UC Irvine
UC Irvine Previously Published Works

Title
Magnetic stimulation of muscle evokes cerebral potentials

Permalink
https://escholarship.org/uc/item/2sf5s3dg

Journal
Muscle & Nerve, 14(8)

ISSN
0148-639X

Authors
Zhu, Yu
Starr, Arnold

Publication Date
1991-08-01

DOI
10.1002/mus.880140806

Copyright Information

Peer reviewed

Somatosensory evoked potentials (SEPs) were recorded from the scalp in man to magnetic stimulation of various skeletal muscles. The potentials consisted of several components, the earliest of which decreased in latency as the stimulated site moved rostrally, ranging from 46 msec for stimulation of the gastrocnemius, to 14 msec for stimulation of the deltoid. Experiments were performed to distinguish the mechanisms by which magnetic stimulation of the muscle was effective in evoking these cerebral potentials. For the gastrocnemius, the intensity of the magnetic stimulus needed for evoking cerebral potentials was less than that required for activating mixed or sensory nerves in proximity to the muscle belly (eg, posterior tibial nerve in the popliteal fossa, sural nerve at the ankle). Vibration of the muscle or passive lengthening of the muscle, procedures which activate muscle spindles, were accompanied by a significant attenuation of the potentials evoked by magnetic stimulation of the muscle. Anesthesia of the skin underlying the stimulating coil had no effect on the latency or amplitude of the early components of the magnetically evoked potentials, whereas electrically evoked potentials from skin electrodes were abolished. Thus, the cerebral potentials accompanying magnetic stimulation of the muscle appear to be due to activation of muscle afferents. We suggest that magnetic stimulation of muscle can provide a relatively simple method for quantifying the function of muscle afferents originating from a wide variety of skeletal musculature

Key words: SEPs • magnetic stimulation • muscle afferent • somatosensory evoked potentials

MAGNETIC STIMULATION OF MUSCLE EVOKES CEREBRAL POTENTIALS

YU ZHU, MD, and ARNOLD STARR, MD

Natural forms of somatosensory stimulation, such as cutaneous deformation by mechanical tap[23,24,32] or air-puff,[21] muscle afferent activation by muscle stretch[1,33] or tendon tap,[12] pain fiber activation by laser heating of skin,[4,7] temperature receptor activation by sudden cooling[16] or by rapid warming,[8] have been used for evoking brain potentials as means of quantifying the function of sensory receptors, their afferent nerve fibers, and their specific central pathways.

Several factors limit the utility of these natural methods for evoking somatosensory evoked potentials. First, the amplitude of the potentials are small relative to those evoked by stimulation of mixed or sensory nerve trunks. Second, the equipment and procedures needed for the delivery of these stimuli, as well as their calibration, are complex. We report, in this article, on the use of magnetic stimulation of the muscle belly in humans as a relatively simple way of activating muscle receptors for evoking brain potentials of robust amplitude.

The application of a focal magnetic stimulus to the body induces an electric field that can depolarize nerve fibers and neurons if its intensity and duration are adequate.[2,18] Somatosensory evoked potentials (SEPs) to intense magnetic stimulation of spinal roots and peripheral mixed nerves have been recently described,[34,36] which did not differ in latency to the potentials evoked by electrical stimulation of the same structures. This report examines the brain potentials accompanying magnetic stimulation of the muscle belly, and defines the relative role of cutaneous and muscle spindle afferent in their generation.

METHODS

Fourteen healthy volunteers (7 men and 7 women), aged 22 to 45 years, were studied after having given informed consent. Subjects were tested while lying prone or supine on a bed, and remained awake through the procedures.

Magnetic stimulation was performed with a Cadwell MES-10 stimulator. A focal point coil with a mean diameter of 4.7 cm was placed tangentially to the skin overlying the gastrocnemius belly. In addition, in 5 of the subjects, stimulation of other muscles was also carried out. A brief pulse, 0.07 msec in duration, up to 3000 V at maximal output, was passed through the coil by the discharge of capacitors. The changing magnetic field, which approached 2.0 Tesla, induced electrical currents within the tissue. The wave form of the magnetic field produced by the Cadwell stimulator is polyphasic. The initial major phase lasts approximately 0.05 msec, followed by a variable number of alternating phases.[9,20] The initial sharp voltage from the baseline represents the relevant stimulus for the tissue. A stimulation rate of 0.7 Hz was used. The transformer in the stimulator overheated during repetitive stimulation at this rate, requiring that it be switched off after every 200 to 300 stimulations for 1 to 3 minutes. The intensity of the magnetic stimulus was defined as a percentage of maximal output.

The subjects' threshold to magnetic stimulation was defined both as the intensity needed for just-detectable perception (sensory threshold or ST), and as the just-visible detection of a movement of the muscle beneath the magnetic coil (motor threshold or MT). Cerebral evoked potentials were usually recorded at 40% to 50% of maximum output (2 to 2.5 MT, or 2 to 2.5 ST), unless otherwise specified.

A high amplitude artifact, lasting approximately 20 msec, accompanied the appliation of the magnetic stimulus. The artifact reflected the spread of the magnetic flux of the stimulus to involve the recording wires resulting in an induced current that was detected by the amplifiers. Twisting the recording wires together and aligning the recording wires transverse to the magnetic coil plane was useful in reducing the amplitude of the artifact.[26] In addition, turning the magnetic coil 180°, for half of the sweeps in each average, effectively reversed the phase of the induced current flow allowing cancellation in the averaging process of the artifact. This reversal did not affect the latencies or amplitudes of cerebral potentials to stimulation of the gastrocnemius muscle.

The discharge of the electrical current through the magnetic coil is acompanied by a clicking sound. The cerebral evoked potentials accompanying magnetic stimulation of the gastrocnemius muscle was found to be unchanged in the presence of a masking noise (2 subjects). Thus, all studies except for threshold estimations were done without noise masking.

Percutaneous electrical stimulation with bipolar electrodes was performed over the posterior tibial nerve (PTN) at the ankle, immediately posterior to the medial malleolus, and at the popliteal fossa. A 0.2-msec square pulse of constant current was delivered at a rate of 0.7/sec. The intensity was adjusted to 30% above the motor threshold of the abductor halluces.

Recording electrodes were Ag/AgCl disks, 8 mm in diameter, attached with electrode cream to the skin. Electrode impedances were maintained close to one another and measured below 2 kOhms. Recording electrodes were placed on thoracic spinous processes, T12, referenced to T10; on the scalp, 2 cm posterior to the Cz position of the 10–20 International System referenced to Fpz. A ground electrode was placed either on the scalp between the pair of recording electrodes during magnetic stimulation, or between stimulation and recording electrodes during electrical stimulation. The potentials were amplified with a gain of 500,000 using a band-pass of 1 to 1500 Hz (6 dB/octave slope), and averaged (usually 100 to 200 trials) with a time base of 120 msec, including a 12-msec pre-stimulus baseline. Duplicate averages were made for each condition. A potential of positive polarity at grid 1 of the amplifier was reflected by a up-going deflection on the trace.

