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CEREBRAL SOMATOSENSORY POTENTIALS EVOKED BY MUSCLE STRETCH, CUTANEOUS TAPS AND ELECTRICAL STIMULATION OF PERIPHERAL NERVES IN THE LOWER LIMBS IN MAN

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SUMMARY

Somatosensory cerebral evoked potentials were recorded in man to natural forms of somatosensory stimulation of the lower extremity including stretching of the muscle tendons, tapping on muscle bellies and tapping on cutaneous surfaces. These potentials were compared with those evoked by electrical stimulation of peripheral nerves measuring the amplitudes and latencies of the evoked potential components and defining the effects of stimulus variables on these parameters. Spinal cord potentials could only be detected to electrical stimuli.

Mechanical stimulation of tendons and muscle bellies evoked scalp potentials at latencies earlier than those evoked by electrical stimulation of the peripheral nerve and by cutaneous stimulation at the same level of the leg. Muscle receptors, most probably muscle spindles, are the source of the short latency components obtained by the stretching of tendons and tapping on muscle bellies. The proximal location of these receptors as well as very rapid spinal conduction account for the latency difference.

The potentials were larger to electrical stimulation of nerve trunks than to mechanical stimulation of tendons or skin, suggesting the asynchronous activation of a smaller number of fibres by the latter. Individuals with the largest potentials to one form of stimulation usually had the largest potentials to the other modes of stimulation.

The use of physiological stimuli such as muscle stretch to test the transmission in specific neural pathways might be useful in investigating the processing of relatively selective afferent volleys using noninvasive evoked potential recordings.

INTRODUCTION

The recording of somatosensory evoked potentials (SEPs) has customarily employed percutaneous electrical stimuli to activate mixed or sensory nerves (Jones, 1982). In the last ten years several studies have demonstrated the possibility of eliciting evoked potentials to different types of natural stimulation: mechanical deformation of the skin of the finger (Pratt *et al.*, 1979*a, b, c*, 1980, 1981; Ishiko *et al.*, 1980; Pratt and Starr, 1981), flexion or extension of the ankle joint (Starr *et al.*, 1981) and thermal stimulation (Carmon *et al.*, 1978). These alternative methods

have the advantage of (1) providing information about the function of the receptors and terminal nerve fibres which electrical stimulation of nerve trunks bypasses and (2) activating relatively specific types of neural elements and central pathways. The resulting evoked potentials, while of small amplitude, reflect the activity of a relatively homogeneous neural population compared with the potentials obtained after electrical stimulation of a mixed or even motor or sensory peripheral nerve. Clinical uses of naturally evoked somatosensory potentials have been infrequent due, in part, to their small amplitude but also to the lack of systematic knowledge of factors contributing to their latencies and amplitudes. This study examines in man the surface-derived cerebral potentials evoked by mechanical stimulation of tendons, muscles and skin at several levels of the lower limb, and compares natural potentials with the potentials evoked by the customary electrical stimulation of nerve trunks.

METHODS

Subjects

Evoked potentials were recorded in 8 healthy subjects (4 men and 4 women) aged 19 to 30 years. Informed consent was obtained for participation in the study. They were tested in a sound-attenuating chamber while reclining on a bed. The ankle and knee joints were fixed at 90 deg by a plastic mould to restrict movements during testing. Skin temperature of the foot was monitored and maintained between 31 and 35° C.

Stimulation and Recording Procedures

Percutaneous electrical stimulation with bipolar electrodes was performed over (1) the tibial nerve at the ankle immediately posterior to the medial malleolus; (2) the deep peroneal nerve at the ankle between both malleoli; (3) the sural nerve at the ankle immediately posterior to the lateral malleolus; (4) the tibial nerve at the popliteal fossa; (5) the common peroneal nerve at the head of the fibula; and (6) the femoral nerve at the inguinal ligament. The intensity of the stimuli was adjusted to elicit a just visible twitch of the appropriately innervated muscles without causing pain. The intensity for the sural nerve was three times the sensory threshold except in thin subjects in whom this intensity also activated the motor fibres of the tibial nerve. The stimulus to the sural nerve was diminished until the muscles of the foot no longer twitched.

Muscle stretch from mechanical stimulation of the tendon was produced by a moving coil vibrator. The spindle of the vibrator was attached to a T rod with the horizontal portion placed in contact with the tendon. Activation of the vibrator resulted in a downward rod movement of 4 to 5 mm. The sound produced by the vibrator was masked by white noise from a headphone applied over the ears of one subject. As there were no differences between recordings obtained with and without masking, it was not used during the rest of the study. For the Achilles tendon the site stimulated was 3 to 6 cm proximal to its insertion; the tendon of the tibialis anterior muscle was tapped at the ankle and the tendon of the quadriceps just below the patella at the insertion of the ligamentum patellae in the tibia. Mechanical stimulation of the muscle was produced by tapping on the skin overlying the middle of the belly of the lateral head of the gastrocnemius, the tibialis anterior and the rectus femoris. Mechanical stimulation of the skin was produced by tapping on the skin overlying the lateral malleolus and the tibial tuberosity, avoiding movements of the joints. The rod was placed 3 mm above the skin surface and activation of the vibrator resulted in a gentle 'tap'. All mechanical stimuli were produced by activating the moving coil vibrator with a 70 to 100 ms duration square wave pulse. In one subject displacement of the tendon was recorded with a force displacement transducer (FDT) attached in parallel with the

rod. The onset of the displacement occurred between 1 to 3 ms after activating the vibrator and peak displacement occurred by 10 ms (fig. 1). Two sets of 700 stimuli were performed for each peripheral nerve stimulation. This number of trials resulted in 20 per cent or less amplitude difference in the scalp P1-N1 components on repeated measures in the same subject at the same session. Two sets of 2000 stimuli were required for each mechanical stimulus to fulfil this same standard.

SEPs were recorded between an electrode on the scalp at Cz (according to the 10-20 system) referenced to the forehead (Fpz). Electrode impedances were below 5 k Ω . The Fpz site was chosen as reference since it was relatively isopotential after 23 ms (Achilles tendon tap) or 26 ms (tibial nerve) when referred to a noncephalic reference such as the shoulder. Earlier components were detected, possibly reflecting activity in subcortical structures or spinal cord. Nerve root and spinal cord potentials to electrical stimulation were recorded between an electrode over L1 referenced to electrodes over L5 or the anterior superior spine of the iliac crest. However, no clear spinal potentials could be defined to mechanical stimulation even using 10 000 stimuli. Subjects were grounded by a metal plate strapped to the leg proximal to the knee.

