the muscle afferent volleys which elicit short latency reflexes are not homogenous and can activate oligosynaptic pathways in addition to monosynaptic pathways (Burke, 1983). Tarkka (1986) suggested that there is sufficient time for transmission in oligosynaptic pathways both in electrically and mechanically elicited short latency reflexes. The short latency reflex following cutaneous nerve electrical stimulation occurred in the muscles of the foot with an onset latency of about 55 msec (Tarkka, 1986).

Long latency reflex response (supraspinal pathway)

Jenner and Stephens (1979, 1982) showed that damage to the motor cortex and dorsal columns results in exaggeration of the initial excitatory component of the cutaneous reflex (E1) and also in complete loss of the normal short latency inhibitory component (I1) and the prominent long latency excitatory component (E2) in small muscles of the hand. They concluded that the later components of CMRs are of supraspinal origin requiring transmission of afferent impulses through the dorsal column, a relay in the sensori-motor cortex and descending transmission to the lower motoneurone pool by way of the corticospinal tract.

Ohki,Suzuki, Ugawa, Uesaka, Sakai and Kanazawa (1994) investigated excitability changes of the motor cortex associated with the E2 phase of cutaneous reflexes in the first dorsal interosseous muscle using transcranial electrical and magnetic stimulation of the motor cortex in humans. They suggested that the motor cortical excitability is increased in association with the E2 phase. They supported the hypothesis of Jenner and Stephens (1979, 1982) that the E2 phase involves transmission in a transcortical pathway.

Comparison of latencies and central delay of CMRs in upper and lower limb

Jenner and Stephens (1982) showed that cutaneous reflex responses could be elicited in the human first dorsal interosseous in upper limb and extensor digitorum brevis in lower limb following electrical stimulation of the digital nerves of the index finger and second toe respectively. CMRs in FDI were triphasic, consisting of E1, I1, E2 whereas in EDB were biphasic, consisting of E1 and E2. They estimated the central delay of E1 in FDI to be 2.4 to 6.2 msec and 0.6 to 4.1 msec for EDB. Differences in latency between short and long latency excitatory components of the CMRs in FDI and EDB muscles ranged from 16 to 18 msec and 27 to 32 msec respectively. They concluded that for the foot, as in the hand, the central delay for the first excitatory component is so short that it must be a relatively simple spinal response.

These differences in latencies of short latency reflexes are consistent with differences in nerve conduction velocities, the length of the limb, and known synaptic delays. The short latency reflex latencies are also dependent on the individual distance of the stimulation and recording sites from the spinal cord

Central delay in CMRs pathway

Choa and Stephens (1982) reported the central delays for the different components of the reflex response to be 0-6 msec for E1, central delay for E2 should be 24-36 msecs , 38-48 msec for E2′ and 45-60 msec for I2 respectively. Jenner and Stephens (1982) showed that the estimated central delay for the initial excitatory components of the cutaneous reflex in extensor digitorum brevis muscle of the lower limb in normal subjects ranged from 0.6 to 4.1 msec with a mean of 2.3 msec.

Afferents responsible for reflex activation of motoneurones or eliciting CMRs

Little is known about the reflex pathways from cutaneous afferents to spinal motoneurones in normal subjects. The afferent axons in the digital nerves arise largely from receptors in the skin and to a lesser extent, from deeply situated receptors such as those in joint capsules and periosteum. The digital nerves do not contain fibres from muscle spindles and it is unlikely that Golgi tendon organs make a significant contribution, since these structures are usually found at the junction of a muscle belly with its tendon (Caccia et al, 1973, Jenner and Stephens, 1982, Chen and Ashby, 1993). Stimulation of mixed nerves like the median nerve activates both cutaneous, muscle and joint afferents (Upton, McComas and Sica, 1971).

Caccia et al (1973) suggested that the earliest excitatory and inhibitory (E1 and I1) responses in the small muscles of the hand are probably mediated by the smaller group II and group III afferent fibres. They believed the I2 inhibition results from activity in the larger group II fibres which are connected to cutaneous mechanoreceptors especially those in the tips of the fingers and thumb. They proposed that this late inhibitory reflex may operate through the fusimotor system.

Garnett and Stephens (1980) stated that the afferents responsible for the lowest threshold inhibition and excitation have been assumed to belong to fast conducting mechanoreceptor in the group II range.

Cody et al (1988) found that the cutaneomuscular reflexes in the extensor and flexor muscles of the wrist are mediated by large, myelinated cutaneous afferents.

When the circulation of the limb is arrested by a sphygmomanometer

cuff, conduction block occurs first in large fibres (Seneviratne and Peiris, 1968). The effects of ischaemia on the cutaneous reflexes in the small muscles of the hand was studied by Chen and Ashby (1993). They reported that the I1 and E2 components were mediated by larger fibres with faster conduction velocities than those responsible for the I2 components. Williams and Hayward (1981) also observed that the I2 from the digital nerve stimulation had a high threshold.

Distribution of the CMRs

Cutaneomuscular reflexes in the muscles of the upper limbs

The few reports dealing with CMRs in hand and arm muscles present a relatively uniform picture (Caccia et al, 1973, Garnett and Stephens, 1980). Digital nerve stimulation evokes alternating excitation and inhibition in hand muscles beginning with latencies at about 40 msec.

Caccia et al (1973) showed cutaneous reflexes in small muscles of the hand, abductor pollicis brevis, abductor digiti minimi, flexor digitorum muscles, biceps and triceps by electrical stimulation of the digital nerves and the finger tip. CMRs in human first dorsal interosseous muscle were recorded following electrical stimulation of the digital nerves of the index finger (Garnett and Stephens, 1980, Jenner and Stephens, 1982). CMRs from flexor and extensor muscles of forearm were recorded following electrical stimulation of the fingers (Issler and Stephens, 1983, Evans, Harrison and Stephens, 1989).

Becker, Hayashi, Lee and White (1987) recorded a complex reflex response during voluntarily contraction of flexor carpi radialis after electrical stimulation of nerves supplying the skin of the fingers.

The EMG reflex responses in antagonistic muscles of the human wrist flexor and extensor muscles evoked by stimulation of the digital nerves and the superficial radial nerve (Cody et al, 1988). Chen and Ashby (1993) recorded the CMRs in proximal and distal muscles of the upper limb, first dorsal interosseous, abductor pollicis brevis, forearm flexors and extensors, biceps, triceps, and in the posterior deltoid following stimulation of digital nerves of the middle finger.

Cutaneomuscular reflexes in the muscles of the lower limbs

Non-noxious electrical stimulation of the posterior tibial, peroneal and sural nerves produced polyphasic cutaneomuscular reflexes consisting of short and long latency excitations in anterior tibial muscles in man (Tarkka and Larsen, 1985).

In normal subjects, innocuous electrical stimulation of the digital nerves of the second toe evokes a polyphasic reflex activity in the intrinsic foot muscles such as extensor digitorum brevis (Jenner & Stephens, 1982) and in the leg muscles of the lower limbs, anterior tibial muscle, soleus and quadriceps (Choe and Stephens, 1982; Rowlandson and Stephens, 1985 a and b, Gibbs et al, 1993). Burke, Dickson and Skuse (1991) recorded CMR responses in tibialis anterior, soleus, biceps femoris and vastus lateralis. In the adults, CMRs consist of an early excitation (E1) followed by a reduction (I1) and a later period of excitation (E2) (Choa and Stephens, 1982). The first two components are believed to be mediated by activity in the oligosynaptic spinal pathways whereas the second excitatory component (E2) requires the integrity of the dorsal columns, sensorimotor cortex and corticospinal tract (Jenner and Stephens, 1982, Choe and Stephens, 1982; Rowlandson and Stephens, 1985 a and b; Gibbs et al, 1993).

Do cutaneous afferents play a role in motor control?

In the human lower limb afferents from cutaneous mechanoreceptors can affect the discharge of α-motoneurones by a direct reflex action (Hagbarth, 1960, Delwaide, Crenna and Fleron, 1981, Meinck et al, 1981; Jenner and Stephens, 1982, Kanda and Sato, 1983) or indirectly by action on γ motoneurones (Aniss, Diener, Hore, Burke and Gandevia, 1990) and interneurones in spinal reflex pathways (Pierrot-Deseilligny et al, 1981).

During bipedal stance cutaneous mechanoreceptors in the foot are subjected to intense forces that vary with body sway; during walking these forces vary with the phase of locomotion. Cutaneous afferents are therefore well placed to provide tonic modulation of motoneurone discharge during stance and phasic modulation during walking (Burke et al, 1991). Afferents from cutaneous mechanoreceptors may play a significant role in the feedback control of bipedal stance and locomotion.

CMRs are also prominent in the upper limb. Garnett and Stephens (1980) investigated the reflex responses of single motor units in the human first dorsal interosseous muscle following cutaneous afferent stimulation of the index finger. They concluded that some cutaneous afferents must activate interneurones with varied patterns of projections to the motoneurones. They also stated that for the cutaneous reflex pathways there are differences in the balance of excitatory and inhibitory sets of interneurones impinging on FDI motoneurones which can be related to the type of motor unit innervated. Their belief that these reflexes can be elicited from skin areas whose natural stimulation can be expected to be associated with movements involving the

respective muscles emphasises the important role that cutaneous input plays in modifying motor outflow during movement. Direct experiments on primates have demonstrated the presence of cells in the sensori-motor cortex that are active during individual finger movements. These cells are excited by cutaneous input from the fingers at very short latency by way of the dorsal columns and whose axons pass through the pyramids to excite motoneurones innervating hand muscles (Lemon, 1981).

Jenner and Stephens (1982) concluded that the transmission along the dorsal columns and the activity of the cortical cells accounts for the prominent late excitatory component of the cutaneous reflex (E2) which can be elicited from FDI muscle by stimulation of the digital nerves of the index finger. The spinal pathways mediating the E1 component are more active during grips than during relatively independent finger movements in childhood (Evans, Harrison and Stephens, 1986, 1988). They concluded that the pathways mediating the E2 component are more active in relatively independent finger movements than grips for a variety of muscles controlling finger movements. Cody et al (1988) revealed the contribution of these reflexes to the normal control of movements.

1.4. Anatomy

The following section of anatomy is based on the book, "Electrodiagnosis, An Anatomical and Clinical Approach" edited by Jennifer Chu- Andrews and Robert J. Johnson (1986).

Sensory innervation of foot and hallux

The tibial nerve arises from the same roots as does the common peroneal nerve except that one more root is added; thus its origin is from L4, L5, S1, S2 and S3. In the popliteal fossa the tibial nerve usually gives off three articular branches to the knee joint and five muscular branches to the plantaris, the medial and lateral gastrocnemius, the soleus and the popliteus. A cutaneous branch is also given off within the fossa. This nerve is the medial sural cutaneous or sural nerve. The medial sural cutaneous nerve unites with the peroneal communicating branch from the lateral sural cutaneous nerve which arises from the common peroneal nerve to form the sural nerve. The lateral sural cutaneous nerve distributes its fibres to the posterolateral aspect of the upper half of the leg . The sural nerve continues distally to pass immediately behind the lateral malleolus and then curves forward along the lateral side of the dorsal aspect of the foot. When the tibial nerve reaches the level of the medial malleolus, it passes deep to the flexor retinaculum and gives off the medial calcaneal branch, which pierces the retinaculum and supplies the skin of the heel. Before the nerve emerges from the lower end of the tunnel, it has divided into the medial and lateral plantar nerves. The cutaneous branches of the lateral plantar nerve supply the lateral portion of the sole of the foot, the fifth toe and half of the fourth toe. The medial

plantar nerve descends toward the sole of the foot and gives a branch of supply to abductor hallucis. As the nerve crosses the tendon of the flexor digitorum longus at about the level of the navicular bone, it gives off a proper digital branch to the medial side of the great toe. On its way to the hallux, this branch supplies both heads of the flexor hallucis brevis and gives cutaneous branches to the medial two thirds of the sole of the forefoot. Two or three centimetres further forward, the continuing trunk of the medial plantar nerve divides into three common digital nerves that pass toward the first, second and third interdigital clefts and then divide into proper digital branches, each supplying its respective side of the toes. The proper digital branch to the medial side of the fourth toe communicates with the proper digital branch from the lateral plantar nerve, which passes to the lateral side of that toe. As the common digital nerves reach the level of the heads of the metatarsal bones, each divides into two proper digital nerves. Each proper digital nerve then passes forward into its respective toe. The level of division of the common digital nerves is at approximately the anterior or distal border of the deep transverse metatarsal ligaments, in each interspace between the heads of the metatarsals.

Functional anatomy of the muscles of the lower limb

Tibialis anterior

This muscle is located on the anterior aspect of the leg. It is the first muscle about one or two centimetres lateral to the anterior border of the tibia. The action of this muscle is dorsiflexion and inversion of the foot. It is innervated by the deep peroneal nerve.

Triceps surae

Triceps surae muscles are located on the posterior aspect of the leg and consist of three muscles, medial and lateral gastrocnemius and soleus. The action of gastrocnemius is plantar flexion of the foot and assistance in flexion of the knee. The belly of the soleus can be seen on either side of the Achilles tendon at a level slightly below the lower margin of the lateral head of the gastrocnemius during plantar flexion of the foot. The action of soleus is plantar flexion of the foot.

Quadriceps

Quadriceps muscles are located on the anterior aspect of the thigh and is composed of four muscles, rectus femoris, vastus lateralis, vastus medialis and vastus intermedius. The action of rectus femoris is extension of the knee and flexion of the hip. The action of the quadriceps is extension of the knee joint.

Hamstrings

The hamstring muscles are located on the posterior aspect of the thigh and consist of semitendinosus, semimembranosus and biceps femoris. Semitendinosus and semimembranosus are flexors of the knee joint and also internal rotators of the leg when the knee is partially flexed. When acting from below on the fixed leg, the muscles are extensors of the hip joint, pulling the pelvis into an upright or extended position after bending forward. Biceps femoris is an extensor of the hip joint when acting from below with the leg fixed and is involved in flexion of the knee joint and external rotation of the leg on the thigh when the knee is partially flexed.

Abductor hallucis

Abductor hallucis is located on the medial side of the foot approximately 1 cm inferior and 1 or 2 cm posterior to the tuberosity of the navicular bone. This will be just anterior to an imaginary line drawn through the anterior margin of the medial malleolus. The action of abductor hallucis is abduction of the great toe at the metatarsophalangeal joint and aiding flexion at this joint.

1.5. Physiology of sensory functions in man

Sensory experiences occur when stimuli excite sensory receptors.
Sensory receptors are sensitive transducers of energy. Different forms of energy are transformed by the nervous system into different sensations or sensory modalities. There are four distinct somatic modalities: touch, which is elicited by mechanical stimulation of the body surface; proprioceptive sensations, elicited by mechanical displacement of the muscle and joints; pain, elicited by noxious stimuli; and thermal sensations elicited by cool and warm stimuli. Each somatosensory modality is mediated by a separate class of receptors. These receptors have reflex effects on spinal motoneurones mediated by interneurones.

Morphology of the cutaneous receptors

The morphology of the cutaneous receptors was investigated by Andres and Bochum (1973) and Iggo (1982). They classified the cutaneous receptors as functioning as mechanoreceptors, thermoreceptors and nociceptors. The mechanoreceptors may have receptors associated with the epidermis (e.g. Merkel receptors), in the outer layers of the dermis (e.g. Meissner corpuscles, Krause end bulbs), or the deeper layers of the skin (e.g. Pacinian corpuscle).

1. Cutaneous mechanoreceptors. These receptors mediate the sensation of touch and can be divided into two major functional groups according to the way they respond to sustained stimuli.

A. Slowly adapting receptors

a. The Merkel cells and Merkel disks. These form a characteristic unit in the mammalian epidermis and are present in both glabrous and hairy skin. These slowly adapting type I mechanoreceptors (Iggo, 1966) such as Merkle's receptor in the superficial glabrous skin adapt slowly to sustained stimulus.

b. Ruffini endings. These are spindle shaped structures which lie in the epidermis both in human glabrous and hairy skin. Each Ruffini ending is supplied by one myelinated axon. The Ruffini endings have been identified as the receptors of the slowly adapting type II cutaneous receptors in the cat (Iggo, 1966). Similar functional properties have been found for mechnoreceptors in human skin, both hairy and glabrous (Vallbo and Hagbarth, 1968).

c. Free nerve endings. These are free or naked sensory endings. Unmyelinated receptors are the most difficult cutaneous receptors to study morphologically. The cutaneous unmyelinated afferent fibres innervate a variety of receptors: C-mechanoreceptors, C-nociceptors and C-thermoreceptors.

B. Rapidly adapting receptors

a. Pacinian Corpuscle. This is a large cutaneous mechanoreceptor and has the typical structure of an encapsulated receptor. The function of the

Pacinian corpuscle is to act as a very rapidly adapting mechanoreceptor. The afferent units are present in human skin (Vallbo and Hagbarth, 1968) and their physiological characteristics indicate that they mediate the human sense of vibration.

b. Meissner corpuscle. These are relatively large receptors occupying the dermal ridges in primate glabrous skin. They are largest in human tissue. The function of Meissner's corpuscles in man is to act as rapidly adapting receptors (Vallbo and Hagbarth, 1968) and they respond at the onset and often also at the end of a period of mechanical stimulation. They do not respond throughout the whole duration of the stimulus

c. Hair follicle mechanoreceptors. The principal mechanoreceptor of the hairy skin is the hair follicle receptor. Hair follicle mechanoreceptors range in size from simple follicles to the morphologically very complex sinus hairs. The function of hair follicles is to respond to hair movement.

d. Krause endings. These are in the form of globular or spherical end bulbs in man and monkeys. The function of Krause endings is to act as cold receptors. The afferent axons are myelinated and 5-7 μm in diameter.

Subcutaneous tissue beneath both hairy and glabrous skin contains two types of mechanoreceptors: the Pacinian corpuscle, a rapidly adapting receptor, and Ruffini's corpuscle, a slowly adapting receptor. The axons of all these receptor types are myelinated.

2. Cutaneous nociceptors. Little is known about the morphology of nociceptors. They have small myelinated or unmyelinated axons. These respond selectively to stimuli that can damage tissue. Three types of nociceptors can be distinguished on the basis of the response to different stimuli. Aδ mechanical nociceptors are supplied by finely myelinated axons and activated by strong mechanical stimulation. Thermal nociceptors respond selectively to heat or cold and the C polymodal nociceptors are supplied by unmyelinated fibres and respond to several different kinds of noxious stimuli such as mechanical, thermal and chemical stimuli.

3. Cutaneous thermoreceptors. The two types of thermoreceptors in the skin are cold receptors and warm receptors. Cutaneous cold receptors are activated from approximately 1° to 20° C below normal skin temperature. Cold receptors are supplied by small myelinated axons. Warmth is mediated by a separate population of thermal receptors that are selectively activated by a range of temperatures between approximately 32° C and 45°C. Warm receptors are supplied by unmyelinated axons.

Physiological classification of sensory nerve fibres in man

Nerve fibres can be classified according to fibre diameter, thickness of the myelin sheath, and speed of conduction of the nerve impulse. In general, the greater the diameter of the fibre, the thicker the myelin sheath and the faster the conduction velocity. A system of nerve fibre classification, pertaining only to sensory fibres, is frequently used by sensory physiologists and includes four groups I, II, III, and IV. These are differentiated chiefly on the basis of fibre size and conduction velocity as well as the origins of the afferent.

Muscle afferents: These afferents are mainly in groups I and II. Group Ia fibres originate from the primary afferents or annulospiral ending of muscle spindles and group Ib fibres originate from Golgi tendon organs. Group II fibres originate from secondary afferents of muscle spindles.

Cutaneous afferents: These afferents consist of group II, III and IV. Group II fibres are myelinated fibres originating from discrete cutaneous tactile receptors, pressure receptors and Pacinian corpuscles. Group III fibres are small, lightly myelinated fibres mediating touch, pressure, pricking pain sensations and temperature. Group IV fibres, which are unmyelinated fibres, transmit pain, itch, and temperature. Table 1 summarises the histological and functional classification of the cutaneous fibres.

Table 1: Histological and functional classification of skin receptors. The material for this table is from chapter 34 of the book "Principles of Neural Science" edited by Kandel and Schwartz (1985)

Functional	Histological	Receptors	Fibre	Quality
Rapidly adapting	Pacinian	Mechanoreceptor	Aβ (II)	vibration
	Meissner	Mechanoreceptor	Aβ (II)	flutter
	Krause	Mechano-thermal	Aδ (III)	cold
	Hair follicle	Mechanoreceptor	Aδ (III)	flutter
Slowly adapting	Merkel	Mechanoreceptor	Aβ (II)	steady skin indentation
	Ruffini	Mechanoreceptor	Aβ (II)	steady skin indentation
	Naked Endings		C (IV)	slow burning pain,Warmth
Nociceptors		Mechanical	Aδ (III)	sharp, pricking pain
		Thermal & Mechanical	Aδ (III)	sharp, pricking pain
		Thermal & Mechanical	C (IV)	slow, burning pain
		Polymodal	C (IV)	slow, burning pain

Conduction velocity of sensory nerve fibres in the lower limb in man

From human investigations, it is known that the conduction velocities of the nerve fibres are different from those in animals. O'Sullivan and Swallow (1968) stated that the human sural nerve contains essentially two types of fibre. The first belonging, to group II (A beta), is formed by axons whose diameter lies between 9 and 13 μm; the second, belonging to group III (A delta), has axons with diameters between 3 and 6 μm. There are few C fibres in the human sural nerve. Conduction velocities of group II and III fibres are from 30 to 55 m/sec and 7.5 to 30 m/sec respectively. Lovelace, Myers and Zablow (1973) measured the sensory conduction velocities between the ankle and knee of tibial and common peroneal nerves in man. They reported sensory velocities of 49.7 ± 2.3 m/sec for posterior tibial nerve and 50.8 ± 5.5 m/sec for peroneal nerve. Group I muscle afferents in the tibial nerve conduct about 5-10 m/sec faster than cutaneous afferent fibres in the sural nerve in human lower limb.(Burke, Skuse and Lethlean,1981, Burke, Gandevia, McKeon and Skuse, 1982, Gandevia, Burke and McKeon, 1982, Burke et al, 1983). The conduction velocities of the muscle and cutaneous afferents in tibial nerve were also reported by Macefield, Gandevia and Burke (1989) to be 54.7 ± 3.4 and 52.8 ± 3.2 m/sec respectively. Shefner and Logigan (1994) also reported the mean conduction velocities for Ia muscle afferent fibre was 57.6 m/sec and 55.1 m/sec for group II cutaneous afferents in the human sciatic nerve. The conduction velocity of C fibres in peroneal and saphenous nerves was reported at a range of 0.4-1.8 m/s in the lower limb in man (Torebjork and Hallin, 1976). Table 2 summarises the conduction velocity of cutaneous fibres in man.

Table 2. Classification of sensory nerve fibres in man. The data are based on the works of Torebjork, H. E., and Hallin, R. G. (1976), Martin, J. H., and Jessell, T. M. (1985), Shefner, J., and Logigian, E. L. (1994)

Sensory fibres	Sensory fibres	Estimated largest fibre diameter (μm)	Estimated fastest conduction velocity (m/s)	Quality
A-α	I	10	58	The primary afferents of muscle spindle
A-β	II	9	56	The secondary afferents of muscle spindle, touch, pressure receptors and pacinian corpuscles
A-δ	III	5	25	Small, lightly myelinated fibres; touch, pressure, pain and temperature
C	IV	1.5	2	Unmyelinated pain and temperature fibres

1.6. Aims of study

In the present thesis, an attempt was made to investigate the nature of cutaneomuscular reflexes in the different muscles of the lower limb and identify the latency and central delay of the different components of CMRs in tibialis anterior. Several investigations were performed to identify the factors influencing the nature and magnitude of the CMRs by posing the following questions:

Do cutaneomuscular reflexes change with graded increases of intensity of stimulation?

The effect of graded increases in stimulus intensity up to 3.5 PT was investigated on the magnitude of the CMRs in the tibialis anterior. The effect of repetitive stimuli on the size of the CMRs in the tibialis anterior was also examined.

Does the nature of CMRs alter with different sites of stimulation?

An attempt was made to produce cutaneomuscular reflexes in the tibialis anterior by using different skin sites of the toes and foot. In addition, CMRs were recorded simultaneously in up to 4 muscles.

Does the magnitude of CMRs change with increasing background force?

An attempt was made to observe the effect of the background activity from low level of activity at 5% MVC to higher level of activity at 30-40% MVC on the magnitude of the CMRs in the tibialis anterior.

Does posture alter the cutaneomuscular reflexes in the lower limbs?

The influence of body position or task-dependent effects on the size of the CMRs in the tibialis anterior was investigated during sitting and standing.

Does skin cooling change the nature of CMRs?

The effect of skin cooling of the foot on the magnitude of the CMRs in the tibialis anterior was investigated . The effects of skin cooling on the afferent volleys were also examined. Similarly, surface neurograms were used to record the afferent input via the tibial nerve to measure whether any effects of skin cooling were due to a peripheral or central phenomenon.

METHODS

2. Methods

2.1. Subjects

Recordings were made from neurologically normal male subjects aged between 20 and 38 years. Details of the subject's age and height are given in table 3. The experimental procedures were fully explained to the subjects beforehand. They were free to discontinue the experiment at any time without giving a reason. The experimental protocols were approved by the West Ethical Committee from Greater Glasgow Health Board.

2.2. Positions of Subjects

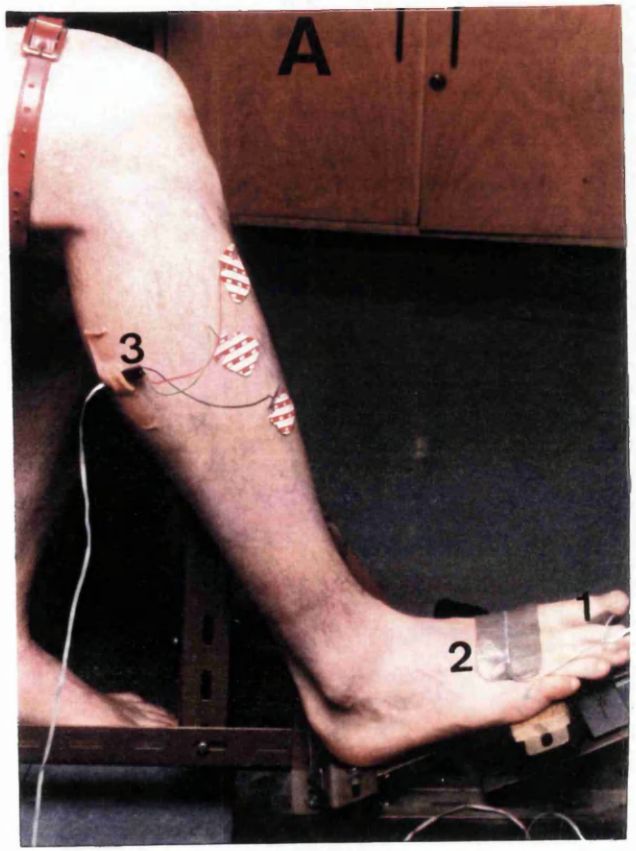
Experiments were performed with subjects in one of 3 positions:

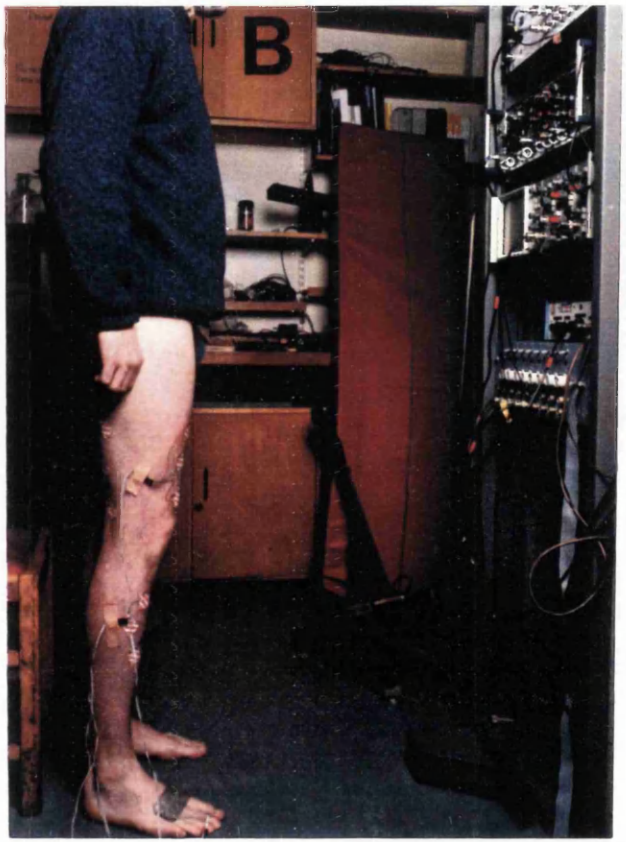
1. Seated in a relaxed semi-reclined position with one leg fixed to a supporting metal frame by straps to prevent lateral or medial rotations. The hip and knee were held at 110° of flexion and ankle at 90° of flexion throughout the experiment.
2. Standing with the hip in a neutral position, knee in full extension and ankle at 90° of flexion. In this position, cutaneomuscular reflexes were elicited whilst subjects leaned slightly forwards or backwards by about 5-15 degrees.
3. Lying relaxed with the hip in extension, knee in extension and ankle in neutral position (figure 1).

Subject Number	Age (years)	Height (cm)
1	37	165
2	37	172
3	36	185
4	36	182
5	33	185
6	33	160
7	21	167
8	20	170
9	21	175
10	38	175
11	20	183
12	35	185
13	22	185
14	21	170
15	21	175
16	21	165
17	22	168
18	21	173
19	21	180
20	21	183
21	21	185
22	22	175
23	29	170
24	22	175
25	30	170
26	22	185
27	22	180
28	39	173
29	38	175
30	21	165

Mean ± SD	**26.7 ± 7.2**	**175.2 ± 7.4**
Range	**20-39**	**160-185**

Table 3. The ages and heights of the 30 experimental subjects used in these investigations.


