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Authors
Cohen, LG
Starr, A

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Article abstract—We recorded potentials evoked by specific somatosensory stimuli over peripheral nerve, spinal cord, and cerebral cortex. Vibration attenuated spinal and cerebral potentials evoked by mixed nerve and muscle spindle stimulation; in one subject that was tested, there was no effect on cutaneous input. Presynaptic inhibition of Ia input in the spinal cord and muscle spindle receptor occupancy are probably the responsible mechanisms. In contrast, muscle contraction attenuated cerebral potentials to both cutaneous and muscle spindle afferent volleys; central mechanisms modulating neurons in the dorsal columns nuclei, thalamus, or cerebral cortex are probably responsible.

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Vibration and muscle contraction affect somatosensory evoked potentials

Leo G. Cohen, MD, and Arnold Starr, MD

When vibration is applied to tendons, muscle spindles are activated in cats[1-3] and humans,[4] accompanied by inhibition of H and muscle stretch spinal reflexes.[5] Two main mechanisms are probably involved: presynaptic inhibition in the spinal cord[5] and peripheral receptor occupancy.[4]

During movement, there is also an attenuation of the transmission of somatosensory afferent information in the nervous system, a process that has been termed "gating."[6-10] Animal experiments suggest that the locus of this gating occurs within the CNS,[6-8] but it is not known where gating of human afferent somatosensory volleys occurs during muscle contraction.

We therefore studied the effects of vibration and contraction on different types of somatosensory afferent input (mixed nerve, muscle spindles, cutaneous nerve, and cutaneous receptors) by recording evoked potentials over peripheral nerve, spinal cord, and cerebral cortex to electrical stimulation of the posterior tibial nerve (Ia fibers[11]), to tendon taps (muscle spindles[12]), to mechanical taps to the skin (cutaneous mechanoreceptors[13]), and to electrical stimulation of the sural nerve (cutaneous afferent fibers[14]).

Methods. *Subjects.* Subjects were young and healthy students, 18 to 23 years old. They were tested lying in bed in a sound-attenuating chamber. The ankle was fixed at 90° by a mold designed to restrict movements of the foot. Skin temperature was monitored and maintained between 31 and 35 °C.

Stimuli. Percutaneous electrical stimulation was delivered over the posterior tibial nerve (PTN) immediately posterior to the medial malleolus and over the sural nerve (SN) posterior to the external malleolus. The intensities of the stimuli were adjusted to the threshold for eliciting a visible twitch of the muscles innervated by PTN and to three times the sensory threshold for the SN. Mechanical taps were delivered to the Achilles tendon for stretching muscle spindles and to the skin overlying the external malleolus for cutaneous stimulation. A moving coil vibrator was activated by a 70 to 100 msec duration square-wave electric pulse, resulting in a downward movement of 4 to 5 mm of a rod attached to the vibrator. The vibrator's spindle was attached to a T-rod, with the horizontal portion placed in contact with the skin overlying the Achilles tendon 3 to 6 cm proximal to its insertion. For skin stimulation, the rod was 3 mm from the skin overlying the external malleolus to avoid moving the foot after contact with the rod.

Recording and analysis. Somatosensory cortical potentials were recorded from an electrode over the scalp at Cz (according to the 10—20 system), referenced to a forehead electrode (Fpz). Spinal cord activity was recorded from an electrode placed over L1, referenced to the iliac crest. Popliteal nerve-evoked potentials were recorded with a monopolar needle electrode located near the nerve, referenced to a subcutaneous electrode in the popliteal fossa. Subjects were grounded by a metal plate strapped to the leg, proximal to the knee. Skin electrode impedances were below 5 KOhm. Amplification of 500,000 was used, with a bandpass of 30 to 1,000 Hz (6 dB down points).

The potentials evoked by mechanical and electrical stimulation were averaged over a 100 msec 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 poten-

From the Department of Neurology, University of California, Irvine, CA.

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Address correspondence and reprint requests to Dr. Starr, Department of Neurology, University of California, Irvine, CA 92717.