Peak latencies and peak-to-peak amplitude of the evoked potential components were measured from a computer display using a cursor. The components of the evoked potential were designated by their polarity (P or N for positive or negative), and their approximate peak latencies in msec. The t-test for related measures between the means was performed to evaluate the significance of differences. Pearson product moment correlations were used to evaluate the relationship between the height of the subject and the latencies. Differences were considered significant at a $P < 0.05$ level.

Scalp topography of SEPs following magnetic stimulation over the gastrocnemius, or electrical stimulation to the PTN at the knee, or at the ankle, were studied in 2 subjects. An array of 16 electrodes referenced to linked-ears served to create amplitude distributions over the scalp at vari-
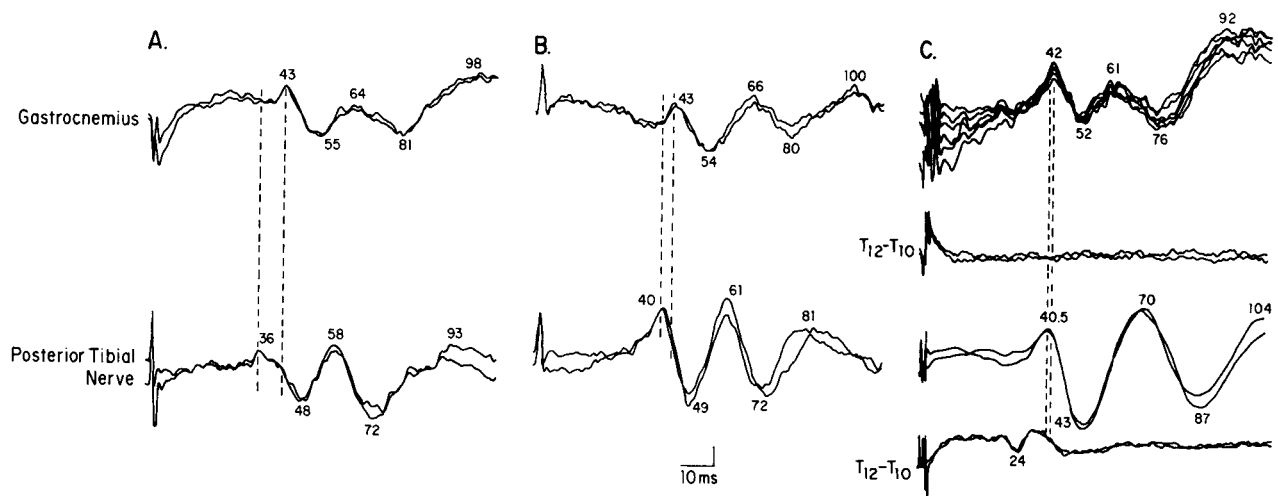
FIGURE 1. Examples from 3 subjects of cerebral [(A), (B), and (C)] and spinal [(C) only] potentials to magnetic stimulation of gastroc-nemius (upper traces) and to electrical stimulation to the PTN at the ankle (lower traces). The vertical lines indicate the peak latencies of P40 for magnetic stimulation of the gastrocnemius and P37 for PTN electrical stimulation, respectively. The upper traces in (C) show the replicability in one subject of cerebral potentials evoked by magnetic stimulation of gastrocnemius. Spinal responses recorded from T12 to T10 to magnetic stimulation of the gastrocnemius or to PTN electrical stimulation are shown in (C), below their corresponding scalp recordings. Each duplicate average consists of 120 sweeps, except for T12 to T10 recordings in which each average consists of 500 sweeps. Vertical calibration: 1 μV for cerebral potentials and 0.5 μV for spinal potential, respectively. Positivity at grid 1 of the amplifier for evoked cerebral and spinal potentials is displayed upward in this and all subsequent figures.

ous peak latencies. The 12-msec pre-stimulus pe-riod was used to define the amplitude of the selected peaks.

Several procedures were used to distinguish the mechanisms by which magnetic stimulation of the muscle was effective in evoking cerebral po-tentials. These included (1) the influence of mus-cle vibration (5 subjects), (2) passive change in muscle length (5 subjects), and (3) cutaneous

nerve blockade (2 subjects). Vibration was pro-duced by activating a rod with a ring 4 cm in di-ameter at its tip, with sinusoidal frequencies be-tween 20 and 120 Hz. The ring was applied on the skin overlying the gastrocnemius muscle belly, 2 cm rostral to the site of application of the mag-netic coil. The displacement of the rod during vi-bration was 5 mm. Maintained passive dorsiflexion and plantar flexion of the ankle was performed

Table 1. Comparison of peak latencies and peak-to-peak amplitudes of SEPs to magnetic stimuli to the gastrocnemius muscle and electrical stimuli to the posterior tibial nerve at the ankle in 14 normal subjects.

	P40	N50	P60	N70	P100
A. Peak latency (mean ± SD, in msec)					
Magnetic stim.	40.2 ± 2.7	52.0 ± 6.5	59.7 ± 7.5	72.9 ± 7.2	101.1 ± 10.2
Electrical stim.	37.3 ± 2.5	48.6 ± 2.2	58.0 ± 3.7	72.5 ± 6.8	92.2 ± 5.6
Latency difference					
(magnetic − electrical)	3.0 ± 2.1	3.7 ± 5.4	1.7 ± 7.9	0.7 ± 6.4	3.8 ± 6.8

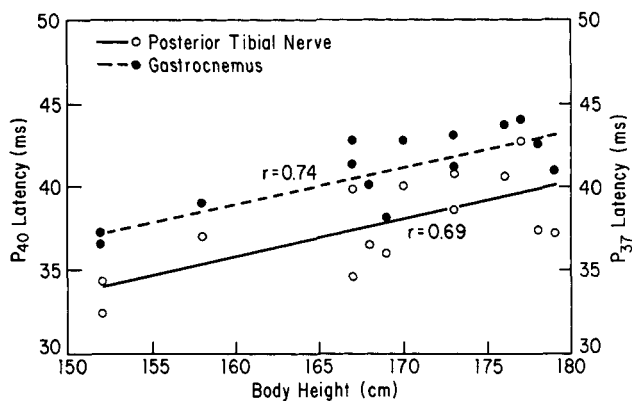|	P40–N50	N50–P60	P60–N70	N70–P100
B. Peak-to-Peak amplitude (mean ± SD, in μV)				
Magnetic stim.	2.64 ± 0.91	1.99 ± 0.96	2.88 ± 1.26	4.02 ± 1.68
Electrical stim.	3.34 ± 0.96	3.02 ± 1.35	4.25 ± 1.55	4.88 ± 2.01
Amplitude ratio				
(magnetic/electrical)	0.79	0.33	0.67	0.82

FIGURE 2. The relationship between height and peak latencies for magnetic stimulation of the gastrocnemius (filled circles) and P37 for PTN electrical stimulation at the ankle (open circles).

manually by the experimenter. A nerve block (1.5% procaine) was made of the lateral femoral cutaneous nerve at the inguinal ligament that produced anesthesia of the skin overlying the lateral portion of the thigh. The effects of this anesthesia on the potentials evoked by both magnetic stimulation of the belly of the vastus lateralis muscle, and electrical stimulation of the skin surface that was anesthetized, were examined.