A
3
2
1


B

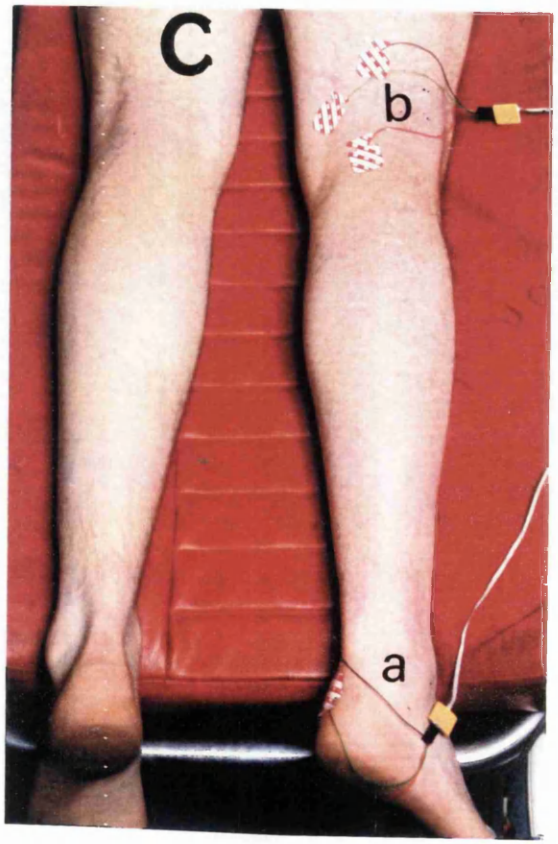

C
b
a

Figure 1. These photographs show the position of the subjects during experiments. A. sitting position , B. standing position and C. lying position. In the seated position, the leg was fixed to a supporting metal frame. The cathode (1) was placed on the skin of plantar surface of the hallux and the anode (2) on dorsum of the foot. Recording electrodes (3) were placed proximally on the bellies of the anterior tibial muscles and the reference electrode about 4 cm distal to the recording electrode. The ground electrode was attached to the skin between the recording and reference electrodes. In the standing position, subjects leaned slightly forwards or backwards by about 5-15 degrees. In the lying position, the recording electrodes were placed on the skin overlying the posterior tibial nerve just behind the medial malleolus at the ankle joint (a), and the popliteal fossa at the knee joint (b).

Subjects were advised to keep their body positions stable and as relaxed as possible. In addition, surface electromyogram (EMG) and force recordings were used to confirm the absence of significant muscle activity. The foot was strapped to a metal plate which was mounted on a strain gauge transducer to allow measurement of plantar flexion and dorsiflexion torques. Figure 1 shows the lower limb position while seated. In all experiments, the force signal was displayed to the subject on an oscilloscope. The temperature of the lab was controlled between 22-25°C.

2.3. Multi-Channel EMG System

2.3.1. Recording Surface Electromyography (EMG)

The EMG was recorded from several muscles of the right lower limb, tibialis anterior muscles (TA), gastrocnemius (GAS) and abductor hallucis (AH) in the sitting position, TA ,GAS, quadriceps (QUAD) and hamstrings (HAMS) in the standing position (backward and forward leans). EMG was recorded with metal foil Littman Electrodes (3M Ltd). These are disposable single-use adhesive electrodes (2x2 cm) for diagnostic purposes. They were attached over the belly of muscles with the recording electrode placed proximally and the reference electrode about 40 mm apart from the centre of the distal to the centre of the recording electrode. The ground electrode was attached to the skin between the recording and reference electrodes. The location of these electrodes is shown in figure 2. The skin over the recording site was

swabbed with alcohol prior to attachment of electrodes in an attempt to reduce resistance between the skin and electrodes. The position of the electrodes was adjusted with the subject at rest i.e. no muscular activity in the muscles under examination to ensure background noise was as small as possible. The electrodes were connected to a preamplifier (gain x1000) positioned within a few millimetres of the skin surface by the bare end of thin wires and from there signals entered the multi-channel EMG system which has an opto-isolation stage to ensure safety for the subject. This system provided a 50 Hz notch filter to reduce mains noise and band pass filtered the signal between 10 Hz-5 KHz. It also provided additional amplification. The amplified filtered EMG was digitised by a CED 1401+ interface. This provided 12 bit voltage resolution at a sampling frequency of 1000 Hz per channel.

The digitised EMG channels were rectified and peristimulus time averages of 500 sweeps were calculated using CED Sigavg software. Each average typically showed 100 msecs before the stimulus and 200 msecs after. Rectified integrated EMG or force output was also displayed on an oscilloscope (Tektronix Guernsey Ltd, model 5103 N) to allow visual feedback of muscle activity for the subject. The general experimental set up is illustrated in figure 3.

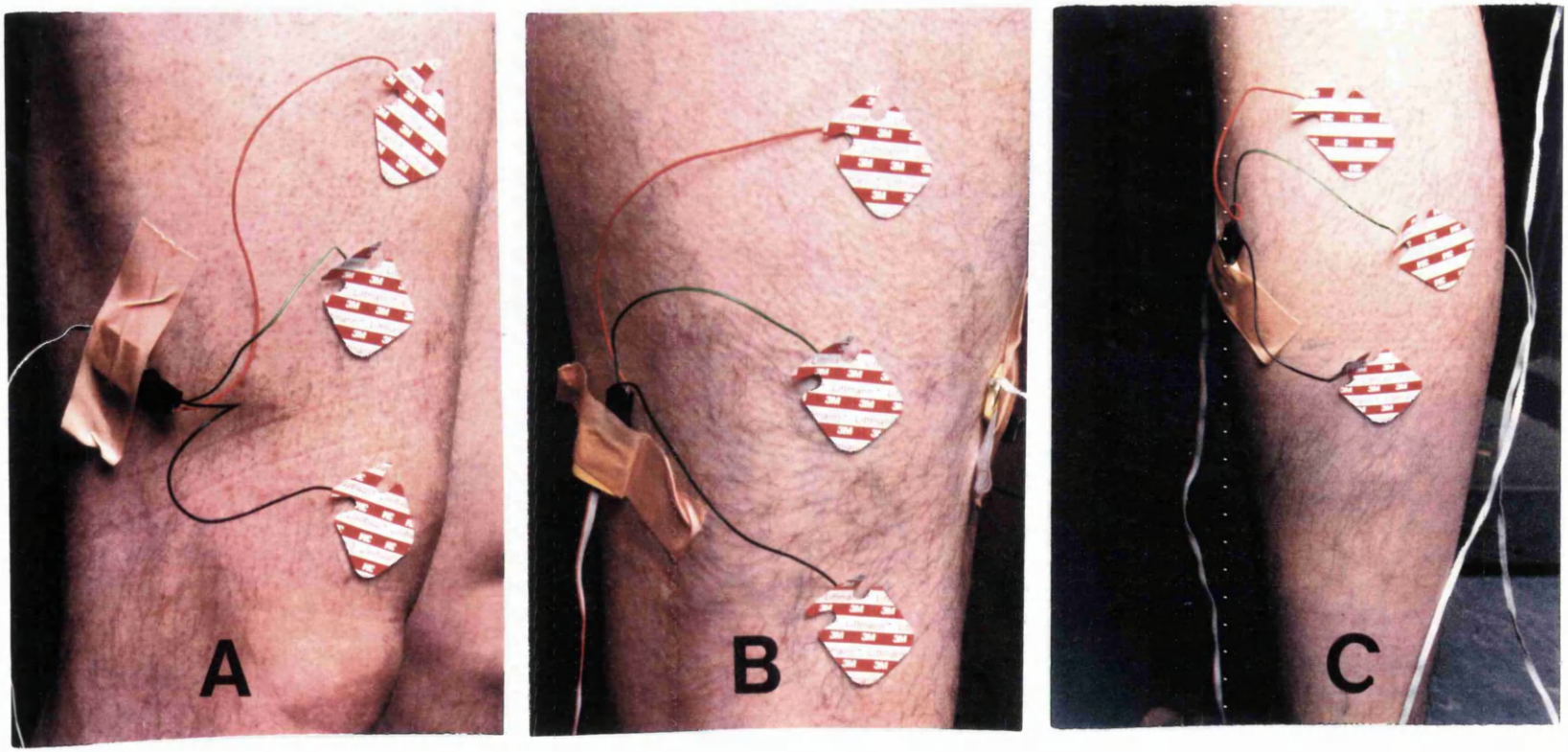

A
B
C

Figure 2: These photographs show the location of the recording electrodes on the bellies of the other muscles of the lower limb, A. quadriceps, B. hamstrings and C. gastrocnemius. The recording electrode was placed proximally on the belly of the muscles and the reference electrode about 4 cm distal to the recording electrode. The ground electrode was attached to the skin approximately between the recording and reference electrodes.

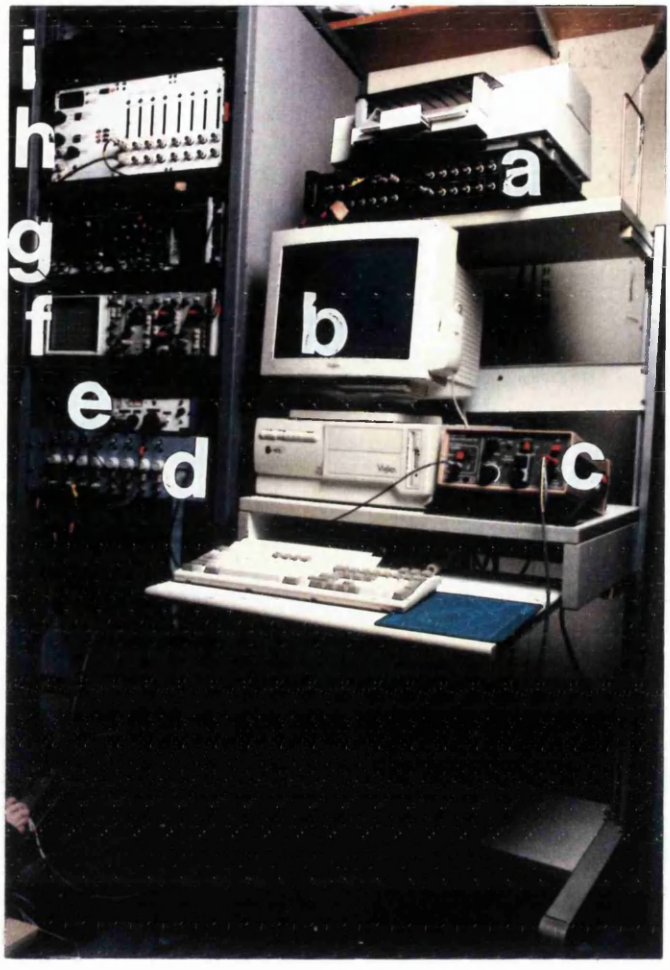


Figure 3. The general experimental set up. The instruments shown include: a. 1401 + Interface, b. IBM PC, C. Stimulator (Digitimer DS7), d. EMG, e. Transducer Amplifier, f. Oscilloscope, g. Neurolog (Pulse Generator), h. PCM8 video adapter, i. Video Recorder.

2.3.2. Surface Neurogram

Surface neurograms were used to examine the afferent volleys set up by skin stimulation. The recording proceeded in a manner similar to that described in section 2.3.1 but this time the subject lay relaxed to ensure no EMG activity would be picked up by the electrodes. The subject lay in a prone position to improve the signal to noise ratio during recording of the compound sensory nerve action potentials. In this posture, interference from muscle electrical activity was kept at a minimum (Jenner and Stephens, 1982). Surface electrical activity from the skin overlying the posterior tibial nerve just behind the medial malleolus at ankle joint and popliteal fossa at knee joint of the right leg was recorded using the same equipment as previously. The recording electrode was placed over the tibial nerve and the reference electrode placed 4 cm proximally or laterally along the nerve separated by a ground electrode in an attempt to reduce background noise. This is the optimal configuration described by Edurdo and Burke (1988). They concluded that the distortions were least with the 4 cm inter-electrode separation, particularly for short conduction distances. The signal was amplified x100 by the multi-channel EMG system giving a total amplification of x 10^5, because the SNAP is much smaller than the EMG from the active muscle. The signals were digitised with a PCM8 video adapter and stored on video tape (Sony, SLV-373 UB) for off-line analysis. The signals were replayed through Neurolog filters module (NL 125/126). This provided an active notch filter of 50-60 Hz and band pass filtered

the signal between 100 Hz-2 KHz. The signals were then digitised and averaged using CED 1401+ interface and Sigavg software.

2.3.3. EMG and Neurogram Averaging Technique

In order to identify and measure the latency and magnitude of the reflex components and improve the signal to noise ratio, peristimulus time averaging was used over 500 sweeps. The duration of each sweep was 300 msecs, 100 msecs before the stimulus and 200 msec after. The sampling rate was 1000 Hz. The neurogram required a post stimulus average of 4000 sweeps. The duration of each sweep was 30 msecs and the sampling rate was 5000 Hz.

2.4. Electrical Stimulation

2.4.1. Type of Stimulating Electrodes

Two types of stimulating electrodes were used :

A. Ring Electrodes

These are stimulating electrodes designed to fit around the digit like a ring to allow stimulation of the digital nerves (Medelec type, part number 16639, E/DS-K). Two ring electrodes were attached to the great toe with the cathode proximal and the anode distal to the interphalangeal joint. A photograph of the arrangement is shown in figure 4.

B. Pad Electrodes

Discs electrodes 50 mm in diameter or rectangular electrodes (90 mm x 50 mm) were used to stimulate skin. These were woven stainless steel mesh electrodes secured to the skin by an integral adhesive conductive gel (Axelgaard Manufacturing Co Ltd). The disks were used as cathodes, the rectangles were used as anodes.

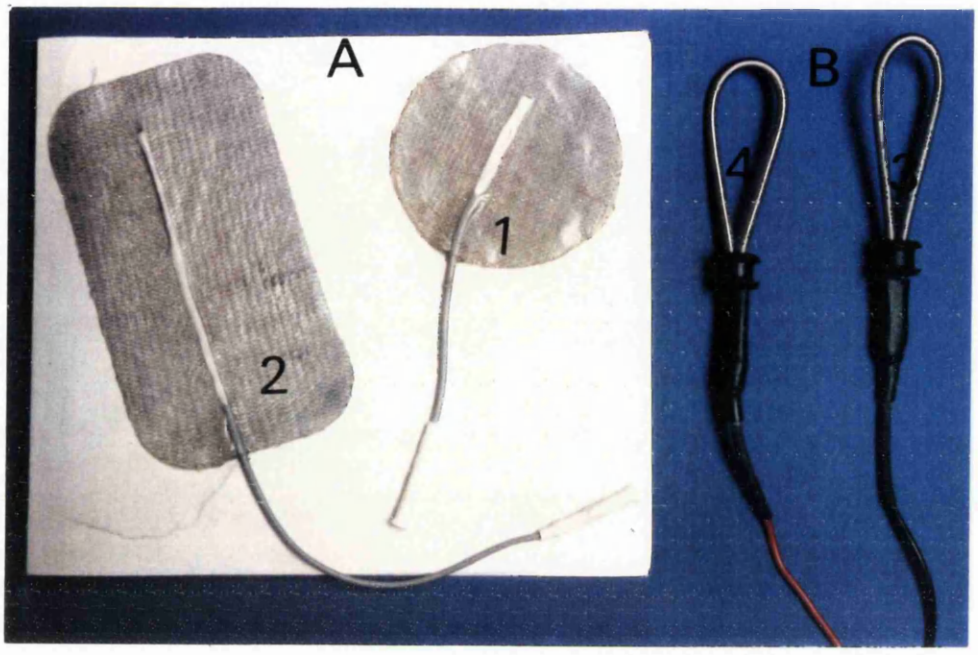


Figure 4. The two types of stimulating electrodes used in experiments described in section 2.4. A: Pad electrodes. B: ring electrodes. In A, the disks electrodes, 50 mm in diameter, were used as cathodes (1) and the rectangular electrodes (50 mm x 90 mm) were used as anodes (2). In B, two ring electrodes were attached to the great toe with the cathode (3) proximal and the anode (4) distal to the interphalangeal joint.

2.4.2. The Location of Stimulating Electrodes

Stimuli were applied at various points on the lower limb. The cathode was a 50 mm diameter disc placed over:

i. Skin covering plantar surface of hallux.

ii. Skin covering plantar surface of heel.

iii. Skin covering lateral border of the foot in the sural region.

iv. Skin covering plantar surface of the foot at mid-foot.

v. Skin covering anterior aspect of leg on anterior tibial muscles on the lower third of the shank.

The anode was rectangular 90 mm x 50 mm, and it was placed over the dorsum of the foot in i and iv and 4 cm distal to the cathode in ii, iii and v.

2.4.3. Stimulus Intensity

The experimental protocol involved stimulation of skin covering the different areas of the foot at different intensities from perceptual threshold up to 3 PT. Such a stimulus is not painful and produces a readily identifiable reflex response after a few minutes of averaging.

In each case the maximum stimulus intensity was kept sub motor threshold to avoid direct activation of skeletal muscle. The stimulus intensity was up to three times perceptual threshold (PT) for the hallux, up to 2 PT for the plantar surface of the foot, heel , sural region and 1.5 PT for skin covering the anterior tibial muscles.

2.4.4. Constant Current Stimulator

A Digitimer DS7 constant current high voltage stimulator (Digitimer Ltd) provided pulses of brief duration for percutaneous stimulation during the investigation of the cutaneomuscular reflexes. Pulse durations of 100 μsec were used to minimise any discomfort to the subject. The output current was continuously variable over the range 0-100 mA from a source voltage continuously variable up to 400 V. With constant current stimulators, the voltage changes according to skin impedance to keep the amount of current delivered constant. The stimulus was delivered through an isolation transformer to ensure safety for subjects and to reduce the amplitude of the stimulus artefact. In addition, the DS7 stimulator provides very fine control of the output current via a multi-turn potentiometer. This enables very precise estimation of threshold currents. Figure 5 shows the calibration curve for the stimulator.

2.4.5. Stimulus Timing

The DS7 stimulator was triggered by a digital timing unit (NL 300) which also triggered the CED 1401+ interface (average) at the same time. The stimulus frequency was about 2 Hz. The precise frequency was 1.93 Hz. This was chosen for subject comfort, to allow a 300 msecs sweep to be recorded and to avoid synchronisation at sub-harmonics of the mains frequency. When the surface neurogram was investigated, the stimulus frequency was about 10 Hz. This allowed a shorter averaging period, but the higher frequency made it possible to collect many more repetitions for averaging.

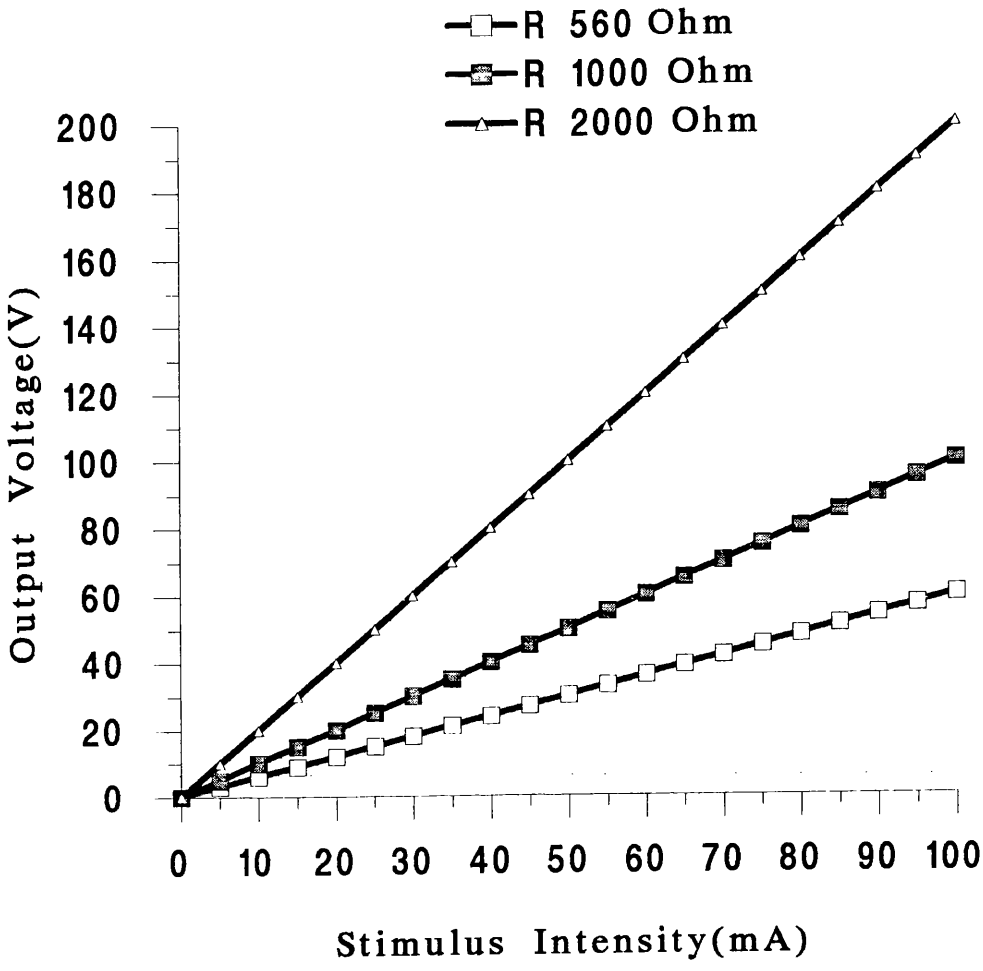


Figure 5: This figure shows calibration of the stimulator with different load resistances; 560 Ω (white squares), 1000 Ω (grey squares) and 2000 Ω (white triangles). The response is linear at each resistance (r^2=1).

2.5. Data Capture

CED 1401+ interface displays and averages the raw data. It also performs full wave rectification of incoming data and averages it.

2.5.1. Digitisation and Analysis of the Data

The amplified filtered EMG, or surface neurogram, were digitised by a CED 1401+ interface. This was described earlier in section 2.3.1. Data files were stored on disc and transferred to Excel spreadsheets for further analysis. First of all, the presence or absence of a reflex component was judged by eye; the presence was indicated by an obvious deviation from the mean level of EMG before and following the reflex response. In addition, cumulative sums of deviations from the mean EMG were calculated. The raw signals and the cusum were subjected to the statistical tests described below.

2.5.2. 95% Predictive Interval

The procedure for calculating the 95% confidence interval was based on the method of Chen and Ashby (1993). The averaged rectified EMG was displayed with a 95% predictive interval based on the mean of the background of the prestimulus period ± two standard deviations. An example of this is shown in figure 6.

2.5.3. The Cumulative Sum

This technique was introduced by Ellaway (1976, 1978). It is a simple statistical procedure that enables small changes in a data series to be observed amongst a large scatter. The cusum is the cumulant of deviations from the EMG. Thus even small changes are detected because their sum becomes large. In this application, the baseline was the average of the EMG during the 100 ms preceding the stimulus; hence the cusum always had the value of 0 at the time the stimulus was applied. The cusum is derived by subtracting a reference level from each datum point and summing these differences consecutively. This can be expressed mathematically as follows:

$$S_i = \sum_{x=1}^{i} (x_i - k)$$

Where S_i is the cusum series and k is the reference level or control mean, is subtracted from each of the series of points (x_i) on the average of EMG. An example of the technique is shown in figure 6. The gradient of the cusum represents the difference between the reference mean and the mean of the period of changes and thus changes in the cusum gradient are indicative of changes in the mean level. The advantage of the cusum is that it smoothes the data without any alteration or distortion. Therefore, the real worth of the cusum lies in amplifying and highlighting regions of change. It can also be useful in the measurement of latency. The disadvantage of the cusum is that it is much affected by short periods of great variation such as those arising from stimulus artefacts. Therefore, cusum calculation was suspended during the period

of stimulation so as to prevent the stimulus artefact introducing error. It is appropriate, therefore, always to analyse cusum traces in the context of the original data.

2.5.4. Statistical Analysis

The aim of statistical analysis is to compare the latencies and the magnitudes of the different components (E1, I1 and E2) of CMRs during different experiments i.e., sitting and standing. In order to compare the groups, Student's t-tests were carried out and $P<0.05$ set as the significance level.

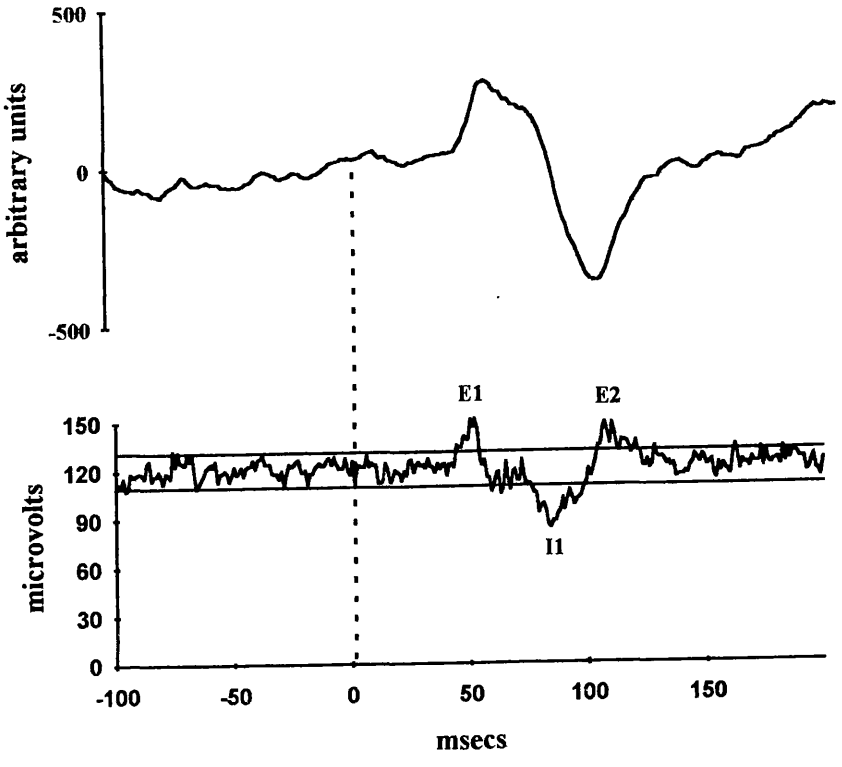


Figure 6. The upper trace shows the cumulative sum (cusum) of deviations from the mean prestimulus EMG. The lower trace shows the average of 500 rectified responses recorded in anterior tibial muscles by electrical stimulation of the plantar surface of the hallux at three times perceptual threshold . The dotted line shows the time of stimulation at 0 msec. The period of 100 msecs before the stimulus was used to calculate the 95% predictive confidence interval (mean background EMG ± 2 SD) and these are shown as the horizontal lines above and below the EMG. The different components of CMRs (E1, I1 and E2) are beyond the 95% predictive confidence interval. A positive slope in the cusum indicates increased EMG activity and a negative slope indicates reduced EMG.

2.6. Measurements

2.6.1. Maximal Voluntary Contraction (MVC)

Before each experiment, maximal voluntary contraction (MVC) or maximal EMG activity was measured. A strain gauge with maximum rating of 20 Kg (Rs, 632-742) was used to measure the relatively low torque associated with dorsiflexion and plantar flexion of ankle joint. The foot was strapped on to a foot plate mounted on the transducer. The strain gauge was calibrated to confirm its linearity. These data are shown in figure 7. The subject was instructed to keep a constant force (10-15% of MVC) and received visual feedback of the force output on an oscilloscope. Alternatively rectified integrated EMG was provided as an approximate indication where the force could not be measured easily e.g. in standing. Each set of recordings lasted about 4 minutes. In order to avoid muscle fatigue, there was a rest of about 3 minutes between each sequence of the experiments.

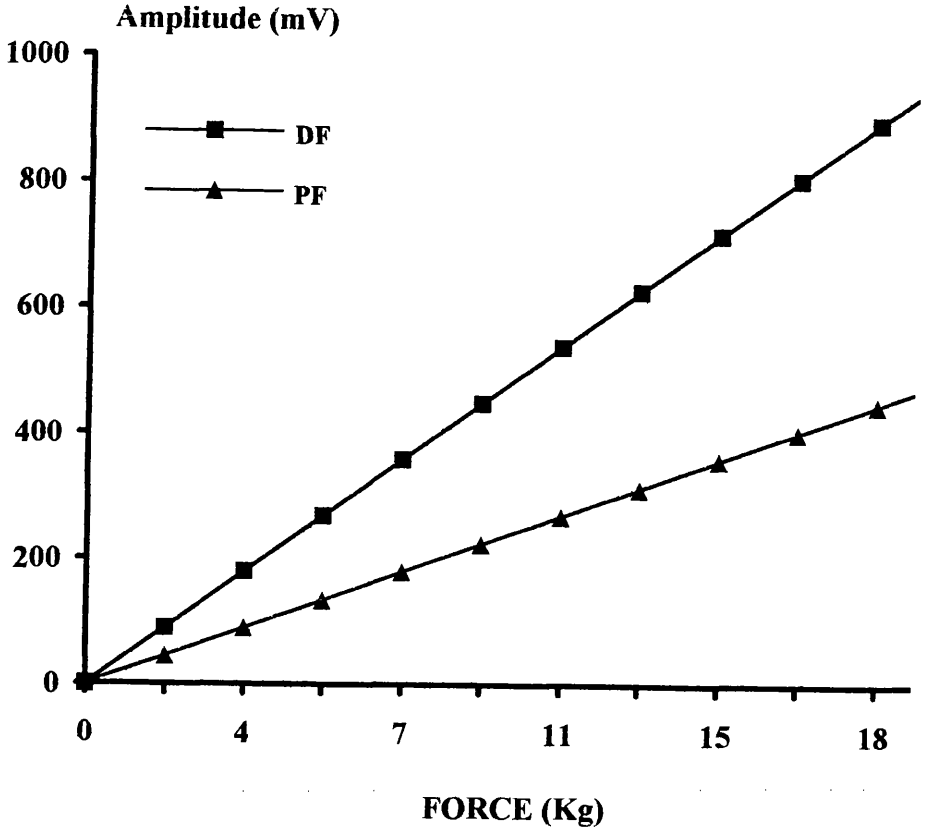


Figure 7. The calibration of the strain gauge for dorsiflexion (black squares) and plantar flexion (black triangles). The response is linear in both directions of force application (r^2>0.99).

