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Permalink
https://escholarship.org/uc/item/1qd88670

Journal
Clinical Neurophysiology, 62(2)

ISSN
1388-2457

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Publication Date
1985-03-01

DOI
10.1016/0168-5597(85)90022-x

Peer reviewed

ABOUT THE ORIGIN OF CEREBRAL SOMATOSENSORY POTENTIALS EVOKED BY ACHILLES TENDON TAPS IN HUMANS [1]

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In a previous study we showed that mechanical taps on the Achilles tendon evoked cerebral potentials at an earlier latency (32 msec) than did electrical stimulation of posterior tibial nerve at the same level of the leg, the ankle (38 msec) (Cohen et al. 1985). In the studies presented in this paper we attempt to define the receptors activated by tendon taps that provide the afferent input for such short-latency evoked potentials recorded from the scalp. There are at least 5 types of receptors involved in transducing mechanical taps to the skin or tendons to neural inputs: primary and secondary muscle spindles, Golgi tendon organs, joint and cutaneous receptors (Jaeger et al. 1982). While a variety of direct recording methods from nerve fibers can be used to characterize these afferents in animals, these techniques cannot be applied to humans. Instead, indirect evidence from the effects of ischemia (Lewis et al. 1937), temperature changes (Eldred et al. 1960), muscle contraction (Vallbo 1973; Cohen and Starr 1985) and vibration (DeGail et al. 1966; Hagbarth and Eklund 1966) on the cerebral potentials offers possible experimental approaches to separate the various afferent contributions to tendon tap evoked potentials. For instance, when a sphygmomanometer cuff is inflated to above systolic pressure around a limb ischemic hypoxia begins distally and pro-

gresses proximally. Large fibers are affected before small ones and sensory before motor fibers (Leksell 1945; Laszlo 1966; Torebjörk and Hallin 1973). Ice cubes applied over a limb can reduce cutaneous temperature rapidly (2–3 min) and affect surface receptors, but longer periods of cooling (15–20 min) are necessary to reduce intramuscular temperature and the sensitivity of receptors within the muscle (Eldred et al. 1960; Abbruzzese et al. 1980). Muscle contraction strongly activates muscle spindles through fusimotor activity (Vallbo 1973), thereby modifying afferent inputs from this source as well as affecting the transmission of sensory input within the central nervous system (Cohen and Starr 1985). Finally, vibration applied to tendons of a muscle strongly activates muscle spindles (Hagbarth 1973) and produces presynaptic inhibition of IA afferents (Desmedt 1983).

This study examines in humans the effects of ischemic hypoxia and cooling of the leg, muscle contraction and vibration on cerebral potentials evoked by Achilles tendon taps and posterior tibial nerve stimulation in order to obtain indirect evidence leading to the identification of the afferent sources for Achilles tendon tap evoked potentials.

Methods

Subjects

Subjects were young and healthy students between 15 and 25 years old. They were tested while lying on a bed in a sound attenuating chamber. The ankle of each subject was fixed at 90° by a mold designed to restrict movements of the foot.

Stimuli

Mechanical taps were delivered over the Achilles tendon and the skin overlying external malleolus. For this purpose a moving coil vibrator was activated by a 70 msec duration square wave electric pulse resulting in a downward movement of the rod attached to the vibrator of 4–5 mm. The vibrator's spindle was attached to a T rod with the horizontal portion placed in contact with the skin overlying Achilles tendon 3–6 cm proximal to its insertion. For cutaneous mechanical stimulation the rod was placed 3 cm above the skin surface and activation of the vibrator resulted in a gentle 'tap.' Percutaneous electrical stimulation was delivered over the posterior tibial nerve (PTN) immediately posterior to the medial malleolus and over the sural nerve (SN) at the ankle posterior to lateral malleolus. The intensities of the stimuli were adjusted to just elicit a visible twitch of the appropriately innervated muscles (PTN) and to 3 times sensory threshold (SN). Occasionally using this intensity for the SN also activated motor fibers of PTN. The stimulus strength was diminished until the muscles of the foot no longer twitched. Two sets of 2000 stimuli and two sets of 700 stimuli were delivered for each tendon tap and PTN somatosensory evoked potentials (SEPs), respectively. This number of trials resulted in 20% or less amplitude difference in the scalp P1-N1 components on repeated measures in the same subject at the same session.