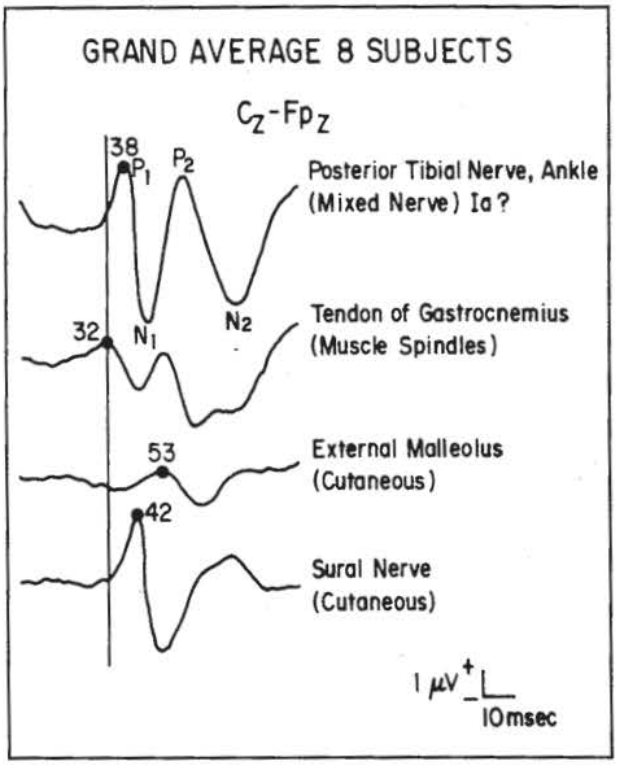

Figure 1. Grand average of cerebral potentials to mechanical and electrical stimulation at the ankle. The different components are labeled by their polarity at the vertex and their sequence (P1, N1, P2, and N2). The latencies of P1 component to each form of stimulation are indicated over the corresponding peaks. A vertical line has been placed at the P1 latency evoked by tendon taps.

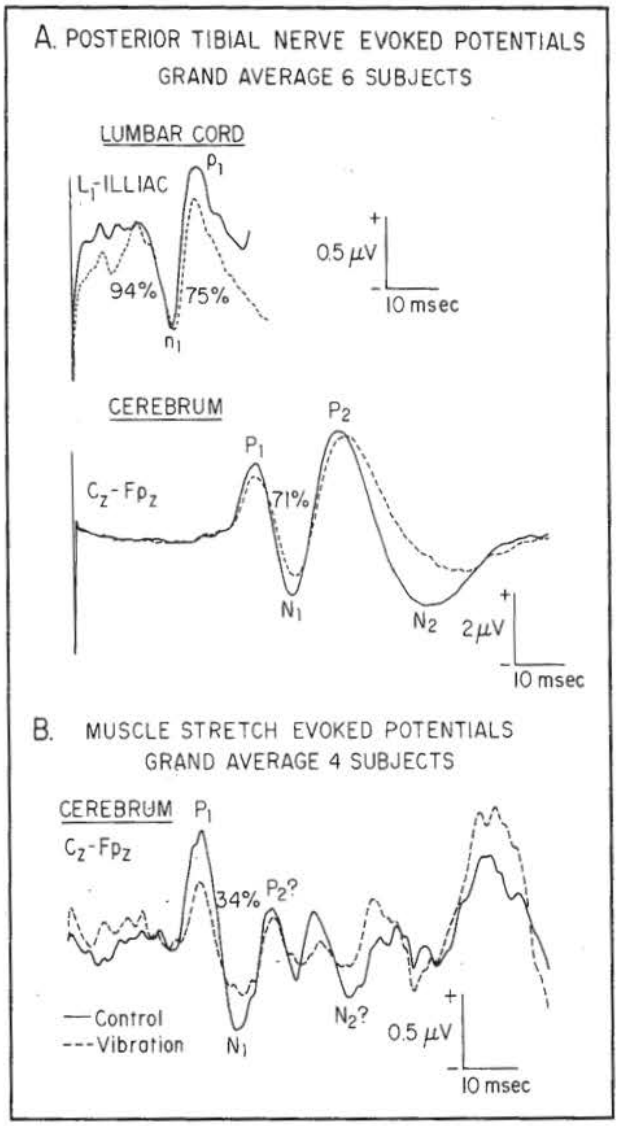

Figure 2. The effects of vibration on A, lumbar and cerebral potentials to posterior tibial nerve stimulation, and B, cerebral potentials to muscle stretch. In this and all subsequent figures, the percentages refer to the amplitudes of the potentials recorded during vibration or muscle contraction (dashed lines) in reference to controls (solid lines) in the same subject and session without interference. The percentages refer to the change in the P1-N1 amplitude compared with the controls.