2.6.2. Measurement of Perceptual Threshold (PT)

The subject's perceptual threshold was measured by gradually increasing the stimulus intensity from zero until the separate pulses could be distinguished. Thresholds showed some inter-subject variation on stimulation of hallux. The thresholds for experiments using the Axelgaard electrodes are shown in table 4 and thresholds with the ring electrodes are shown in table 5. Thresholds were higher when the ring electrodes were used. The mean threshold was 10.8 ± 2.9 mA for Axelgaard electrodes and 14.1 ± 3.3 mA for Medelec ring electrodes. These values were significantly different ($P<0.001$). The threshold did not change by more than 2 mA throughout the course of any experiments in one subject. The perceptual threshold was rechecked prior to each recording.

Number	PT (mA)
1	12
2	13.3
3	6
4	12
5	13
6	11
7	7.2
8	15
9	7.5
10	11
11	14
12	11
13	11
14	13
15	10
16	12
17	5
18	13
19	16.5
20	13
21	11.5
22	11
23	10
24	11
25	13
26	11.7
27	16.5
28	16.5
29	13
30	10

Mean ± SD	**11.7 ± 2.8 mA**
Range	**5-16.5 mA**

Table 4. The perceptual threshold of different subjects on stimulation of the hallux when pad electrodes (Axelgaard) were used.

Number	Pad Electrodes PT (mA)	Ring Electrodes PT (mA)
1	12	17
2	13.3	16
3	6	8
4	12	16
5	13	14
6	11	10.5
7	7.2	11
8	15	18
9	7.5	13
10	11	17
Mean ± SD	**10.8 ± 2.9 mA**	**14.1 ± 3.3**
Range	**6-15 mA**	**8-18 mA**

Table 5. The perceptual threshold of different subjects on stimulation of the hallux when either pad electrodes (Axelgaard) or ring electrodes (Medelec) were used.

2.6.3. Measurement of latency and Nerve Conduction Velocity

Measurement of the latency was based on the method of Evans et al (1990). The latency of the E1 component of cutaneomuscular reflexes was measured in milliseconds from stimulus artefact to the first sign of inflexion from the mean background EMG level. The I1 and E2 latencies were measured where the trace crossed the mean EMG level. An example of this is shown in figure 8.

The latency of the sensory nerve action potential in the posterior tibial nerve was measured at the ankle joint and the popliteal fossa from the stimulus artefact to the onset and peak of the major negative phase. The distance between the stimulus point and the recording electrode was divided by the difference of the latencies to give the conduction velocity. The measurement to the peak of the negative potential was used when a difference in latencies between two points was needed. This measurement eliminates errors induced by the presence of stimulus artefacts.

2.6.4. Measurement of amplitude of components of the CMR

CMRs were identified by comparison of the averaged rectified EMG of 100 msecs before and 200 msecs after stimulation. The area of the E1, I1 and E2 components were measured and normalised with respect to background i.e. the magnitude was expressed as a percentage of modulation of background activity. In this analysis, the E1, I1 and E2 components of the averaged rectified EMG signal are dealt with separately. An example is shown in figure 9. These measurements allowed a statistical comparison of responses compared to control values.

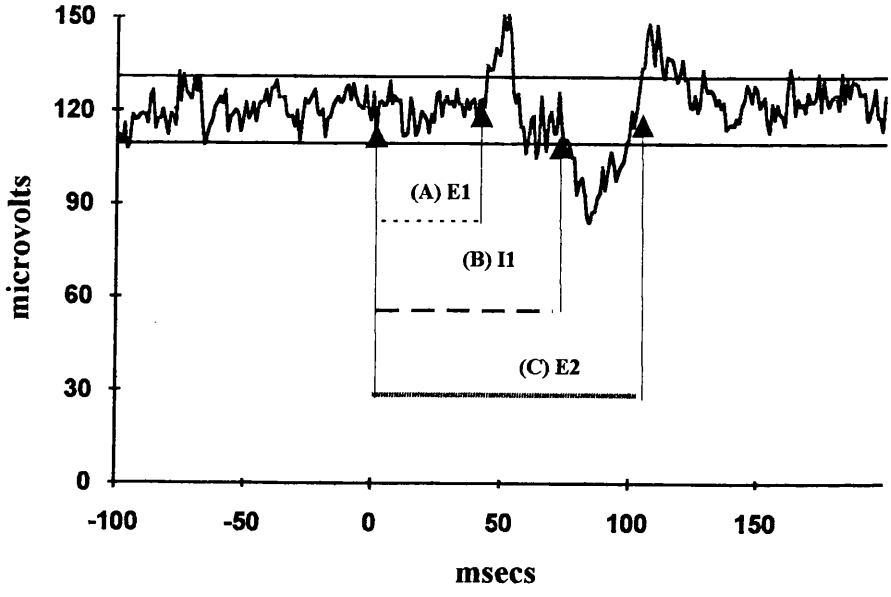


Figure 8. The average of 500 rectified responses recorded in the anterior tibial muscles by electrical stimulation of the plantar surface of the hallux at three times perceptual threshold . The time of stimulation is at 0 msec. The period of 100 msecs before the stimulus was used to calculate the 95% predictive confidence interval (mean background EMG ± 2 SD) and these are shown as the horizontal lines above and below the EMG. The different components of CMRs (E1, I1 and E2) are beyond the 95% predictive confidence interval. The latency of E1 (A) was measured from the time of stimulation to the first sign of inflexion from the mean background EMG level. The latencies of I1 (B) and E2 (C) components were measured where the trace crossed the mean EMG level.

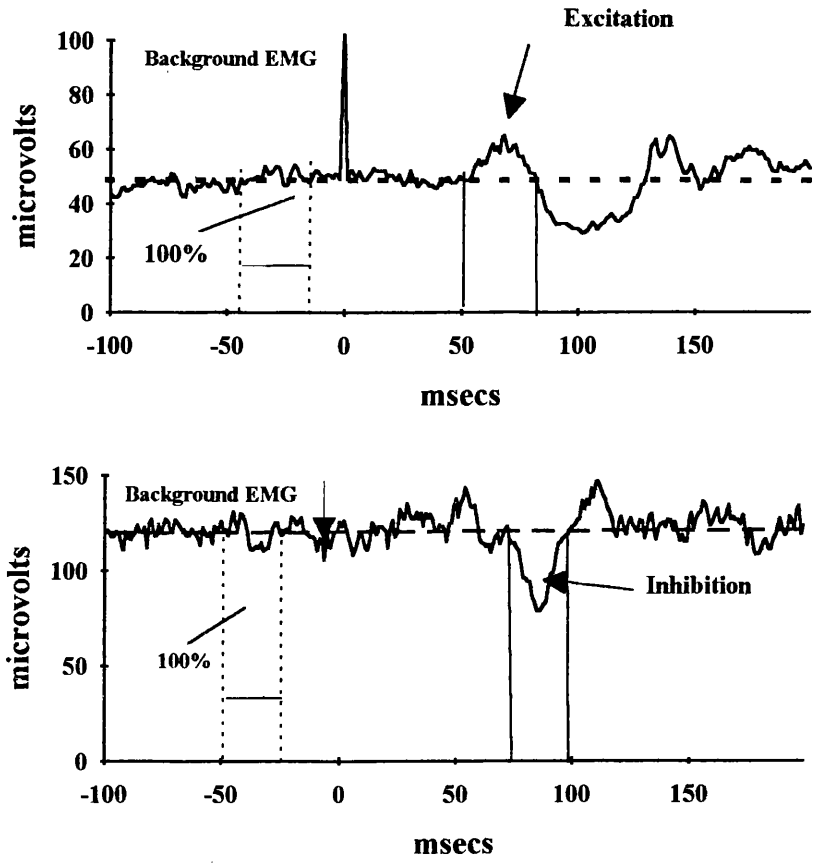


Figure 9. The method of measurement of the magnitude of excitatory and inhibitory components of CMRs in averaged rectified EMG. The area of the E1, I1 and E2 components were measured and normalised with respect to background EMG i.e. the amplitude was expressed as a percentage of modulation of background activity. The peristimulus period was used as the background activity and was deemed to equal 100 %. In the upper trace, the excitation response was greater than 100 % and in the lower trace, the inhibition response was below 100 %.

2.6.5. Measurement of amplitude of sensory nerve action potentials

The amplitude of the surface neurogram was measured from the peak of the initial positive phase to the peak of the major negative phase. The data was again normalised by deeming the control amplitude to equal 100% and expressing the cooled and re-warmed amplitudes as a percentage of control in skin cooling experiments.

2.7. Experimental procedure

2.7.1. Increasing the number of stimuli

The effect of the increasing the number of stimuli on the magnitude of cutaneomuscular reflexes was tested using the experimental procedure described earlier in sections 2.3 and 2.4. Cutaneomuscular reflexes were elicited by 2 or 3 shocks at intensity up to 3 PT with 5 msecs intervals. This stimulation did not evoke painful sensations. However, the addition of a fourth pulse changed the character of the sensation in many subjects.

2.7.2. Intensity of contraction

The effect of intensity of contraction on the size of cutaneomuscular reflexes in tibialis anterior was investigated. The experimental procedure was described earlier in sections 2.3 and 2.4. Cutaneomuscular reflexes were elicited by single shock while voluntary contractions were made at 5%, 10% & 20% of maximal voluntary contraction.

2.7.3. Skin cooling

The effects of skin cooling on the magnitude of different components of cutaneomuscular reflexes of the tibialis anterior and on the amplitude of the surface neurogram of the tibial nerve was investigated. The general experimental procedure was described earlier in section 2.3 and 2.4. The whole of the foot up to the ankle was placed inside a polythene bag and then placed inside a bucket of crushed ice. The skin temperature was closely monitored throughout with a mercury thermometer on the skin

surface. The skin was cooled until the temperature dropped to 12 to 14 ° C at the skin / bag interface. This was found to remain constant after a cooling period of 4 to 5 minutes. Cutaneomuscular reflexes were elicited by stimulation of the hallux with paired electrical pulses of 0.1 msec duration with 5 msecs interval, with an intensity up to three times perceptual threshold at a frequency of about 2 Hz during control, periods after cooling and after recovery.

RESULTS

3. RESULTS

Results will be presented for experiments investigating cutaneomuscular reflexes in different muscles of the lower limb. The sequence of the experimental results is:

1. The effects of the site of stimulation on CMRs in TA.
2. Variations in perceptual threshold.
3. CMRs in the other muscles of the lower limbs.
4. The effects of stimulus intensity and number of the stimuli on CMRs in TA.
5. The effects of background contraction (force) on CMRs in TA on stimulation of hallux.
6. The effects of posture on CMRs in TA.
7. The effects of skin cooling on CMRs in TA and tibial nerve neurograms.

3.1. The nature of the CMRs

Electrical stimulation applied to patches of skin covering foot especially to the hallux, produced changes in the averaged surface EMG signal recorded during steady voluntary contraction of the muscles of the lower limb. Responses were particularly strong in the anterior tibial muscles. The method of recording introduced by Gassell & Ott (1970) was used. In this method, the EMG activity is first rectified and then trigger averaged. The amplitude of the rectified EMG is proportional to the intensity of the contraction. In the absence of a cutaneous stimulus, the EMG averaged over 500 repetitions will approximate to a smooth

horizontal trace. When a stimulus is delivered any excitatory effects will cause the averaged trace to rise. Conversely, any inhibitory effects will cause a temporary reduction in EMG activity. An example is shown in figure 10. The upper panel of figure 10 shows the average of 500 repetitions without cutaneous stimulation. The record is flat showing no modulation of EMG. The lower panel is the result of a similar experiment with shocks applied to the heel at twice perceptual threshold. The time of stimulation can be clearly identified from the stimulus artefact at 0 msec. The averaged EMG in the 100 msecs before stimulation is used to calculate the 95% predictive confidence interval. There is a period of increased EMG which exceeds the confidence interval at about 60 msec, though the rise begins earlier at 52 msec. This is followed by a longer latency reduction and a subsequent second rise. Most CMRs displayed these polyphasic excitations and inhibitions.

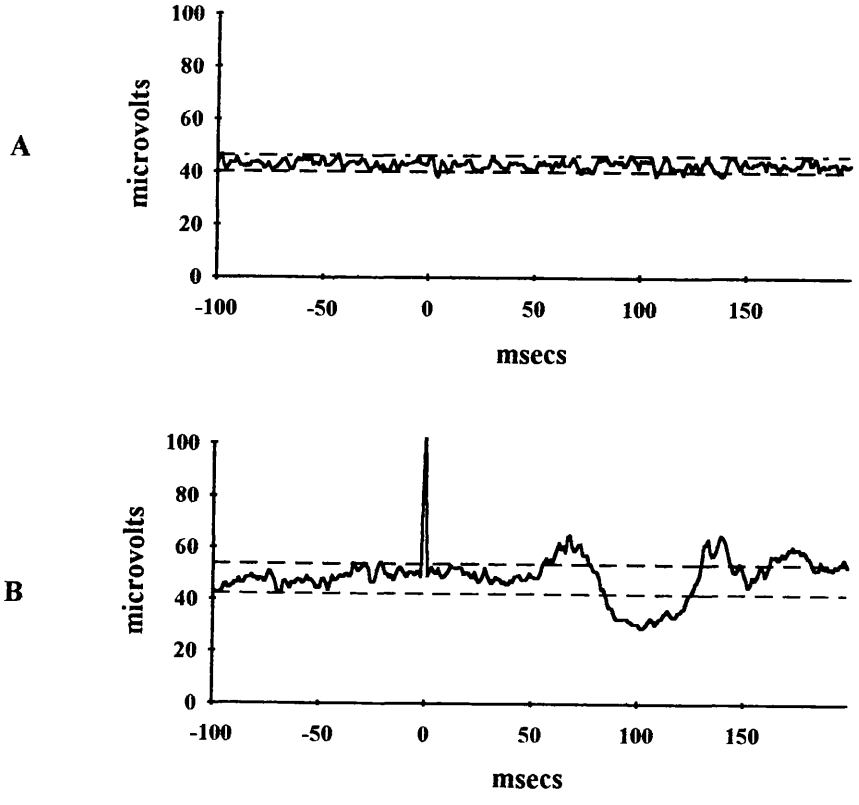


Figure 10. This shows the average rectified EMG recorded from tibialis anterior during a constant low force contraction. Each trace is the average of 500 repetitions.

A: Averaged EMG during control conditions without cutaneous stimulation. Note there is no modulation of EMG.

B: averaged EMG when a single shock at twice PT is applied to the heel. The stimuli is delivered at 0 msec and a stimulus artefact is obvious. Subsequent reflex excitations can be seen as increases in EMG. There is also a period of inhibition. The horizontal dashed lines show 95% confidence intervals based on the pretrigger EMG.

CMRs in TA elicited by stimulation of the hallux

Experiments were performed in 30 neurologically normal subjects to investigate the effects of electrical stimulation of skin covering plantar surface of the hallux at intensities up to 3 PT in tibialis anterior electromyogram.

The CMRs typically has three identifiable components comprising; an initial short latency increase in EMG (the E1 component), followed by a decrease, (the I1 component), followed by a second increase, the E2 component. Figure 11 illustrates a typical series of CMRs. There is an initial excitation some 45 msec after the stimulus. This is followed by inhibition at 65 msec and a second excitation at 80 msec. In about half of the subjects, a later inhibitory phase followed these reflex responses.An example is shown in figure 12. The upper panel shows the cumulative sum of deviations (cusum) from the mean prestimulus EMG. The slope in the cusum can be used to aid the measurement of the latency of the responses and also to make the small responses easier to detect. The lower panel shows the late responses of cutaneomuscular reflexes in tibialis anterior by electrical stimulation of hallux at three times perceptual threshold. The latency of these late responses often exceeded than 100 msecs and showed considerable variations. These responses have been labelled E1, I1, E2 and I2 following the convention introduced by Caccia et al (1973) and Stephens & Usherwood (1976).

The E1 response in tibialis anterior appeared in 18 out of the 30 subjects with a mean latency around 47.7 ± 3 msecs (mean ± SD). The inhibitory response (I1) was the first response in some of the subjects and

appeared in only 16 out of 30 with a mean latency around 69.3 ± 9.8 msecs. The most stable response in most of the subjects was E2 with a mean latency around 79.3 ± 10.4 msecs . The I2 response was the most variable in latency, magnitude and appearance. Table 6 shows the summary data of frequency of occurrence of each reflex component and their latencies. The latencies of each component of the cutaneomuscular reflexes were significantly different. Unpaired t-tests gave $p<0.001$ when the latencies of E1 and I1 and E2 and I2 were compared. The difference in the mean latencies of I1 and E2 was also significant ($p<0.01$).

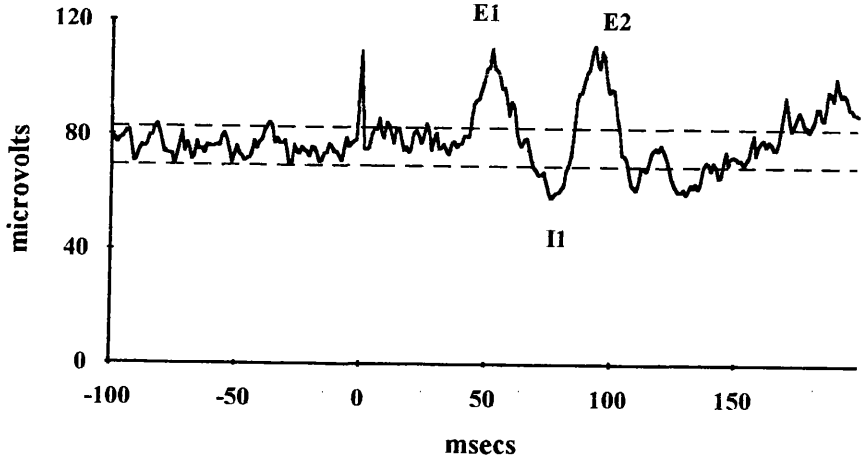


Figure 11. Average of 500 rectified responses recorded in anterior tibial muscles by electrical stimulation of patches of skin covering hallux at intensity up to three times perceptual threshold. The stimulus artefact shows the time of stimulation at zero time. The pattern of responses is triphasic, consisting of an early excitation with a latency around 45 msecs, followed by an inhibition with a latency around 65 msecs and a late period of excitation with a latency around 80 msecs.

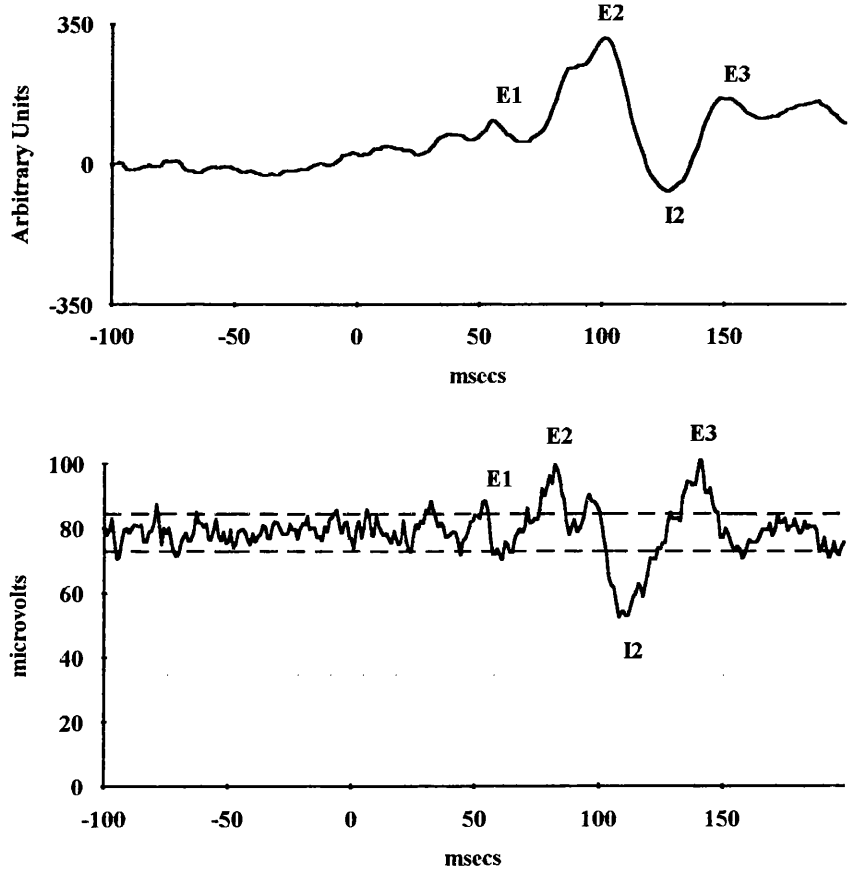


Figure 12. Lower trace shows the late responses of cutaneomuscular reflexes in tibialis anterior by electrical stimulation of hallux at three times perceptual threshold. The latencies of these late responses (I2 and E3) were variable, but often exceeded 100 msecs. The stimulation is at zero time. Upper trace shows the cumulative sum of deviations (cusums) from the mean prestimulus EMG. The positive and negative slope in the cusum can be used to aid the measurement of the latencies of the responses.

Subject	E1 (msec)	I1 (msec)	E2 (msec)	I2 (msec)
1	50	84		
2			68	86
3	45	65	88	
4	45	75	95	
5	45	67	85	97
6	46	69	94	102
7	42	64	98	
8	45		75	95
9			69	
10	55	76		
11			65	80
12		50	75	
13	50		75	
14		72		
15	47	54		
16			80	107
17			74	84
18		56	78	
19	45		67	77
20		70	87	
21			71	87
22		76		113
23	49		63	103
24			80	95
25	50			117
26	49	79	91	104
27	50		76	102
28	49	70	90	
29	49			90
30	48	82		
Frequency	**18/30**	**16/30**	**22/30**	**16/30**
Mean ± SD	**47.7 ± 3**	**69.3 ± 9.8**	**79.3 ± 10.4**	**96.2 ± 11.6**
Range	**42-55**	**50-84**	**63-98**	**77-117**

Table 6. This table shows latency of different components of CMRs in tibialis anterior on stimulation of hallux at 3 PT.

CMRs in TA elicited by stimulation of the heel

Experiments were performed in 12 neurologically normal subjects to investigate the effect of stimulation the skin covering the plantar surface of the heel at an intensity up to 2 times perceptual threshold on CMRs in the anterior tibial muscles . The range of stimulus intensities available for eliciting cutaneomuscular reflexes at this site is restricted by the possibility of initiating muscle contraction of the intrinsic muscles of the foot by stimulation of the tibial nerve.

Muscle contraction will present severe problems for these experiments since any reflex effects could be due to modulation of muscle afferent activity rather than due to cutaneous afferent stimulation. The stimulus intensity was always kept below threshold for motor fibres.

As with stimulation of the hallux, the most common pattern of responses was triphasic with an early excitation at around 45-50 msecs followed by an inhibition with a latency around 70 msecs. The I1 response was the most stable in appearance. A later period of excitation appeared at a latency around 90 msecs. An example is shown earlier in figure 10.

Table 7 shows the latency of different components of CMRs in tibialis anterior on stimulation of heel at 2 PT. The latencies of each component of the cutaneomuscular reflexes were significantly different. Unpaired t-test gave $P<0.001$ when the latencies of E1, I1 and E2 were compared. There is no significant differences between the latencies of different components of the CMRs on stimulation of hallux and heel (unpaired t-test, $p>0.05$).

Subject	E1 (msec)	I1 (msec)	E2 (msec)
1	52	82	
2	46	72	86
3	42	60	95
4		50	75
5		68	
6		65	
7	45	65	80
8	51	72	105
9	49	81	107
10		52	74
11			
12		75	
Frequency	**6/12**	**11/12**	**7/12**
Mean±SD	**47.5±3.8**	**67.5±10.5**	**88.9±13.7**
Range	**42-52**	**50-82**	**75-107**

Table 7. This table shows the latency of different components of CMRs in tibialis anterior on stimulation of plantar surface of the heel at 2 PT.

CMRs in TA elicited by stimulation of the lateral border of the foot

Experiments were performed on 9 neurologically normal subjects to investigate the effect of stimulation of skin covering the lateral border of the foot on tibialis anterior at intensity up to two times perceptual threshold. The range of stimulus intensities at this site is restricted by possibility of initiating muscle contraction by stimulation of the lateral branches of the peroneal nerve. Therefore, stimulus intensity was always kept below threshold for motor fibres.

The most common pattern of responses seen was biphasic, consisting of an early inhibition with a mean latency of 59.8 ± 7.4 msecs. This was followed by a period of excitation with a mean latency of 79.9 ± 9.3 msecs. In two subjects, a later period of inhibition appeared at a latency around 95 msecs. An example is shown in figure 13.

Table 8 show the summary data of latencies of components of CMRs in tibialis anterior by stimulation of sural. The latencies of each component of the cutaneomuscular reflexes were significantly different. Unpaired t-test showed that the latencies of the I1 components were significantly shorter in duration than the latencies of the E2 components ($p<0.001$).

Subject	E1 (msec)	I1 (msec)	E2 (msec)	I2 (msec)
1	49	62	95	
2		51	74	
3		55	68	95
4		50	75	97
5		67	76	
6		54		
7		70	87	
8		65		
9		64	84	
Frequency	**1/9**	**9/9**	**7/9**	**2/9**
Mean ± SD		**59.8 ± 7.4**	**79.9 ± 9.3**	
Range		**50-70**	**68-95**	**95-97**

Table 8. This table shows the latency of different components of cutaneomuscular reflexes in tibialis anterior on stimulation of lateral border of the foot at twice the perceptual threshold.

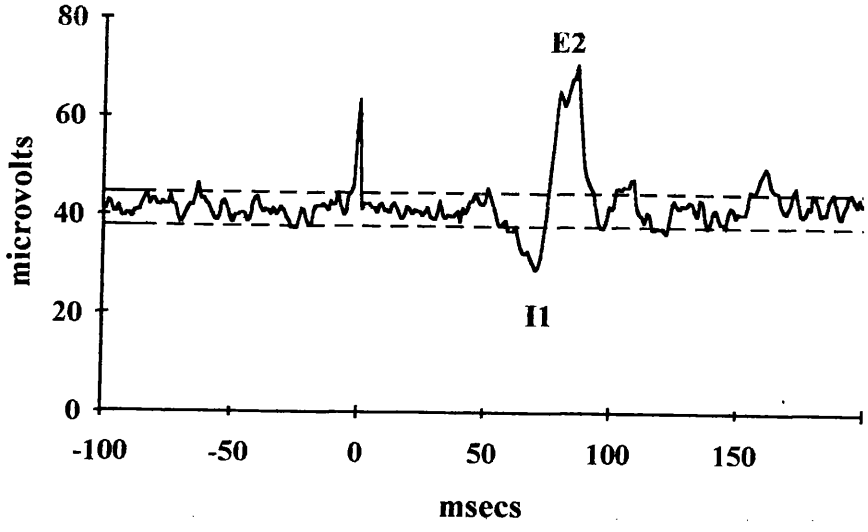


Figure 13. Average of 500 rectified responses recorded in anterior tibial muscles by electrical stimulation of patches of skin covering lateral border of foot at intensity up to two times perceptual threshold. The stimulus artefact shows the time of stimulation at 0 msec. The pattern of responses is biphasic, consisting of an early inhibition with a latency around 50 msecs, followed by an excitation with a latency around 75 msecs.

CMRs in TA elicited by stimulation of the plantar surface of foot

Cutaneomuscular reflexes were also elicited by stimulation of the skin covering the plantar surface of the foot with intensities up to 2 PT in 5 subjects. This elicited triphasic CMRs in tibialis anterior. The range of stimulus intensities available for eliciting cutaneomuscular reflexes at this site is restricted by possibility of initiating muscle contraction of the intrinsic muscles of the foot by stimulation of the plantar nerves. The stimulus intensity was always kept below threshold for motor fibres. The most common pattern of responses was triphasic consisting of an early excitation with a mean latency around 42.8 ± 2.2 msecs, then a period of inhibition with a mean latency around 61.8 ± 3.3 msec followed by a later period of excitation with a mean latency around 83.6 ± 5 msecs. An example is shown in figure 14.

Table 9 shows the latency of different components of CMRs in tibialis anterior on stimulation of mid-foot at 2 PT. The responses in subject 5 are unusual. He has the usual sequence of waves but each occurs much later than in the other subjects. The reasons for this are not known and these values have been omitted from the calculation of the mean latencies.The latencies of each component of the cutaneomuscular reflexes were significantly different. The latencies of E1, I1 and E2 were found to be significantly different when subjected to unpaired t-tests ($p<0.001$).

Subject	E1	I1	E2
1	44		85
2	45	60	85
3	40	65	85
4	41	58	75
5	65	95	140
6	44	64	88
Frequency	**5/6**	**4/6**	**5/6**
Mean ± SD	**42.8 ± 2.2**	**61.8 ± 3.3**	**83.6 ± 5**
Range	**40-45**	**58-65**	**75-88**

Table 9. This table shows latency of different components of CMRs in tibialis anterior on stimulation of skin covering plantar surface of the foot. The data for subject 5 has been omitted from the calculation of mean and standard deviations.