The potentials were amplified with a gain of 500 000 using a band pass of 30-3000 Hz (6 dB/octave slope) and averaged over a 102.4 ms time period with a dwell time of 0.2 ms and 512 points per channel. A duplicate of each average was made to assess reproducibility. The averaged potentials were plotted (positivity at the 'active' electrode of the differential configuration was displayed in an upward direction) and stored for further analysis. Amplitudes and latencies of the various components of the recorded potentials were measured from a computer display with a cursor. Latencies were measured from the onset of the electrical pulse activating the peripheral nerve or the mechanical vibrator to the peak of the various components. Amplitudes were measured in one or both of two ways: (1) absolute amplitudes between baseline and positive or negative peaks; (2) differential amplitudes between the positive peak and immediately following negative peaks. The main intent of this paper is to compare the latencies and amplitudes of components evoked by different forms of stimulation at several sites along the leg (initial positivity, initial negativity, etc.). Since these components have different latencies for each of the 14 stimuli applied, we have designated the components with their polarity (P or N for positive or negative) and their sequence (i.e., 1, 2 for first, second, etc.). The *t* test for related measures between the means was performed to evaluate differences.

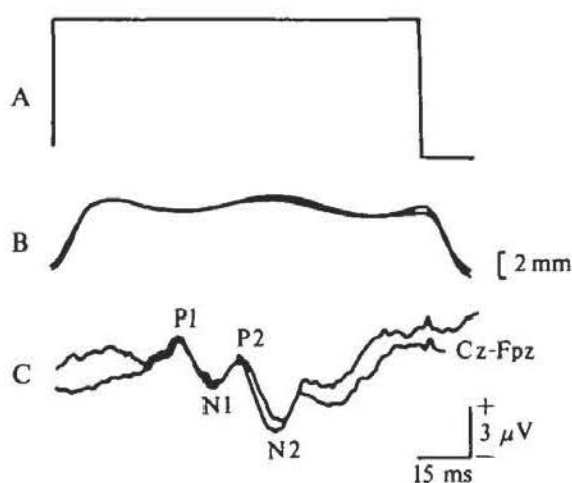


FIG. 1. A shows the stimulus pulse applied to the vibrator, B, the mechanical displacement of the tendon and C, two superimposed averages of cerebral evoked potentials (Cz-Fpz) to Achilles tendon tap stimulation. The components of the evoked potentials are labelled according to their polarity and sequence (P positive, N negative).

RESULTS

Mechanical deformation of the Achilles tendon evoked reproducible scalp potentials with an initial positive component peaking at 31.7 ± 3.1 ms (P1) followed by negative, positive, negative components at 43.5 ± 3.8 ms (N1), 53.5 ± 2.5 ms (P2) and 65.3 ± 4.5 ms (N2), respectively (fig. 1). There was a clear correlation between the latency of P1 component to both Achilles tap and tibial nerve stimulation at the ankle and body height in each subject. Fig. 2A shows these relationships in 11 subjects and fig. 2B the relation between P1 latencies to Achilles tap and to tibial nerve stimulation at the ankle. We concentrated our analysis on the initial positive-negative components and studied the influence of several stimulus variables on these components.

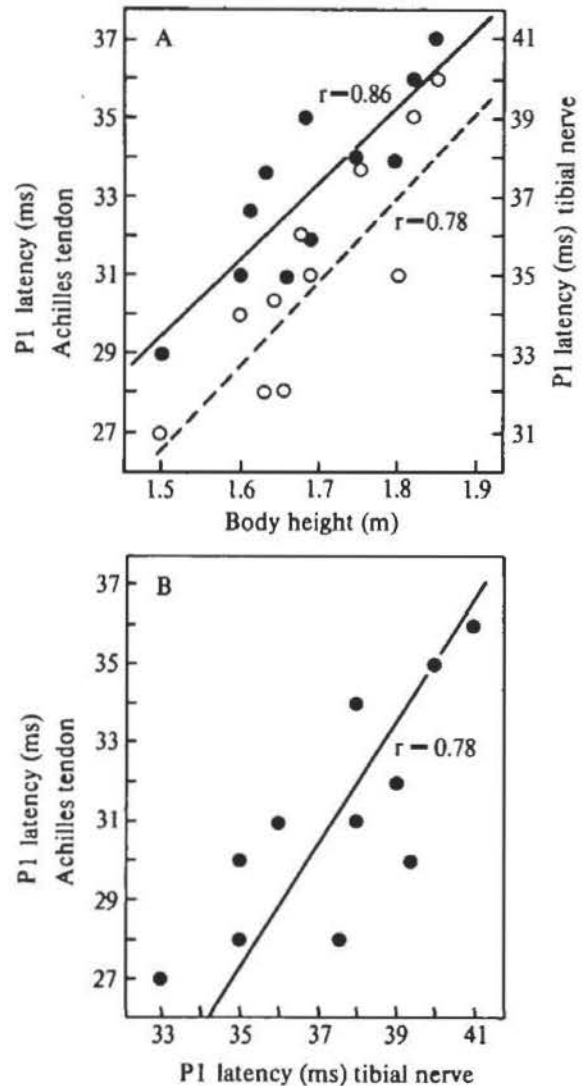


FIG. 2. A shows the relationship between height and P1 latency of the cortical potentials evoked by stimulation of the tibial nerve at the ankle (filled circles) and the Achilles tendon (open circles). In B, the relationship between P1 latencies to tibial nerve stimulation and to Achilles tendon tap stimulation are compared.

Effects of Stimulus Parameters

Repetition rate. In the one subject tested, the amplitude of the P1-N1 components decreased in a fairly linear manner as stimulus rate increased from 1 to 6 Hz (fig. 3A, C).

Intensity. As the extent of the vibrator excursion was reduced, the amplitude of the P1-N1 components decreased in the same subject (fig. 3B, C). The effective dynamic range of the stimulus over which the amplitude of the cortical potentials was affected was approximately 20 dB.