tials were recorded by an X-Y plotter (positivity at Cz displayed upwards) and stored on disks for further analysis. Amplitudes of cerebral evoked potentials and latencies of the various components of the recorded potentials were measured from the computer screen 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 evoked potentials were measured in one or both of two ways: (1) absolute amplitudes between baseline and positive or negative peaks, and (2) differential amplitudes between each component and the immediately following component of opposite polarity. Amplitudes of lumbar and peripheral nerve-evoked potentials were measured between the baseline and first negativity (n1), and between first negativity and subsequent positivity (n1-p1). Amplitudes of potentials recorded during vibration and muscle contraction are expressed as a percentage of control recorded in the same subject during the same session. Three considerations show that the signals picked up with scalp electrodes to Achilles tendon taps were of neural origin: (1) they had a well-defined scalp distribution[12]; (2) the differential recording montage used in this study (C_z-F_{pz}) would have severely attenuated the detection of muscular artifact originating from the calf; and (3) during muscle contraction, when spinal muscle reflexes are enhanced, cerebral potentials were attenuated.

Vibration interference. Vibration was produced with a second vibrator activated by a frequency generator at 60 Hz and applied over the heel at the insertion of Achilles tendon on the calcaneus bone. This site proved to be effective for transmitting vibration to the muscles in both the foot and in the calf. Vibration interference started 30 seconds before

Table 1. Effects of vibration on SEP

A. Amplitudes of cerebral and lumbar potentials to posterior tibial nerve stimulation expressed as percentages of control values (100%)

	Cerebral						Lumbar	
Subjects	P1	N1	P2	N2	P1-N1	P2-N2	n1	n1-p1
1	0*	82	75	113	74	100	76	74
2	66	200	90	65	82	77	90	77
3	53	86	82	72	79	78	89	75
4	63	58	72	48	66	59	106	85
5	54	56	80	50	61	62	104	79
6	73	57	65	62	68	64	101	69
$\bar{X}$	51.4 ± 26.2	90 ± 55.4	77.4 ± 8.8	68.4 ± 23.6	71.6 ± 7.9	73.4 ± 15.3	94.3 ± 11.4	75.1 ± 6.6
$t =$	4.5	0.4	6.2	3.2	8.7	4.2	1.2	9.1
$p <$	0.01	NS	0.01	0.025	0.01	0.01	NS	0.01

B. Amplitudes of cerebral potentials to muscle stretch expressed as percentages of control values (100%)

	Cerebral					
Subjects	P1	N1	P2	N2	P1-N1	P2-N2
1	0*	41	28	54	14	41
2	0*	34	48	39	14	95
3	27	36	100	108	33	51
4	82	77	66	40	78	43
$\bar{X}$	27.2 ± 38.5	47.1 ± 20	60.4 ± 30.4	60.5 ± 32.4	34 ± 30.1	57.7 ± 25
$t =$	3.7	5.2	2.6	2.4	4.3	3.2
$p <$	0.025	0.01	0.05	0.05	0.025	0.025

* The component could not be distinguished from baseline.

recording the potentials to the various somatosensory stimuli. To increase the efficiency of the vibratory stimulus on muscle spindles in the gastrocnemius-soleus muscles, the ankle was passively dorsiflexed.

Muscle contraction interference. Isotonic active contraction of the ipsilateral and contralateral gastrocnemius-soleus and flexors of the toes was exerted against a load averaging 10 kg. The effects of increasing the load from 1 to 15 kg, as well as the effects of isometric muscle contraction on the evoked potentials, were tested. They were compared with the effects produced by passive flexion and extension of the foot.

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

Results. The different stimuli applied at the ankle evoked cerebral potentials consisting of a sequence of positive and negative components whose latencies differed with the type of stimulation (figure 1). The latency of the first positivity was shortest after gastrocnemius-soleus muscle stretch (32 msec), followed by posterior tibial nerve stimulation (38 msec), sural nerve (42 msec), and finally cutaneous tapping on the external malleolus (53 msec). Details of these various potentials are contained in a separate report.[12] Both vibration and muscle contraction affected amplitudes, but not latencies, of the evoked potentials.