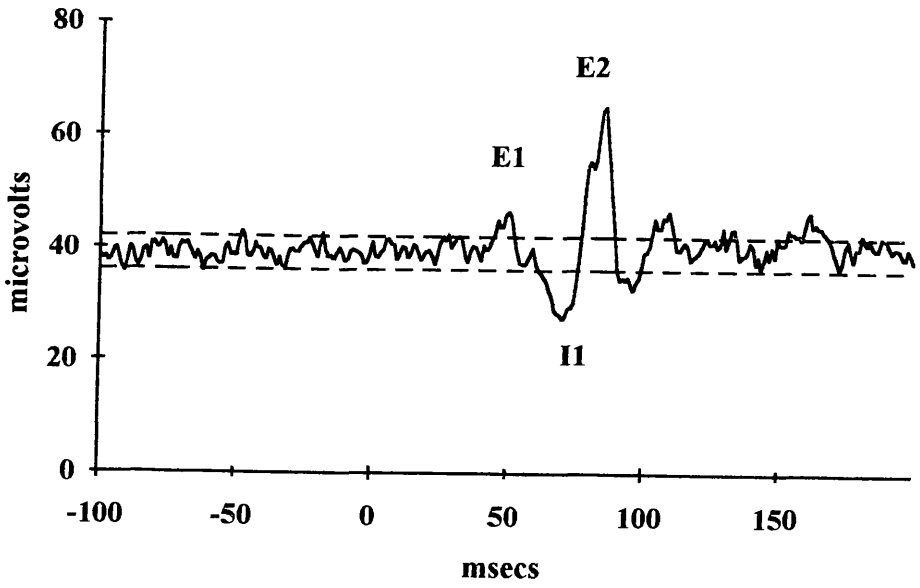


Figure 14. Average of 500 rectified responses recorded in anterior tibial muscles by electrical stimulation of patches of skin covering plantar surface of foot at intensity up to twice perceptual threshold. The pattern of responses is triphasic, consisting of an early excitation with a latency around 45 msecs, followed by an inhibition with a latency around 60 msecs and a late period of excitation with a latency around 80 msecs.

CMRs in TA elicited by stimulation of skin covering lower third of the shank

Experiments were performed in 5 neurologically normal subjects to investigate the effect of stimulation of patches of skin covering anterior aspect of lower third of the shank on tibialis anterior. The range of stimulus intensities available for eliciting cutaneomuscular reflexes in this area was restricted by possibility of initiating direct muscle contraction.

The most common pattern of responses was triphasic consisting of an early excitation with a mean latency around 38.5 ± 2.4 msecs, then a period of inhibition with a mean latency around 70.3 ± 4.1 msecs followed by a later period of excitation with a mean latency around 86.3 ± 21.9 msecs. An example is shown in figure 15.

Table 10 shows the latency of different components of CMRs in tibialis anterior on stimulation of lower third of the shank at 1.5 PT. The latencies of each component of the cutaneomuscular reflexes were significantly different when subjected to t-test ($P<0.001$).

Subject	E1 (msec)	I1 (msec)	E2 (msec)
1	40	75	100
2		70	
3	40	65	98
4	39		61
5	35	71	
Frequency	**4/5**	**4/5**	**3/5**
Mean±SD	**38.5±2.4**	**70.3±4.1**	**86.3±21.9**
Range	**35-40**	**65-75**	**61-100**

Table 10. This table shows latency of different components of CMRs in tibialis anterior on stimulation of skin covering lower third of the shank.

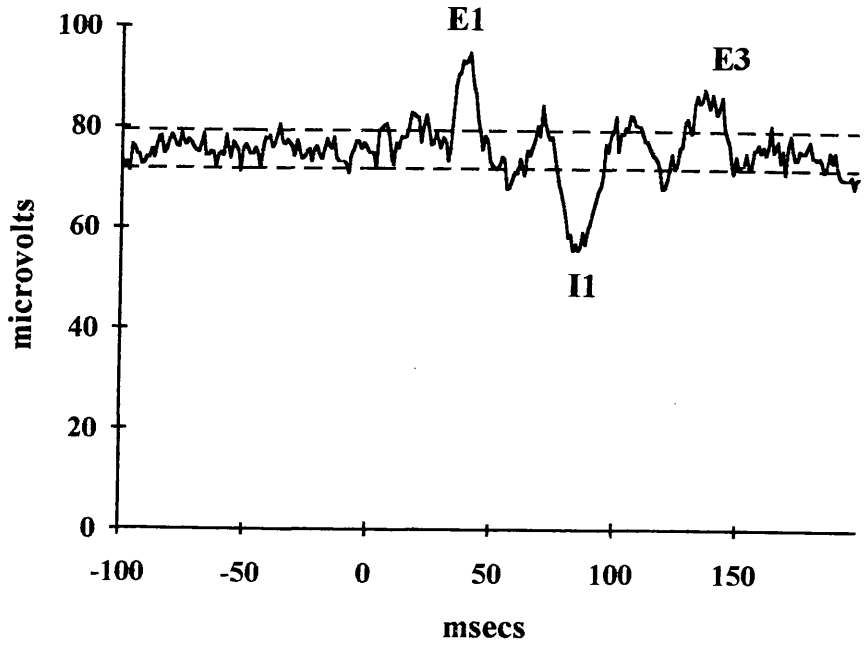


Figure 15 Average of 500 rectified responses recorded in anterior tibial muscles by electrical stimulation of patches of skin covering lower third of the shank at intensity up to one and half times perceptual threshold. The time of stimulation is at 0 msec. The response is triphasic, consisting of an early excitation with a latency around 40 msecs, followed by an inhibition with a latency around 70 msecs and a late period of excitation with a latency around 85 msecs. There is no sign of direct muscle stimulation which might be expected to appear as a brief increase in EMG within a few milliseconds of the stimulus. A later excitatory component E3 appears at a latency around 130 msec.

3.2. Variations in perceptual thresholds

Perceptual thresholds for hallux stimulation varied between subjects as shown in table 4 in the methods section. In addition, in any one subject the thresholds were different at different sites of stimulation. Summary data showing the mean perceptual threshold currents is presented in table 11. The perceptual thresholds of the hallux were significantly less than all other sites of stimulation (unpaired t-test, $P<0.0001$). Perceptual threshold for stimulation of the heel were significantly higher than for the mid-foot and the shank (unpaired t-test, $P<0.05$).There are no significant differences between the perceptual threshold of the sural, mid-foot and the shank (unpaired t-test, $P>0.05$).

Whilst thresholds were different between subjects and between sites within the same subject, the value was almost constant at any one site during any single experiment. Perceptual thresholds were measured several times during each experiment and variations were less than 10%.

Site of Stimulation	Number of Subjects	PT (mA) mean ± SD	PT (mA) Range
Hallux	30	11.7 ± 2.8	5-16.5
Heel	12	31.8 ± 6.5	24-44
Sural	9	25.9 ± 7.3	10.1-36
Sole of the foot	6	23.2 ± 3.7	17-27
Skin covering TA	5	22.6 ± 4.9	18-30

Table 11. This table shows the mean (± SD) and range of perceptual thresholds for the different site of stimulation.

Responses in TA

There was considerable variability in the form of the CMR in different subjects. Few of the subjects showed all 4 components of CMRs for all 5 sites of stimulation. Examples of this variation are shown in figures 16 and 17. Figure 16 shows a CMR with only a pure excitation with a latency of 67 msecs. There are no significant earlier responses in this subject. Figure 17 shows a CMR elicited by similar stimulation in a second subject. In this case the CMR consists of a single inhibition at 70 msecs.

Table 12 A, B show summary data from experiments on 30 subjects over 3 years. It shows the frequency of occurrence of each component of CMR for 5 sites of stimulation. These are expressed as the number of observations in table 12A and as a percentages in table 12B. The number of subjects and the intensity of stimulation varies because of the progressive changes in experimental protocols.

Each site of stimulation produced CMRs in tibialis anterior. However, the nature of CMR differs. For example, stimulation of hallux regularly produces all 4 components, though E2 is the most consistent and was observed in 73% of the experiments. Whereas stimulation of the sural site rarely generates E1 responses though it elicits I1 in all cases. Stimulation of the heel and shank causes E1, I1 and E2 regularly but never caused I2 responses.

Figure 18 shows the influence of site of stimulation on anterior tibial muscles at an intensity up to three times perceptual threshold in one subject. Different skin areas of lower limb were stimulated, the plantar surface of hallux at 3 PT, the plantar surface of heel at 2 PT, the skin

covering lateral surface of foot at 2 PT, the skin covering lower third of the shank over the anterior tibial muscles at 1.5 PT. An early excitation appeared with a latency around 40-50 msecs on stimulation of hallux and skin covering tibialis anterior, an early inhibition appeared on stimulation of sural nerve at a latency around 50 msecs. Later periods of inhibition and excitation appeared on stimulation of all the different skin areas tested. Of the 4 identifiable CMR components, the longest latency response I2 is also the least consistent. The second inhibitory components of CMRs is relatively infrequent for all sites except the hallux where it was seen in 53% of tests. It is never observed in more than half the subjects even on stimulation of the hallux where it is elicited most consistently. Late excitations were elicited most regularly from the skin of the mid-foot, sural and hallux regions where the frequencies were 100 %, 78 % and 73 %, respectively. In general E1, I1 and E2 components were regularly observed though there are exceptions, e.g. sural stimulation very rarely elicits E1 in tibialis anterior.

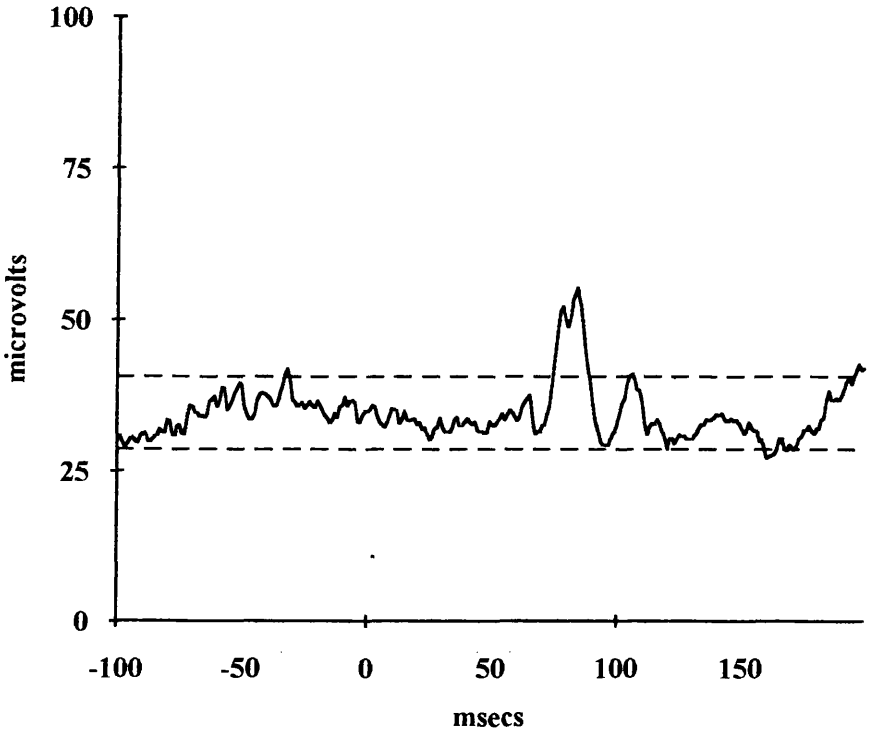


Figure 16. This shows the pure excitation in tibialis anterior by stimulation of hallux at 2 PT. The stimulation is at 0 msecs.

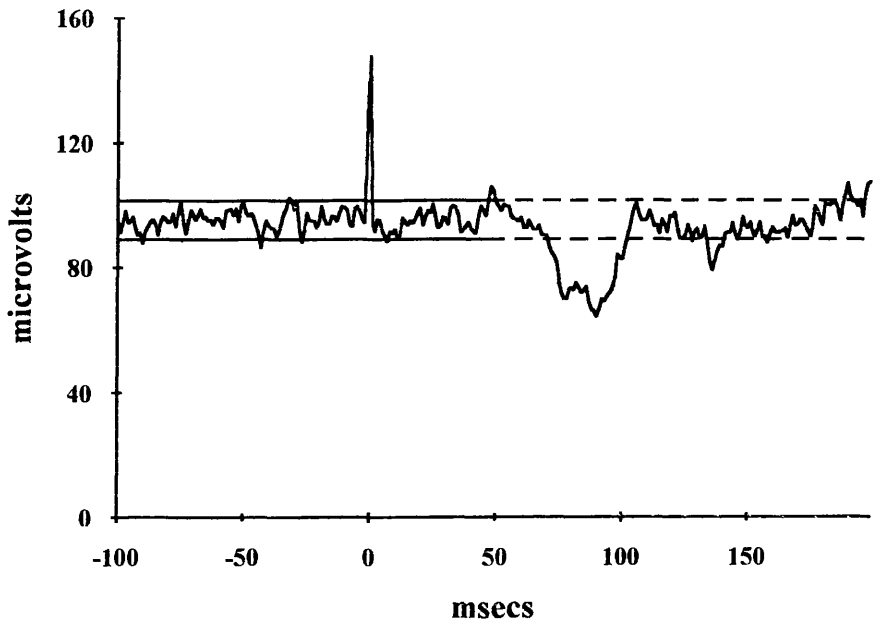


Figure 17. This shows the pure inhibition in tibialis anterior by stimulation of hallux at 2 PT. The stimulation is at 0 msecs.

A:

Site of Stimulation	E1	I1	E2	I2
Hallux	18/30	16/30	22/30	16/30
Heel	6/12	11/12	7/12	0/12
Sural	1/9	9/9	7/9	2/9
Mid-foot	5/6	4/6	6/6	1/6
Shank	4/5	4/5	3/5	0/5

B:

Site of Stimulation	E1	I1	E2	I2
Hallux (3 PT)	60 %	53 %	73 %	53 %
Heel (2 PT)	50 %	92 %	58 %	0 %
Sural (2 PT)	11 %	100 %	78 %	22 %
Mid-foot (2 PT)	83 %	67 %	100 %	17 %
Shank (1.5 PT)	80 %	80 %	60 %	0 %

Table 12: This table shows a summary of number of experiments in which each responses was observed (A) and this frequency expressed as a percentage of the responses (B). All data comes from tibialis anterior on stimulation of the specified skin areas.

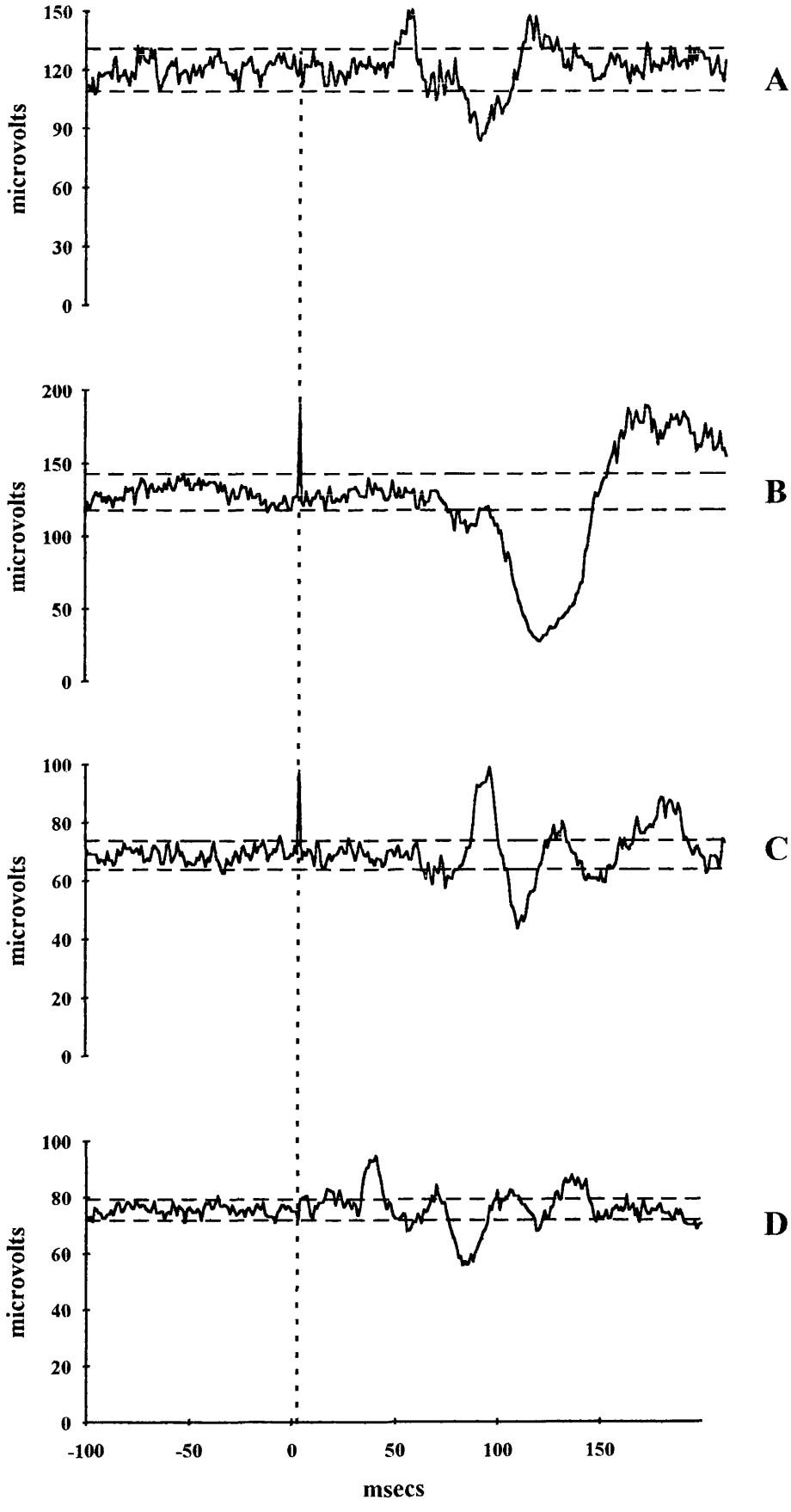

150
120
90
60
30
0
microvolts
A
200
150
100
50
0
microvolts
B
100
80
60
40
20
0
microvolts
C
100
80
60
40
20
0
microvolts
D
-100
-50
0
50
100
150
msecs

Figure 18. This figure shows the influence of the site of the stimulation at intensity up to three times perceptual threshold on CMRs in the anterior tibial muscles in one subject. Different skin areas of lower limb were stimulated: plantar surface of hallux (A), plantar surface of heel (B), skin covering lateral border of foot (C), skin covering lower third of the shank over anterior tibial muscles (D). The dotted line shows the time of stimulation at zero msecs. An early excitation appeared with a latency around 40-50 msecs in A and D, an early inhibition appeared on C at a latency around 50 msecs. Later periods of inhibition and excitation were elicited by stimulation of each of skin area.

Latency of responses

The mean latencies of components of the CMRs in tibialis anterior are shown in table 13. The early excitatory component of CMRs appeared at a latency around 40-50 msec for all sites of stimulation. There are significant differences between the latencies of E1 on stimulation of the hallux compared with mid-foot and shank (unpaired t-test, $p<0.01$ and $p<0.001$, respectively).

The first inhibitory component of the CMR appeared at a latency between 60-70 msecs for all 5 sites of stimulation. The only significant differences between the latencies of the I1 components occur when the effects of stimulation of hallux and sural were compared (unpaired t-test, $p<0.05$). The second excitatory component of CMR appeared at a latency around 80-90 msecs on all site of stimulation. The latencies of E2 components of all 5 sites of stimulation were not significantly different (unpaired t-test, $p>0.05$).

Site of Stimulation	E1 (ms)	I1 (ms)	E2 (ms)	I2 (ms)
Hallux	47.7 ± 3	69.3 ± 9.8	79.3 ± 10.4	96.2 ± 11.6
Heel	47.5 ± 3.8	67.5 ± 10.5	88.9 ± 13.7	-
Sural	*49*	59.8 ± 7.4	79.9 ± 9.3	*96 ± 1.4*
Mid-foot	42.8 ± 2.2	61.8 ± 3.3	83.6 ± 5	-
Shank	38.5 ± 2.4	70.3 ± 4.1	86.3 ± 21.9	-

Table 13: This table shows the latencies of responses (mean ± SD) in tibialis anterior by stimulation of different areas of the foot. The numbers in italics indicate that only one or two subjects showed E1 and I2 respectively on sural stimulation.

3.3. Cutaneomuscular reflexes elicited by stimulation of the hallux in the other muscles of the lower limb

CMRs were recorded in several muscles. Since each muscle has to be activated voluntarily , it is rarely possible to make concurrent records from several muscles simultaneously.

CMRs in gastrocnemius elicited by stimulation of the hallux

Experiments were performed on 10 subjects to study CMRs in gastrocnemius muscles on stimulation of patches of skin covering plantar surface of the hallux at intensity up to three times perceptual threshold. Table 14 shows a summary of latencies of CMR components. The pattern of response in gastrocnemius is different to that in TA. Comparison with table 6, which shows the data for CMRs in TA and table 14, reveals that E1 responses in gastrocnemius are less frequent and of significantly longer latency than those in TA ($p<0.05$, unpaired Student's t test). There is a clear difference in the I1 response which is more frequent in gastrocnemius than in TA, though there is no significant difference in latency ($p>0.05$, unpaired non-parametric, Mann-Whitney test). Six subjects showed CMRs in gastrocnemius which began with an initial period of inhibition showing that this is not a consequence of an initial E1 response.

E2 responses were substantially less frequent in gastrocnemius than in TA, though there is no significant differences in latency ($p>0.05$, unpaired Student's t test). No CMR ever began with an E2 response. E2 in gastrocnemius was always preceded by I1. Figure 19 shows a CMR in gastrocnemius which consists of a strong initial I1 with a weak

subsequent late excitation. Figure 20 shows a very weak E1 response, which is barely significant, followed by clear I1, E2 and I2 responses. An even later excitation (E3) can be seen in this subject.

Subject	E1 (msec)	I1 (msec)	E2 (msec)	I2 (msec)
1	-	72	-	-
2	-	75	-	125
3	-	75	-	-
4	57	79	92	100
5	50	72	-	-
6	50	-	-	-
7	-	75	95	-
8	-	70	80	-
9	-	75	91	-
10	50	80	-	-
Frequency	**4/10**	**9/10**	**4/10**	**2/10**
Mean±SD	**51.8 ± 3.5**	**74.8 ± 3.2**	**89.5± 6.6**	**112.5 ± 17.7**
Range	**50-57**	**72-80**	**80-95**	**100-125**

Table 14. This table shows the latency of the different components of CMRs in gastrocnemius on stimulation of hallux at 3 PT.

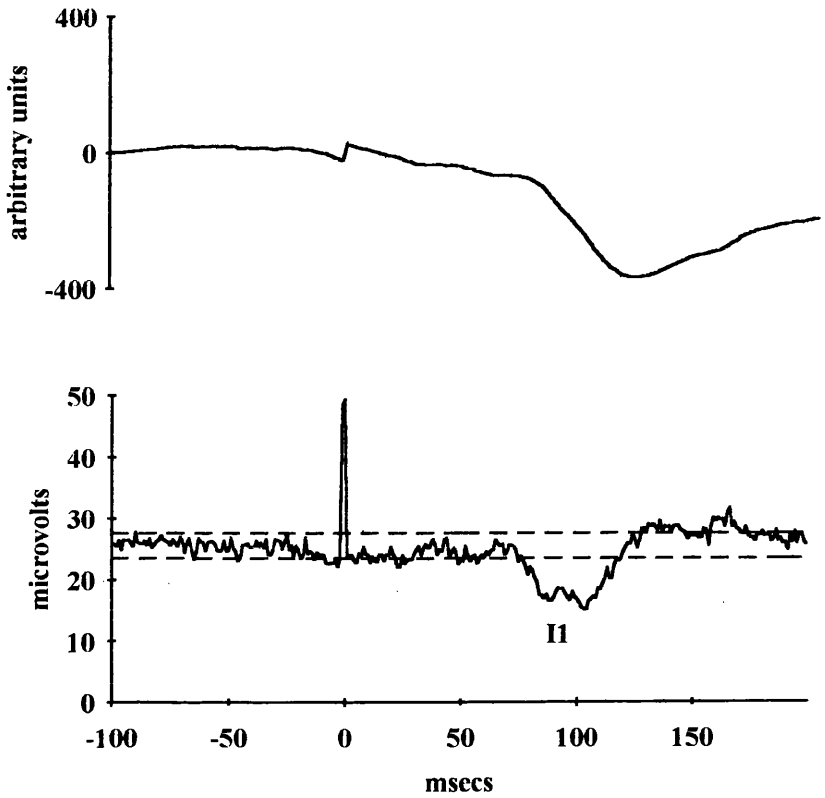


Figure 19. Average of 500 rectified responses recorded in gastrocnemius muscles after electrical stimulation of plantar surface of the hallux at two times perceptual threshold. The stimulus artefact shows the time of stimulation at 0 time. Upper trace shows the cumulative sum of deviations from the mean prestimulus EMG. The negative slope in the cusum shows the strong inhibition and the positive slope in the cusum indicates a weak subsequent of late excitation E3 at a latency more than 120 msecs following inhibition. This has too long a latency to be considered a simple E2 response.

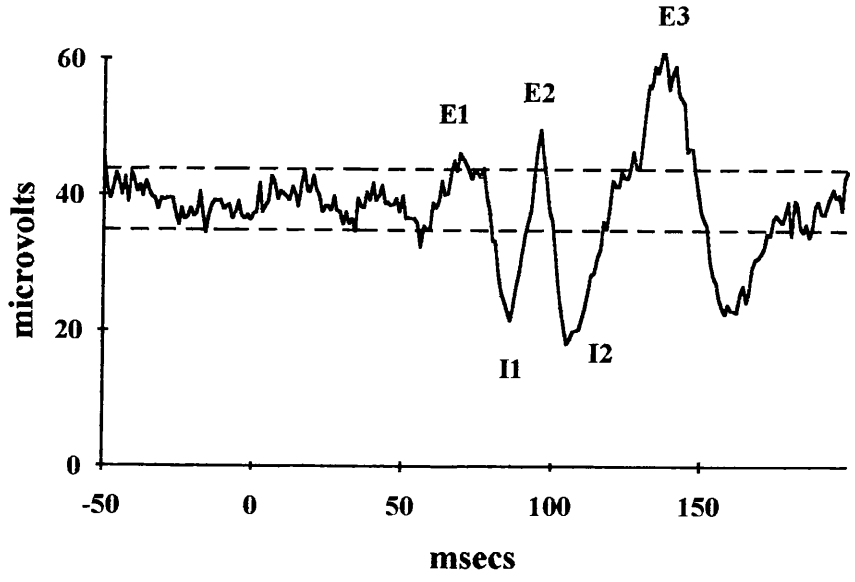


Figure 20. Average of 500 responses recorded in gastrocnemius muscle after electrical stimulation of plantar surface of hallux at three times perceptual threshold in the standing position during leaning forward. The time of stimulation is at 0 msec. All components of CMRs E1, I1, E2, I2 are present. A later excitatory component E3 is appeared at a latency around 120 msecs.

CMRs in quadriceps muscles elicited by stimulation of hallux

Experiments were performed in 10 normal subjects to investigate the effects of stimulation of skin covering plantar surface of hallux on quadriceps muscles at an intensity up to three times perceptual threshold. These experiments were performed with the subject standing erect. An example is shown in figure 21. Table 15 shows a summary of latencies of components of CMRs.

Like CMRs in TA and gastrocnemius muscles the overall frequency of CMR components is about 50 %. The unusual observation here is that 4 of the 10 subjects had no CMR at all whilst 5 showed two components.

In gastrocnemius and TA muscles all subjects showed some feature of the CMRs complex, though rarely all components. The response in quadriceps muscles was variable. In some subjects there was an initial excitation and in others an inhibition. The mean latency of the inhibitions was 70.2 ± 4.9 msec (mean ± SD, n=5/10) and the latency of the excitations was 74 ± 15.4 msec (n=6/10). There is no significant differences between the latencies of two responses ($p>0.05$, unpaired non-parametric, Mann-Whitney test) but both are substantially longer than would be consistent with a spinal segmental pathway.

Subject	Inhibitions	Excitations
1	65	57
2	75	98
3	65	81
4	72	60
5	-	-
6	-	80
7	74	68
8	-	-
9	-	-
10	-	-
Frequency	**5/10**	**6/10**
Mean±SD	**70.2 ± 4.9**	**74 ± 15.4**
Range	**65-75**	**57-98**

Table 15. This table shows latency of different components of CMRs in quadriceps muscles on stimulation of hallux at 3 PT.

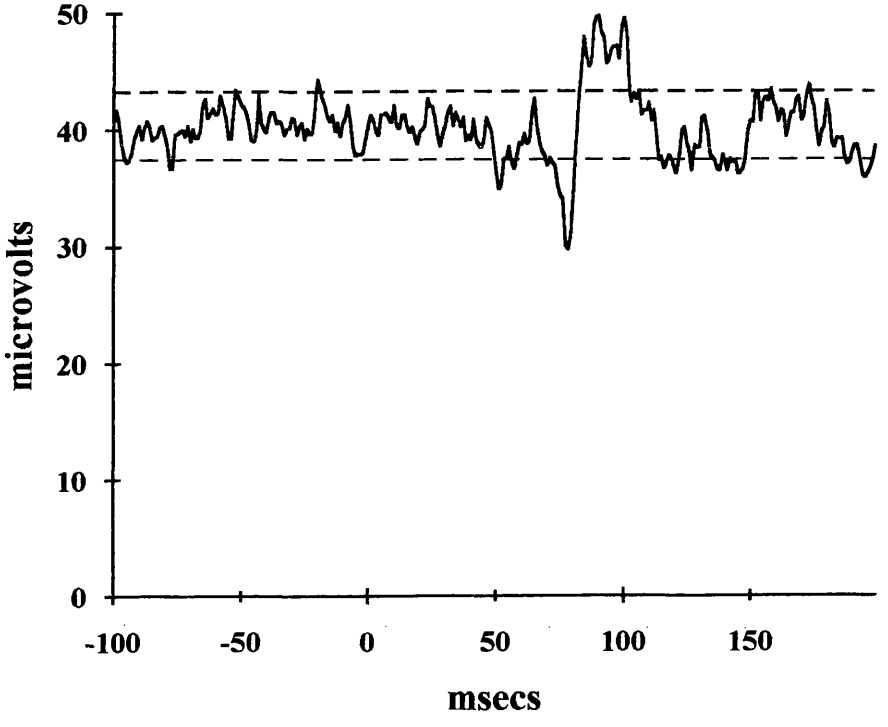


Figure 21. This trace shows the average of 500 responses recorded in quadriceps muscle during stimulation of the plantar surface of hallux at 3 PT in standing position during a backward lean. The stimulation is at 0 msecs. A period of inhibition at 65 msecs is followed by an excitation at 81 msecs.