Rise and fall times. The rise time of the pulse applied to the vibrator changed the acceleration of the mechanical tap in the one subject tested and affected both the amplitude and latency of the evoked potentials. Slowing acceleration from $0.08 \text{ m}\cdot\text{s}^{-2}$ to $0.008 \text{ m}\cdot\text{s}^{-2}$ caused an increase in latency from 32 ms to 44 ms and 65 per cent diminution of amplitude (fig. 4A, C). It is interesting that for an acceleration faster than $0.035 \text{ m}\cdot\text{s}^{-2}$ the amplitude of P1-N1 did not change, whereas for an acceleration slower than $0.035 \text{ m}\cdot\text{s}^{-2}$ there was a profound

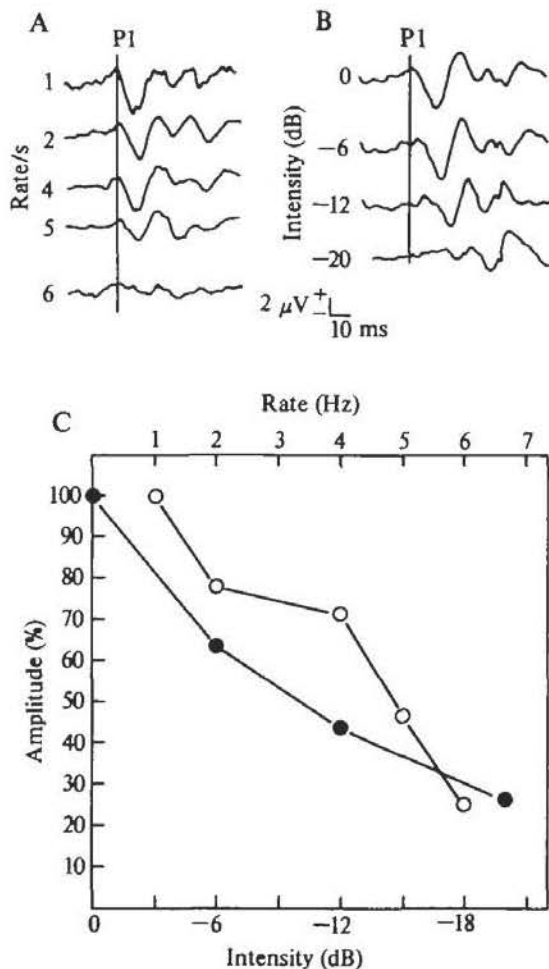


FIG. 3. A shows the effects of repetition rate and B, the intensity of the mechanical stimuli on the amplitude of Achilles tendon tap evoked potentials in one subject. The initial scalp positivity P1 is marked by a vertical line. In C, the graph shows the amplitude of the initial positive-negative complex (P1-N1) as a function of the repetition rate (open circles) and intensity of the mechanical taps (filled circles). The amplitude at the highest intensity (0 dB) and slowest rate (1 Hz) was set to be 100 per cent and the amplitudes of the other intensities and rates adjusted accordingly.

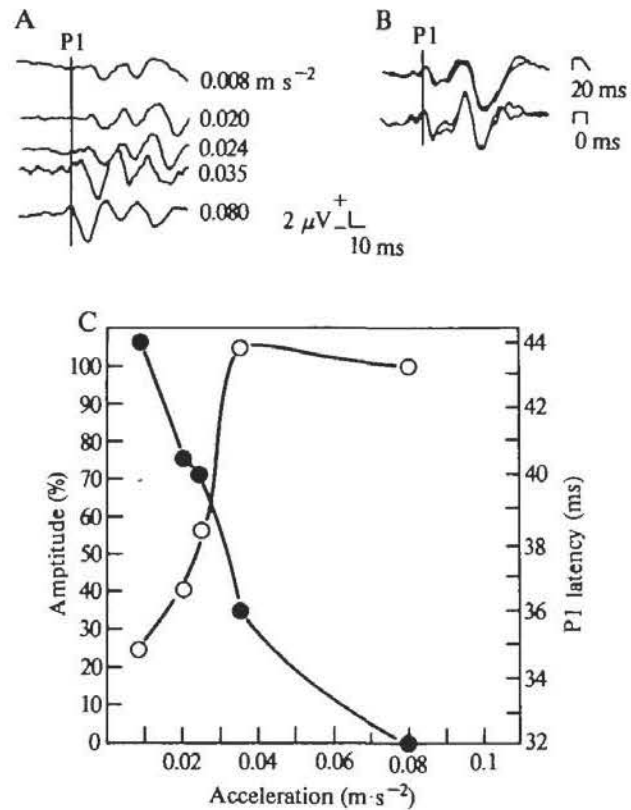


FIG. 4. Effects of changing the rise time (A) and fall time (B) of the pulse applied to the vibrator and consequently its acceleration and deceleration on potentials evoked by Achilles tendon taps in one subject. In C, the amplitude (filled circles) of the initial positive-negative component (P1-N1) and the latency (open circles) of the P1 component is plotted as a function of acceleration of the mechanical stimulus. Slowing acceleration increased the latency and diminished the amplitude of the evoked potentials while changing the deceleration of the taps had no effect.

decrement in amplitude. Changing the fall time of the pulse and thus the rate of release of the mechanical tap had no effect on the potentials since the components all occurred before the release (fig. 4B).

Site of stimulation. In one subject, taps were delivered at different positions along the longitudinal axis of the Achilles tendon beginning at the heel, extending up the tendon and ending on the gastrocnemius-soleus muscle belly (triceps surae) (fig. 5). Even though these stimuli were delivered over a 25 to 30 cm distance, their evoked potentials had the same conformation and latencies but some amplitude differences. The largest potentials were obtained when tapping on the Achilles tendon and the triceps surae muscle belly. The smallest were obtained when tapping at the union of the tendon and muscle or at the calcaneus bone.

Angular incidence of the tap. In one subject taps were delivered to the Achilles tendon at different angles of incidence in both the longitudinal and perpendicular axes (45, 90 and 135 deg) without affecting the amplitudes or latencies of the potentials.

Position of the subject. In one subject Achilles tap SEPs were recorded with the subject in three different positions: (1) lying prone; (2) sitting on a chair; and (3) lying on his side. There were no consistent amplitude or latency differences between these recordings.