Effects of vibration. Vibration applied over the ipsilateral heel diminished moderately (to 71.6 ± 7.99%; $p < 0.01$) the P1-N1 component of cerebral potentials to PTN stimulation in six subjects (figure 2A, cerebrum). The positive potentials recorded over the lumbar region also decreased (to 75.16 ± 6.64%; $p < 0.01$), whereas the immediately preceding negative component was unchanged (figure 2A, lumbar cord, and table 1A). In addition, the PTN potentials recorded from the popliteal fossa in one subject were unaffected. Vibration of the limb contralateral to the one being tested did not affect lumbar or cerebral potentials to PTN stimulation. Frequencies between 40 and 80 Hz had the greatest effect on the amplitude of the P1-N1 component of SEP after stimulation to the PTN (figure 3). We therefore used 60 Hz in our studies. The amplitude of the cerebral P1-N1 component also decreased as the vibrator excursion was increased (figure 3), and we used the highest possible excursion of the vibrator (5 mm) as the interfering stimulus. The results (table 1) indicate that the effects of vibration on diminishing the amplitude of PTN sensory volleys recorded over the cerebrum and lumbar regions occur distal to or at the site of generation of the positive component of lumbar SEP, but proximal to the peripheral nerve or nerve root.

Vibration also diminished the P1-N1 component of cerebral potentials evoked by muscle stretch (to 34 ± 30%; $p < 0.025$) in four subjects (figure 2B). The