Recording and analysis

Somatosensory cortical activity was recorded from an electrode over the scalp at Cz (according to the 10-20 system) referenced to a forehead electrode (Fpz). Subjects were grounded by a metal plate strapped to the leg proximal to the knee. Impedances were below 5 kΩ. Amplification of 500,000 was used with a bandpass of 30–1000 Hz (6 dB down points). The potentials evoked by mechanical and electrical stimulation were averaged over a 100 msec time period using a dwell time of 0.2 msec and 512 addresses per channel. A duplicate of each average was made to assess reproducibility. The averaged potentials were recorded by an X-Y plotter (positive upwards) and stored on disks for further analysis. Amplitudes and latencies of the various components of the recorded potentials for each subject were measured from a computer display with a cursor. Latencies were measured from the onset of the electrical pulse delivered to the peripheral nerve or to the mechanical vibrator to the peaks of the various components. Amplitudes of cerebral SEPs were measured in one or both of two ways: (1) the amplitude difference between the baseline and each positive or negative peak and (2) the amplitudes between each positive peak and the immediately following negative component. Amplitudes of potentials recorded during vibration, muscle contraction, ischemia and cooling of the leg are expressed as percentages of controls recorded in the same subject during the same session. For some of the figures grand averages of the individual evoked potentials were formed.

Data analysis

Separate t tests for paired and unpaired comparisons were performed to evaluate differences between means.

Ischemic hypoxia

After recording control cerebral potentials to Achilles tendon taps and PTN stimulation, a pneumatic tourniquet 10 cm wide was placed around the ankle 1 cm proximal to the site of electrical stimulation of the PTN. The cuff was inflated to 80 mm Hg above the systolic blood pressure. Evoked potentials were recorded in 3 subjects (2 males, 1 female) and continuously monitored for 25–30 min after the cuff was inflated and for 10 min after its release. The same procedure was then performed with the cuff placed proximally just above the knee. Because of the pain induced at this location only one subject was able to tolerate the ischemia from the cuff placed at the knee to complete the experiment. Shortly after the cuff was inflated, the subject felt tingling and numbness distally. After some minutes the same area became painful, particularly during ischemia at the knee. The electric shocks to PTN stimulation became uncomfortable whereas the appreciation of mechanical stimuli to the Achilles tendon was unchanged. After releasing the cuff, the subject felt strong paresthesias in the ischemic areas that disappeared within a few minutes.

Cooling of the leg

The effects of cooling of the leg were tested in one female. The leg between knee and ankle was surrounded by a plastic bag containing ice cubes. Simultaneous recordings of cutaneous and intramuscular (gastrocnemius-soleus muscle) temperature were obtained from two thermocouples: one placed over the skin and the other in the tip of a 3.5 cm needle in the gastrocnemius-soleus muscle belly. Evoked potentials were recorded repeatedly during the cooling process. A sense of coldness was felt by the subject during the first few minutes of exposure which was replaced during the next 10 min by discomfort that then persisted until the ice pack was removed.

Muscle contraction

The effects of muscle contraction on cerebral potentials evoked by PTN stimulation were tested in 8 subjects (4 males and 4 females), and on cerebral potentials evoked by Achilles tendon taps in 3 subjects (2 males and 1 female). Isotonic active contraction of the ipsilateral gastrocnemius-soleus and flexors of the toes was exerted against a load averaging 10 kg.

Vibration

The effects of vibration on cerebral potentials evoked by PTN stimulation were tested in 6 subjects (3 males and 3 females), and on cerebral potentials evoked by Achilles tendon taps in 4 subjects (2 males and 2 females). Vibration was produced with a second vibrator activated by a frequency generator at 60 Hz. The displacement of the rod attached to this vibrator was 5 mm and it was applied over the Achilles tendon or the heel. In order to increase the efficiency of the vibratory stimulus on muscle receptors in the gastrocnemius-soleus muscle, the ankle was passively dorsiflexed.