The H-reflex was recorded to both magnetic stimulation and electrical stimulation of the PTN at the popliteal fossa in 3 subjects. They lay supine on the bed with the examined leg flexed 120° at the knee joint. The magnetic coil was positioned with the edge of the coil tangential to the course of the nerve. The active recording surface electrode was placed on the soleus belly with the reference electrode on the Achilles tendon. The ground electrode was placed near the active recording electrode. The recorded signals were filtered with a band-width of 30 to 3000 Hz. The H-reflex from soleus muscle was also recorded to electrical stimulation with 1.0-msec square pulses to the PTN at the popliteal fossa in the same subjects.

RESULTS

General Description. Magnetic stimulation, applied to the belly of the gastrocnemius muscle at a rate of 0.7 Hz, evoked cerebral potential recorded between Cz' and Fpz (Fig. 1), consisting of several components: P40, N50, P60, N70, and P100. SEPs to stimulation of the PTN at the ankle showed a similar sequence of components that peaked on average from 0.2 to 8.9 msec earlier than the magnetically evoked SEPs (Fig. 1, Table 1). For both types of stimulation, there was a linear correlation between body height and latency of the initial positive component (P40 for magnetic stimulation of the gastrocnemius, and P37 for PTN electrical stimulation, Fig. 2). The amplitudes of the cerebral components evoked by magnetic stimulation of the gastrocnemius were 33% to 82% of the comparable PTN-evoked potentials. In particular, the amplitude of the early P40/N50 component to magnetic stimulation was approximately 80% of
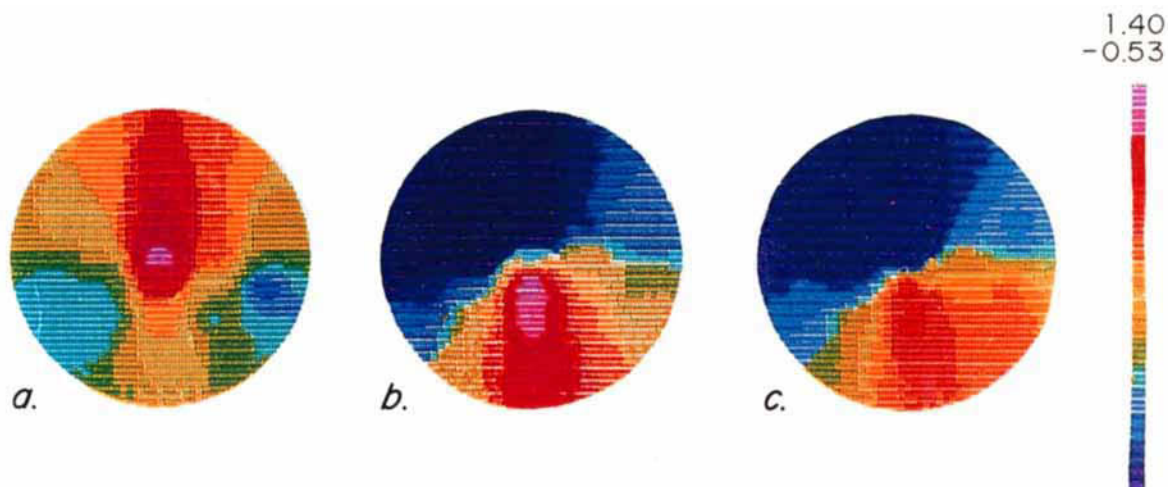

FIGURE 3. Topographic maps of SEPs following magnetic stimulation to the right gastrocnemius **(a)**, electrical stimulation to the right PTN at the ankle **(b)**, and at the popliteal fossa **(c)**, in one subject. These maps were sampled at the peak latencies of the initial prominent positivity of cerebral evoked potentials: 43 msec for the stimulation to gastrocnemius, 39 msec for the PTN at the ankle, and 31 msec for the PTN at the popliteal fossa. Each sampling run consists of 500 sweeps.
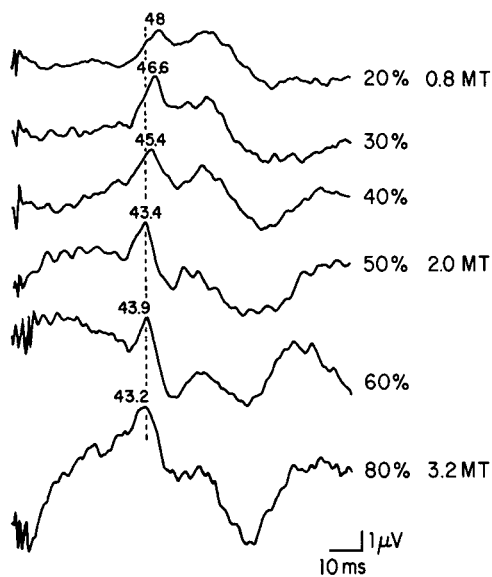
FIGURE 4. The effects of the intensity of stimulation on the amplitude and latency of cerebral potentials evoked by magnetic stimulation to gastrocnemius in 1 subject. The P40 is marked by a vertical line. The motor threshold, identified by visual inspection, was 25% of maximum output. Each trace is the sum of two consecutive averages containing a total of 320 sweeps.

the same complex evoked by electrical stimulation. It was not possible to record a potential over the lumbar region to magnetic stimulation of the gastrocnemius muscle in the 3 subjects tested, whereas these same subjects all had clear lumbar potentials to PTN stimulation (Fig. 1C).

SEPs evoked by magnetic stimulation of the gastrocnemius muscle, were very replicable with regard to form and latency within the same subject. Figure 1C contains 6 superimposed averages from the same subject tested several times in one day, as well as over several different days. The latencies of the components hardly varied, whereas their amplitudes could differ by as much as 20%. The scalp distribution of the P40 potential evoked to magnetic stimulation of the gastrocnemius mus-

cle peaked at the midline and tailed off symmetrically on either side (Fig. 3a).

In contrast, the P37 to PTN stimulation at the ankle, or the P31 to PTN stimulation at the knee, were asymmetrically distributed over the scalp, being positive in the midline ipsilateral to the stimulus site, and isopotential or even negative over the contralateral hemisphere (Figs. 3b and c).