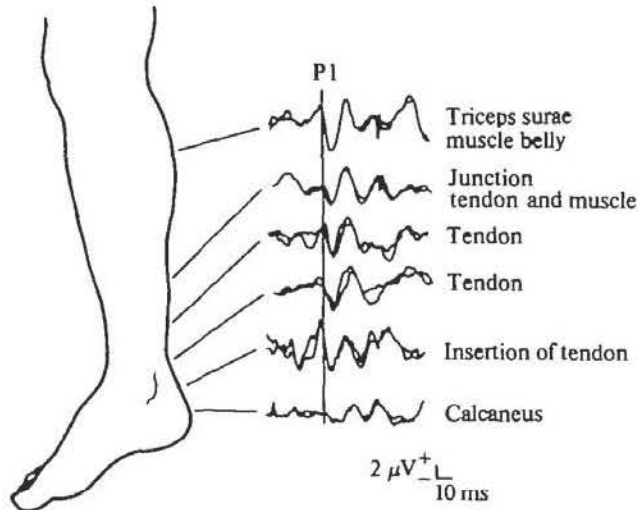


FIG. 5. Shows the effects of delivering taps at different positions along the longitudinal axis of the heel and calf in one subject on the evoked potentials recorded over the scalp (Cz-Fpz). The initial component of the potentials was maximal when stimulating the tendon or the muscle belly and minimal when tapping over the calcaneus. The initial positivity, P1, is marked by a vertical line.

Replicability of the potentials. When the same subject was tested at different times on the same day as well as on different days the potentials had the same general morphology though their amplitude varied slightly (by less than 20%) (fig. 6).

Potentials Evoked by Mechanical Stimulation of Tendon, Muscle and Skin and by Percutaneous Electric Stimulation of Peripheral Nerves

Activation of the somatosensory system from the lower limb by several different methods, namely mechanical deformation of tendon, muscle, skin or electrical stimulation of nerve trunks, evoked similarly shaped potentials from the scalp.

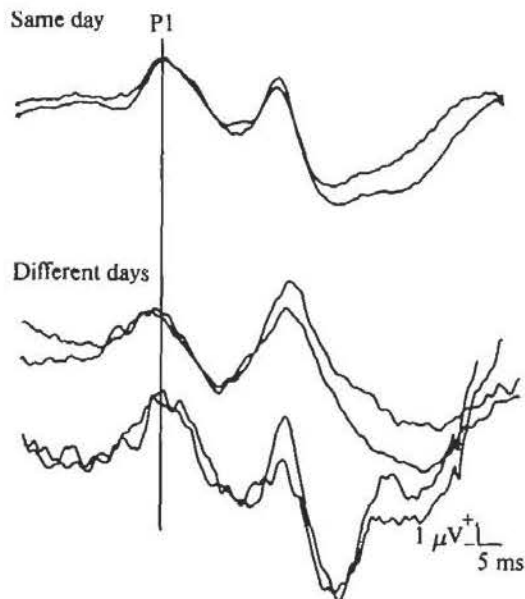


FIG. 6. Replicability of cerebral evoked potentials to Achilles tendon taps in two different sessions on the same day and on different days in the same subject. Each average consists of 2000 stimuli.

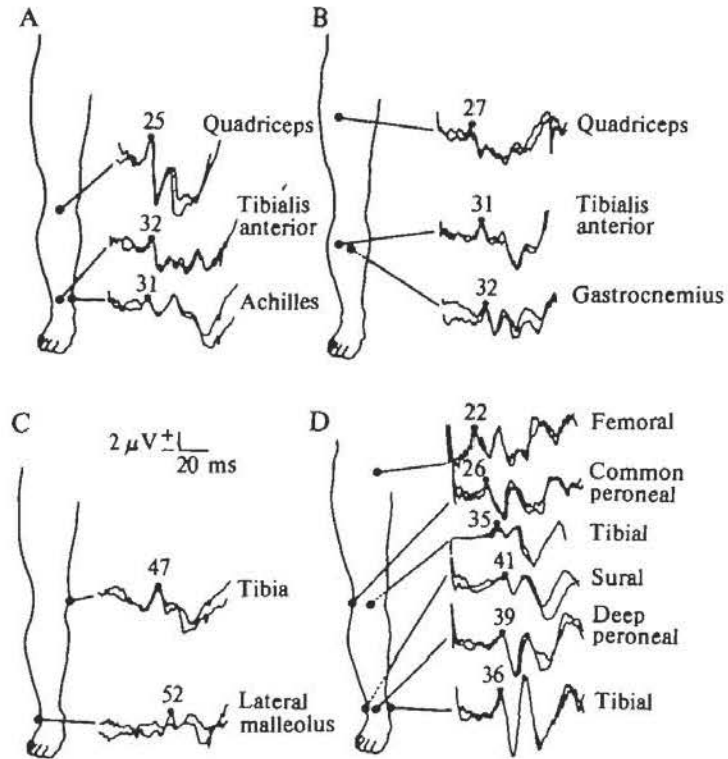


FIG. 7. Somatosensory potentials evoked by different stimuli in a single subject. A, mechanical stimulation of tendons. B, mechanical stimulation of muscle bellies. C, mechanical stimulation of skin overlying bone. D, electrical stimulation of nerves. The latencies of the P1 component recorded over the scalp (Cz-Fpz) to each form of stimulation is indicated over the peak. Averages of 700 stimuli for electrical stimulation and 2000 stimuli for mechanical stimulation are superimposed.

There was an initial positive peak followed by a sequence of alternating polarity components. Both the latencies and amplitudes of these components varied as a function of the level of the limb stimulated (e.g. ankle vs knee) as well as the particular type of somatosensory stimulus employed. Fig. 7 contains the averaged potentials from a single subject to the various stimuli applied to the ankle, knee and groin and fig. 8 the grand average from 8 subjects. The quantitative measures of latencies and amplitudes of these various potentials are in Table 1.

In summary, the amplitudes of the P1-N1 components evoked by electrical stimulation of the nerve trunks were considerably larger (4–10 μV) than those evoked by tendon taps (2–6 μV), muscle belly taps (1–2 μV) and cutaneous taps (2 μV). As would be expected, the latencies of the initial scalp positive component decreased as the site of stimulation on the lower limb was moved proximally.

Relation between Potentials Evoked by Mechanical Stimulation of the Tendon, Muscle and Skin and Potentials Evoked by Electrical Stimulation of Peripheral Nerves

Fig. 9 shows that stimulation at a given level of the leg, for example at the ankle, evoked potentials whose latencies differed in a systematic manner depending on the mode of stimulation. The latencies were shortest for tendon taps, intermediate for whole nerve stimulation and longest for cutaneous stimulation (*see also* Table 1).