Results

Fig. 1 shows the grand average of cerebral potentials evoked by electrical and natural stimulation at the ankle in 8 subjects. The components
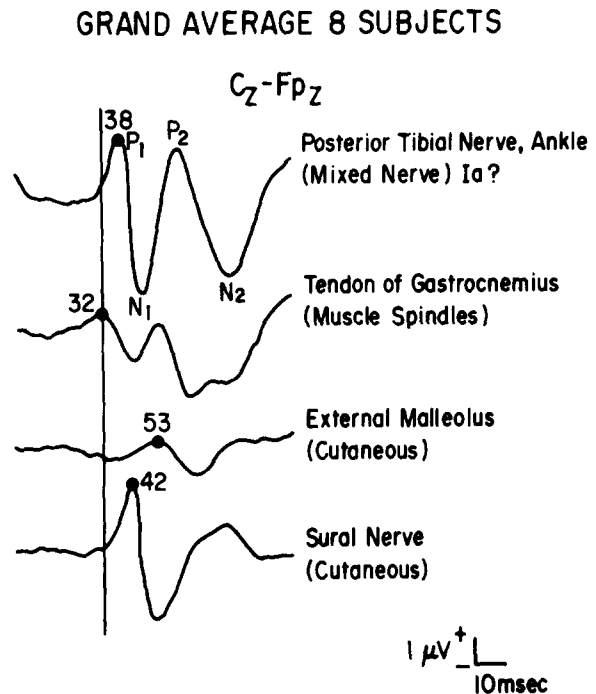

GRAND AVERAGE 8 SUBJECTS

C_z-Fp_z

Fig. 1. Grand average of cerebral potentials to mechanical and electrical stimulation at the ankle. The components are labeled by their polarity at the vertex and their temporal sequence (P1, N1, P2 and N2). 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 latency evoked by tendon taps.

are labeled by their polarity, P or N (positive or negative at the vertex electrode) and their order of appearance (1, 2, ...). Achilles tendon taps evoked the earliest cerebral response (32 msec), then electrical stimulation of posterior tibial nerve (38 msec), sural nerve (42 msec) and finally cutaneous taps on the skin overlying external malleolus (53 msec). Details of these various potentials can be found in an earlier report (Cohen et al. 1985).