CMRs in hamstring elicited by stimulation of hallux

Similar experiments were performed to investigate CMRs in the hamstrings. These data were not obtained concurrently with the quadriceps data, though some subjects are common to both experiments. Table 16 shows the summary latencies of components of CMRs in hamstrings muscles by stimulation of hallux. The responses in hamstring were organised and showed an initial period of inhibition with a latency of 68.4 ± 5.9 msec (mean± SD, n=7) followed by a subsequent excitation with a latency of 78 ± 4.7 msec (n= 5). The mean latency of the inhibitions is significantly shorter than the mean of the excitations ($p<0.05$, unpaired Student's t-test).

There is no significant differences between the latencies of quadriceps and hamstrings responses ($p>0.05$, unpaired non-parametric, Mann-Whitney test). These latencies are similar to those in quadriceps and too long to suggest a spinal reflex. A typical hamstring CMR is shown in figure 22.

Subject	Inhibitions	Excitations
1	63	-
2	74	84
3	65	75
4	64	76
5	-	-
6	78	-
7	71	82
8	-	-
9	-	-
10	64	73
Frequency	**7/10**	**5/10**
Mean±SD	**68.4 ± 5.9**	**78 ± 4.7**
Range	**63-78**	**73-84**

Table 16. This table shows latency of different components of CMRs in hamstrings on stimulation of hallux at 3 PT.

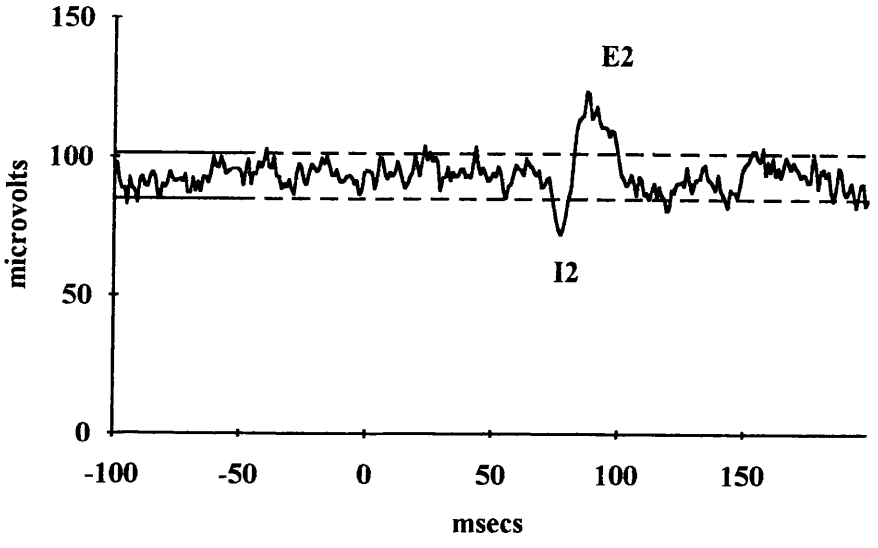


Figure 22. This shows the average of 500 rectified responses recorded in hamstrings muscles by electrical stimulation of plantar surface of hallux at three times perceptual threshold in standing position during forward lean. The stimulation is at 0 msecs. The inhibition at 71 msecs followed by a later excitation at 82 msecs.

CMRs in Abductor hallucis muscle elicited by stimulation of hallux

Cutaneomuscular reflexes on stimulation of hallux were also elicited in abductor hallucis. As seen in table 17, the general pattern of responses in abductor hallucis consists of an early excitation with a mean latency of 55 ± 6.7 msec (mean ± SD, n= 4) followed by a later period of inhibition and excitation. An example of this is shown in figure 23. There is a significant difference between the latencies of E1 in TA (47.7 ± 3 msec) and abductor hallucis (55 ± 6.7 msec) ($p<0.05$, unpaired non-parametric, Mann-Whitney test). The difference of about 7 msecs in E1 latency in TA and abductor hallucis is due to increased efferent distance of about 300 mm and it can be safely assumed that the E1 in abductor hallucis is of spinal origin.

Subject	E1 (msec)	I1 (msec)	E2 (msec)
1	49	72	-
2	50	80	-
3	-	81	89
4	63	78	-
5		60	94
6	58	-	84
Frequency	**4/6**	**5/6**	**3/6**
Mean±SD	**55 ± 6.7**	**74.2 ± 8.8**	**89 ± 5**
Range	**49-63**	**60-81**	**84-94**

Table 17. This table shows latency of different components of CMRs in abductor hallucis on stimulation of hallux at 3 PT.

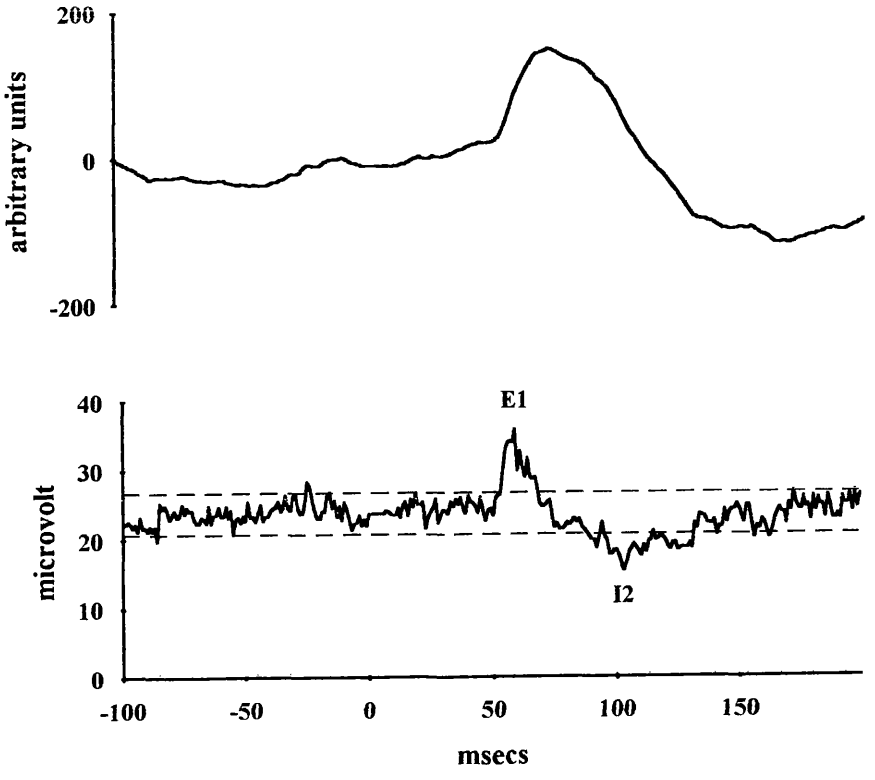


Figure 23. Average of 500 responses recorded in abductor hallucis muscle following stimulation of hallux at 3 PT. The time of stimulation is at 0 msecs. The upper trace shows the cumulative sum of deviations from the mean prestimulus EMG. The latency of first excitatory component is around 50 msecs. The negative slope in the cusum indicates a later period of inhibition at 72 msec.

3.4. Effects of intensity of stimulus on cutaneomuscular reflexes in tibialis anterior

Experiments were performed in 11 neurologically normal subjects to investigate the effect of graded increases in stimulus intensity from perceptual threshold up to three and half times perceptual threshold. Stimulation was applied to the hallux. CMRs were recorded from TA.

Stimulation at perceptual threshold elicited a response in only one subject. Graded increases in intensity up to two times perceptual threshold evoked CMRs in all but one subject. In 4 subjects the response was biphasic excitation/inhibition which could not be separated by electrical thresholds. An example is shown in figure 24. The upper panel shows a CMR elicited by stimulation at 2 PT. There is a clear I1 response preceded by a barely significant E1. When the stimulus intensity is increased to 3 PT the magnitudes of all components increases but their sequence and latency is unchanged.

The lowest threshold response was pure inhibition in 6 subjects and pure excitation in one subject. Figures 25 and 26 show pure excitation and inhibition in two subjects. Stimulation at higher intensities caused mixed excitations and inhibitions in all subjects. In figure 25 the I1 response becomes significant only at intensities above 2 PT. The subject whose data is shown in figure 26 displayed pure inhibitions at intensities of 3 PT or less. With more intense stimulation a secondary late excitation develops. The magnitude of both excitations and inhibitions increased with more intense stimulation. These magnitudes are expressed as a percentage modulation of the mean EMG area. Figure 27 shows pooled

data from 11 subjects. Inhibitions tended to reach a maximum between 2-3 times perceptual threshold whereas excitations increased progressively with increasing stimulus intensity. Most subjects reported a change in the perceived sensation at intensities above 3.5 times perceptual threshold and more intense stimuli were not investigated.

The magnitude of excitation, expressed as percentage of background, increased significantly ($p<0.001$, paired Student's t-test) from 8.8 ± 5% (mean ± SD) at 2 PT to 18.3 ± 11.5% at 3.5 PT. The magnitude of inhibition increased from -9.4 ± 3.3% at 1.5 PT to -20.2 ± 8.3% at 3 PT ($p<0.01$, paired Student's t-test). There is no significant change in the magnitudes of CMRs at stimulus intensity greater than 3 PT ($p>0.05$, paired Student's t-test).

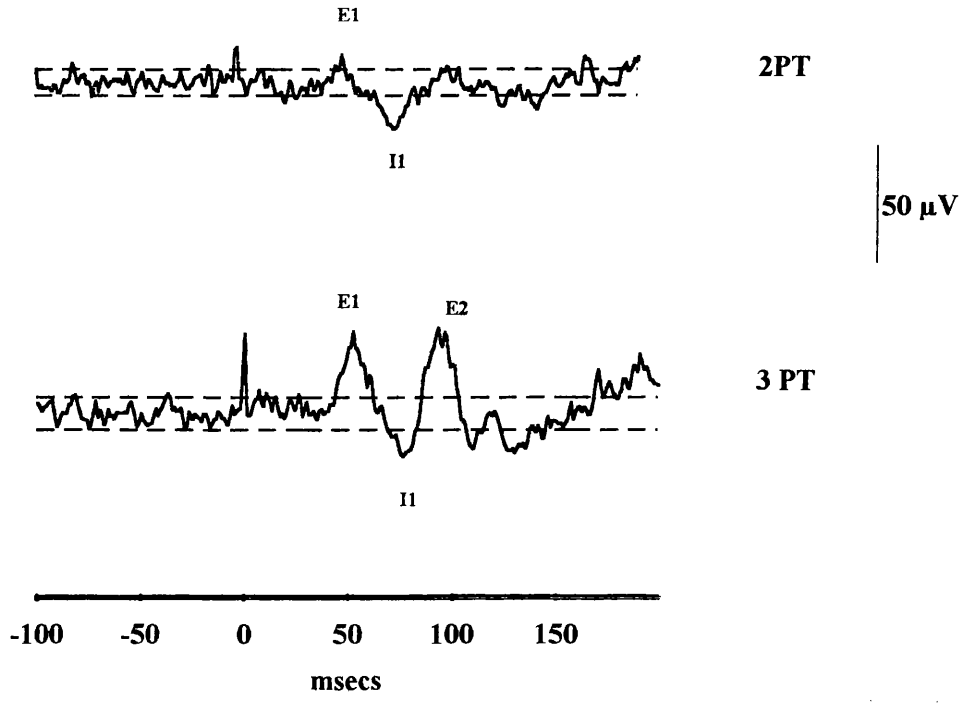


Figure 24. This figure shows an example of mixed excitations and inhibitions in TA by electrical stimulation of the hallux. The intensity of stimulus increases from 2 PT to 3 PT. The stimulus artefact shows the time of stimulation exactly at 0 msec. The vertical bar shows the voltage calibration. Upper panel shows an initial inhibition and a low amplitude of excitation at 2 PT. Lower panel shows a mixed excitation and inhibitions at 3 PT.

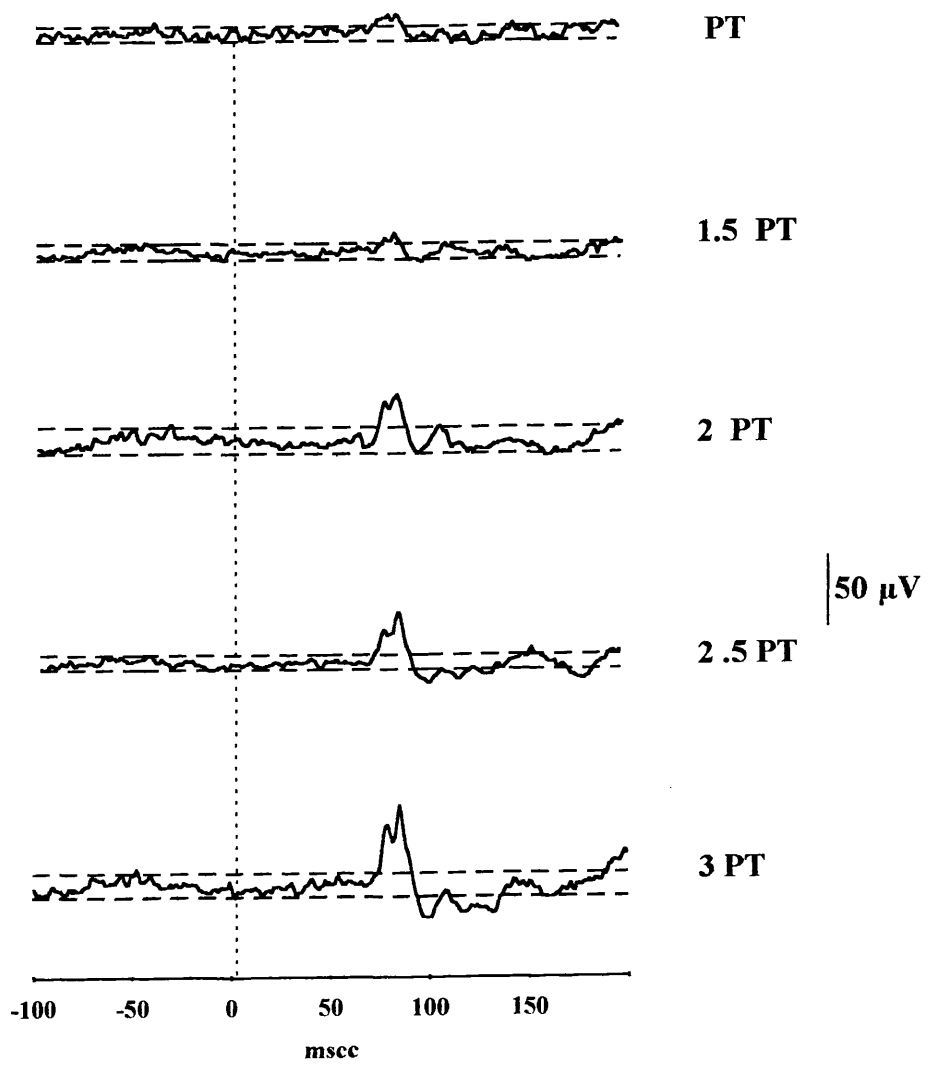


Figure 25. This figure shows an example of pure excitation in TA by electrical stimulation of the hallux. The intensity of stimulus increases from perceptual threshold up to 3 PT. The dotted line shows the time of the stimulation at 0 msec. The vertical bar shows the voltage calibration. The magnitude of the excitation increased as the stimulus intensity goes up to 3 PT.

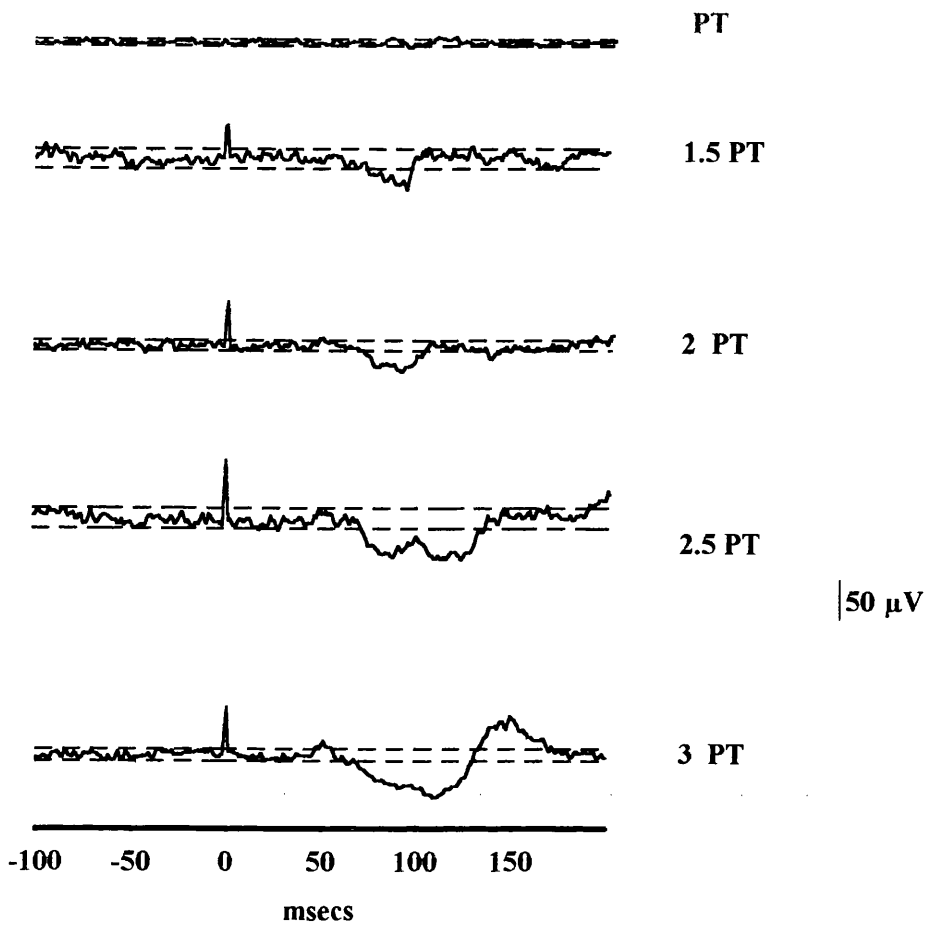


Figure 26. This figure shows an example of pure inhibition in TA following electrical stimulation of hallux. The intensity of stimulus increases from perceptual threshold up to 3 PT. The stimulus artefact shows the time of the stimulation at 0 time. The vertical bar shows the voltage calibration. The magnitude of the first inhibitory component increased progressively as the stimulus intensity increases.

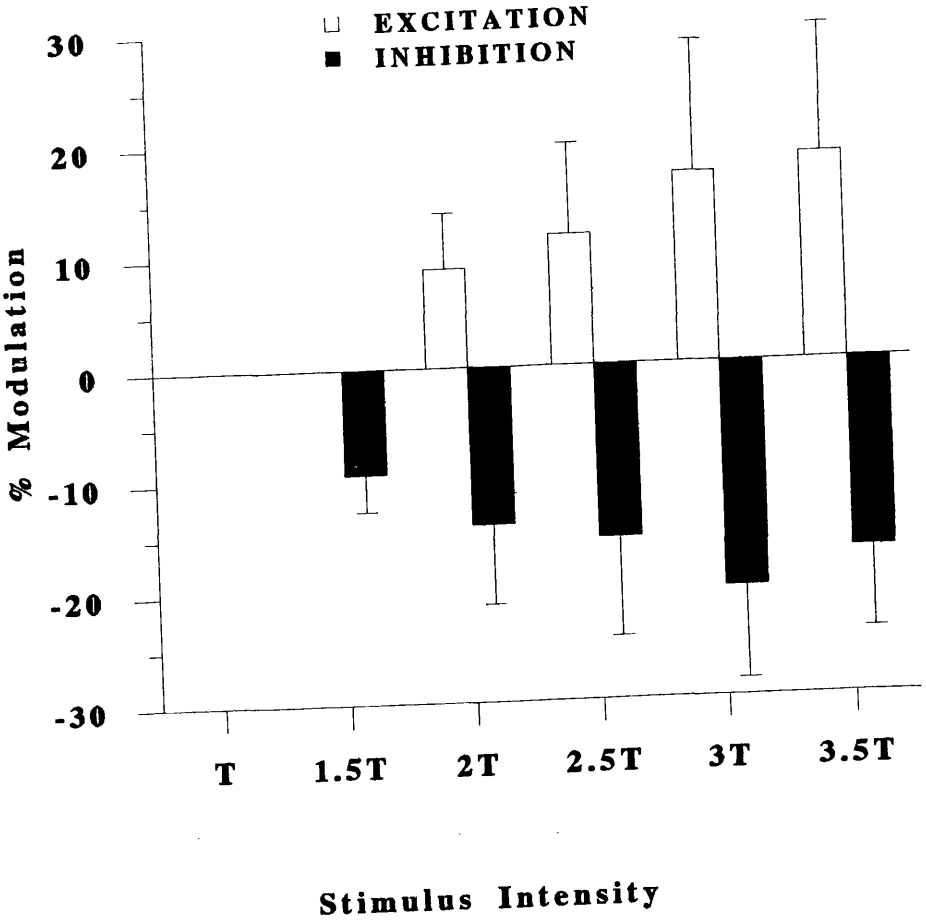


Figure 27. This figure shows the mean and standard deviation of the amplitude of the excitatory and inhibitory components of cutaneomuscular reflexes expressed as percentage modulation of background EMG at different stimulus intensities (n=11). The magnitude of excitation increases as the stimulus intensity goes up to 3.5 PT but the magnitude of the inhibition tended to reach maximum between 2-3 PT.

Temporal summation in the CMRs pathway

An additional series of experiments was performed in 10 normal subjects to investigate the effects of summation in the CMR pathway. These experiments delivered 2 or 3 stimuli at intervals of 5 msec whilst measuring the magnitude of the different components of the CMRs on stimulation of hallux. The stimulus intensity was up to 3 PT. In general, the magnitude of the CMR components increased with additional pulses. An example is shown in figure 28. The latencies of the various components did not change significantly when a second or third pulse was added to the stimulus train. The inhibitory component of the CMRs was significant with single shock stimulation and grew progressively larger as a second or third shock were delivered.

The amplitude of the excitatory components of CMRs also increased significantly when a second or third pulse was added to the train.

Figure 29 shows pooled data from 10 subjects. The addition of a second pulse 5 msecs after the first increased the amplitude of the first excitatory component from 7 ± 2.9 % to 17.6 ± 9.5 % and a third pulse caused an increase of 33.6 ± 16.9 %. The addition of a second pulse 5 msecs after the first increased the amplitude of the first inhibitory component from -10 ±5.3 % to -21.4 ± 6.8 % and a third pulse caused an increase to -24.6 ± 6.7 %. These effects were statistically significant when tested with paired Student's t test ($p<0.01$) for first excitatory and inhibitory components. The amplitude of the second excitatory component increased significantly ($p<0.01$) from 6 ± 2.5% to 13.4 ± 4.1% when a second stimulus pulse was added to the train.

There was no significant differences in the magnitude of inhibitory and excitatory components elicited by 2 or 3 pulses ($p>0.05$).

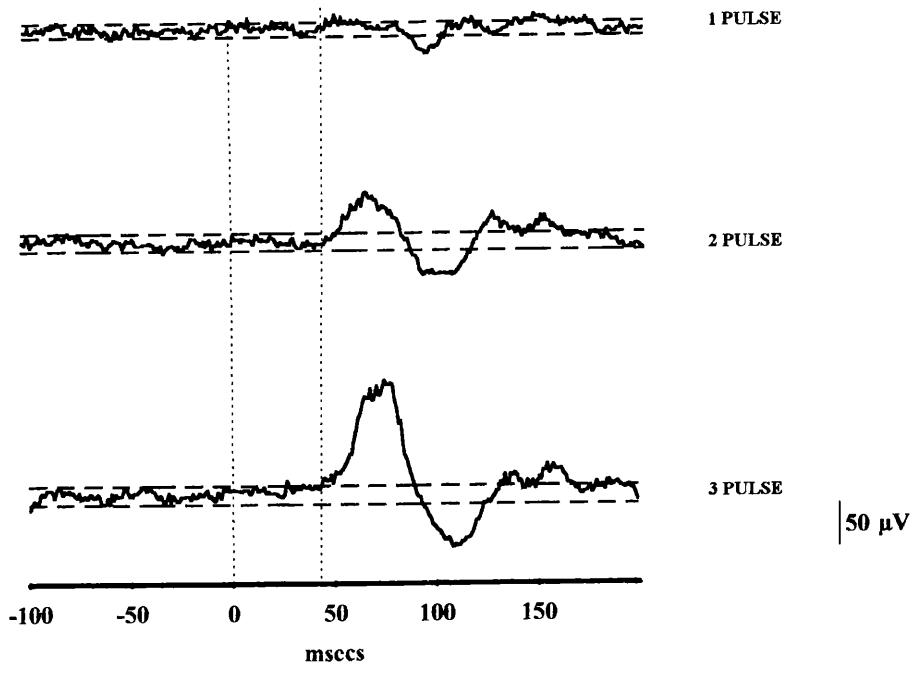


Figure 28. This figure shows the effect of increasing the number of pulses in each episode of stimulation on the size of CMRs elicited in TA by stimulation of the hallux at intensity up to 3 PT. Each trace shows the average of 500 rectified EMG responses. A single pulse delivered in upper trace, 2 pulses in middle trace, and 3 pulses in lower trace. The interval between pulses is 5 msecs in each case. The first dotted line shows the time of the stimulation at 0 msec , the second dotted line shows the time of the beginning of the responses. With single pulse, the inhibitory component was outside of the 95% predictive confidence interval .The magnitude of the responses increases but the latencies remain unchanged.

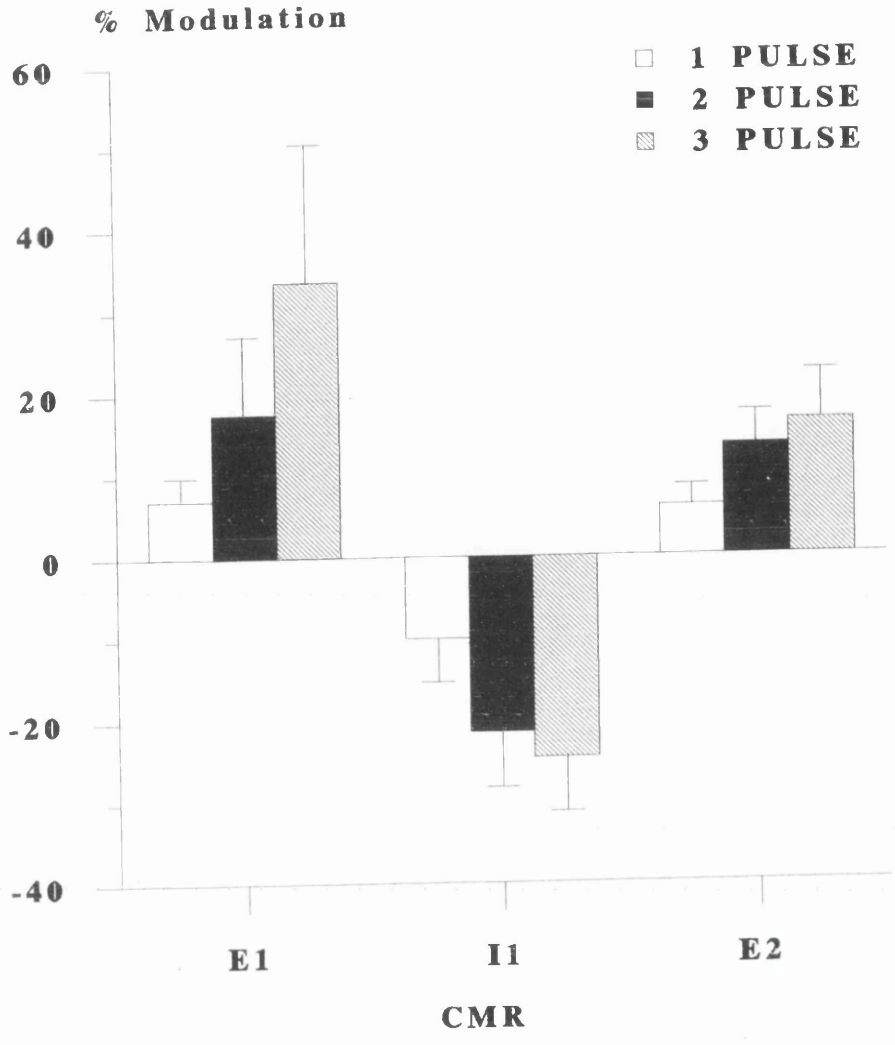


Figure 29. This figure shows the pooled data from 10 subjects. The mean and standard deviation of the amplitude of the first excitatory component expressed as percentage modulation of background EMG, increased significantly ($p<0.01$) when a second or third stimulus pulse was added to the train. The amplitude of the first inhibitory component increased significantly ($p<0.01$) when a second stimulus pulse was added to the train. The amplitude of the second excitatory component increased significantly ($p<0.01$) when a second stimulus pulse was added to the train. There was no significant differences between two pulses and three pulses for the magnitudes of first inhibitory and second excitatory components of CMRs ($p>0.05$).

3.5. The effects of the intensity of contraction on the size of cutaneomuscular reflexes in tibialis anterior

CMRs were identified as modulation of the averaged rectified EMG. The extent to which such modulations appear may depend on the degree to which the motoneurone pool is activated. This hypothesis was tested by investigating the effect of background force on the magnitude of CMRs. Experiments were performed in 7 neurologically normal subjects by recording cutaneomuscular reflexes in tibialis anterior and stimulation of the skin covering hallux. Where the background EMG was small or absent, it was not always possible to identify all components of each CMRs. A typical series of CMRs elicited against background force between 5 and 40% of MVC is shown in figure 30.

Stimulation of the hallux never evoked EMG activity if the muscle was initially relaxed. However, when a weak 5% MVC background is present a small but significant E2 response appears. At 10% MVC the E2 response is larger but retains a similar latency and appearance. At 20% MVC the E2 may be marginally smaller than at 10% MVC and by 40% MVC the reduction is clear. The CMR displays a late excitation which begins at about 175 msec when the background is 10% or 20%. This is absent in the 5 and 40% trials where the E2s appear relatively weak.