The intensity of magnetic stimulation of the gastrocnemius muscle affected both the amplitude and latency of the evoked potentials (Fig. 4). In the subject portrayed in Figure 4 the latency of the initial peak, P40, was reduced approximately 5 msec and its amplitude (P40 to N50) increased approximately 3-fold when the stimulator output was raised from 20% to 50% of maximum. Increasing the stimulus intensity above 50% of maximum resulted in little further change in both latency and amplitude of the P40 component. Table 2 contains latency and amplitude measures of the P40 component for 3 subjects tested at two stimulus intensities, 30% and 50%.

Cerebral potentials accompanied magnetic stimulation of muscles widely distributed over the body (Fig. 5). The latency of the initial positive component of the cerebral potentials shortened as the site of stimulation moved rostrad: from 40 msec for the muscles of the leg, to approximately 30 msec for the paraspinous lumbar muscles, to 20 msec for the deltoid muscle. Note, that for the latter muscle, an even earlier negative component at 14 msec could be defined.

Mechanisms by Which Magnetic Stimulation of the Muscle Belly Evokes a Cerebral Potential

Stimulation of Cutaneous Receptors. The early (P25, P38) cerebral potentials, evoked by magnetic stimulation of the skin surface overlying the vastus lateralis belly, were essentially unchanged by a procaine nerve block of the lateral femoral cutaneous nerve that resulted in anesthesia of the skin underlying the coil (Fig. 6A). The modification of

Table 2. Comparison of P40 latencies and P40–N50 amplitudes of SEPs to magnetic stimuli to the gastrocnemius at the intensities of 30% and 50% of maximal output in 3 subjects.

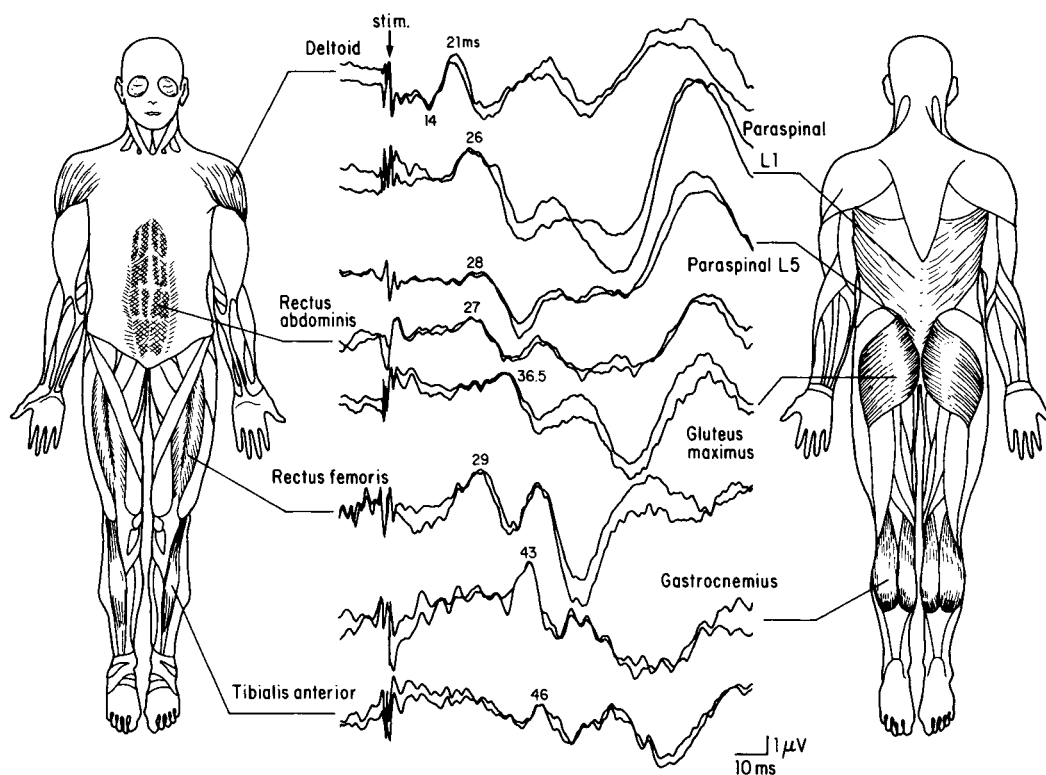|	P40 latency (ms)			P40–N50 amplitude (µV)		
Subject	30%	50%	Difference (50% − 30%)	30%	50%	Difference (50% − 30%)
1	46.6	43.4	3.2	1.4	3.2	1.8
2	44.2	40.5	3.7	1.3	3.8	2.5
3	41.3	38.4	2.9	1.0	2.9	1.9

FIGURE 5. Cerebral potentials following magnetic stimulation to a variety of muscles in 1 subject. Recordings were made between Cz' referenced to Fpz, except for deltoid muscle stimulation during which recordings were made between the scalp 2 cm lateral to Cz', contralateral to the stimulated side, referenced to earlobe (A2 of the 10–20 International System). Note that the initial deflection of cerebral potentials following magnetic stimulation to the deltoid is of negative polarity. Each average consists of 120 sweeps.

the late components suggests that cutaneous afferents are one of the contributors to these late components.

In contrast, electrical stimulation of this same

area through bipolar skin surface electrodes (separation 3 cm) at an intensity of 3ST (defined before nerve block) failed to elicit any cerebral potentials after anesthetic block of the lateral
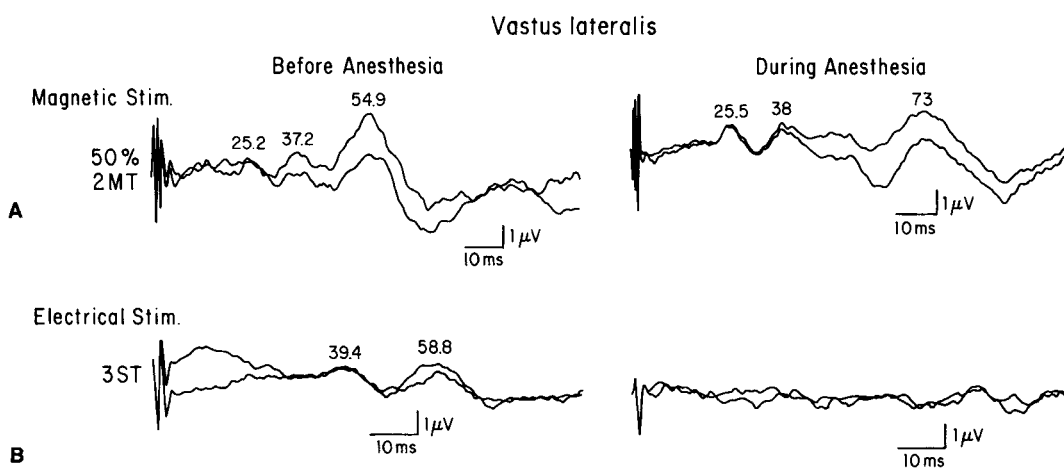

FIGURE 6. To show the cerebral potentials evoked by magnetic stimulation of the skin overlying the vastus lateralis **(A)**, and by electrical stimulation of the same area **(B)**, before and during procaine nerve block of the lateral femoral cutaneous nerve. Each of duplicate averages consists of 200 sweeps.

femoral cutaneous nerve (Fig. 6B). Thus, the cerebral potentials evoked by magnetic stimulation applied to the skin overlying a muscle belly is not due to activation of cutaneous receptors, whereas the potentials evoked by electrical stimulation of this same skin area reflect activation of superficial cutaneous receptors.