Ischemic hypoxia

Ischemic hypoxia at the ankle did not affect amplitudes or latencies of cerebral potentials evoked by Achilles tendon taps (Fig. 2A). In contrast, ischemic hypoxia at the ankle was accompanied by an attenuation of the amplitude of the cerebral potentials evoked by posterior tibial nerve stimulation in the 3 subjects (P1-N1 component diminished to 34%, 42% and 52% respectively) and
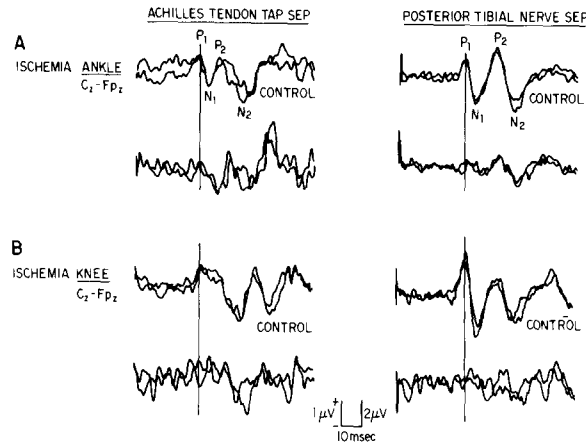
Fig. 2. Effects of ischemia on cerebral SEP in 2 different subjects (A and B) when the cuff was located just above the ankle (A) and just above the knee (B). A vertical line has been placed at the P1 latency of potentials recorded during control condition. Posterior tibial nerve evoked cerebral potentials were affected by ischemia at both levels (ankle and knee). Achilles tendon tap cerebral SEPs were only affected when the cuff was inflated above the knee resulting in delayed latency of P1, poor reproducibility of N1 and the disappearance of P2 and N2. Note the different vertical calibrations for Achilles tendon tap and PTN evoked potentials.
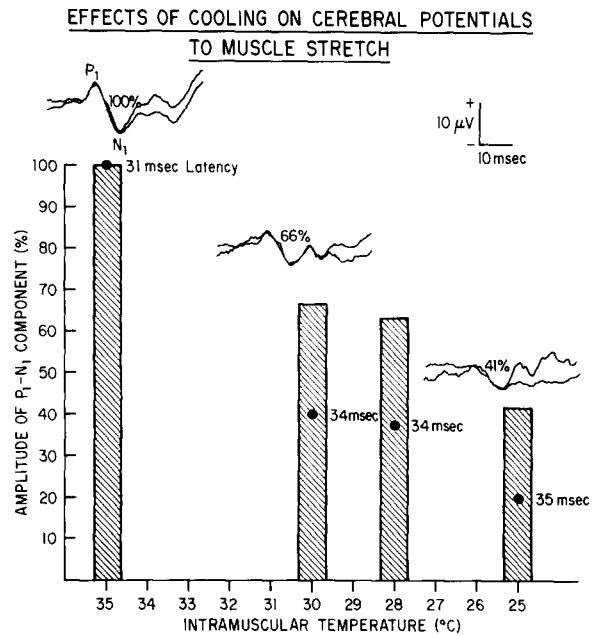

Fig. 3. Effects of cooling of the leg on latencies and amplitudes of cerebral potentials evoked by Achilles tendon tap stimulation. The amplitude of the P1-N1 ccomponent 3 min after positioning the plastic bag containing ice cubes did not change in relation to controls and was set to be 100%. At this time skin temperature had already dropped to 25°C (from control of 35°C) while intramuscular temperature was still 35°C. As muscle temperature diminished there was a progressive attenuation of the evoked potentials. Intramuscular temperatures of 30, 28 and 25°C corresponded with skin temperatures of 22, 19 and 17°C respectively. The latency of the P1 component at each temperature is shown by a filled circle. Examples of the potentials at 3 different temperatures are located above the appropriate columns.

a delay in latency of the P1 component (3 msec, 2 msec and 2.6 msec respectively) (Fig. 2A). These results suggest that the receptors responsible for Achilles tendon taps evoked potentials lie proximal to the ankle and are affected by ischemia of the ankle and foot whereas the fibers of the posterior tibial nerve afferent volleys are affected since they lie distal to the cuff.

When the pressure cuff was moved proximally to the knee and inflated to produce ischemic hypoxia of the leg, both the Achilles tendon tap and PTN evoked potentials were affected (Fig. 2B). To Achilles tendon taps there was a clear attenuation of the amplitude of the P1, a delay in its latency (14 msec) and a failure to replicate the other. components. PTN evoked potentials during ischemia at the knee had a corresponding attenuation of the amplitude (P1-N1 component diminished to 48%), a delay in latency (4 msec of the P1 component) and a failure to evoke the other components. These results show that the receptors activated by tendon taps that provide the input for

evoking cerebral potentials are situated between the ankle and the knee.

Cooling of the leg

Cooling of the leg produced a rapid decrease in skin temperature (10°C in 3 min) at a time when intramuscular temperature had not changed. Intramuscular temperature was affected only later, dropping from 35°C during control recordings to 30°C, to 28°C and to 25°C after 10, 15 and 20 min, respectively. Latencies and amplitudes of cerebral potentials evoked by Achilles tendon taps were modified only when the intramuscular temperature dropped (Fig. 3). Thus, structures deep in

muscles or bone are the sites of receptors responsible for providing input for tendon tap evoked potentials.