With background of about 5% of MVC, the mean amplitude of the E2 was 22% ± 14.3%. Increasing the background to 10% of MVC increased the E2 amplitude to 34.9% ± 14.2%. This increase was statistically significant when tested with paired Student's t-test ($p<0.01$). CMRs were usually at their greatest at this contraction level and further force

increases to 20% of MVC caused a slight reduction in E2 amplitudes to 24.6% ± 15.3% ($p>0.05$). Increasing the background force to 30-40 % of MVC abolished the E2 components in some of the subjects. Figure 31 shows a summary plot of these data.

The magnitude of inhibitory responses could be increased by increasing background force. An example of this is shown in figure 32. At 10% MVC there is a prominent E2 response followed by a smaller, but still significant inhibition. However at 20% MVC the inhibition is substantially larger and has an earlier onset. This has eroded the excitation almost completely. Note that the clear-cut increase in E2 with increasing force shown in figure 30 does not suffer this erosion because of the absence of a pronounced inhibition in that subject.

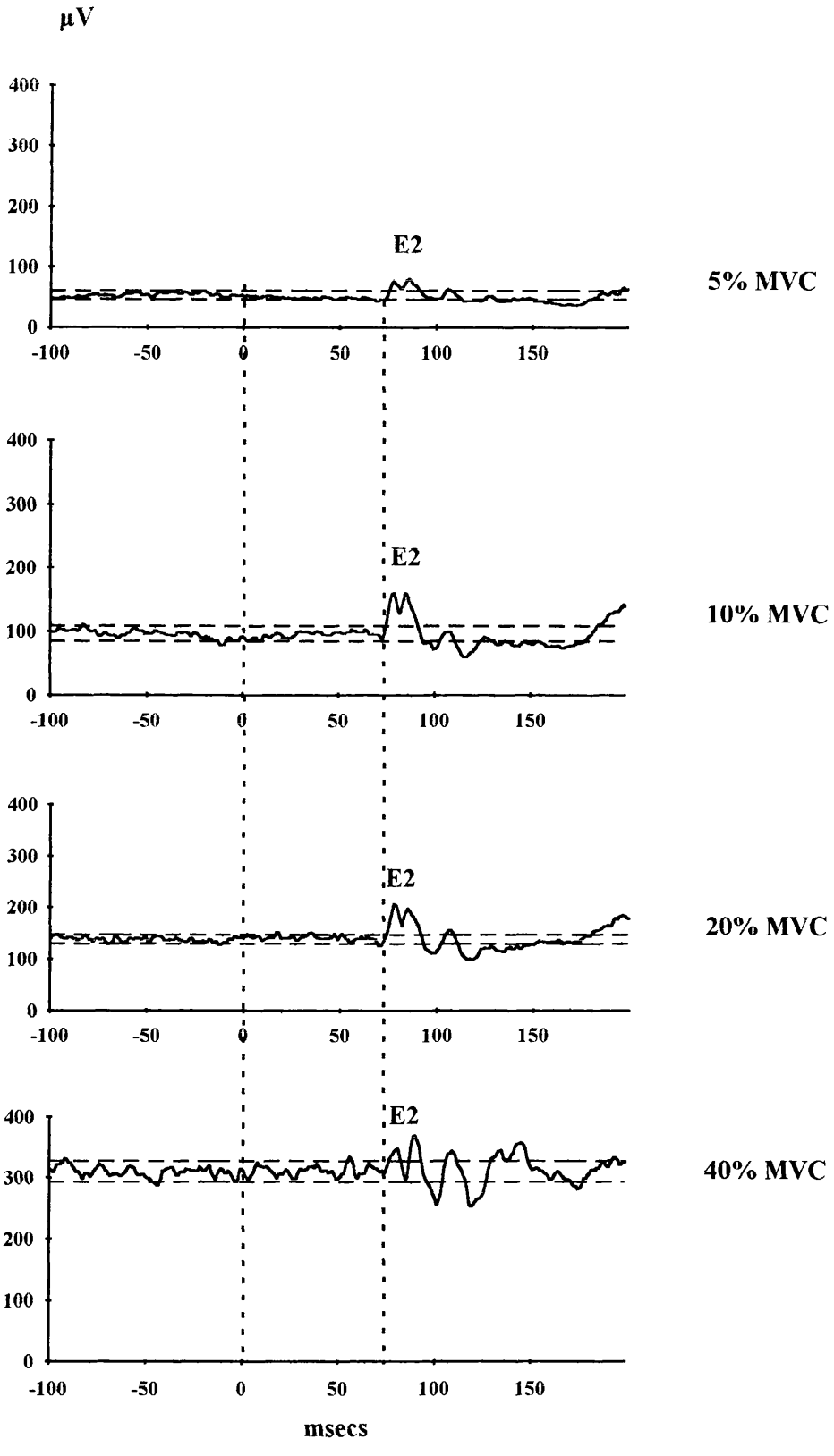

µV
400
300
200
100
0
E2
5% MVC
-100
-50
0
50
100
150
10% MVC
20% MVC
40% MVC
msecs

Figure 30. This figure shows the effect of increasing the background force on the size of cutaneomuscular reflexes elicited by stimulation of the hallux at intensity up to three times perceptual threshold in tibialis anterior. Each trace shows the average of 500 EMG responses. The first vertical dotted line shows the time of stimulation at 0 msec. The second dotted line shows the time of the beginning of the responses. The magnitude of the E2 increases up to 10% of MVC but further force increases to 40% of MVC diminished significantly the size of the excitatory component. The latencies of the responses remain unchanged.

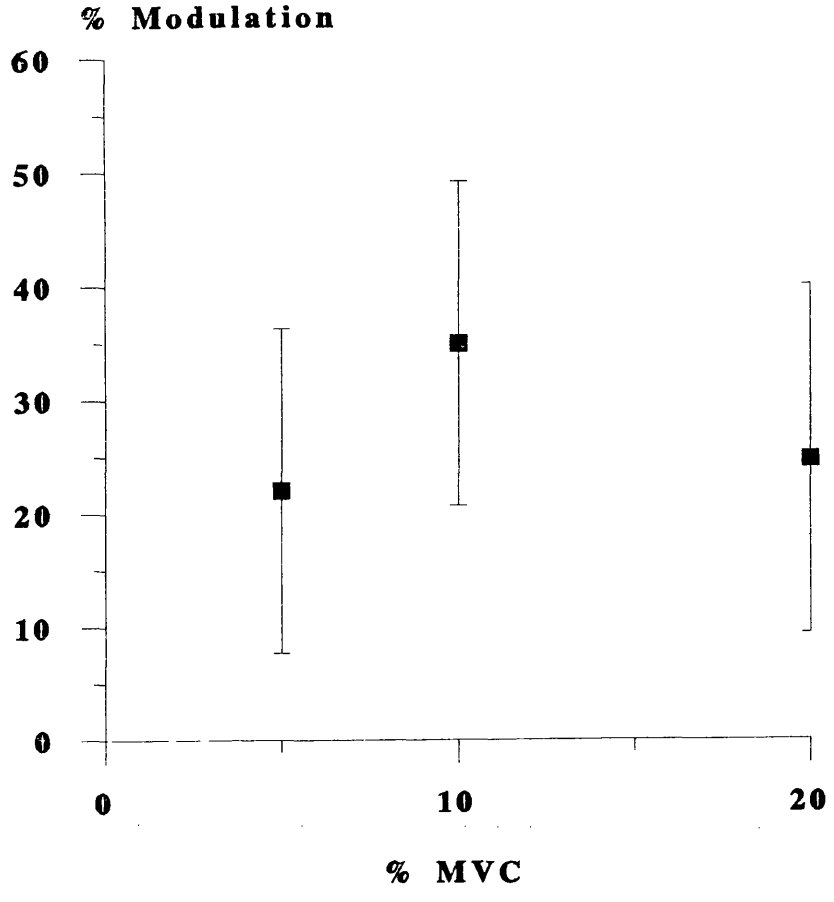


Figure 31. This figure shows the mean and standard deviation of the amplitude of the second excitatory component of CMRs expressed as percentage modulation of background EMG (n=7). The amplitude increased significantly ($p<0.01$) with increasing the background activity up to 10% of MVC, but increasing the level of background activity to 20% of MVC caused a slight reduction in E2 amplitude.

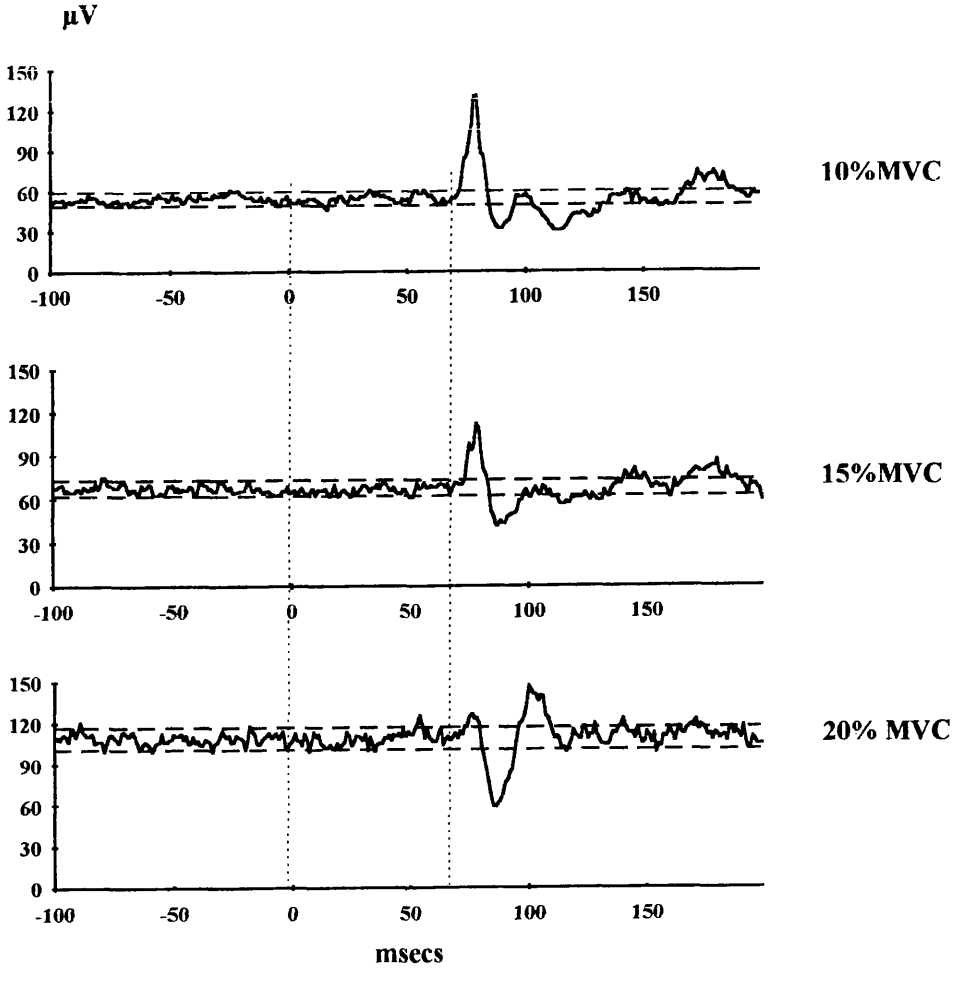


Figure 32. This figure shows the effect of increasing the background force on the amplitude of inhibitory components of CMRs in tibialis anterior. The first dotted line shows the time of stimulation exactly at 0 msec and the second dotted line shows the time of the beginning of the responses. Increasing the force up to 20% of MVC, decreased the magnitude of the excitatory component significantly. However, the magnitude of the inhibitory component increased. Note that the latency of the inhibitory component changes and it has an earlier onset at higher forces.

3.6. The effects of the posture on the size of cutaneomuscular reflexes in tibialis anterior

Experiments were performed in 5 neurologically normal subjects to investigate the effect of posture on the magnitude of CMRs. Identical stimulation/recording protocols were followed to elicit CMRs in TA whilst subjects were seated or stood erect. Since ankle dorsiflexion force could not be measured, in these experiments the background EMG activity was set to be the same during sitting and standing at about 10% of maximal activity. This was achieved during standing by asking volunteers to lean backwards slightly.

The magnitude of the second excitation (E2) was consistently larger when elicited in seated subjects. This is illustrated in figure 33. The mean E2 amplitude changed from 38.8 ± 27.9 % in the sitting position to 17.6± 13.6 % in standing position. These effects were statistically significant when tested with paired Student's t-test ($p<0.05$).

There were no significant differences between the amplitudes of the earlier E1 and I1 components ($p>0.05$). This can be seen in figure 33 where both E1 and I1 are absent in both positions. In this case the later inhibitory and excitatory components are much less modified by position than is the E2.

Figure 34 shows the comparison of the magnitude of the different components of the CMRs in seated and standing positions. The latency of the different components of the cutaneomuscular reflexes did not change significantly with changes in position.

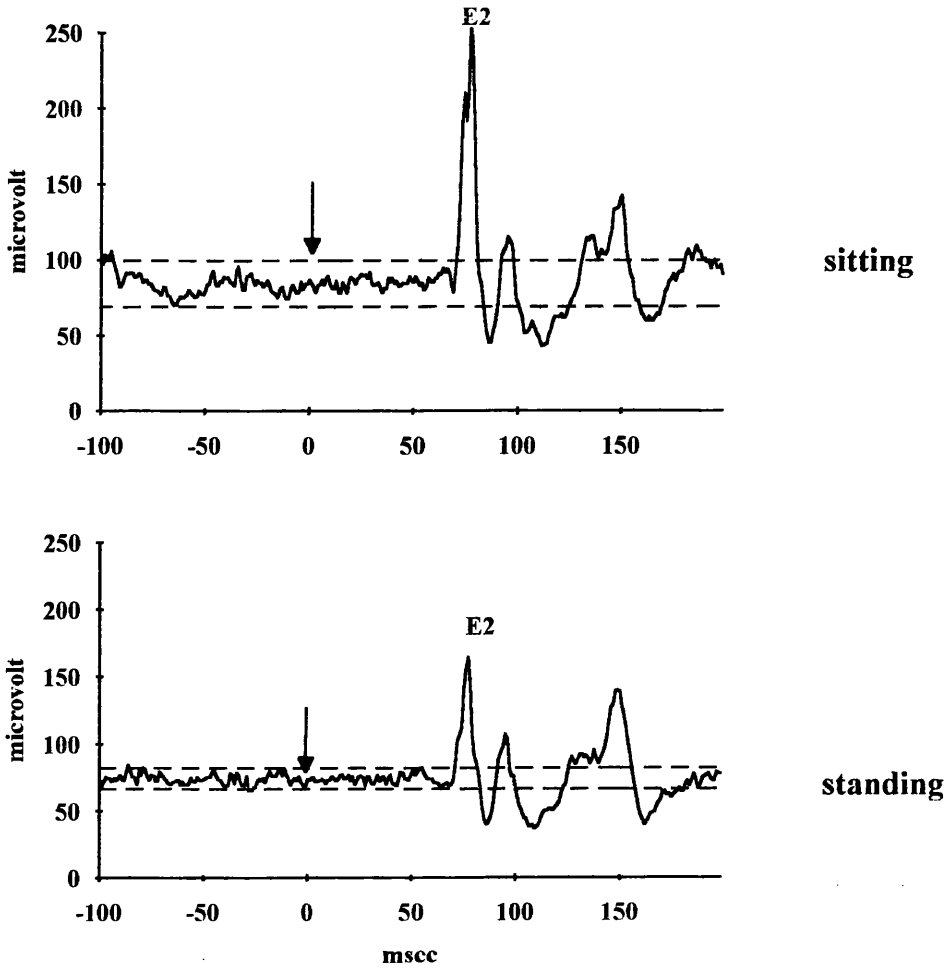


Figure 33. This figure shows the effect of posture on the magnitude of CMRs in sitting and standing positions. The subjects were asked to maintain the same background activity during sitting and standing at about 10 % of maximal EMG activity. Each trace shows the average of 500 rectified EMG responses. The arrows show the time of stimulation exactly at 0 time. The horizontal lines above and below the average of rectified EMG shows the 95% confidence interval. The magnitude of the second excitatory component (E2) decreased significantly from sitting position to standing position ($p<0.05$).

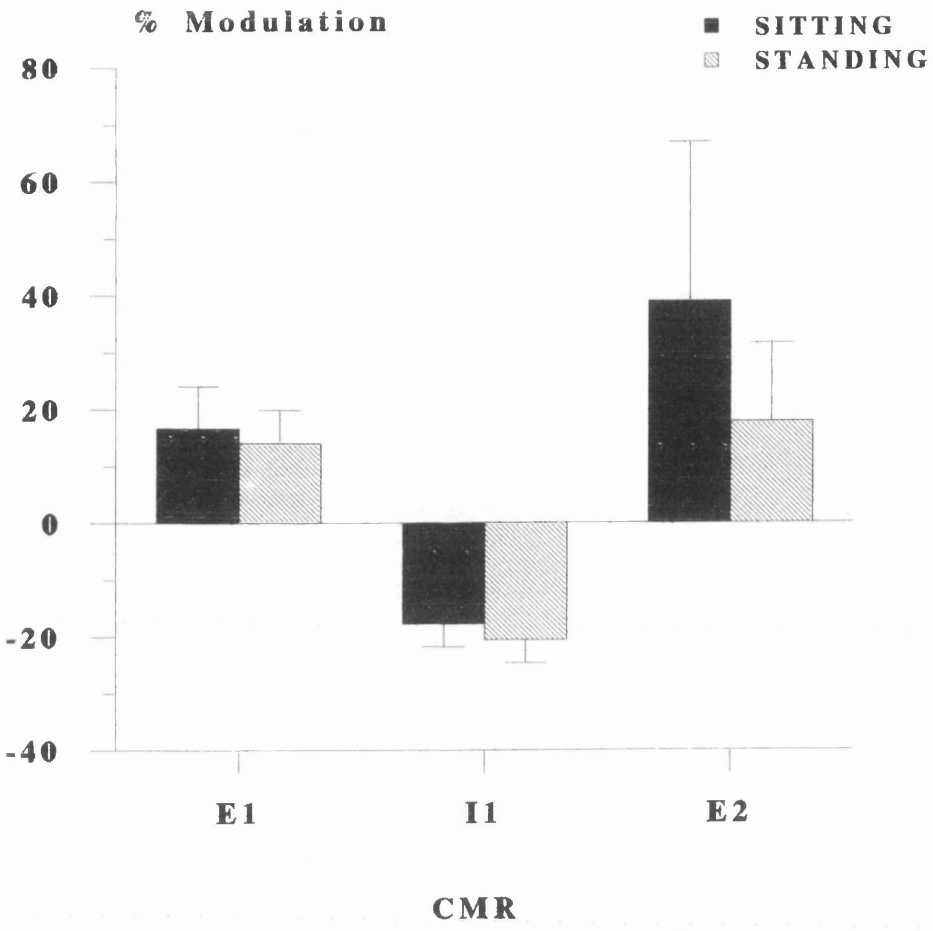


Figure 34. This figure shows the mean and standard deviation of the amplitude of the different components of CMRs expressed as percentage modulation of background EMG in sitting and standing positions (n=5). There were statistically significant differences between the amplitude of the second excitatory components of CMRs during sitting and standing (Student's t test, $p<0.05$). There was no significant difference in the amplitude of the earlier components ($p>0.05$).

The effect of backward and forward leans on the magnitude of CMRs in anterior tibial muscles

Experiments were performed in 5 neurologically normal subjects to investigate further the effect of standing posture on the size of cutaneomuscular reflexes in tibialis anterior. The experiment was conducted in the standing position during backward and forward leans. An example of typical modulation of CMRs is shown in figure 35. CMRs were elicited by stimulation of the hallux in both positions. The intensity of the background EMG is very different. It is more than ten times greater during the backward lean than during the forward lean. This reflects the postural use of TA. It was impossible to match EMG levels in the two positions. All components of the CMR are larger during backward leans. This remained true even when the responses were expressed as percentage modulation of the background activity. This can be expected to minimise the effects of changing background EMG. Figure 36 shows the mean amplitude of different components of CMRs in tibialis anterior expressed as percentage modulation of background in two different body positions for all 5 subjects. The mean amplitude of first excitation was 20.8 ± 12.7 % during backward leans and 5 ± 3% during forward leans. The mean amplitude of first inhibition was -19.1 ± 5.9 % during backward leans and -6.3 ± 8.9 during forward leans. The mean amplitude of the second excitatory components was 22.4 ± 15.5% during backward leans and 9.8 ± 16.6 during forward leans. There are significant differences between the amplitudes of the different components of the CMRs during backward and forward leans ($p<0.05$, paired Student's t-test). No significant differences were found

between the latencies of all components of CMRs during backward and forward leans ($p>0.05$, paired Student's t test).

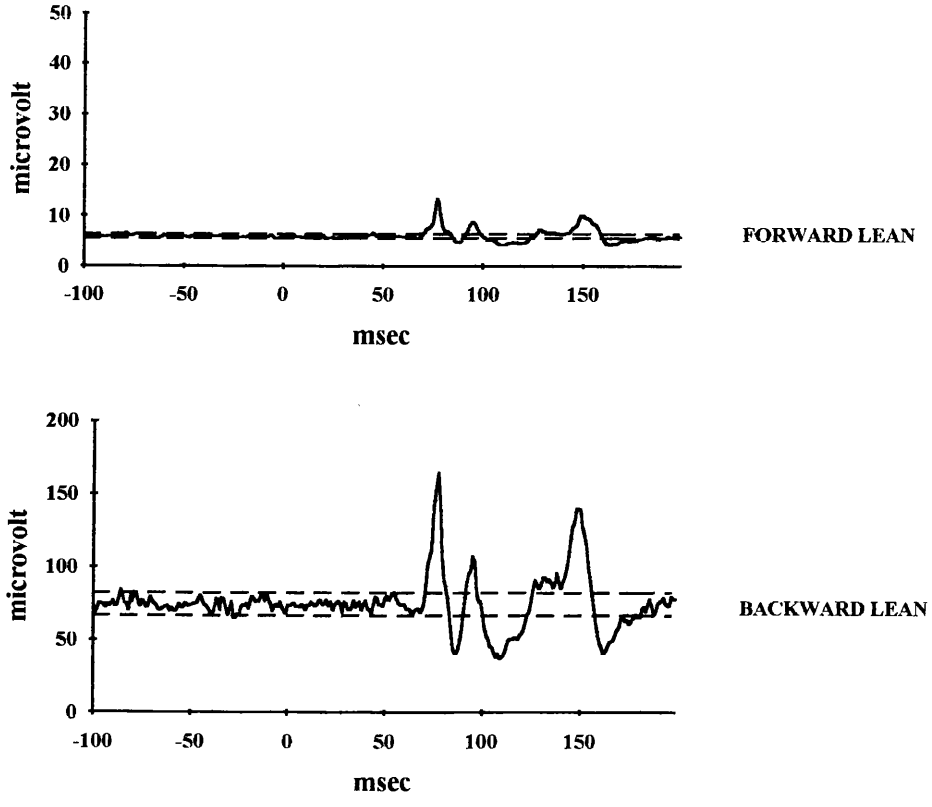


Figure 35. This figure shows the average of 500 rectified EMG responses recorded in tibialis anterior on stimulation of hallux at intensity up to 3 PT during backward and forward leans in standing position. The stimulation is at 0 msecs. The magnitude of the reflex activity is larger during backward leans than during forward leans. Not only the size of the different components of cutaneomuscular reflexes changed but also the background activity. Note the different scales for the averaged rectified EMG for each case.

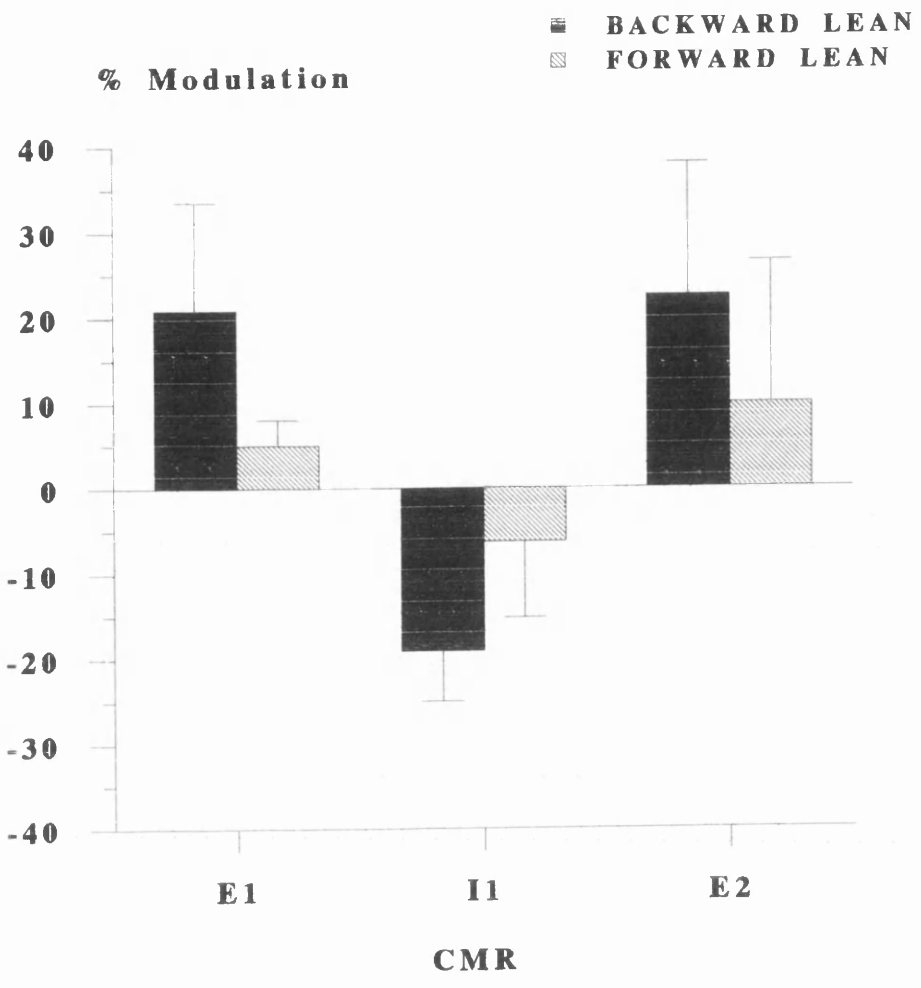


Figure 36. This figure shows the mean of the amplitude expressed as percentage modulation of the background EMG of the different components of the CMRs in tibialis anterior during backward and forward leans in the standing position (n= 5). The magnitude of the responses is larger during backward lean ($p<0.05$).

3.7. The effects of skin cooling on the magnitude of the cutaneomuscular reflexes in tibialis anterior

The question of the origin of the polyphasic CMR responses is still unanswered. Does one afferent volley generate a segmental EMG response or are distinct afferent populations responsible for each component? A further attempt to investigate this was made by cooling the skin before stimulation in the hope that different afferent populations might show differential sensitivities to cold block.

Experiments were performed in 10 neurologically normal subjects. The effect of skin cooling on the magnitude of the cutaneomuscular reflexes in tibialis anterior elicited by stimulation of hallux was tested.

Nine subjects displayed E1 responses and these were all reduced or abolished by cooling the skin to 12-14°C by application of ice packs. All 10 subjects showed I1 responses and these were again decreased in amplitude or abolished. Nine of the ten subjects had E2 responses and these were reduced in amplitude or abolished. Figure 37 shows a typical example of these results. The cutaneomuscular reflexes in anterior tibial muscles were elicited during three different stages, before and during skin cooling and on rewarming of skin. The upper panel shows the CMRs in control stage. The first excitation appears at a short latency of about 50 msecs after stimulation followed by a late period of inhibition and excitation. Middle panel shows the CMRs during skin cooling. The excitatory and inhibitory components disappeared during the cooling stage. Lower panel shows the CMRs on rewarming of skin. Rewarming the skin restored the CMRs and the magnitude of the responses often exceeded control amplitudes.

Figure 38 shows the CMRs in another subject in whom the nature of the CMR changed with cooling. The upper panel shows the CMRs in control stage. Clear E1 and I1 and a late excitatory response can be seen. The lower panel shows the CMRs during cooling stage. The E1 component has disappeared but the I1 component is still present and significant. The late excitatory component of CMRs has also disappeared. This abolition of the E1 response with residual I1 by cooling was seen in 3 subjects, therefore the excitations are easier to abolish than the inhibitions.

The amplitude of the early excitatory response, expressed as percentage of background, decreased significantly from 11.3% ± 5.3% in control conditions to 0.5 ± 0.7% during skin cooling. The inhibitory response was also reduced from -20 ± 7.2% to -7.5 ± 6.4%. The inhibitions are on average larger than the excitations. These effects were statistically significant when tested with paired Student's t-test ($p<0.01$). Rewarming the skin restored the CMRs. The CMRs often appeared larger than control on rewarming but no significant differences were found between the amplitudes of controls and recovery CMRs ($p>0.05$). Figure 39 shows the magnitudes of different components of CMRs during control, cooling and recovery.