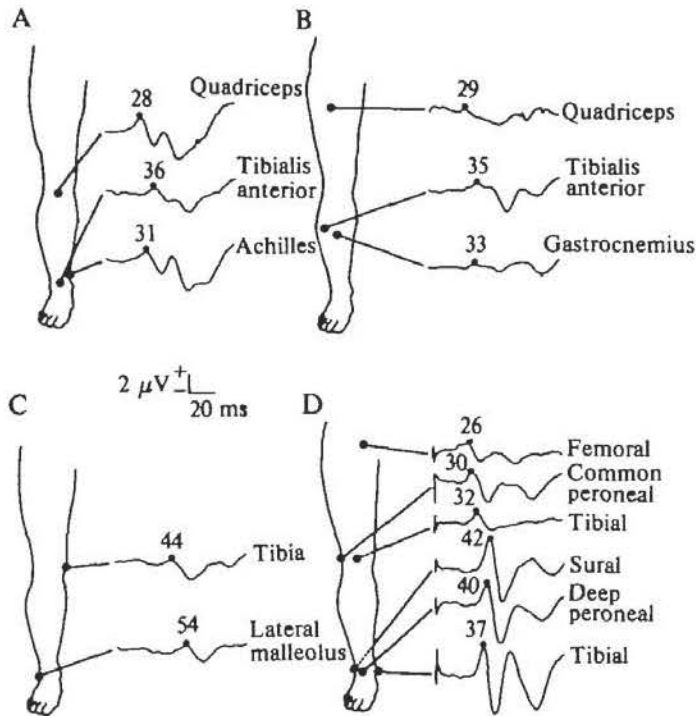


FIG. 8. Grand averages of potentials evoked by different stimuli in 8 subjects. A, mechanical stimulation of tendons. B, mechanical stimulation of muscle bellies. C, mechanical stimulation of skin overlying bone. D, electrical stimulation of nerves. The latencies of the P1 component recorded over the scalp (Cz-Fpz) to each form of stimulation is indicated over the peak.

TABLE 1. LATENCIES AND AMPLITUDES* OF P1 AND P1-N1 COMPONENT OF CEREBRAL POTENTIALS EVOKED BY VARIOUS SOMATOSENSORY STIMULI (8 SUBJECTS)

	P1			P1-N1	
	Latency	Amplitude	% Amplitude	Amplitude	% Amplitude
Nerves					
Tibial at ankle	37.5 ± 2.6	3.9 ± 2.1	100	10.0 ± 7.5	100
Sural at ankle	42.1 ± 1.4	4.2 ± 2.9	107	7.2 ± 7.9	71
Deep peroneal at ankle	40.8 ± 2.3	3.3 ± 2.0	84	8.3 ± 8.0	82
Tibial at popliteal fossa**	32.7 ± 2.1	2.1 ± 1.2	53	5.8 ± 4.0	58
Common peroneal at fibula	30.6 ± 3.3	2.8 ± 1.4	71	5.1 ± 5.4	51
Femoral at inguinal ligament	26.2 ± 3.0	1.9 ± 0.8	48	4.1 ± 3.2	41
Tendon taps					
Achilles	31.7 ± 3.1	1.1 ± 1.5	27	3.2 ± 3.1	32
Tibialis anterior	36.2 ± 2.6	0.3 ± 1.3	9	2.9 ± 2.4	29
Quadriceps	26.5 ± 3.6	2.2 ± 2.7	56	6.1 ± 5.7	60
Muscle taps					
Gastrocnemius	33.6 ± 2.7	0.7 ± 1.0	17	2.2 ± 2.6	21
Tibialis anterior	35.2 ± 3.8	1.1 ± 1.2	27	1.8 ± 2.7	18
Quadriceps	29.6 ± 5.1	0.5 ± 1.1	12	2.6 ± 2.1	26
Cutaneous taps					
Tibia	44.0 ± 11.3	0.4 ± 1.4	10	2.0 ± 1.5	20
Lateral malleolus**	54.1 ± 3.8	0.1 ± 0.8	3	1.9 ± 1.0	19

* The amplitude of potentials evoked by stimulation of the tibial nerve at the ankle was set at 100 per cent. ** n = 7.

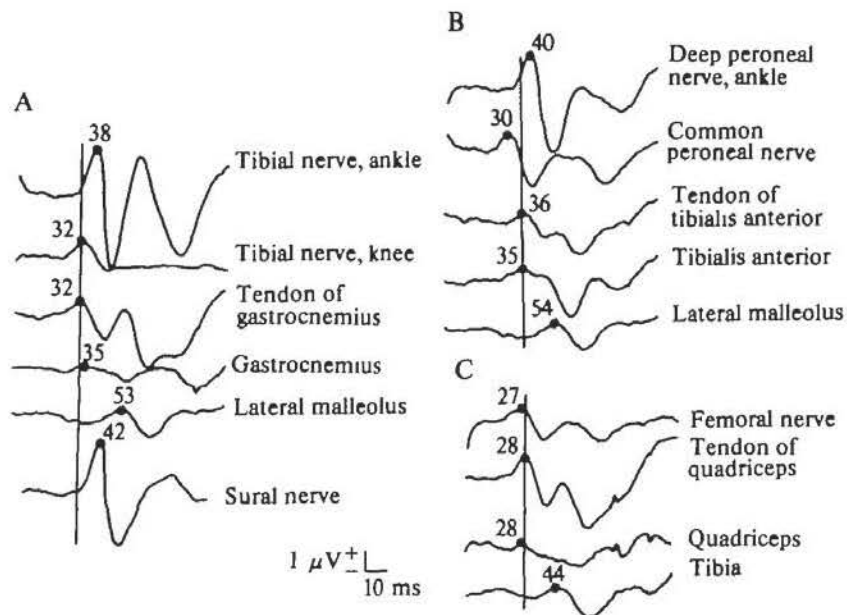


FIG. 9. Grand average of cerebral potentials to mechanical and electrical stimulation at different levels of the leg. A, posterior aspect of the ankle. B, anterior aspect of the ankle. C, knee. The latencies of P1 component to each form of stimulation are indicated over the corresponding peaks. A vertical line has been placed at the P1 latencies evoked by tendon taps at each level.