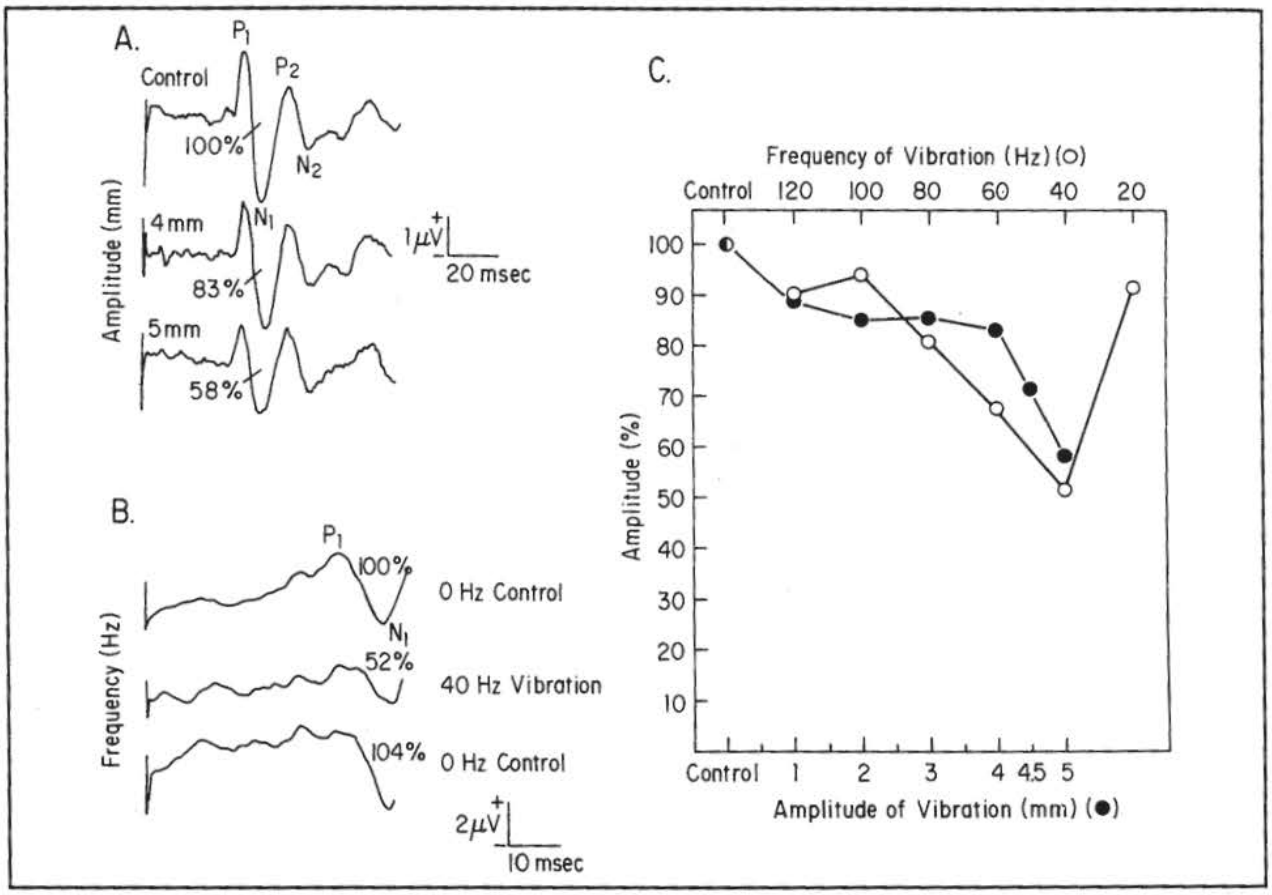

Figure 3. A, the effects of varying the amplitude, and B, the frequency of vibration, on the amplitude of posterior tibial nerve-evoked potentials. In C, the graph shows the amplitude of the initial positive-negative complex (P1-N1) as a function of the amplitude (●——●) and frequency (○——○) of vibration. The amplitude of the P1-N1 component in the absence of vibration ("control") was set to be 100%.

attenuation of muscle stretch-evoked cerebral potentials was greater than that of PTN-evoked cerebral potentials, but only the diminution of the P1-N1 component reached statistical significance (to 34% versus to 71%; $p < 0.01$) (table 1B).

In contrast, the cerebral potentials evoked by cutaneous stimulation (mechanical taps to skin) or electrical stimulation of the sural nerve did not change during vibration in the one subject tested (no. 1 in table 1), although that subject had demonstrated a clear attenuation of potentials evoked by stimulation of PTN and muscle stretch.

Effects of muscle contraction. Isotonic contraction of gastrocnemius-soleus muscle and flexors of the toes did not alter the lumbar potentials evoked by ipsilateral PTN stimulation, but did exert a moderate attenuation on the P1-N1 component of the cerebral potentials to the same stimulus (to 63.2 ± 15.8%; $p < 0.01$, eight subjects) (figure 4A, table 2A). Similarly, isotonic active contraction of the gastrocnemius-soleus muscle and flexors of the toes *contralateral* to the stimulated PTN slightly attenuated the P1-N1 component of cerebral potentials to PTN stimulation (to 72.9 ± 10.5%; $p < 0.025$), without affecting lumbar-evoked potentials (figure 4B, table 3). In the one subject tested, the slope of the attenuation of the P1-N1 component was linear as a function of the extent of the force of contraction (figure 5).

Muscle contraction also affected the amplitude of the other types of SEPs. The amplitude of P1-N1 component of cerebral potentials evoked by stretch of the gastrocnemius-soleus was diminished (to 22 ± 21.9%, $p < 0.025$) by isotonic contraction of these same muscles (figure 6A). The attenuating effect of muscle contraction on muscle stretch-evoked potentials was much greater than on the potentials evoked by PTN stimulation (P1-N1 component diminished to 22% versus to 63%, $p < 0.01$) (table 2B). Muscle contraction was also associated with an attenuation of the P1-N1 component of the potentials evoked by stimulating the cutaneous sural nerve in four subjects (to 68.4 ± 12.4%, $p < 0.01$, figure 6B), without affecting the amplitudes of the lumbar potentials evoked by the same stimulus (two subjects) (table 2C).

These results indicate that muscle contraction, in contrast to vibration, exerts an inhibitory effect proximal to the lumbar cord on cerebral potentials

Table 2. Effects of ipsilateral gastrocnemius-soleus muscle contraction on SEP

A. Amplitudes of cerebral and lumbar potentials to posterior tibial nerve stimulation expressed as percentages of control values (100%)

	Cerebral						Lumbar
Subjects	P1	N1	P2	N2	P1-N1	P2-N2	n1-p1
1	59	82	86	95	74	90	87
2	50	66	40	74	59	65	NT
3	43	97	100	91	64	100	NT
4	49	89	115	100	81	106	NT
5	49	51	49	63	50	57	48
6	83	78	64	91	80	80	100
7	66	56	67	104	65	54	94
8	49	24	40	22	34	29	NT
$\overline{X}$	56 ± 13	68 ± 23.6	70.2 ± 27.9	80.1 ± 27	63.2 ± 15.8	72.6 ± 26.1	82.3 ± 23.4
$t =$	9.5	3.8	3	2.1	6.5	2.9	1.5
$p <$	0.01	0.01	0.01	0.05	0.01	0.025	NS