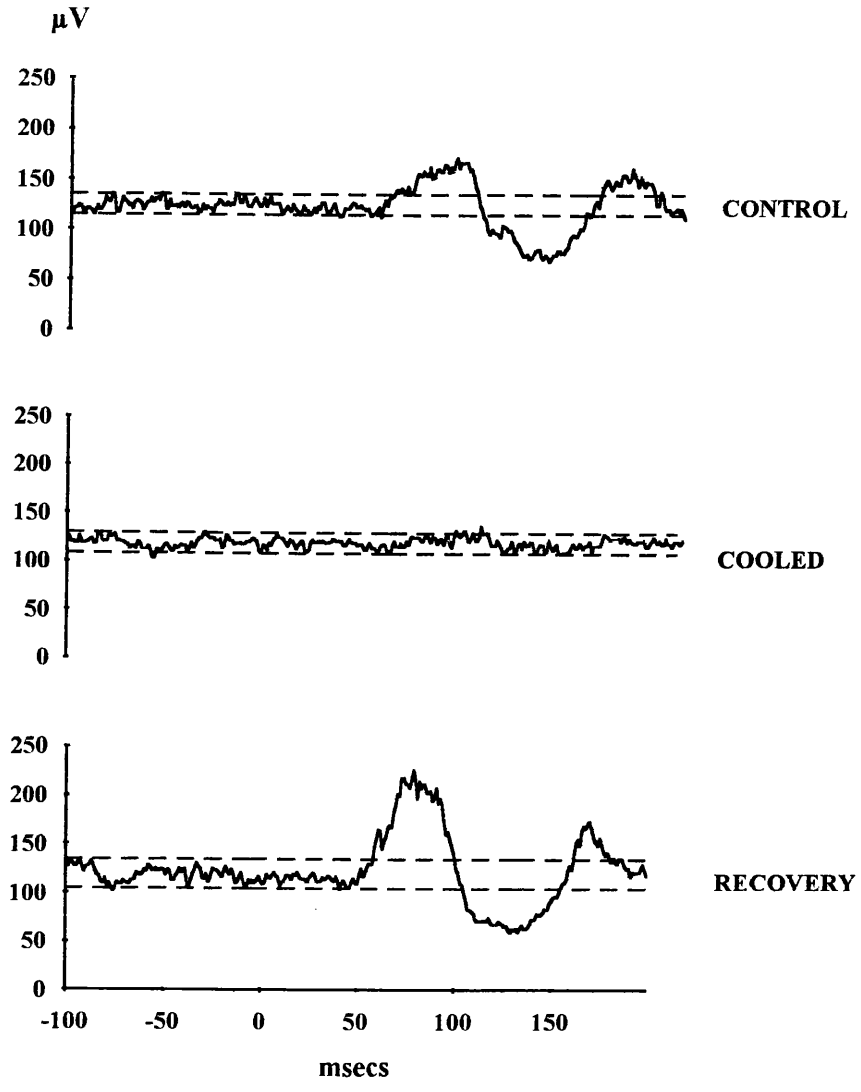


Figure 37. This figure shows CMRs in TA elicited during three different stages before and during skin cooling and on rewarming of skin. The upper panel shows the CMRs in control conditions. The stimulation is at 0 msec. The first excitation appears at a short latency about 50 msec after stimulation followed by a period of inhibition and excitation. Middle panel shows the CMRs during cooling stage. The excitatory and inhibitory components were disappeared. Lower panel shows the CMRs on rewarming of skin. Rewarming the skin restored the CMRs and the magnitude of the responses to control values.

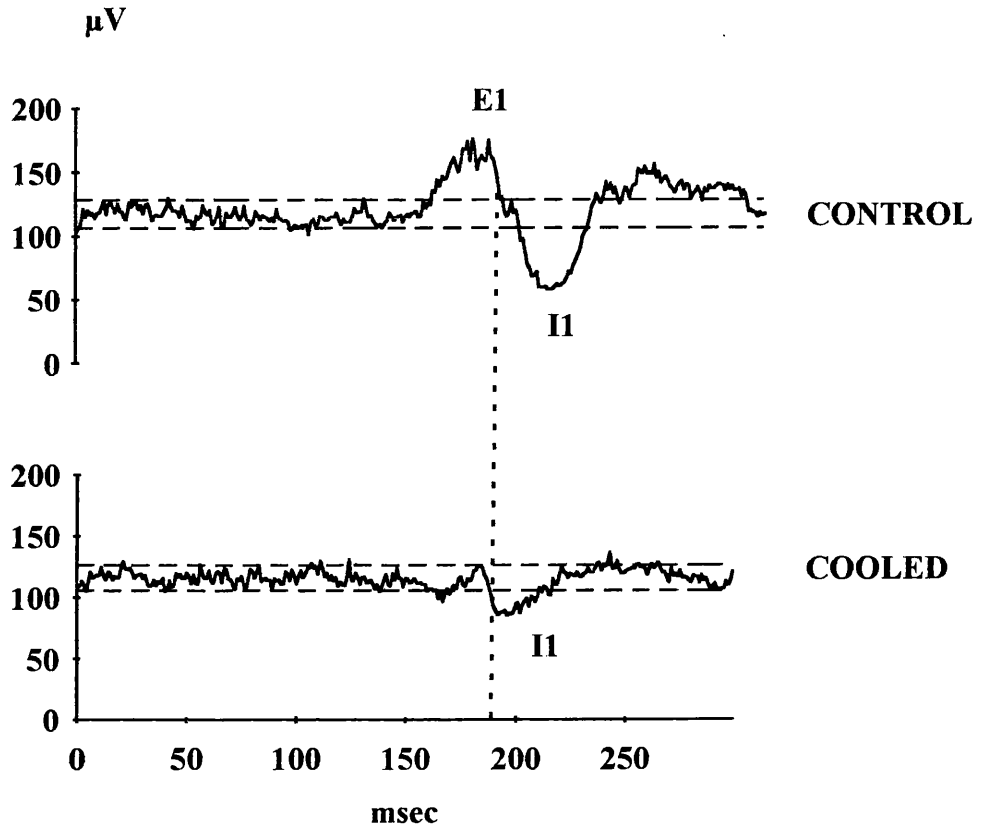


Figure 38. This figure shows CMRs in anterior tibial muscles elicited during control and cooling stage. The stimulation is at 100 msec. The upper panel shows the CMRs in control stage. The horizontal bars above and below the averaged rectified EMG shows the 95% confidence interval. The lower panel shows the CMRs during cooling stage. The dotted line shows the time of inhibitory component of CMRs. The E1 component disappeared but the I1 component is still present and significant. Note that the late excitatory component of CMRs also disappeared.

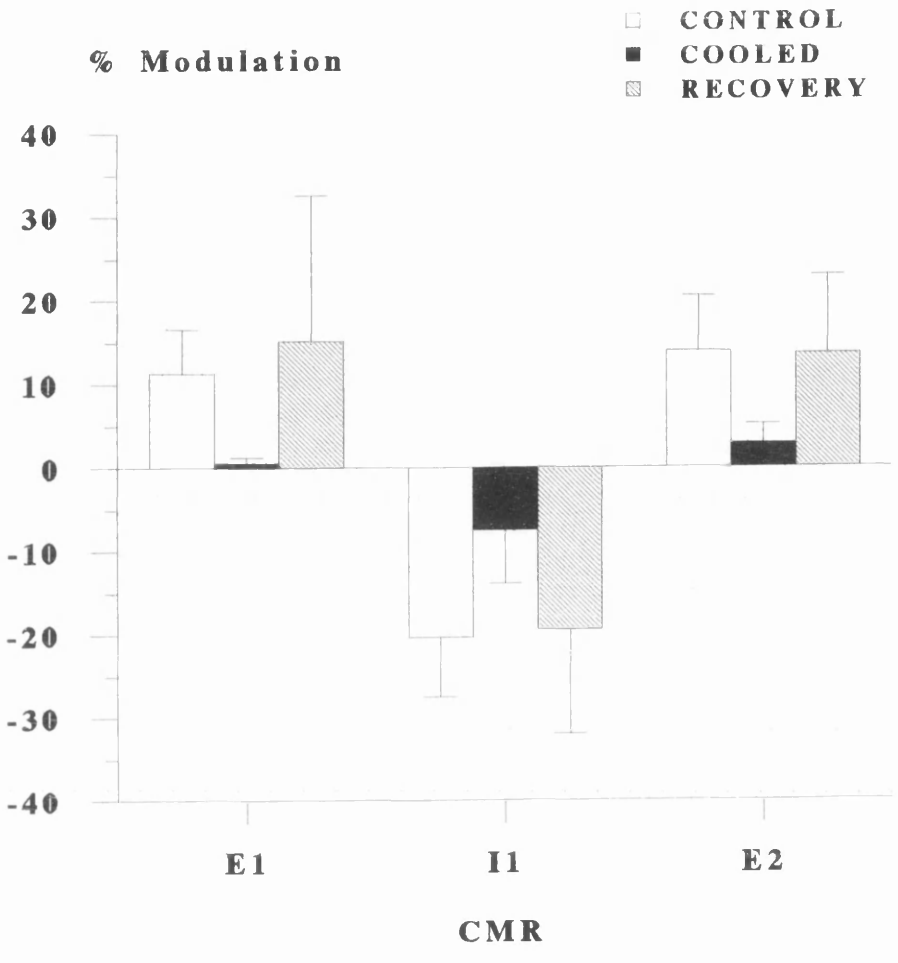


Figure 39. This figure shows the pooled data from 10 subjects. The amplitude of CMRs expressed as percentage modulation of background EMG. The mean amplitude (SD) of the E1 response decreased significantly from 11.3 ± 5.3 % in control conditions to 0.5 ± 0.7% during skin cooling. The I1 response was also reduced by ice application from -20.3 ± 7.2 % to -7.5 ± 6.4 %. Rewarming the skin restored the magnitude of the responses to control values.

The effect of skin cooling on tibial nerve neurogram

The effects of skin cooling on the evoked responses in the tibial nerve was tested in experiments on 6 subjects from those who participated in the experiments described in the previous section. The tibial neurograms were not recorded at the same time as the CMRs. Recording the neurogram required at least 4000 repetitions to be averaged rather than the 500 required for the CMR. The neurograms were recorded with higher stimulus frequencies to ensure the data was collected in a reasonable time. Details of the recordings are given in the methods.

Typical neurograms are illustrated in figure 40. Here the upper and lower panels show the recordings made at the ankle and in the popliteal fossa respectively. The form of the volley changes with the different recording conditions but the beginning and peak of the volley can be identified. The range of latencies, 4.5 - 6 msec at the ankle and 8.6 - 10.2 msec at the knee suggest that the conduction velocity of the fastest axons must be about 50 m/sec.

The amplitude of the neurograms was significantly reduced, and in some cases abolished, by cooling of the hallux. The data shown in figure 41 shows a case where the volley is almost completely abolished by cooling to about 12^{o} C. The amplitude is restored during skin rewarming. There was commonly a short period of increased neurogram amplitude as the skin temperature was allowed to recover. The summary data for peak to peak neurogram amplitudes of all six subjects is shown in figure 42. The reduction in amplitude during skin cooling to 12-14^{o} C is highly significant ($p<0.01$, paired Student's t-test). The amplitude was restored to control values after rewarming the skin.

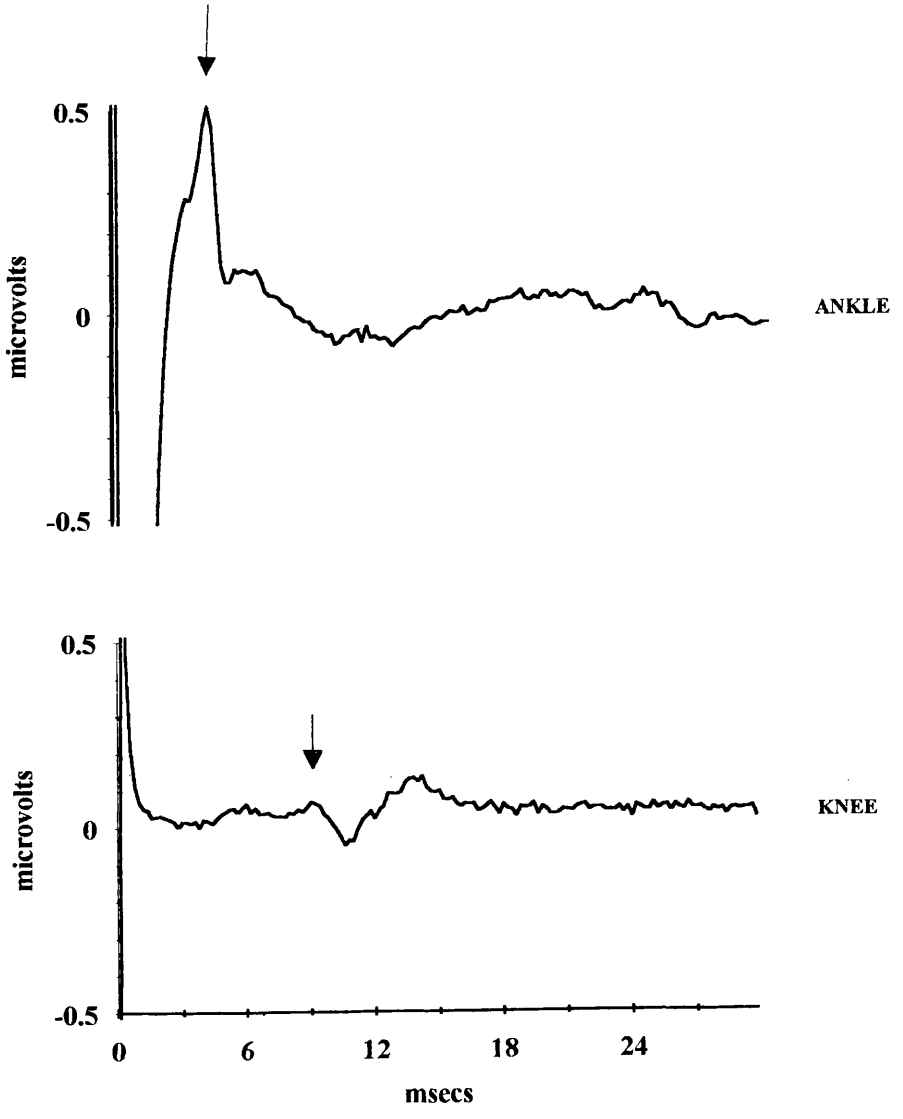


Figure 40. This figure shows the average of 4000 sweeps of tibial nerve neurogram recorded at ankle joint behind the medial malleolus and at the knee joint in the popliteal fossa. The hallux was stimulated at intensity up to three times perceptual threshold. In normal conditions, the fastest fibres conducted at about 50 m/s.

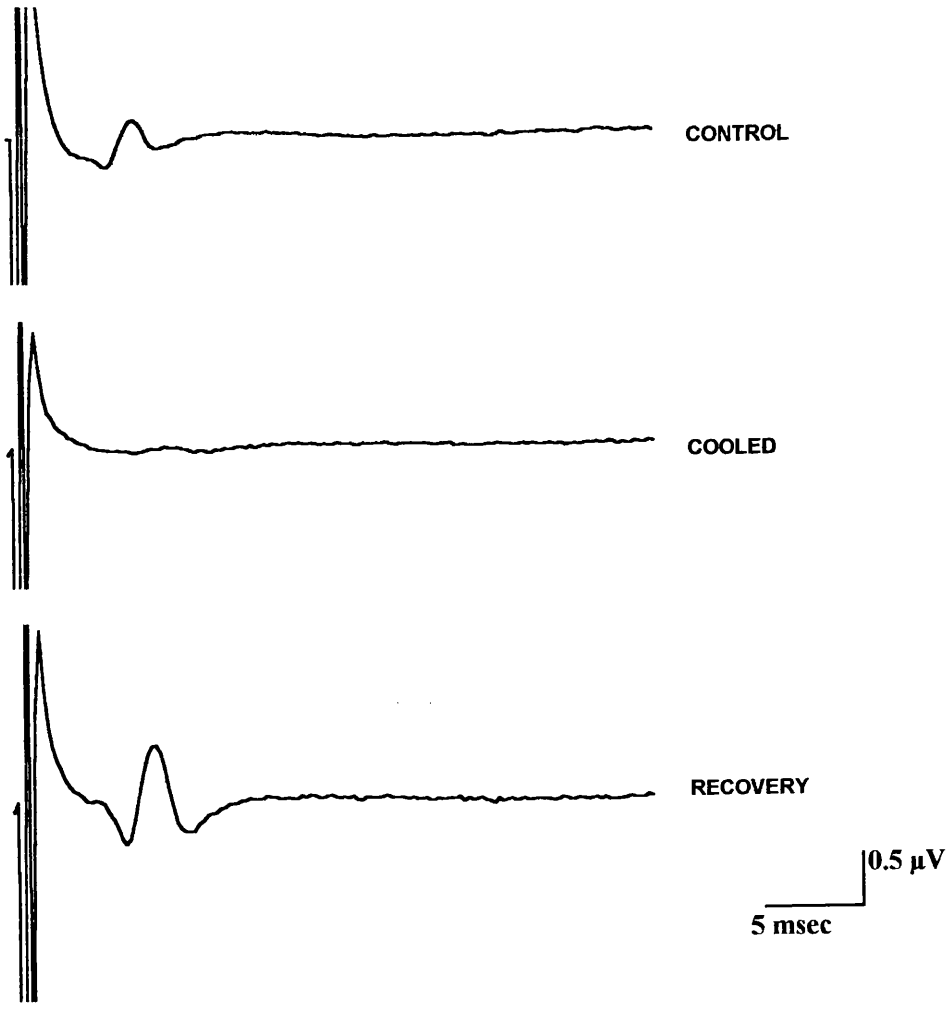


Figure 41. This figure shows the average of 4000 sweeps of tibial nerve neurogram recording at the ankle on stimulation of hallux at three times perceptual threshold in three different stages, control, cooling and recovery. The upper panel shows the average surface electroneurogram in control condition. The middle panel shows the electroneurogram during skin cooling condition. The application of ice almost completely abolished the sensory volleys in this subject. The lower panel shows the averaged surface electroneurogram on rewarming of skin.

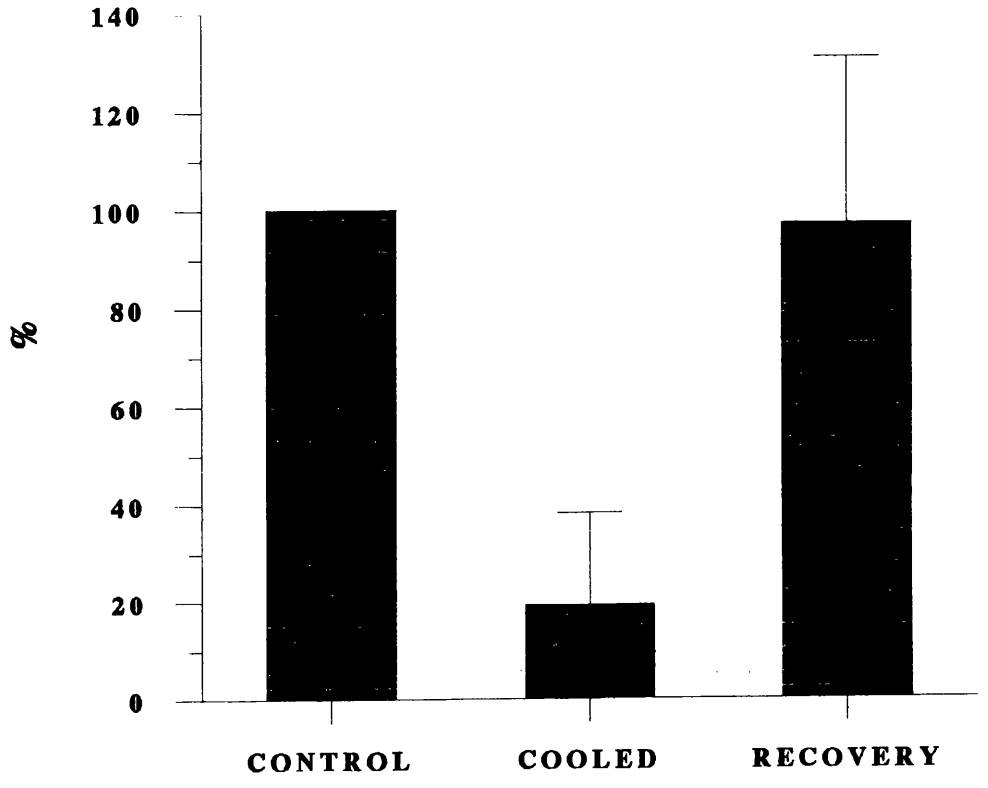


Figure 42. This figure shows the mean amplitudes (SD) of the afferent volleys were normalised to control values (n = 6). In cooling stage, the amplitude was sharply reduced to about 20 % of its original amplitude. Rewarming the skin restored the magnitude of the responses to control values.

DISCUSSION

4. Discussion

4.1. Statement of results:

Experiments were performed in 30 subjects, using 5 sites of stimulation, and recording CMRs from 5 muscles of the lower limb. In all these tests, there were only 9 occasions on which CMRs could not be elicited. Thus CMRs seem to be prominent features of the sensorimotor function of normal individuals.

Tables 11,12, and 13 summarise the results of these experiments. CMRs in TA are almost universally present in all experiments. There was only one stimulation site in one subject which did not give rise to a CMR in TA. In contrast , CMRs were much less frequently observed in quadriceps and hamstrings. There was great variability in the pattern of responses in tibialis anterior. Of the thirty subjects tested, ten had triphasic responses, ten had biphasic responses and ten had some other combination of excitations and inhibitions. The mean latency of the earliest excitatory component of the CMR is about 50 msec which is consistent with segmental spinal latency. The later excitatory components of CMRs have latencies of about 80 msec which permit a supraspinal pathway for these components.

4.2 The nature of the responses

Innocuous electrical stimulation of the cutaneous afferents of finger and toes is known to produce polyphasic reflex responses in the upper and lower limbs of the human subjects (Jenner & Stephens, 1979, 1982, Gibbs et al, 1993). These responses were identified from trigger averaged electromyograms.

Gibbs et al (1993) found that triphasic reflex responses in the muscles of the lower limb were elicited by stimulation of the second toe at 2 times perceptual threshold. An initial increase in mean rectified EMG (E1) was followed by a period of reduction in EMG (I1), and then a second increase in EMG. They reported that 3 out of 10 subjects showed an E1 response, 9 of 10 subjects showed an I1 and 8 out of 10 subjects showed an E2. The frequency of the responses in tibialis anterior on hallux stimulation in the present findings are 18 out of 30 for E1, 16 out of 30 for I1, 22 out of 30 for E2 and 16 out of 30 for I2 (table 12A). These differences in the frequency of the responses may be due to different sites of stimulation or the fact that their subjects were examined in the lying position, whilst those described here were performed in seated position.

The results reported in this thesis show that innocuous electrical stimulation of patches of skin covering plantar surface of hallux, heel, sural, mid-foot and shank can elicit polyphasic cutaneomuscular reflexes in different muscles of the lower limb, tibialis anterior, gastrocnemius, quadriceps, hamstrings and abductor hallucis.

There was considerable variability in the form of the CMR between the subjects. Each site of stimulation produced CMRs in anterior tibial

muscles, although the nature of CMR differs. The pattern of responses in TA was different in different subjects. In general, they showed a polyphasic pattern of responses consisting of excitations and inhibitions (E1, I1, E2). In almost half of the subjects a later period of inhibition (I2) was observed following stimulation of the hallux.

Stimulation of hallux regularly produced all 4 components, though E2 was the most consistent and was observed in 73% of the experiments. Stimulation of the sural site rarely generated E1 responses though it elicited I1 in all cases. Stimulation of the heel and shank elicited E1, I1 and E2 regularly but never elicited I2 responses.

Of the 4 readily identifiable CMR components, the I2 was the least frequently observed. This was true for all stimulation sites. Even stimulation which generates the strongest CMRs, elicited I2 responses on only 53% of tests. Late excitations (E2) were elicited most regularly from the skin of the mid-foot, sural and hallux regions where the frequencies were 100 %, 78 % and 73 %, respectively. In general E1, I1 and E2 components were regularly observed though there are exceptions, e.g. sural stimulation very rarely elicits E1 in tibialis anterior. The most common initial response in anterior tibial muscles on sural stimulation was an I1 component, which occurred in 8 out of 9 subjects. This was followed by a later period of excitation, E2 component, in 7 out of 9 subjects. An E1 response was observed in one subject. This agrees with Burke et al (1991) who reported the reflex response in TA on stimulation of the sural nerve at 2-4 times perceptual threshold consists predominantly of an inhibition followed by a period of late excitation. Stimulation of the sural nerve at an intensity of 1.5 times the sensory

threshold elicited less uniform responses in tibialis anterior in comparison with the posterior tibial nerve and common peroneal nerve (Tarkka, and Larsen, 1985). In their experiments, monophasic responses, showing only a distinct reduction in EMG activity, was observed in six of the total of 15 recordings and long latency excitatory response was observed in 9 recordings.

4.3. Latency of responses

In the present experiments, the early excitatory components of the CMRs (E1) in anterior tibial muscles on stimulation of hallux appeared at a mean latency of 47.7 ± 3 msec. The first inhibitory component of CMRs (I1) appeared at a mean latency of 69.3 ± 9.8 msec. The second excitatory component of CMRs (E2) appeared at a mean latency of 79.3 ± 10.4 msec. The second inhibitory component of CMR (I2) had a mean latency of 96.2 ± 11.6 msec. Tables 6-10 in results section show the latency of the different components of the CMRs in tibialis anterior on different stimulation sites.

Central delay and nerve conduction velocity

In the present experiments, the nerve conduction velocity of the posterior tibial nerve was measured by stimulation of the hallux and recording volleys in the posterior tibial nerve at the ankle joint and the popliteal fossa with stimulus intensities up to 3 PT. The fastest afferent responsible for eliciting CMRs conducted with a mean latency of

59.8 ± 4.2 m/s for onset conduction velocity and 48.5 ± 3.3 m/s for peak conduction velocity from hallux to ankle. These are in agreement with previous reports. The conduction velocity of sensory and motor nerves of posterior tibial and common peroneal nerves was measured by Lovelace et al (1973). They stimulated the posterior tibial nerve at the base of the first phalanx of great toe and recorded at ankle joint and popliteal fossa. They reported the velocity for sensory fibres in the posterior tibial nerve from ankle to knee of 49.7 ± 2.3 m/s and a velocity of motor fibres in the peroneal nerve from ankle to knee of 47.4 ± 4.4 m/s. The conduction velocity of the fastest cutaneous afferents in the human tibial nerve has been measured by Macefield et al (1989) at 52.8 ± 3.2 m/s. The present findings suggest that E1 is mediated by fast-conducting fibres with a short central delay. The afferent time from the hallux to the spinal cord was estimated by dividing the distance between the hallux and L4 roots approximately 135 cm by the mean conduction velocity of the afferent fibres. The efferent time from the spinal cord to tibialis anterior was estimated by dividing the distance between the L4 roots and mid-point of TA, approximately 84 cm by the mean conduction velocity of the efferent fibres. The central delay for different components of CMRs can be calculated by subtracting the sum of the afferent and efferent times from the latency of that component. The following formula is used to measure the central delay.

Central Delay = Latency of CMR - (Afferent time + Efferent time)

The predicted afferent times of about 27.8 msec and efferent times of

about 17.7 msec, leave a central delay of about 2.2 ms for E1 components. The short central latency for E1 indicates that this part of reflex mediated by a spinal segmental pathway.

If Shahani's (1970) conduction velocity figures (35-40 m/s) for A delta fibres are used in the calculation of central delay in the present experiments, the predicted afferent time is about 10 msec longer. However, it seems unlikely that responses evoked only just above perceptual threshold will be due to anything other than the largest afferent fibres and this would seem to strongly suggest a role for the largest cutaneous afferents in eliciting the earliest components of CMRs. As a consequence, the results on the central delays of different components of CMRs were in general agreement with those of Choa and Stephens (1982).

Short latency reflex response

Non-noxious stimulation of the second toe elicited short latency reflex responses in the intrinsic muscles of the foot such as extensor digitorum brevis and muscles of the leg such as tibialis anterior, and soleus in normal subjects (Gibbs et al, 1993, Choa, and Stephens, 1982, Jenner, and Stephens, 1979). Gibbs et al (1993) reported the latency of the first excitatory component of CMRs (E1) for tibialis anterior in a range of 43-63 msec and for the first inhibitory component of CMR (I1) in a range of 48-72 msec. They believed that the E1 and I1 components are mediated via spinal pathways. Burke et al (1991) stated the latency of the first inhibitory component (I1) of CMRs in tibialis anterior on stimulation of sural at about 60-65 msec after the onset of the stimulus train and he concluded that activity occurring 60-65 msec after the onset

of the stimulus train must be transmitted through a spinal reflex pathway.

The latencies of early components of CMRs in the present findings are in a range of 42-55 msec for E1 and 50-84 msec for I1. The latencies of E1 and I1 are in general agreement with those of Gibbs et al (1993), Burke et al (1991) and Tarkka et al (1986) and suggest that the two early components of CMRs have a spinal pathway.

Long latency reflex response

Gibbs et al (1993) reported the latency for the E2 component in tibialis anterior to be in the range of 61-83 msec and concluded that these late responses have a supraspinal pathway. Burke et al (1991) suggested that latencies of 85-90 msec for CMRs in tibialis anterior on stimulation of sural nerve are sufficient to allow a transcortical reflex response. He also stated that a long latency alone does not necessitate that the reflex volley traverses a long loop-latency supraspinal circuit. It is known that segmental reflex responses in spinalised human subjects can have very long latencies. Sural nerve stimulation can evoke EMG activity in lower limb muscles at latencies of 130-450 msec (Roby-Brami & Bussel, 1987).

The latency of the second excitatory component of CMRs (E2) in the present findings is in the range of 63-98 msec with a mean latency of 79.3 ± 10.4. E2 components were evoked in some subjects at a stimulus intensity above perceptual threshold. This indicates that afferent fibres responsible for E2 may belong to group II afferent fibres. The long central delay of about 33.8 msec for E2 indicates that this part of the

reflex is mediated by a supraspinal pathway. This is in general agreement with the findings of Burke et al (1991), Gibbs et al (1993) and Chen and Ashby (1993).

The latency of the second inhibitory component (I2) of CMRs in the tibialis anterior is in the range of 77-117 msec with a mean latency of 96.2 ± 11.6 msec. The nature of the neural pathway responsible for the long latency of the I2 reflex is unknown. Williams and Hayward (1981) suggested that the I2 components had a high threshold on stimulation of the digital nerves. Chen and Ashby (1993) showed that the I1 and E2 components were mediated by larger fibres with faster conduction velocities than those responsible for the I2 components in small muscles of the hand. However, using Shahani's (1970) conduction velocity (35 m/s) of group III afferent fibres for calculation of central delays for I2 components reveals that I2 has a long central delay about 40 msec. This indicates that I2 components may involve slower conducting afferents and pathway.