Muscle contraction

Active contraction of gastrocnemius-soleus muscle and flexors of the toes resulted in the marked attenuation of the amplitude of cerebral potentials evoked by Achilles tendon taps (P1-N1 component diminished to $22 \pm 22\%$, $P < 0.025$, Fig. 4A) and a moderate attenuation of the amplitude of cerebral potentials evoked by posterior tibial nerve stimulation (P1-N1 component diminished to $63 \pm 15.8\%$, $P < 0.01$, Fig. 4B).

Fig. 4C illustrates the mean amplitude changes of the different components of the cerebral potentials during muscle contraction. It is evident that the influence of muscle contraction was greater on

attenuating Achilles tendon tap evoked potentials than on attenuating the potentials evoked by posterior tibial nerve stimulation ($P < 0.05$ for all cerebral components, Fig. 4C).

Vibration

Vibration of the Achilles tendon was accompanied by a reduction of the amplitude of cerebral potentials evoked by both Achilles tendon taps (P1-N1 component diminished to $34 \pm 30\%$, $P < 0.025$, Fig. 5A) and posterior tibial nerve stimulation (P1-N1 component diminished to $71.6 \pm 7.9\%$, $P < 0.01$, Fig. 5B).

Fig. 5C plots the mean amplitude changes induced by vibration on the different components evoked by the two stimuli. The attenuating effect of vibration on Achilles tendon tap evoked potentials was greater than on the potentials evoked by
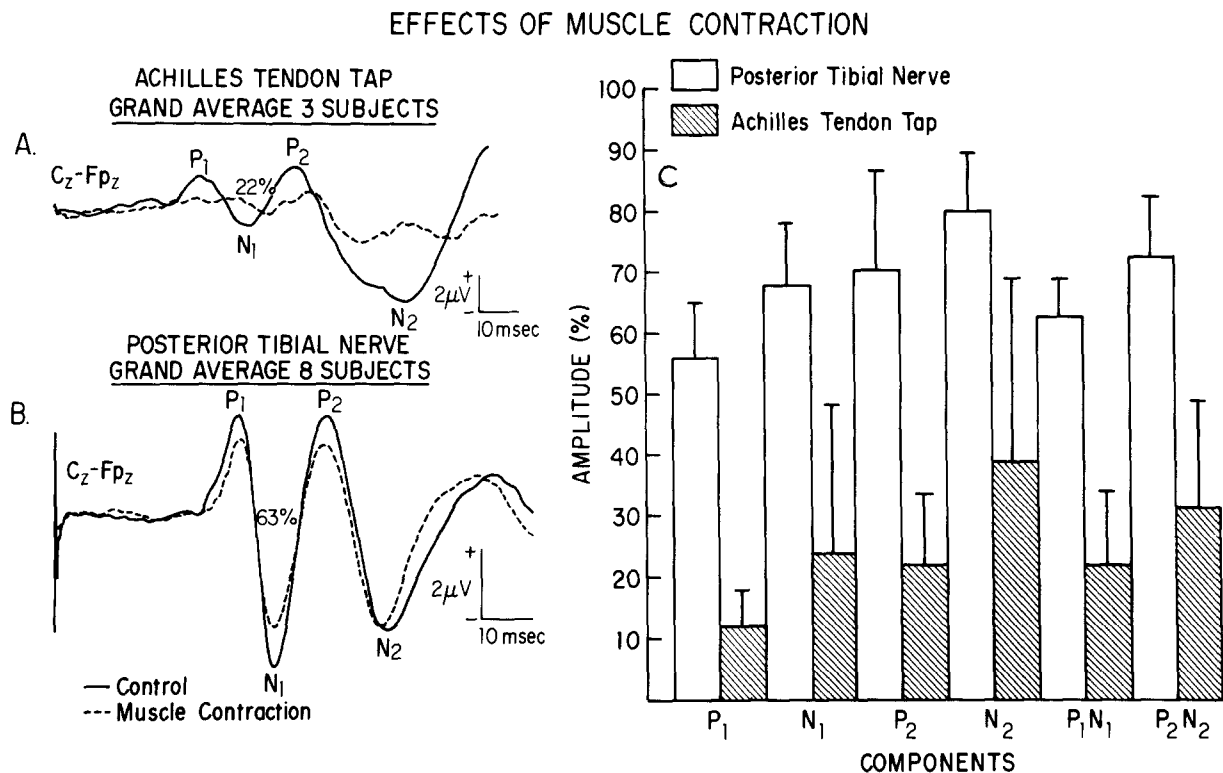