Stimulation of the posterior sector of the ankle. Fig. 9A contains the grand average of potentials evoked in 8 subjects by the various stimuli to the posterior portion of the ankle. Achilles tap evoked an initial scalp positive component 5.8 ± 1.2 ms before the initial positive component evoked by tibial nerve stimulation at the same level of the ankle ($P < 0.01$) and by 10.4 ± 2.6 ms before the initial positive component evoked by sural nerve stimulation at the same level ($P < 0.01$). This is unexpected since the mechanical stimulation, to be effective, must first be transduced by the skin, tendon and muscle receptors. Yet in spite of this transduction process, the latency to tendon percussion was shorter than to stimulation both of the tibial and the sural nerve at the same level. In fact, the latency of the scalp positivity to Achilles tendon tap was not significantly different from the latency to tibial nerve stimulation at the popliteal fossa, almost 30 cm rostrally, and to tapping on the belly of the triceps surae. These results suggest that percussion of the tendon at the ankle activates receptors proximal to the ankle to account for the shortened latency of its initial evoked potential component recorded from the scalp. In contrast, tapping on the skin overlying the lateral malleolus at the ankle evoked an even later initial positive scalp component (22.4 ± 3.3 ms) indicating that the initial scalp positivity evoked by tendon percussion did not derive from activating cutaneous afferents.

In one subject, Achilles tendon taps evoked the largest amplitude of P1 component at Pz, decreasing in a symmetrical fashion as electrodes were displaced

from that site. In contrast, electrical stimulation of tibial and sural nerves evoked the largest amplitudes of P1 component at Pz but showed ipsilateral amplitude enlargement, as reported by Cruse (Cruse *et al.*, 1982) (fig. 10). The latency of the positive component P1 from an Achilles tendon tap at the different electrode locations was within 1 ms of its latency at Cz. Thus the scalp distribution amplitude data indicate that the generators for tibial nerve and Achilles tendon tap SEPs differ. Furthermore, the latency difference is a distinguishing characteristic of these two systems and not a technical limitation due to location of the scalp electrode.

Stimulation of the anterior sector of the ankle. Fig. 9B contains the grand average of potentials evoked by the various stimuli in 8 subjects in the anterior sector of the ankle. Tibialis anterior tendon taps evoked an initial scalp positivity 4.6 ± 2.4 ms before the positive component to anterior tibial nerve stimulation ($P < 0.005$). Again the latency of potentials evoked by tendon taps was not significantly different from that obtained after tapping directly on the tibialis anterior muscle belly (1.0 ± 2.8 ms). Cutaneous tap on the malleolus of the ankle evoked a later first scalp positivity (17.9 ± 4.0 ms) and peroneal nerve stimulation at the head of the fibula an earlier one (5.6 ± 3.2 ms, $P < 0.005$). This latter finding differs from those obtained with the Achilles and quadriceps tendons in which tap stimuli evoked potentials with a similar latency to that observed in stimulation of nerves situated proximally (i.e. tibial nerve at the popliteal fossa and femoral nerve at the inguinal ligament).

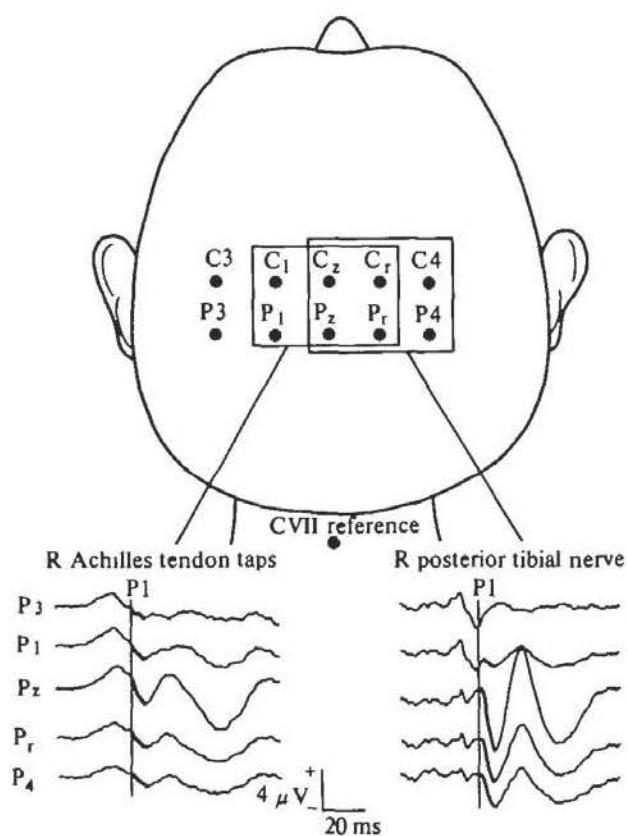


FIG. 10. Scalp distribution of cerebral potentials evoked by stimulating right tibial nerve at ankle and right Achilles tendon tap in one subject. The drawing over the scalp show the electrode positions where the amplitude of P1-N1 component was at least 80 per cent of the amplitude recorded at Cz for each modality. The coronal array of parietal electrodes (P₃, P₁: middle point between P₃ and P_z, P_z, P_r: middle point between P_z and P₄, and P₄) referenced to the skin over the seventh cervical vertebra (CVII) show the different scalp distribution for tibial nerve stimulation (clearly ipsilateral) and Achilles tendon tap (symmetrical) cerebral potentials. Vertical lines were drawn at the peak of the P1 component for each modality. Note that before the P1 component there are additional positive deflections in the scalp recordings reflecting subcortical or spinal components.

Stimulation at the knee. Fig. 9C contains the grand average of potentials evoked by the various stimuli in 8 subjects at the level of the knee. Tapping on the quadriceps tendon evoked an initial positivity 6.2 ± 5.6 ms before electrical stimulation of the tibial nerve at the same level at the popliteal fossa ($P < 0.025$, fig. 9A). There was no significant difference between the latencies to quadriceps tendon stimulation and to electrical stimulation of the femoral nerve 40 to 50 cm proximally at the inguinal ligament. There was no significant difference either between latencies of first positivity to tapping on the belly of the quadriceps muscle and to tapping on the tendon of the quadriceps. Cutaneous stimulation of the tibial tuberosity evoked potentials that occurred later (17.5 ± 4.0 ms).

Relation between Amplitudes of the Scalp Components Obtained to Stimulation of Nerve Trunks and Tendon Taps

There was considerable intersubject variation in the amplitude of the potentials evoked by all forms of stimulation. The standard deviation of amplitudes was usually close to the mean amplitude (*see* Table 1). This variability did not derive from technical considerations such as placement of stimulating electrodes, or transduction of the mechanical stimulus since those individuals with the largest potentials to one form of stimulation usually had the largest potentials to other modes of stimulation (Table 2).