B. Amplitudes of cerebral potentials to muscle stretch stimulation expressed as percentages of control values (100%)

	Cerebral					
Subjects	P1	N1	P2	N2	P1-N1	P2-N2
1	0*	0*	0*	0*	0*	0*
2	13	71	37	87	44	66
3	23	0*	30	28	22	29
$\overline{X}$	12.1 ± 11	24 ± 41.2	22.4 ± 19.6	38.3 ± 44.3	22 ± 21.9	31.6 ± 32.8
$t =$	13	3.2	6.8	2.4	6.1	3.6
$p <$	0.01	0.05	0.025	NS	0.025	0.05

C. Amplitudes of cerebral potentials to sural nerve stimulation expressed as percentages of control values (100%)

	Cerebral						Lumbar
Subjects	P1	N1	P2	N2	P1-N1	P2-N2	n1-p1
1	69	85	138	12	77	118	NT
2	12	86	85	88	50	89	NT
3	0*	91	0*	0*	72	0*	110
4	62	80	80	85	74	83	105
$\overline{X}$	36.1 ± 34.9	85.6 ± 4.3	75.9 ± 56.9	46.4 ± 46.9	68.4 ± 12.4	72.7 ± 50	107.5 ± 3.5
$t =$	3.7	6.5	0.8	2.2	5.1	1	
$p <$	0.025	0.01	NS	0.1	0.01	NS	

* The component could not be distinguished from baseline.
NT Not tested.

evoked by both cutaneous and Ia afferent volleys.

Passive flexion and extension of the ankle. The effects of maintained passive flexion and extension of the ankle were tested in one subject. Neither flexion nor extension was associated with any change in the amplitudes and latencies of PTN-evoked potentials.

Discussion. These results show that vibration and muscle contraction inhibit afferent volleys in the somatosensory system by effects at different locations and through different mechanisms.

Vibration selectively attenuated potentials arising from activation of Ia afferents, ie, muscle stretch and PTN-evoked potentials,[11] but not those derived from cutaneous stimulation. The frequency of vibration employed (60 Hz) provides sustained activation of muscle spindles to discharge Ia afferents, but is less effective in activating cutaneous inputs.[5,15,16]

Vibration did not modify PTN-evoked potentials at the popliteal fossa or the initial negative component of the lumbar potentials, but it did attenuate the subsequent positive component of lumbar potentials. These findings support the idea that potentials recorded over the lumbar spine are probably the sum of at least two generators. The initial negative component, which did not change with vibration, is attributed to the nerve root volley as it enters the spinal cord. The following positivity, which suffered an attenuation, is attributed to generators within the spinal cord. Thus, vibration initiates an inhibitory process affecting afferent input within the spinal cord. This process is frequency- and amplitude-dependent (figure 3), and is localized to input from the