4.4. Distribution of the cutaneomuscular reflexes in the different muscles of the lower limb

Following mild, non-noxious electrical stimulation of patches of skin covering plantar surface of hallux, CMRs responses were found in several different muscles of the lower limb. Responses were recorded in tibialis anterior, gastrocnemius, quadriceps, hamstring and abductor hallucis. This distribution of reflex activity indicates that non-noxious cutaneous input arising from the great toe has a widespread action modulating motor activity in the lower limb. CMRs responses were more commonly observed in small muscles acting on great toe such as abductor hallucis and muscles acting on ankle joint such as tibialis anterior and gastrocnemius. Responses were seen less frequently in proximal muscles such as quadriceps and hamstring.

The magnitude of the CMRs varied between subjects. This variation can not be easily explained by different sensitivities of cutaneous afferents to stimulation, since the intensity of stimulation is related to perceptual thresholds. Neither can the extent of muscle activation provide an explanation since subjects produced equivalent forces normalised to their individual maximum. The variation in reflex magnitude must be explained by the relative strength of cutaneous afferent projection to motoneurone pools. This projection is clearly stronger in some individuals than others and the strength could not be related to any previous motor history of the subjects.

There are only few previous reports about the CMRs in the muscles of the lower limb. Gibbs et al (1993) recorded cutaneomuscular reflexes in the different muscles of the lower limb, extensor digitorum brevis,

anterior tibial muscles, soleus, quadriceps and erector spinae on stimulation of the second toe at intensity of 2 PT. Burke et al (1991) recorded CMRs from the ipsilateral muscles of the lower limb anterior tibial muscles, soleus, biceps femoris and vastus lateralis. They stimulated the sural nerve with needle electrodes by short trains of five pulses, each of duration 0.2 ms (train duration 12 ms) with an interpulse interval of 3 ms at intensity 2-4 times perceptual threshold.

Tarkka and Larsen (1985) recorded the CMRs in tibialis anterior on stimulation of mixed nerve (posterior tibial and peroneal) and sural nerve.

The results in this thesis are in general agreement with those of Tarkka and Larsen (1985), Burke et al (1991), and Gibbs et al (1993).

It is surprising that Burke et al (1991) used 5 pulses in a train at 300 Hz at a level 2-4 times perceptual threshold in order to activate afferents from cutaneous mechanoreceptors. They described the sensation produced as intense paraesthesiae of short duration, but they also report contralateral reflex effects which might suggest activation of a flexor withdrawal, crossed extensor reflex pathway. However, they stated that this type of stimulation was not perceived as painful by their subjects. Gibbs et al (1993) restricted their stimuli to 2 PT and reported no contralateral effects. In the experiments in this thesis, the stimulus intensity never exceeded 3 PT and was never described as painful. Indeed, when repetitive stimuli were delivered in trains at 200 Hz, the nature of the sensation changed, when a 4th pulse was added to the train. No CMRs were elicited with trains of this length or longer because of the possibility of activating nociceptive pathways.

4.5. Origin of the polyphasic CMRs

In the present study, studies of the electroneurogram suggested that the earliest components of CMRs were mediated by large, myelinated cutaneous fibres, probably group II fibres which conduct at about 50 m/s. These are considerably faster than the group III fibres at about 35 m/s (Shahani, 1970). However, the later components of CMRs elicited at 3 PT could contain components due to activation of group III afferents. It is clear that later CMR components can be elicited by the largest cutaneous afferents. Figure 25 showed an E2 response when the stimulus was just at threshold and it was very unlikely that there was any group III involvement. At higher intensities, the possibility of an Aδ contribution cannot be eliminated. Group III fibres are also thought to be responsible for the classical flexor reflex (Kugelberg, 1948). This reflex pathway seems to depend on the number of stimuli delivered and their frequency as discussed in Section 4.4. This evidence would suggest that the cutaneomuscular reflexes are distinct from the protective noxious reflexes first demonstrated by Sherrington (1910). This evidence is also supported by the association of tactile mechanical sensations with CMRs and pain perception with flexor reflexes.

The question of the origin of the polyphasic CMRs is still unanswered. Does one afferent volley generate the polyphasic CMRs? Alternatively, are distinct components of the afferent volley associated with E1, I1, E2? Two types of experiment were performed to investigate this problem. Firstly, attempts were made to separate the CMR components by graded electrical stimulation. Secondly, the analysis of frequency of responses showed which components occurred in isolation and which were associated.

Response threshold

The literature contains very few reports of experiments examining the reflex effect of innocuous stimulation. Hagbarth (1960) used stimulation which was certainly above 4 times perceptual threshold and often up to maximally tolerable levels. Valuable information can however be gained using lower intensities of stimulation which elicit mechanical sensations rather than pain.

Garnett and Stephens (1980) reported the cutaneomuscular reflexes in the first dorsal interosseous muscle in the upper limb on non-noxious stimulation of the index finger at intensity between 2 and 5 times perceptual threshold. They observed powerful reflex effects elicited at these level of stimulus intensity. Twice perceptual threshold stimulation has also been used by others (Jenner and Stephens, 1979, 1982, Choa and Stephens, 1982, Gibbs et al, 1993).

No account in the literature details responses to stimulation at perceptual threshold. In the present experiments there was only one subject who showed the weak response at perceptual threshold. This is shown in figure 25. The question which arises here is what exactly the perceptual threshold represents? Perceptual threshold was taken to be the point at which the subject could feel each pulse distinctly. It would be expected that the principal afferent fibres in patches of the skin covering the foot would be Group II, III and IV fibres. The largest and most susceptible of these fibres would be Aβ fibres. It is unlikely that Aδ and C fibres respond at perceptual threshold.

The surface neurogram recordings suggest that the conduction velocity of the afferent nerve fibres which are responsible for eliciting the CMRs are

at about 50 m/s. The conduction velocity of the group III fibres was measured between 35-40 m/s by Shahani (1970). These group III fibres are too slow to be associated with the E1 or I1 components but the timing does not eliminate them from eliciting E2 or I2 components.

The results in figure 27 show that at stimulation intensities of 1.5 PT it is possible to elicit small inhibitions without significant accompanying excitations. With more intense stimulation mixed excitations and inhibitions are generated. This suggests that on average, inhibitions are associated with the very lowest threshold cutaneous afferents, whilst excitatory responses require activation of slightly higher threshold afferents. It seems unlikely that responses evoked only just above perceptual threshold will be due to anything other than the largest afferent fibres and this would seem to strongly suggest a role for the largest cutaneous afferents in eliciting the earliest components of CMRs.

Mixed polyphasic CMRs

Caccia et al (1973) showed that in small muscles of the hand, such as abductor pollicis brevis, increasing the stimulus intensity increased both the excitatory and inhibitory components of the cutaneous reflexes. Figure 24 showed that when the stimulus intensity was increased to 3 PT the magnitudes of all components of polyphasic CMRs in tibialis anterior increased but their sequence and latency was unchanged. This is consistent with the findings of Meinck, Benecker, Kuster and Conrad, (1983) who showed that in normal subjects, stimulation of medial plantar nerve evoked cutaneomuscular reflexes in tibialis anterior. They reported that enhancement of excitatory and inhibitory reflex phases was

observed with increasing stimulus strengths. In general, the more intense the cutaneous stimulation the greater the probability of this stimulus producing a reflex response; in particular, the amplitude of the CMRs components increases with increasing intensity of stimulation.

Caccia et al (1973) recorded the sensory nerve volleys of the median nerve during eliciting CMRs in abductor pollicis brevis simultaneously. He reported that the size of the afferent volleys changed as the stimulus intensity increased. Therefore, one possibility for increasing the size of the different components of the CMRs is by increasing the stimulation intensity, so that the number of afferents fibres involved in the cutaneous reflexes is increased.

Inhibitory component of CMRs as a real response

The nature of the first inhibitory component of CMRs is worth consideration. Is it a real inhibition or merely a disfacilitation following E1, a post-reflex silent period? The subjects whose data is shown in figure 26 had purely inhibitory responses at intensities up to 2 PT. Thus in this case at least the appearance of an I1 does not cause a subsequent E2. Neither does the I1 have to be preceded by an E1.

The population of inhibitory interneurones mediating the short latency inhibitory components of the cutaneous reflex in the upper and lower limbs are normally subject to tonic facilitation from inputs whose activity is dependent upon the functional integrity of the motor cortex (Jenner and Stephens, 1982). Two classes of inhibitory interneurones in cat lumbosacral cord are known to be excited by cutaneous stimuli; the reciprocal Ia inhibitory interneurone and the Ib inhibitory interneurones.

These same cells are also known to receive excitation from the rubrospinal and corticospinal tracts (Lundberg, 1975).

The intermediate latency of I1 suggests that it involves a long loop within the spinal cord. Group II afferents are known to excite both Ia and Ib inhibitory interneurones in the spinal cord (Lundberg, 1975). Cutaneous projections to Ib interneurones have been demonstrated in man (Cavallari, Fournier, Katz, Malmgren, Pierrot-Deseilligny and Shindo, 1985). Cutaneous afferents also project to propriospinal neurones in man but these projections are generally inhibitory (Pierrot-Deseilligny, 1989). It may be that the I1 component of the cutaneous reflex response reflects activity in a pathway involving these Ia and Ib inhibitory interneurones (Rowlandson and Stephens, 1985a).

In the present experiments, in some subjects, I1 was the first response in tibialis anterior without any preceding excitatory component. Thus, the conclusion must be that the I1 is genuinely part of the initial reflex response. Similar conclusions for excitations can be drawn for the subject in figure 25. Here E2 responses occur without preceding E1 or I1 components. In addition the data in figures 38 and 39 show that the inhibitory responses persist after abolition of excitations by skin cooling. All of these observations support the belief that the E1, I1, E2 responses are independent and so can not be a sequence of responses triggered by a homogenous afferent volley. The afferents associated with inhibitions must be in the group II range but slightly higher in threshold and more resistant to cold block than those causing the excitations.

Temporal summation in the CMRs pathway

Single pulses and short trains often fail to evoke consistent reflex effects because of lack of temporal facilitation (Caccia et al, 1973; Conrad and Aschoff, 1977; Willer, Boureau and Albe-Fessard, 1978, Garnett and Stephens, 1980). This is specially true for flexion reflexes. CMRs can often be elicited by single pulses but short trains of 2 or 3 pulses elicit stronger responses as shown in figure 28.

The effect of increasing the number of pulses on the cutaneous reflexes has been investigated by several authors. Gupta and Harrison (1988) studied the properties of the various components of the CMRs in intrinsic hand muscles by using trains of stimuli of 1-6 shocks (inter-shock interval; 5 ms) and by systematically changing the stimulus strength. They concluded that the various components of the reflex exhibited differing sensitivities to different stimulation paradigms.

Burke et al (1991) used short trains of five pulses, each of 0.2 ms duration with an interpulse interval of 3 ms applied through needle electrodes placed close to the sural nerve at the ankle. Stimulus intensity was set to produce intense parasthesia, and pain of short duration at a level 2-4 times perceptual threshold.

In the present experiments, increasing the number of pulses from a single pulse to two or three pulses greatly enhanced the amplitude of the reflex, and could also be tolerated by the subjects although most did report that the sensation had become sharper. These findings suggested that CMRs can be influenced by summation processes of the responses by the cutaneous receptors increasing the afferent input to the spinal cord. The latencies of the various components did not change significantly when a second or third pulse was added to the stimulus.

4.6. Topographic organisation

Variations in perceptual threshold

In the present experiments, the perceptual threshold was different at different sites of the stimulation, hallux, heel, mid-foot, sural and shank (table 11). The hallux has lower perceptual thresholds than the other areas of the foot, while the heel has higher perceptual threshold. The perceptual thresholds currents were different between the subjects. One possible explanation for these differences in current at threshold between the sites could be the difference in the nature of the skin. The hallux is covered by skin thinner than the other areas of the foot, whereas the skin covering the heel is thicker. In addition the density of innervation may vary. While the absolute current requirement varies, stimulation of all areas of the foot evoked CMRs at an intensity of 2 PT. The range of the stimulus current intensity at each site except the hallux is restricted by possibility of initiating muscle contraction by stimulation of the motor fibres in the area. In order to eliminate the effects of the muscle activation the stimulus intensity was always kept below threshold for motor fibres.

The phenomenon of local sign has been investigated exhaustively since it was first suggested by Sherrington (1910). Hagbarth (1960) made a systematic analysis of the receptive fields for various lower limb muscles to nociceptive cutaneous stimulation. He showed wide variation in the reflex patterns according to the site of the stimulus. He concluded that dorsiflexion is a suitable withdrawal movement from a noxious stimulus at the hallux, but inappropriate as a response to stimulation of the heel

where plantar flexion would be more effective. Grimby (1963a) also mapped the skin reflexes of the foot to painful electric shocks in 25 healthy subjects. He described an extensive system of flexor and extensor reactions representing a highly purposeful defence mechanisms for specialised withdrawal. He reported that when the stimulus moved from hollow of foot to the ball of the hallux, the response changed from hallux plantar flexion to hallux dorsiflexion, and when the stimulus moved to plantar surface of the heel, the response changed from ankle dorsiflexion to ankle plantar flexion. Changing the stimulus from the medial sole of the foot to the lateral sole of the foot altered the response of supination to pronation. In general, he concluded that a stimulus applied to the hallux is more likely to excite the tibialis anterior whilst a stimulus applied to the heel is more likely to excite the triceps surae. All of these were studies of withdrawal responses to painful stimulation.

In the present experiments, an attempt was made to investigate how CMRs in TA changed as stimuli were applied to the foot at 4 sites, the hallux, heel, sural, and the shank. The present findings suggested that different areas of skin of the foot produced different CMRs in tibialis anterior. This is illustrated in figure 18. Each of the four sites of stimulation elicits a CMR but the form of the response is different in each case. The most obvious feature is the strong inhibition following the stimulation of the heel. However it is very difficult to predict the mechanical consequence of each of these CMRs on the basis of averaged EMG from a single muscle. The fall in TA EMG in 18B must lead to a reduced plantar flexion torque but in the absence of data from other muscles the functional consequences are unclear. This problem requires further experimental investigation to resolve the matter.

4.7. Background force and CMRs

In the experiments described in this thesis innocuous skin stimulation was never powerful enough to recruit any EMG in an initially inactive muscle. This agrees with Rowlandson and Stephens (1985 b) and Burke et al (1991) who reported that they were unable to record cutaneous reflexes in quiescent muscle. However, all authors agree that CMRs are easily elicited against a background of muscle activity.

If a muscle is tonically activated, rhythmic stimulus repetition results in a successive stabilisation of the reflex response, possibly because of a different pre-set of interneurones or motoneurones or both (Caccia et al, 1973, Desmedt and Godaux, 1976, Meinck et al, 1981).

Quantitative analysis of modulation of tonic EMG during reflexes is subject to several problems (Meinck et al, 1983). The most significant problem arises in coping with background activity. Small reflex effects can appear as large modulations of weak background activity. Similarly, large reflexes can appear small if expressed as a modulation of an intense background. This problem can be minimised if a standardised background is maintained. In order to keep background activity constant, a continuous feedback of EMG activity should be presented to the subject (Godaux and Desmedt, 1975b).

However, one series of experiments was performed to investigate how the amplitude of CMRs change as the intensity of the background increases.

The effect of background force on the late responses of CMRs

Meinck, Kuster, Benecke and Conrad (1985) reported that with increasing the background activity in anterior tibial muscles, the triphasic, shape and the time course of the reflex pattern remained fairly stable but the amplitudes of the individual components were in general augmented. Bruce, Poon and Poon (1991) showed differences in the nature of CMRs elicited in first dorsal interosseous during sustained contractions and during sinusoidal force modulation at 0.5 Hz. This suggests that force changes alone can not explain all the modulation in CMRs.

In this study, an attempt was made to observe the effect of the background activity from low level of activity at 5% MVC to higher level of activity at 30-40% MVC on the magnitude of the cutaneomuscular reflexes in tibialis anterior. Figures 30 and 32 illustrate examples of individual CMR components increasing with background force as described by Meinck et al (1985). Figure 31 shows summary data showing progressive increases in the E2 responses as force increases from 5 % to 10 % MVC.

In the present study, the size of the background contraction against which the CMRs are elicited had a strong effect on the magnitude of the late responses. There is evidence that CMRs are likely to be most effective when muscles are operating in a low force range at about 20% or less of their maximum force. The reasons for this are not clearly established.

Meinck et al (1985) suggested that occlusion between the tonic descending drive and the reflex effects of the afferent volley occurs to a greater extent as the contraction intensity increases.

As a result, occlusion between competing excitatory drives on the motoneuronepool is the most likely cause but the effect of fatigue during sustained higher force contractions can not be excluded.

Another explanation for reduction of CMRs amplitudes in higher force ranges might be the saturation phenomenon. The number of additional motor units recruited for a given increment in force declined sharply at high levels of voluntary force (Milner- Brown, Stein and Yemm, 1973). This suggests that even though the high threshold units generate more tension, the contribution of recruitment to the increase in voluntary force declines at higher force levels. Garnett and Stephens (1980) suggested that large late excitatory responses might be related to the higher threshold motor units which are recruited at higher levels of the force.

An additional problem with amplitude measurements is clear from figure 32. As the force increases, an initially strong E2 appears to become weaker. There is also a progressive increase in the intensity of the subsequent I2. If an excitatory phase is primarily weak, it might be completely occluded by a stronger tonic activity (Caccia et al, 1973).

In this case the inhibition is not only stronger but also begins earlier as force increases. At least part of the decrease in the E2 must be due to interaction with the I2. Thus changes in the onset latency of reflex components might appear as modulation of amplitude in some circumstances.

Ashby, Hilton-Brown and Stalberg (1986) found that cutaneous stimulation of the toes yielded facilitatory responses of tibialis anterior motor units and that this facilitation decreased during strong voluntary

contractions. The findings in the present experiments are consistent with those of the Meinck et al (1985) and Ashby et al (1986). They are similar to the findings of Bruce et al (1991) in the upper limb.

In general, there is a shift from net excitation at low forces to inhibition at high forces. The precise mechanism underlying this is not clear, but it may be that cutaneous inputs have a role in providing inhibitory control during high force contractions. It is unlikely that the effects have described here are due to circulatory arrest in the active muscles, since Stephens and Taylor (1972) indicated that occlusion only occurs at above 40 % of MVC.

Are the CMRs likely to produce significant force changes?

The relationship between the different components of the CMRs and the reflex modulation in muscle force produced by this reflex was investigated in first dorsal interosseous muscle by Harrison (1988). He found that reflex force modulation consisted of three components, excitation, inhibition and excitation. He made an assumption that these three components of muscle force corresponded to the three reflex components of the EMG.

Durbaba, Cynk and Davies (1992) showed that the cutaneomuscular reflex modulates the force of abduction produced by first dorsal interosseous muscle using trains of 1-10 stimuli at twice sensory threshold. They suggested that the modulation of the force is related to the E2 phase of the reflex. Evans, Harries and Harrison (1992) investigated the reflex force modulation in first dorsal interosseous muscle by using trains of stimuli and by systematically changing the stimulus strength and compared variations in amplitude and latency of a given component of the reflex force modulation with the behaviour of a

putative counterpart component in the EMG. They concluded that the first major component of reflex force modulation corresponds to the E2 component of the EMG and not E1. They also showed that the second and third major components of the reflex force modulation correspond to the later EMG activity such as I2 and E3.

Early excitatory and inhibitory components of CMRs are not sufficiently powerful to produce a significant modulation in the force recording (Evans et al, 1992). Subsequent experiments show that CMRs in tibialis anterior do produce force modulation (Venkatesan and Baxendale, unpublished observations).

4.8. Task-dependent changes in CMRs

It is increasingly clear that cutaneomuscular reflexes are not simply stereotyped reactions since their gain is highly task-dependent (Duysens, Tax, Trippel and Dietz, 1993). They stated that the amplitude of the cutaneous reflexes increased during human running as compared to standing position. Gibbs et al (1993) reported that in the muscles of the lower limbs and trunk, the E2 responses are task dependent and widely distributed. They compared the amplitudes of the different components of the CMRs during lying and standing and observed that E2 was significantly larger when the reflex was recorded during voluntary contraction of the muscle in a lying position than when the muscle was

posturally active in a standing position. Task-dependent changes were also investigated by Burke et al (1991) in the ipsilateral tibialis anterior, soleus, biceps femoris and vastus lateralis of the lower limb during different postural and non-postural tasks. They concluded that the reflex pattern in different muscles and within a single muscle may change, depending on the task that the subject was undertaking. They showed that in the lower limb, the response of tibialis anterior to sural nerve stimulation was larger when subjects were standing on an unstable base and the ipsilateral limb was held in flexion than when they were seated or were standing on a platform tilted toe-up. They considered the early components, with mean latencies of 53 and 71, msec as spinal reflexes. The latency of their second component is equivalent to E2 in the present findings.

In this study, the effect of sitting and standing on the size of the CMRs in tibialis anterior was investigated at the same level of background EMG. There was no significant differences between the amplitude of E1 and I1 whether tested in a seated or a standing position. This is shown in figure 34. However, the E2 responses are significantly greater whilst sitting. The present findings are in general agreement with those of Gibbs et al (1993) and Burke et al (1991) which showed that E2 phase was significantly larger when the reflex was recorded during voluntary contraction of the muscle than when the muscle was active posturally. This effect was clearly evident in figure 33. It might be that the supraspinal pathways responsible for second excitatory component of the CMRs are more active during voluntary contraction of lower limb muscles than when these muscles are active posturally. The responses in the upper limb are broadly similar to those described in the lower limb (Evans et al, 1986, 1988).

The effect of backward and forward leans on CMRs in TA

It is well known that cutaneous reflex responses may be dependent upon the behavioural context in which they are elicited. The question here is, are CMRs modulated by body position during forward and backward leans or is this change in CMRs due to variations in the background force?

In the present study the intensity of the background EMG was very different in the two different trunk positions. The EMG in TA is more than ten times greater during the backward lean than during the forward lean. This is clear in figure 35. Whilst all components of the CMRs were larger during backward lean, they were seen against a more intense background. When the amplitude of the CMR was expressed as a percentage of background EMG, as in figure 36, it was clear that CMRs were significantly greater during backward leans, even allowing for the greater EMG activity.

Figure 30 and 31 in Section 4.7 showed that the magnitude of the late responses of CMRs increased with the intensity of the contraction up to 10 % of MVC. It was not possible to measure the forces developed by TA during standing and so ultimately it is impossible to decide if the CMR modulation was due to force or postural changes on the basis of these experiments. However, since force changes of 5-10 % of MVC produce modulations of CMRs of approximately 35% in E2 and inclination of the trunk gives about the same magnitude of change in E2, it is clear that neither effect completely dominates the other.

4.9. Skin cooling and CMRs

There are no reports about the effects of skin temperature on the cutaneomuscular reflexes in the literature. Most previous investigations of the influence of skin cooling on motoneuronal excitability have centred on H-reflex testing which is thought to be a good indicator of alpha motoneurone excitability and tests of tendon jerk reflexes which reflect both alpha and gamma efferent fibres excitability. Bell and Lehmann (1987) showed no change in H-reflexes or tendon jerk in normal subjects during skin cooling. They concluded that cutaneous receptors have no influence on the excitability of alpha motoneurones and therefore that skin cooling alone would not influence muscular function. In addition, Mai, Pedersen and Arlien-Soborg (1976) found no clear-cut effects on H reflexes when skin was cooled in a group of multiple sclerosis patients.

The results in this thesis leave no doubt that skin cooling decreased the magnitude of cutaneomuscular reflexes. This is clearly shown in figures 37, 38 and 39. In some subjects the whole CMR was abolished. In others there was a residual inhibitory component during cooling. The most likely explanation of this lies in a cold block of cutaneous afferents as shown in figure 41. The possibility of additional central effects can not be completely eliminated by these experiments, but taken in conjunction with the results of Bell and Lehmann (1987) and Mai et al (1976), it is likely that central effects will not be significant. This may explain the variable effects on muscle tone reported to accompany cooling of skin with ice in physiotherapy practice (Knuttson and Mattsson, 1969). If an

area of skin which generates net excitation in a motoneurone pool is cooled, then the effect should be a reduction in muscle tone. Conversely, if an area of skin which generates net inhibition in a motoneurone pool is cooled, then the effect should be an increase in muscle tone. Thus the variation in muscle tone might be explained by CMR activity rather than any other cause.

4.10. Possible application techniques

Investigation of CMRs might be extended to clinical investigation as a diagnostic tools and also in clinical treatment such as physiotherapy. EMG testing of cutaneous reflexes is promising as a new investigative approach. Rowlandson and Stephens (1985b) believed that testing of CMRs had promise as a potential diagnostic tool. They suggested that it was limited in cases of gross hemiplegia, but in mild upper motor neurone diseases cutaneous reflex testing provided a simple and painless way of confirming the diagnosis.

Lesions of the sensorimotor cortex or the descending tracts results in a selective suppression of the second excitatory reflex phase and exaggeration of early components of CMRs in the small muscles of the hand in upper limb (Conrad and Aschoff, 1977; Jenner and Stephens, 1979). In patients with well documented lesions affecting either the dorsal column, motor cortex or descending motor pathways the E2 phase

of the cutaneous reflex in FDI was either delayed or absent (Jenner and Stephens, 1979). Chen and Ashby (1993) reported that lesions of the cord or hemisphere caused the early excitatory component of CMRs in small muscles of the hand to appear but the later responses to be abolished. Cutaneous reflex testing has also played a role in suggesting a diagnosis of minimal cerebral dysfunctions for children with learning difficulties (Rowlandson and Stephens, 1985b). They also reported in children with early metachromatic leukodystrophy, the CMRs would show a delayed E1, reflecting the peripheral neuropathy.

In patients with clinical evidence of central nervous lesions affecting the descending motor pathways such as multiple sclerosis, cerebrovascular accidents or spinal cord compression, the reflex response in tibialis anterior becomes monophasic, consisting only of the first short latency excitatory component whose amplitude is commonly exaggerated (Choa and Stephens, 1982). To date there has been little of testing of CMRs in the lower limbs of clinical populations. This is an obvious future development.

Clinical application of CMRs in physiotherapy

Cutaneous stimulation techniques were introduced into physiotherapy and occupational therapy practice (Rood, 1954). These techniques utilise non-noxious mechanical stimulation such as light brushing, icing and scratching of the skin overlying muscle groups whose normal function has been affected by various neurological disorders, such as spinal cord injury, stroke and lower motor neurone disease such as poliomyelitis. In the majority of cases, these techniques are employed in an attempt to

facilitate contraction of flaccid muscle groups.

The application of ice is a commonly used technique by physiotherapists attempting to influence muscle tone. Ice packs initially reduce regional blood flow, pain and relax muscles. In patients with spasticity ice is used in an attempt to reduce muscle tone (Knuttson and Mattssson, 1969) . It is not clear whether this effect is due to thermal or mechanical stimulation. The results of the present studies in surface neurograms have shown that decreased cutaneous afferent inputs to the spinal cord can influence the level of muscle activity and this suggests a possible explanation for the use of ice in attempting to influence muscle tone. Therefore, the reduction in CMRs amplitude may be attributed to the decreased afferent responsiveness. This may provide a neurophysiological explanation for the physiotherapy practice. Present findings also suggested that electrical rather than mechanical stimulation of the skin can change the excitability of motoneurone pools and produce reflex changes in muscle activity.

Proprioceptive Neuromuscular Facilitation (PNF)

Techniques of proprioceptive neuromuscular facilitation may be defined as methods of modifying muscle tone through stimulation of the proprioceptors (Knott and Voss, 1968). During normal movement there is a flow of afferent information present before movement and as feedback during movement. This natural afferent activity may activate, facilitate, or inhibit motor responses. Rood (1967) believed that problems of movement can all be viewed from the point of associated

sensory input. Clinically, cutaneous afferent stimulation has been incorporated into physical medicine and rehabilitation treatments for developmental disabilities and neuromuscular disorders. Cutaneous afferent stimulation is used as a component of proprioceptive neuromuscular facilitation techniques to restore flexibility, strength, and to improve sensory motor integration (Kabat, 1961).

4.11. Future plan

Several possible developments of this project can be suggested.

CMRs in the other muscles

The present experiments showed that ipsilateral CMRs can be evoked in some muscles of the lower limb. It would be useful to investigate CMRs in the other muscles of the lower limb such as gluteal and abductor muscles of the thigh and also in abdominal muscles such as rectus abdominus. In addition, this could be extended to the contralateral limb.

CMRs in neurological dysfunctions

This study has investigated responses in healthy subjects. Valuable information might be gained by examination of cutaneomuscular reflexes in patients with neurological deficits for example in spinal cord injuries such as paraplegic patients and hemiplegia in the case of cerebral dysfunctions.

The effect of diazepam on CMRs

During this project there were two observations that subjects who had taken 10 mg diazepam on the previous day had no CMRs. The same subjects had strong CMRs when tested earlier and on subsequent days. Diazepam is routinely used to control spasticity and it is worthwhile considering an extension of this investigation, particularly since it might yield information about the organisation of the spinal pathway.

The CMRs in locomotion

Duysens et al (1993) investigated the phase-dependent reflex modulation of tibialis anterior and biceps femoris to a 20 msec train of 5 electrical pulses, applied to the sural nerve at different phases of the step cycle in human volunteers running on a treadmill at 8 km/h. They concluded that the magnitude of the late responses were higher during running than during standing. They suggested that movement induced afferent activity from muscle and skin may almost continuously contribute to the ongoing activity during gait. The functional significance of cutaneomuscular reflexes in motor control can be investigated by examining how movement such as locomotion affects them.

CMRs in physiotherapy techniques

Each epoch of stimulation and data capture lasts less than 5 minutes. It is proposed to use these techniques to examine changes in cutaneomuscular reflexes before, during and at intervals after standard physiotherapy cutaneous stimulation techniques. In any one experiment, if the magnitude of the voluntary contraction is kept constant then comparison of the averaged EMG provides an indication of the strength

of the CMR. These experiments will provide information concerning the modulation of motoneurone activity during a period of sustained background activity. However, the CMRs have a functional role in motor control and it is proposed to use the techniques of eliciting the CMRs to examine changes in the amplitude of the CMRs of specific muscles before, during and after proprioceptive neuromuscular facilitation techniques in order to compare the strength of those muscles and to reinforce the motor pathways.

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