TABLE 2. RANK ORDER* OF THE AMPLITUDE OF THE P1-N1 COMPONENT OF THE CEREBRAL POTENTIALS EVOKED BY ELECTRICAL AND MECHANICAL STIMULATION

Rank order	Tibial n, ankle	Sural n	Deep peroneal n, ankle	Tibial n, knee	Peroneal n	Femoral n	Tendon of gastrocnemius	Tendon of tibialis anterior	Tendon of quadriceps
Hona	1	1	1	2	1	1	1	1	1
Lisa	2	3	4	—	4	2	2	2	2
Albert	3	2	2	1	6	8	4	6	4
Yao	4	4	3	3	2	3	3	3	7
Dana	5	6	6	5	3	5	6	4	3
Greg	6	5	5	4	5	4	5	5	8
Lee	7	7	7	—	7	6	8	7	5
Seline	8	8	8	—	8	7	7	8	6

*Larger to smaller. — = no potentials obtained because stimulation at motor threshold was painful.

DISCUSSION

The results of this study show that mechanical stimulation of tendons (Achilles tendon, tendon of the tibialis anterior muscle and tendon of the quadriceps) evoke clear scalp potentials that differ in latency from potentials evoked either by stimulation of nerves at the same level in the leg or adjacent cutaneous areas. The latency difference was as much as 23.7 ± 4.0 ms if the measures were taken at the onset of the initial cortical positive component and as much as 31.7 ± 3.1 ms if the peak of the component was chosen. In this study we have emphasized measurement at the peak because of the ease of its definition.

There are several candidate receptors which could be responsible for the generation of tendon tap evoked potentials. Since the mechanical tap of the tendon stimulates the skin, input from cutaneous receptors could be significant. Cutaneous mechanoreceptors are rapidly adapting (velocity or acceleration detectors) or slowly adapting (displacement detectors) (Iggo, 1974). In our experiments, varying the rise time of the mechanical tap indicated that the receptors providing input for the evoked potentials are extremely sensitive to acceleration and therefore are likely to be fast adapting mechanoreceptors. Fast adapting cutaneous mechanoreceptors are pacinian corpuscles such as Golgi-Mazoni and hair follicle receptors. Most of them would be activated by the tap on the tendon through spread of the mechanical vibration to the surrounding skin. It is unlikely that these cutaneous receptors participate in the generation of the first scalp positivity to tendon taps since mechanical stimulation of the skin overlying bones at the same level of the tendons (i.e. lateral malleolus, tibial tuberosity) evoked potentials whose initial positivity occurred more than 20 ms later than those evoked by tendon taps at the same level of the leg.

Tapping on the tendons could also activate receptors in joints lying close to these tendons. Joint capsules contain free nerve endings, pacciniform, Ruffini type and Golgi type corpuscles. Most of their afferent fibres are group II (except those for Golgi type) (Matthews, 1974). Several reasons are against their participation as a source of tendon tap evoked potentials. First, the joints were fixed by a mould, so that very few of these receptors would be activated by the slight mechanical deformation of the tap. Secondly, the conduction velocity of group II afferent fibres would not result in an initial scalp event to mechanical taps at a latency earlier than that to stimulation of the tibial nerve at the same level. Thirdly, in additional studies we found that ischaemia of the foot produced by a pressure cuff placed just above the ankle did not alter the latency of the Achilles tendon evoked potential while the potentials tibial nerve stimulation at the ankle were abolished, similar to the results of Starr *et al.* (1981) using ankle flexion as a means of stretching the muscles to evoke cerebral potentials.

Golgi tendon organs could also be activated by mechanical tendon taps. They are sensitive receptors of muscle tension that are distributed throughout the body musculature with a density nearly as great as that of muscle spindles (Houk *et al.*, 1980). They are situated at the junction between the muscle tendon and extrafusal muscle fibres (Sweet and Eldred, 1960; Bridgman, 1970; Schoultz and Sweet, 1972; Barker, 1974). They are known to have extremely low thresholds in response to active contraction (Jansen and Rudjord, 1964; Houk and Henneman, 1967) and respond poorly to passive stretch (Houk *et al.*, 1971). In man, tendon organ afferents discharge at long latency to muscle stretch, when the muscle reflex contraction first occurs (Burg *et al.*, 1973). If this result also applied to our experiments, Golgi organs would have been activated 35 to 40 ms after the tendon tap, at the time of the first reflex contraction. Since the initial scalp events to tendon taps can be detected 30 to 40 ms after the tendon tap it is unlikely that activation of

Golgi receptors could contribute to this short-latency event. Although Golgi tendon organs can discharge in response to tendon taps, the stretch of a relaxed muscle is a relatively ineffective stimulus and, consequently, they are unlikely to be a significant source of receptor input from the small mechanical deformation of the tendon used in this study.

Receptors in the muscle itself are the most likely source of afferent input for the tendon tap evoked potentials. The primary muscle spindle endings lie midequatorially, in bag and chain fibres (Matthews, 1972). These receptors are extremely sensitive to a rapidly accelerating stretch (Matthews, 1972) and their signals are conducted along fast Ia fibres. Moreover the central pathways for muscle spindle activity project to the cortex through a rapidly conducting system (Phillips *et al.*, 1971; Lucier *et al.*, 1975; Hore *et al.*, 1976). In an earlier study in man, Starr *et al.* (1981) showed that stimulation of a peripheral nerve fascicle, identified as containing muscle spindle afferents, evoked cerebral potentials at short latency.

The secondary muscular endings lie juxta-equatorially, predominantly in the chain fibres on regions that are well striated (Matthews, 1972) and they have a much lower dynamic sensitivity to tendon taps than do primary endings (Lundberg and Winsbury, 1960; Bessou and Laporte, 1962). Moreover, the peripheral conduction velocity of the group II afferent fibres from secondary endings would not permit such an early cortical potential as that obtained to tendon tap stimulation.

Muscle mechanoreceptors like pacciniform corpuscles are found almost exclusively at the musculotendinous junctions, are smaller than pacinian corpuscles (Matthews, 1972) and in the soleus muscle of the cat represent only 2 per cent of the muscle spindles (Barker, 1962). Occasionally a pacinian corpuscle may be found in the muscle. These receptors could be activated by the mechanical disturbance of the tendon but their contribution to the evoked potential is probably very small.