Stimulation of Peripheral Nerve Trunk(s). When the coil was placed over a superficial mixed nerve, such as the PTN at the ankle, the stimulus strength necessary for provoking either a muscle contraction or a cerebral evoked potential was much higher than when the coil was placed over the muscle itself. In 5 subjects, the threshold for a visible gastrocnemius contraction when the magnetic stimulator was placed over the muscle belly was 26% ± 1.6%, compared with 52% ± 2.0% when the coil was placed over the PTN in the popliteal fossa ($P < 0.001$). Moreover, the threshold for evoking a cerebral potential by magnetic stimulation applied over a cutaneous nerve, such as the sural nerve at the ankle, was approximately double that needed for evoking cerebral potentials when the stimulator was applied over the belly of the gastrocnemius muscle. Thus, it is unlikely that the cerebral potentials accompanying magnetic stimulation over the muscle belly are due to activation of nerve trunks.

Stimulation of the Muscle Leads to Activation of Muscle Receptors. Sustained vibration applied to the muscle belly to activate muscle receptors affected the potentials evoked by magnetic stimula-

tion of that muscle. Vibration applied over the gastrocnemius diminished (up to 44.8% ± 11.4%; $P < 0.05$) the amplitude of the P40–N50 component of cerebral potentials to gastrocnemius magnetic stimulation accompanied by a modest delay in peak latency (Fig. 7A, Table 3). Vibration of the limb contralateral to the stimulated side had no such effect on the cerebral potentials to magnetic stimulation of the gastrocnemius muscle. Vibration frequencies between 40 and 80 Hz were the most effective in attenuating the amplitude of P40–N50 (Fig. 7B). Thus, it is likely that Ia afferents become activated when magnetic stimulation is applied to the muscle belly. This involvement could either be by direct stimulation of muscle receptors and their nerve endings, or by indirect means secondary to an induced contraction of the muscle itself.

These alternatives were examined by testing the effects of passive dorsiflexion and plantar flexion of the ankle on magnetically evoked potentials. These positions should have little effect on the amplitude of the evoked potentials to magnetic stimulation if the magnetic stimulus directly activated the muscle receptors or their afferent. Passive lengthening of the gastrocnemius–soleus, by dorsiflexion of the ankle, was accompanied by an attenuation (54.4% ± 8.2%) of the P40–N50 component of the cerebral potentials evoked by magnetic stimulation of the gastrocnemius (Table 4A). In contrast, there was no change in amplitude of the SEPs to gastrocnemius stimulation
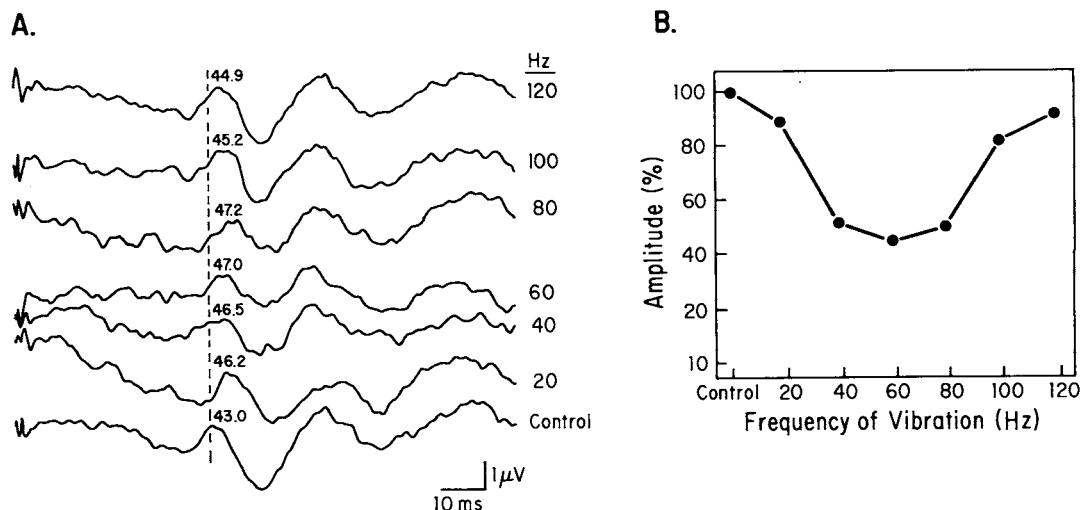
FIGURE 7. (A) shows the attenuation exerted by muscle vibration on cerebral potential evoked by magnetic stimulation of gastrocnemius in 1 subject. The vertical line indicates the peak latency of P40 in the control condition (absence of vibration). **(B)** shows the amplitude of P40–N50 components as a function of the frequency of vibration. The amplitude of the P40–N50 component, in the control condition, was set to be 100%. Each trace is the sum of two consecutive identical averages, and contains a total of 240 sweeps.

Table 3. Effects of vibration at 60 Hz on cerebral potentials evoked by magnetic stimuli to the gastrocnemius muscle.

Subject	Amplitude of cerebrally evoked potentials to gastrocnemius stimulation expressed as percentages of control value (100%)			
	P40–N50	N50–P60	P60–N70	N70–P100
1	52	55	70	30
2	60	48	65	55
3	38	46	51	57
4	31	37	44	35
5	43	47	60	51
Mean ± SD	44.8 ± 11.4	46.6 ± 6.4	58.0 ± 10.5	44.6 ± 13.5
$t =$	10.8	18.5	8.9	9.2
P	0.001	0.001	0.001	0.001

during passive shortening, ie, plantar flexion at the ankle (Table 4B). These same positional changes had no effect on the SEPs evoked by electrical stimulation of the PTN (Fig. 8). Thus, it is likely that magnetic stimulation activates muscle receptors indirectly by causing a muscle contraction, rather than by directly activating the receptors themselves or their nerve endings.