Fig. 4. Attenuation exerted by muscle contraction on cerebral potentials evoked by Achilles tendon taps (A) and by posterior tibial nerve (B) stimulation. The potentials in A and B represent the grand averages from 3 and 8 subjects, respectively. The bar graph (C) contains the mean amplitudes and standard errors of the individual measurements for the different components of these potentials relative to control (100%). The extent of attenuation for all components is greater for Achilles tendon tap potentials than for PTN potentials ($P < 0.05$).
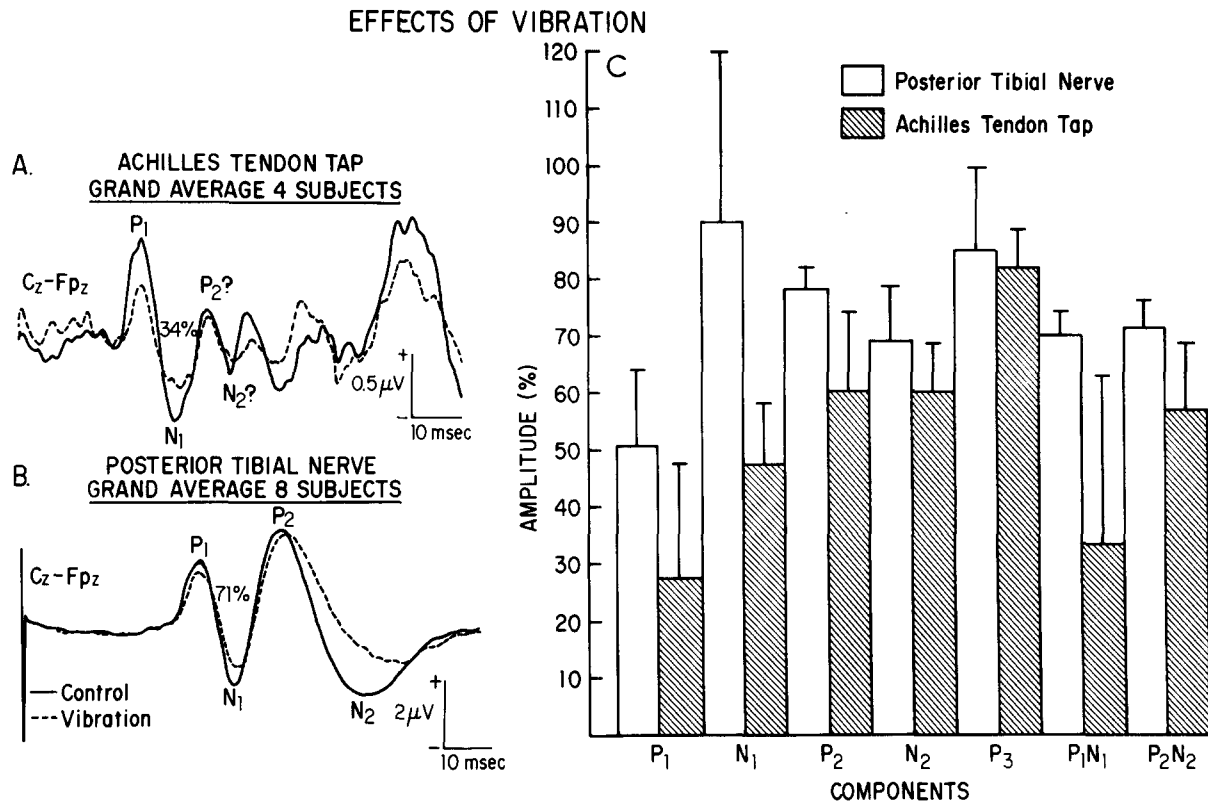
Fig. 5. Attenuation exerted by vibration on cerebral potentials evoked by Achilles tendon taps (A) and posterior tibial nerve (B) stimulation. A and B represent the grand averages of potentials recorded in 4 and 8 subjects respectively. The identification of P2 and N2 components is uncertain in the grand average of A. The bar graph (C) contains the mean amplitudes and standard errors of the individual measurements for the different components of these potentials relative to control (100%). The extent of attenuation for all components is greater for Achilles tendon tap potentials than for PTN potentials but achieves statistical significance for only the P1-N1 amplitude measure ($P < 0.05$).