The finding that the potentials evoked by tendon taps occur earlier than on stimulation of the nerve at the same level of the leg could be due to several mechanisms: (1) the distance that the afferent volley must travel to tendon taps is less than that to stimulation of the nerve since the muscle spindle receptors lie proximal to the place of percussion; (2) the peripheral conduction velocity of the afferent nerve fibres from spindles activated by tendon taps is faster than for those activated by percutaneous electrical stimulation of the nerve; (3) the central conduction velocity of the afferent volley to tendon taps may be faster than to electric stimulation of the whole nerve; and (4) a combination of all or some of these possibilities.

The spindle receptors in the triceps surae muscle belly lie 25 to 30 cm proximal to the site of percussion at the ankle which would account for a decrease of 5.6 ms if the fibres conducted at $53 \text{ m}\cdot\text{s}^{-1}$ (Burke *et al.*, 1981). However, it must also be appreciated that the onset of tendon displacement occurs 1 to 3 ms after activating the vibrator and peak displacement occurs by 10 ms (fig. 1). In addition, there is a time delay involved in the propagation of the percussion wave to activate the spindle receptors (possibly $40\text{--}80 \text{ m}\cdot\text{s}^{-1}$) Brown *et al.*, 1967) and the time for

receptor transduction to occur (Burke, 1983). These latter factors would tend to increase the latency of the evoked potentials by 3.5 to 8 ms, whereas the proximal location of the receptors would reduce it by 5 to 6 ms. Using the 3.5 ms figure for time of stimulus transduction and activation of the spindles we estimate that the proximal location of the muscle spindle receptors probably accounts for approximately half (2.5 ms) of the 5.8 ms latency disparity between tendon tap and nerve stimulation at the ankle. The possibility that differences in peripheral conduction velocity from these two methods of stimulation could account for the latency disparity is not supported by recent data derived from epidural recordings in man (Sherwood *et al.*, 1983). In these subjects the latency difference between the potentials evoked by tendon taps and electrical stimulation of the nerves could be accounted for by the proximal location of the receptors activated by tendon taps.

A final possibility to explain latency differences between tendon tap and nerve evoked potentials is that the conduction velocity of the afferent volley to tendon taps is conducted centrally more rapidly than is the volley to electrical stimulation of the whole nerve. Muscle spindle afferents from triceps surae converge on neurons of the dorsal spinocerebellar tract (Lundberg and Oscarsson, 1960; Kröller and Grüsser, 1982) that conduct through the spinal cord quite rapidly in man ($65\text{--}80\text{ m}\cdot\text{s}^{-1}$, Jones *et al.*, 1982), but must synapse in the cerebellum and thalamus before arriving at the cerebral cortex. In contrast, conduction of input from electrical stimulation of mixed nerves to evoke cerebral potentials depends upon the integrity of the posterior columns (Halliday and Wakefield, 1963; Noël and Desmedt, 1975) which are known to conduct relatively slowly ($30\text{--}50\text{ m}\cdot\text{s}^{-1}$, Jones *et al.*, 1982) but have a direct path to the cerebrum with synapses in the gracile and cuneate nuclei and the thalamus. Thus the shorter latencies of the cerebral potentials to tendon taps compared with those evoked by whole nerve stimulation probably reflects a combination of two factors: the proximal location of the receptors activated by tendon tap stimulation coupled with their conduction along a very rapid spinal pathway.

The basis for ipsilateral scalp distribution of tibial and sural nerve SEPs in contrast with the symmetrical scalp distribution of Achilles tendon tap SEPs is not clear. Certainly such results would be compatible with different orientations of their cortical generators. Cutaneous receptors project mainly to areas 1 and 3b and muscle receptors to area 3a to account for the sural nerve and Achilles tendon tap SEP differences (Phillips *et al.*, 1971; Powell and Mountcastle, 1959). Such cortical differences, however, cannot explain the asymmetry between tibial nerve and Achilles tendon tap SEP scalp distributions because both derive from activation of Ia afferent inputs (Burke *et al.*, 1981).

The amplitudes of the potentials evoked by tendon taps were smaller than those obtained to percutaneous electrical stimulation (Table 1). This may be due to the fewer number of nerve fibres activated by stretching the muscle, as well as their relative asynchrony compared with electrical stimulation of the entire nerve. Individuals with the largest potentials to one form of stimulation usually had the

largest potentials to other modes of stimulation. While it is possible that the number of central neurons and tracts activated by the same stimulus can differ in different subjects it is more likely that there are individual anatomical differences in the cortical dipole orientations similar to those described for the scalp distribution of somatosensory or visual evoked potentials (Halliday, 1982; Seyel *et al.*, 1983). Another possibility is that there are individual differences in the resistive-capacitative properties of the medium between the cortical generators and the surface recording electrodes, resulting in different filtering properties of the volume conductor.

Clinical and Physiological Significance

Somatosensory potentials evoked by electrical stimulation of peripheral nerve trunks and recorded from scalp electrodes are now an accepted diagnostic procedure (Halliday, 1978; Starr, 1978). Potentials evoked by natural activation of somatosensory pathways through cutaneous mechanoreceptors (Pratt *et al.*, 1979c), skin cooling (Chatt and Kenshalo, 1979), noxious thermal stimulation (Carmon *et al.*, 1978) and muscle stretch (Starr *et al.*, 1981) have also been described but thus far have not had clinical applications. This study shows that mechanical stimulation of tendons (Achilles tendon, tendon of the tibialis anterior muscle, tendon of the quadriceps) evoked cerebral potentials that most probably reflect activation of a fast somatosensory pathway originating in muscle spindles and group I fibres. Long-latency muscle reflexes of triceps surae may be demonstrated in humans to abrupt muscle stretch (Berardelli *et al.*, 1982). Abnormalities of long-latency reflexes have been reported in reflex myoclonus (Dawson, 1947; Halliday, 1967; Chadwick *et al.*, 1977; Shibasaki *et al.*, 1978) and parkinsonian rigidity (Tatton and Lee, 1975), but there is no agreement as to the mechanisms responsible for their generation (Marsden *et al.*, 1983). Moreover, it is thought that long-latency reflexes appearing in different muscles may have different origins (Marsden *et al.*, 1983). The simultaneous study of muscle reflexes and muscle stretch evoked potentials might provide additional information as to mechanisms underlying long-latency muscle reflexes and the physiology of motor systems in health and disease. Furthermore, the use of a physiological stimulus such as muscle stretch that tests the transmission in neural pathways under conditions more closely resembling the natural states is, theoretically, more likely to demonstrate functionally important deficits.

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