Furthermore, it was difficult to directly stimulate Ia afferent by the magnetic coil, even when it was placed in close proximity to a mixed nerve. Thus, magnetic stimulation of the PTN in the popliteal fossa at intensities of 50% elicited a small M-response in the soleus, but no H-reflex. When the stimulus intensity was raised above 50% of the maximum, an H-reflex could be obtained (Fig. 9A). In contrast, the H-reflex obtained by electrical stimulation of the same nerve appeared at stimulus levels well below that for evoking an M-response (Fig. 9B). The amplitude of the Hmax

Table 4. Effects of ankle dorsiflexion and plantar flexion on cerebral potentials evoked by magnetic stimulation of gastrocnemius muscle.

| Subject	Amplitude of cerebral evoked potentials to gastronemius stimulation expressed as percentages of control value (100%)			
	P40–N50	N50–P60	P60–N70	N70–P100
A. Dorsiflexion				
1	66	74	66	80
2	52	78	90	72
3	59	90	71	79
4	50	76	85	70
5	45	41	47	52
Mean ± SD	54.4 ± 8.2	71.8 ± 18.3	71.8 ± 17.0	70.6 ± 11.2
$t =$	12.4	4.7	3.7	5.8
P	0.001	0.005	0.05	0.005
B. Plantar flexion				
1	89	95	88	98
2	93	105	112	108
3	109	92	106	115
4	104	110	94	87
5	96	115	92	92
Mean ± SD	98.2 ± 8.2	103.4 ± 9.8	98.4 ± 10.1	100.0 ± 11.5
$t =$	−0.49	0.78	−0.35	0
P	NS	NS	NS	NS

NS = not significant.

A. Muscle
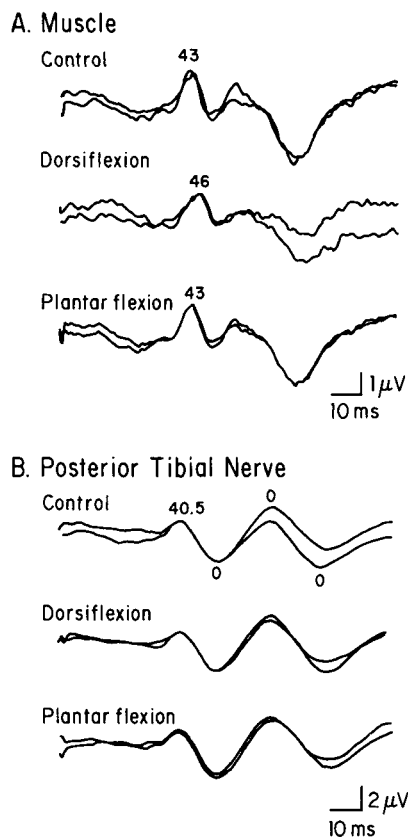
FIGURE 8. To show the effects of passive dorsiflexion and plantar flexion at the ankle on cerebral potentials evoked by magnetic stimulation of gastrocnemius (A), and by electrical stimulation to the PTN at the ankle (B). The vertical lines indicate the peak latencies of P40 for magnetic stimulation of the gastrocnemius, and P37 for PTN electrical stimulation, respectively. Note, in (A), passive dorsiflexion at the ankle diminished the amplitude of P40–N50 component by more than 40% and delayed the peak latency of P40 by 3 msec. Each trace is the sum of two consecutive identical averages, and contains a total of 240 sweeps. Passive dorsiflexion was without effect.

obtained by magnetic stimulation was 60% of that obtained with electrical stimulation (Fig. 9C). Thus, magnetic stimulation applied in the proximity of a mixed nerve activated motor fibers at lower stimulus strengths than required for activating Ia afferent.

DISCUSSION

This study demonstrated that magnetic stimulation applied to the muscle belly can evoke cerebral potentials that are most likely due to activation of muscle afferents secondary to the induced phasic contraction of the stimulated muscle. The method presented provides an opportunity to quantify the function of muscle afferent from muscles widely distributed over the body. Prior work on eliciting

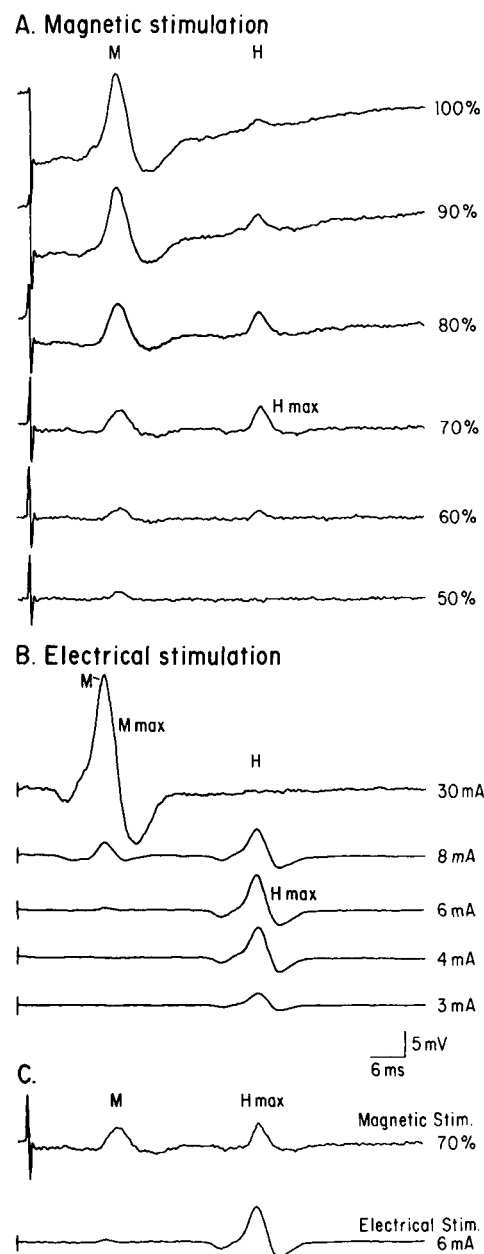## A. Magnetic stimulation

B. Electrical stimulation

FIGURE 9. H-reflexes of soleus in 1 subject elicited by magnetic stimulation (A), and by electrical stimulation (impulse duration: 1 msec) (B), to the PTN at the popliteal fossa. The responses were recorded in response to progressively increasing stimulus intensities with an interval of 10 sec. Mmax was not obtained by magnetic stimulation at maximal stimulus output. Hmax, obtained by the two forms of stimulation, have different amplitudes and are associated differently with the occurrence of the M-response (C). In this recording, negativity at grid 1 of the amplifier is displayed upward.

sensory evoked potentials from muscle afferents have used a rapid change in muscle length as the effective stimulus induced by a phasic movement of a joint,[33] mechanical displacement of the ten-

don,[12] electrical stimulation of muscle afferents through a microelectrode located within a peripheral nerve,[6,33] or intramuscular electrical stimulation with the microelectrode inserted percutaneously at the motor point.[27] The peak latencies of the initial positivity evoked by these different methods applied to the muscles at the same level (ie, the gastrocnemius and tibialis anterior) differed: ranging from 46 msec with mechanical movement of the ankle joint, to 40 msec with magnetic stimulation of the muscle (in the present experiment), to approximately 33 msec with Achilles tendon tap, and 32 msec with direct stimulation of muscle afferents. The latency differences must reflect delays inherent in these various techniques in activating muscle receptor afferents.