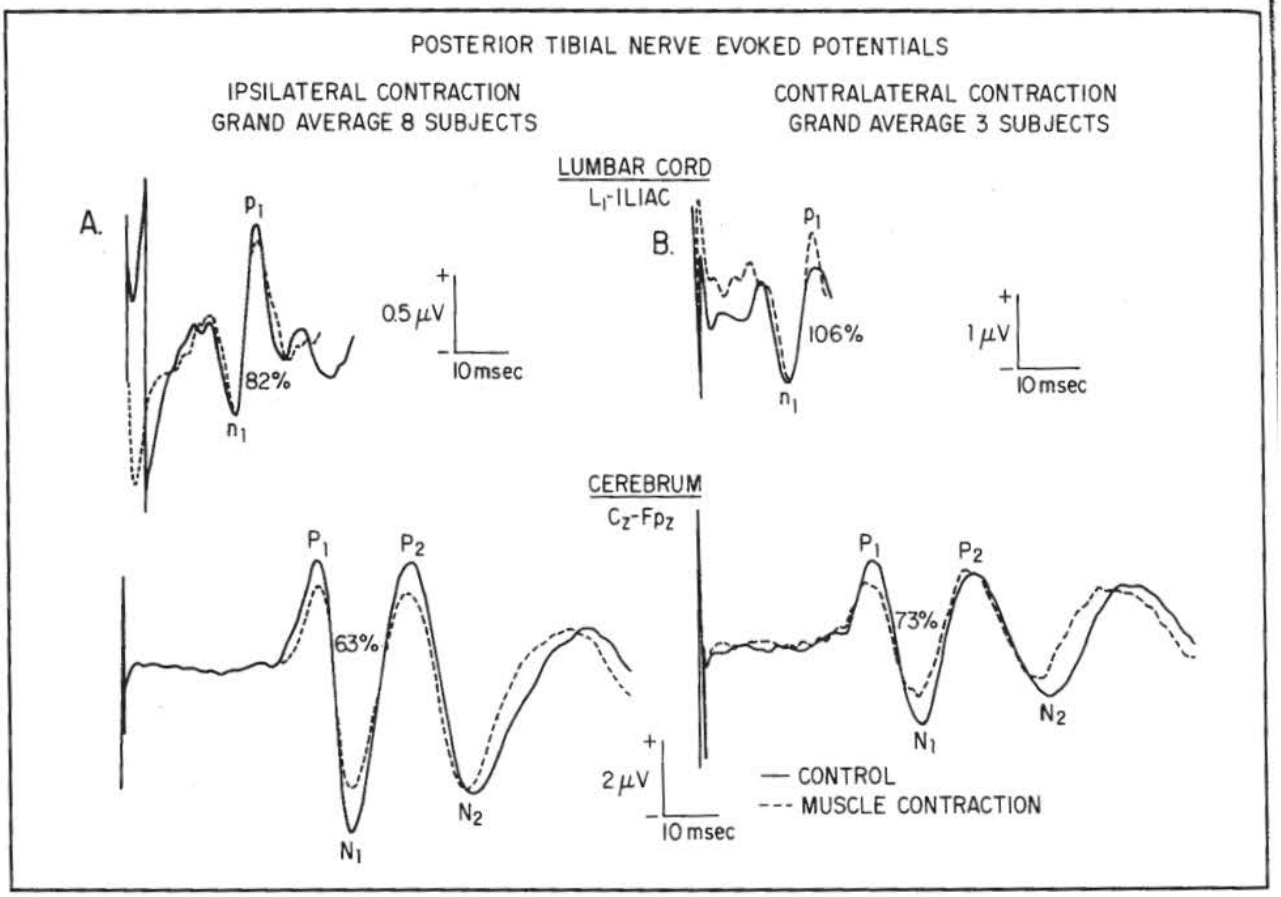

Figure 4. The effects of contraction of A, ipsilateral, and B, contralateral gastrocnemius-soleus muscle and flexors of the toes, on lumbar and cerebral evoked potentials to posterior tibial nerve stimulation.

Table 3. Effects of contralateral gastrocnemius-soleus muscle contraction on SEP

A. Amplitudes of cerebral and lumbar potentials to posterior tibial nerve stimulation expressed as percentages of control values (100%)

	Cerebral						Lumbar
Subjects	**P1**	**N1**	**P2**	**N2**	**P1-N1**	**P2-N2**	**n1-p1**
1	42	78	100	70	66	79	98
2	70	91	100	93	85	96	100
3	71	64	96	87	67	91	120
$\overline{X}$	61 ± 16	78.1 ± 13.4	98.8 ± 2	83.6 ± 12.4	72.9 ± 10.5	88.3 ± 8.7	106 ± 12.1
$t =$	4.1	2.8	1	2.3	4.4	2.3	0.8
$p <$	0.05	NS	NS	NS	0.025	NS	NS

limb being vibrated. A likely mechanism is presynaptic inhibition of spindle afferents in the spinal cord, since it can be induced in humans by vibration in the range of frequencies and amplitudes we used[17] (figure 2). Moreover, vibration-induced presynaptic inhibition does not occur to inputs from the opposite limb.[18]

The afferent volley to PTN stimulation could not have been reduced by receptor occlusion or "line-busy effect" by the steady vibration-induced input, since both peripheral nerve potentials and the negative component of lumbar potentials did not change when the foot was vibrated. However, a peripheral mechanism could have played a role in the attenuation of muscle stretch-evoked potentials, because stretch-evoked potentials were more affected than PTN-evoked potentials; thus, spindles in the stretched muscle could have been occluded by vibration. Unfortunately, we were unable to successfully record potentials from either the peripheral nerve or lumbar region to muscle stretch to test this possibility.