posterior tibial nerve stimulation but reached statistical significance ($P < 0.05$) for only the P1-N1 component.

Discussion

The results of this study demonstrate that the main sources of afferent input for Achilles tendon tap cerebral potentials in humans are located between the ankle and knee deep to the skin and that the potentials are affected by both muscle contraction and vibration.

Among the several candidate receptor sources, primary muscle spindles are particularly sensitive

to a rapidly accelerating stretch of the muscle such as would result from a tap applied to the Achilles tendon and their inputs are conducted rapidly along IA fibers (Cohen et al. 1985). It is the proximal location of these muscle spindle receptors in the gastrocnemius-soleus muscle belly in the leg that probably accounts for the earlier latency at the scalp of potentials evoked by Achilles tendon taps than those evoked by electrical stimulation of posterior tibial nerve at the same level, i.e., the ankle. The attenuation of the tendon tap evoked potentials during muscle vibration probably reflects the fact that muscle spindles are also strongly activated by vibration at 60 Hz (Desmedt 1983) and would therefore be less capable of re-

sponding to the tendon taps because of receptor occupancy, i.e., the so-called 'afferent busy line' phenomenon (Hagbarth 1973). It is also likely that muscle spindles are modified during isotonic contraction by fusimotor activity (Vallbo 1973) thus accounting for the profound attenuation of Achilles tendon tap evoked potentials during muscle contraction. This evidence points to the muscle spindles as the major receptor type contributing to the cerebral potentials evoked by tendon taps. Moreover, the location of the muscle spindles in the belly of the gastrocnemius-soleus muscle, proximal to the ankle, would account for the failure of ischemia applied at the ankle (distal to receptor's location) to modify the tendon tap cerebral potentials. However, when ischemia was applied at the knee and thus proximal to receptors, the potentials were attenuated. Finally, muscle rather than cutaneous receptors seem the most likely source of input causing the cerebral potentials to tendon taps since cooling of the leg only altered these potentials when the temperature deep within the muscle dropped, rather than when the skin alone was cooled.

These results relate to other experiments (Starr et al. 1981) in which direct stimulation of small nerve fascicles innervating muscle spindles in pretibial flexor muscles in humans produced cerebral potentials of the same general form but of shorter latencies (32 msec) than those evoked by tapping on the tendons of these muscles (37 msec). The former value of 32 msec is identical to the 32 msec latency of the cerebral potential obtained to tapping on Achilles tendon described in the present study (Fig. 1). Furthermore, the rate of acceleration of muscle stretch, be it from passive movement of the ankle (Starr et al. 1981) or from Achilles tendon taps (Cohen et al. 1985) is a significant variable affecting the amplitude of the potentials evoked by muscle stretch. The exquisite sensitivity of muscle spindles to acceleration points to their participation as a major source of cerebral potentials evoked by tendon taps and joint position changes.

Secondary muscular endings are unlikely to be a major receptor source for Achilles tendon tap cerebral potentials. Secondary receptors have a lower dynamic sensitivity to muscle stretch than

do primary muscle spindles (Lundberg and Winsburg 1960; Bessoni and Laporte 1962). Also the relatively slow peripheral conduction velocity of group II afferent fibers from secondary endings would not permit the early scalp positivity recorded to tendon tap stimulation.