The use of a magnetic stimulus, applied to the belly of a muscle to activate muscle afferents, has the advantage in that a variety of different muscles can easily be tested and the equipment easily be used. In contrast, the equipment required to produce rapid movement at joints is complex and, for some joints, the arrangement of the instrumentation would be extremely difficult; the accessibility of tendons for mechanical displacement via a tap is easily achieved in only a few muscles; microelectrode stimulation of muscle afferent is an invasive and technically difficult procedure.

The topography of the cerebral potentials evoked by magnetic stimulation of the muscle can be distinguished from the potentials evoked by stimulation of mixed nerves passing close to the muscle under study. The early prominent P40 component following magnetic stimulation of the gastrocnemius was maximal in amplitude at the vertex attenuating symmetrically on either side, similar to the scalp distribution of the initial positivity to mechanical stretch of the gastrocnemius muscle.[11]

In contrast, stimulation of mixed nerves either just distal (PTN at ankle) or just proximal (PTN in popliteal fossa) to the belly of the gastrocnemius, produced early positive components (P37 for PTN at the ankle and P31 for PTN at the popliteal fossa) that have an asymmetrical distribution being maximum in the midline and ipsilateral to the side of stimulation, and becoming isopotential or even negative contralaterally. These differences could be due to differences in the cortical arrangement of inpus arising from muscle afferent from the gastrocnemius, compared with mixed inputs from posterior tibial nerve.

The cortical projection area of sensory afferent from the gastrocnemius is along the curvature of the interhemispheric fissure. The accompanying dipoles project more tangential to the surface than do the dipole generators activated by inputs from the foot, which lie deep within the interhemisphere fissure.[30,31]

The cortical distribution of the initial positive component (P31) to electrical stimulation to PTN at the knee, as shown in Figure 3B, was similar to that of P40 to PTN at the ankle, with the initial positivity amplitude predominance on the side ipsilateral to the stimulus.[3,13,14,27,30] Electrical stimulation to PTN at the knee activates a wide variety of sensory afferents, including those from muscle, and skin and joint from the foot and the calf. Pelosi et al.[30] suggested that the scalp fields accompanying PTN stimulation at the level of the knee represent an interaction of a variety of dipoles. These factors could account for the differences observed between the scalp distributions of mixed nerve and selective muscle afferent input described in the present study.

There are several lines of evidence indicating that magnetic stimulation applied to a muscle belly evoked cerebral potentials due to Ia afferents from muscle receptors. The cerebral evoked potentials following magnetic stimulation were attenuated when vibration was simultaneously applied to the same muscle. The maximum effects were seen with rates close to 60 Hz, a stimulation rate that induces a discharge of muscle spindles.[5,12,15] One of the mechanisms underlying this phenomenon is that vibration activates muscle spindles making them inaccessible to other stimuli, such as that accompanying the discharge of the magnetic coil, ie, the busy line effect.[19] Similarly, passive lengthening of the muscle, which also activates muscle spindles,[29] was also accompanied by an attenuation of amplitude of the potentials evoked by magnetic stimulation over that muscle.

In contrast, shortening of that muscle was not accompanied by any such attenuation of the evoked potentials to magnetic stimulation. Cutaneous afferents contribute little to the early components of the potentials evoked by magnetic stimulation, as was shown by the lack of effect of anesthetic blockade of the lateral femoral cutaneous nerve on the evoked potentials accompanying magnetic stimulation of the vastus lateralis muscle.

Magnetic stimulation of the muscle could directly stimulate muscle receptors and/or their afferent nerve endings, or indirectly activate muscle receptors by inducing a contraction of the muscle itself. Lotz et al.[25] concluded that magnetic stimu-

lation applied to a muscle preferentially activates small intramuscular nerve fibers compared with muscle fibers, because, in the curarized patient, there was no muscle twitch in response to maximal magnetic stimulation over muscle.

In the present experiment, the finding that latency of the cerebral evoked potentials to magnetic stimulation of the gastrocnemius muscle were longer than to stimulation of the PTN distally at the ankle, also suggests that magnetic stimulation activates muscle receptors or their afferent fibers indirectly through muscle contraction. Even when the magnetic stimulus was applied to a mixed nerve, muscle afferents were engaged (as defined by the H-reflex) at intensities above that necessary for motor efferent effects. Thus, magnetic stimulation easily activates motor nerves sufficient to cause contraction and only activate Ia afferent at high stimulus levels.

This is in contrast to electrical stimulation (usually of 0.5 to 1.0 msec in impulse duration) of the nerve, which activates Ia afferents (the H-reflex) at lower stimulus levels than necessary to activate motor efferents.[22] The differences in threshold between sensory and motor nerve fiber activation to magnetic and electrical forms of stimulation are most likely due to differences in their stimulus du-

rations: In magnetic forms of activation, the stimulus is extremely brief (0.05 msec) and has the form of a spike, whereas in electrical forms of activation, the stimulus is usually of long duration (0.2 to 1.0 msec) and has a square-wave shape.[28,35]

We applied a short-duration electrical pulse, 0.04 msec, to the posterior tibial nerve in the popliteal fossa, and found that the threshold for the M and H waves were changed in comparison with the 1-msec pulse used in the experiments. With the very short-duration pulse, the M and H waves appeared at the same threshold.

Mammalian motor units contract at different speeds.[10] In skeletal muscle of man, there is a continuum of motor unit twitch speeds rather than the 2 distinct populations found in other mammals, ie, fast-twitch and slow-twitch motor units. The duration of contraction varies from less than 10 msec for ocular muscle to 100 msec for soleus muscle, depending on the particular motor unit population.[17] This factor may account for the paradoxical findings illustrated in Figure 5 that the peak latencies of the initial positive component of the cerebral potentials do not change entirely in an orderly fashion as a function of the location of the muscle (eg, compare rectus femoris with gluteus maximus).

REFERENCES

1. Abbruzzese G, Berardelli A, Rothwell JC, Day BL, Marsden CD: Cerebral potentials and electromyographic responses evoked by stretch of wrist muscle in man. *Brain Research* 1985;58:544–551.
2. Barker AT, Freeston IL, Jalinous R, Jarratt IA: Magnetic stimulation of the human brain and peripheral nervous system: An introduction and the results of an initial clinical evaluation. *Neurosurgery* 1987;20:100–109.
3. Beric A, Prevec TS: Distribution of scalp somatosensory potentials evoked by stimulation of the tibial nerve in man. *J Neurol Sci* 1983;59:205–214.
4. Bromm B, Treede RD: Human cerebral potentials evoked by CO_2 laser stimuli causing pain. *Exp Brain Res* 1987;67:153–162.
5. Brown MC, Engbeng I, Matthews PBC: The relative sensitivity to vibration of muscle receptors of the cat. *J Neurophysiol (Lond)* 1967;192:773–800.
6. Burke D, Skuse NF, Lethlean AK: Cutaneous and muscle afferent components of the cerebral potential evoked by electrical stimulation of human peripheral nerve. *Electroenceph Clin Neurophysiol* 1981;51:579–588.
7. Carmon A, Dotan Y, Sarne Y: Correlation of subjective pain experience with cerebral evoked responses to noxious thermal stimulation. *Exp Brain Res* 1978;33:445–453.
8. Chartt AB, Kenshalo DR: Cerebral responses to skin warming recording from human scalp. *Exp Brain Res* 1977;28:449–455.
9. Claus D, Murray NMF, Spitzer A, Flugel D: The influence of stimulus type on the magnetic excitation of nerve structure. *Electroenceph Clin Neurophysiol* 1990;75:342–349.
10. Close R: Dynamic properties of mammalian skeletal muscle. *Physiological Reviews* 1972;52:129–197.
11. Cohen LG, Starr A, Pratt H: Cerebral somatosensory potentials evoked by muscle stretch, cutaneous taps and electrical stimulation of peripheral nerves in the lower limbs in man. *Brain* 1985;108:103–121.
12. Cohen LG, Starr A: About the origin of cerebral somatosensory potentials evoked by Achilles tendon taps in humans. *Electroenceph Clin Neurophysiol* 1985;62:108–116.
13. Cruse R, Klem G, Lesser RP, Lueders H: Paradoxical lateralization of cortical potentials evoked by stimulation of posterior tibial nerve. *Arch Neurol* 1982;39:222–235.
14. Desmedt JE, Bourguet M: Color imaging of parietal frontal somatosensory potential fields evoked by stimulation of median of posterior tibial nerve in man. *Electroenceph Clin Neurophysiol* 1985;62:1–17.
15. Desmedt JE: Mechanisms of vibration-induced inhibition or potentiation: tonic vibration reflex and vibration paradox in man, in Desmedt JE (ed): *Motor Control Mechanisms in Health and Disease.* New York, Raven Press, 1983, pp 671–683.
16. Duclaux R, Franzen O, Chatt AB, Kenshalo DR, Stowell H: Responses recorded from human scalp evoked by cutaneous thermal stimulation. *Brain Research* 1974;78:279–290.
17. Freund HJ: Motor unit and muscle activity in voluntary motor control. *Physiological Reviews* 1983;63:387–436.
18. Geddes LA: Optimal stimulus duration of extracranial cortical stimulation. *Neurosurgery* 1987;20:94–99.
19. Hagbarth KE: The effects of muscle vibration in normal

man in patients with motor disorders, in Desmedt JE (ed): *Human Reflexes, Pathophysiology of Motor System, Methodology of Human Reflexes. New Development in Electromyography and Clinical Neurophysiology.* Basel, Karger, 1973, vol 3, pp 428–443.

20. Hallett M, Cohen LG, Nilsson J, Panizza M: Differences between electrical and magnetic stimulation of human peripheral nerve and motor cortex, in Chokroverty S (ed): *Magnetic Stimulation in Clinical Neurophysiology.* Stoneham, MA, Butterworth, 1990, pp 113–119.
21. Hashimoto I: Somatosensory evoked potentials elicited by air-puff stimuli generated by a new high-seed air control system. *Electroenceph Clin Neurophysiol* 1987;67:231–237.
22. Hugon M: Methodology of the Hoffman reflex in man, in Desmedt JE (ed): *New Developments in Electromyography and Clinical Neurophysiology.* Basel, Karger, 1973, vol 3, pp 277–293.
23. Kakigi R, Shibasaki H: Scalp topography of mechanically and electrically evoked somatosensory potentials in man. *Electroenceph Clin Neurophysiol* 1984;59:44–56.
24. Larsson LE, Prevec TS: Somatosensory response to mechanical stimulation as recorded in human EEG. *Electroenceph Clin Neurophysiol* 1970;28:162–172.
25. Lotz BP, Dunne JW, Daube JR: Preferential activation of muscle fibers with peripheral magnetic stimulation of the limb. *Muscle Nerve* 1989;24:568–573.
26. Maccabee PJ, Amassian VE, Cracco RQ, Cadwell JA: Analysis of peripheral motor stimulation in humans using the magnetic coil. *Electroenceph Clin Neurophysiol* 1988;70:524–533.
27. Macefield G, Burke D, Gandevia SC: The cortical distribution of muscle and cutaneous afferent projections from the

human foot. *Electroenceph Clin Neurophysiol* 1989;72:518–528.
28. Panizza M, Nilsson, J, Hallett M: Optimal stimulus duration for the H reflex. *Muscle Nerve* 1989;12:576–579.
29. Patton HD: Reflex regulation of movement and posture, in Ruch TC, Patton HD (eds): *Physiology and Biophysics,* 19th ed. Philadelphia, Saunders, 1965, pp 181–206.
30. Pelosi L, Cracco JB, Cracco RQ, Hassan NF: Comparison of scalp distribution of short latency somatosensory evoked potentials to stimulation of different nerves in the lower extremity. *Electroenceph Clin Neurophysiol* 1988;71:422–428.
31. Penfield W, Rasmussen T: *The Cerebral Cortex of Man. A Clinical Study of Localization of Function.* New York, Macmillan, 1950.
32. Pratt H, Amlie RN, Starr A: Short latency mechanically evoked somatosensory potentials in humans. *Electroenceph Clin Neurophysiol* 1979;47:524–531.
33. Starr A, McKeon B, Skuse NF, Burke D: Cerebral evoked potentials to muscle stretch in man. *Brain* 1981;104:149–166.
34. Tsuji S, Murai Y, Yarita M: Somatosensory potentials evoked by magnetic stimulation of lumbar roots, cauda equine, and leg nerves. *Ann Neurol* 1988;24:568–573.
35. Veale JL, Mark RF, Sandra R: Differential sensitivity of motor and sensory fibres in human ulnar nerve. *J Neurol Neurosurg Psychiatry* 1973;36:75–86.
36. Zarola F, Caramia MD, Mariorenzi R, Desiato MT, Rossini PM: SEPs to median nerve stimulation: A comparative study during electrical and magnetic stimulation in healthy humans. *Electroenceph Clin Neurophysiol* 1990;75:S165 (abstr).