Since mechanical taps stimulate the skin, cutaneous receptors must be considered as a possible source for Achilles tendon tap evoked potentials. However, there are several lines of evidence that argue against this possibility. First, cooling of the skin did not modify Achilles tendon tap cerebral potentials. Second, tapping of the skin overlying external malleolus of the ankle, thus avoiding stretch of the muscles, elicited cerebral potentials whose initial positivity occurred more than 20 msec later than those evoked by Achilles tendon taps at the same level of the leg (Fig. 1). Third, Achilles tendon tap evoked cerebral potentials were attenuated by vibration at frequencies that are relatively ineffective for stimulating cutaneous receptors (60 Hz) (Talbot et al. 1968; Hunt 1974). Thus, it is unlikely that cutaneous receptors participate in the generation of the initial scalp positivity evoked by Achilles tendon taps.

The participation of joint receptors in the generation of Achilles tendon tap cerebral potentials appears improbable. First, the joints were fixed with a mold so that very few, if any, joint receptors could be activated by the mechanical taps. Second, ischemia of the ankle did not modify Achilles tendon tap cerebral potentials making the participation of ankle joint receptors unlikely. Moreover, these ischemia experiments localized the source of Achilles tendon tap cerebral potentials to the limb between the ankle and the knee where joint receptors are not present. Third, the conduction velocity of group II afferent fibers from joint receptors is too slow to allow an initial scalp event to mechanical taps earlier than to stimulation of PTN at the same level.

Finally, while Golgi tendon organs can be activated by tendon taps, they respond poorly to passive stretch (Houk et al. 1971) making it unlikely that these receptors are a significant source of input for the cerebral potentials to Achilles tendon tap stimulation.

In summary, these experiments point to primary

muscle spindles in the proximal gastrocnemius-soleus muscle as the main source of afferent input for evoking cerebral potentials to Achilles tendon taps. In the future, it may be possible with these techniques to study the cerebral events accompanying tendon taps in a variety of neurological conditions similar to the way physicians have traditionally tested the deep tendon reflexes in these different diseases to shed light on some of the neural events accompanying modifications of tendon reflexes.

Summary

This study examines the effects of ischemic hypoxia and cooling of the leg, muscle contraction and vibration on cerebral potentials evoked by Achilles tendon taps and posterior tibial nerve stimulation to obtain indirect evidence leading to the identification of receptors activated by tendon taps.

Experiments performed during ischemia of the leg showed that these receptors lie between the ankle and the knee. Cooling of the leg showed that they are located deep in muscles or bone. Experiments performed during vibration and muscle contraction suggest that muscle stretch receptors provide the afferent input responsible for Achilles tendon tap evoked potentials. All of these experiments point to primary muscle spindles in the proximal gastrocnemius-soleus muscle belly as the main source of afferent input for evoking cerebral potentials to Achilles tendon taps in humans.

Résumé

De l'origine chez l'homme, des potentiels évoqués somatosensoriels aux chocs sur le tendon d'Achille

Cette étude analyse les effets d'une hypoxie ischémique, d'un refroidissement de la jambe, de la contraction et de la vibration musculaire sur les potentiels cérébraux évoqués par chocs sur le tendon d'Achille et par stimulation du nerf tibial postérieur, afin d'identifier indirectement les récepteurs activés lors d'un choc sur un tendon.

Les études effectuées au cours d'une ischémie de la jambe ont permis de localiser ces récepteurs entre la cheville et le genou. L'effet du refroidissement de la jambe les situe en profondeur dans le muscle ou l'os. Enfin, les études effectuées au cours de la vibration et de la contraction musculaire suggèrent que ce sont les récepteurs à l'étirement musculaire qui sont à l'origine des informations responsables des potentiels évoqués aux chocs sur le tendon d'Achille. Toutes ces expériences désignent les terminaisons musculaires fusoriales primaires dans la partie proximale du muscle gastrocnémien-soléaire comme source des messages afférents des potentiels cérébraux évoqués par choc du tendon d'Achille chez l'homme.

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