In contrast, Abbruzzese et al[15] found no changes in cervical spinal cord potentials after median nerve

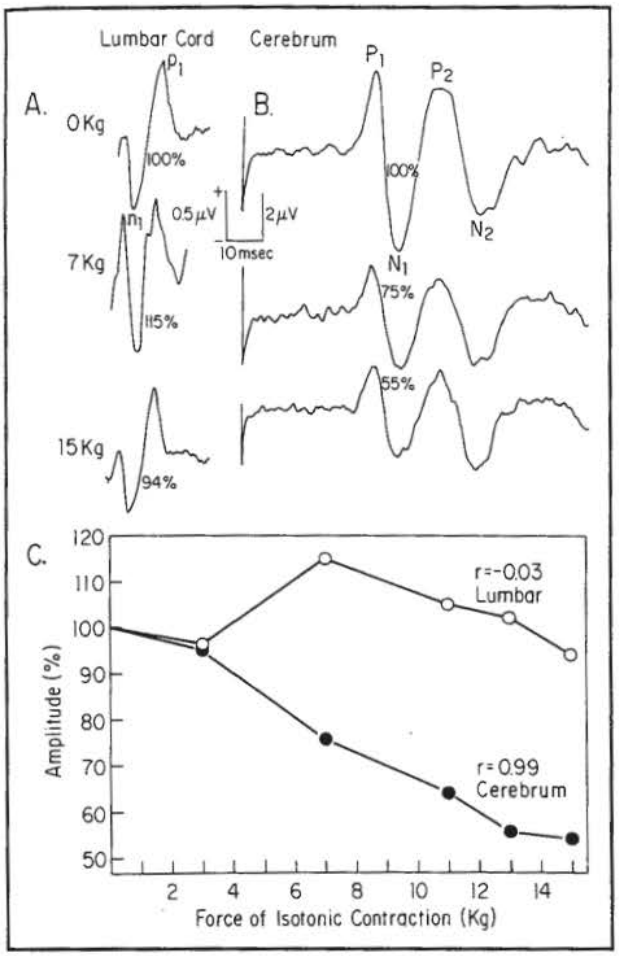

Figure 5. The effects of increasing force of isotonic contraction on A, lumbar, and B, cerebral evoked potentials, to posterior tibial nerve stimulation. In C, the graph shows the measured amplitude of lumbar (n1-p1) (○——○) and cerebral (P1-N1) (●——●) evoked potentials to PTN stimulation as a function of the force of contraction.

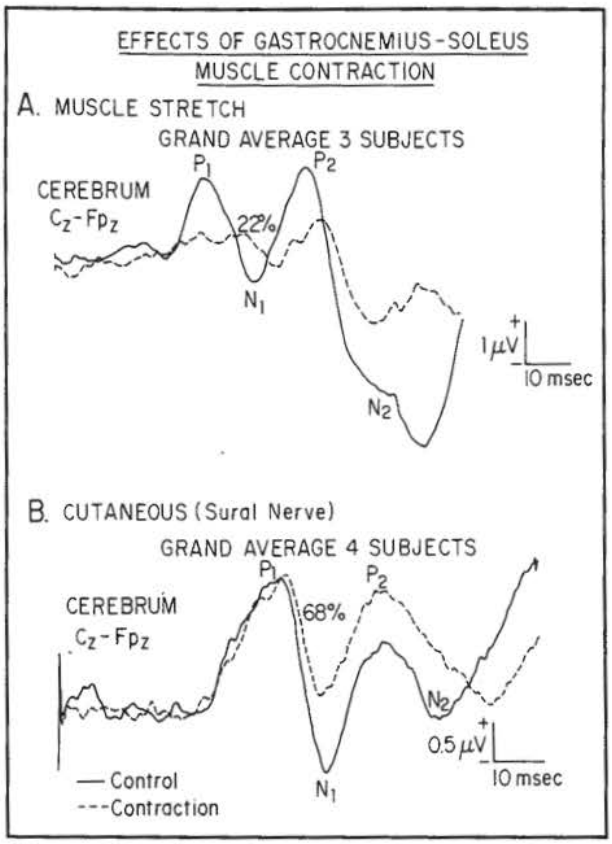

Figure 6. The effects of contraction of gastrocnemius-soleus muscle and flexors of the toes on cerebral potentials to A, muscle stretch, and B, sural nerve stimulation.

stimulation when the stimulated hand was vibrated. Failure to detect inhibition could have been due, as they suggested, to contamination of the cervical potentials by an EMG-induced tonic vibration reflex that reduced clarity of the recordings. Attenuation of cerebral potentials evoked by the same stimulus during vibration was attributed to inhibitory interactions at the thalamocortical level between lemniscal and spinocerebellar inputs.[15] Our results, although not ruling out this possibility, suggest a spinal cord location for the vibration-induced attenuation of sensory input.

In the second part of this study, we found that muscle contraction attenuated cortical SEPs to different somatosensory inputs, including electrical stimulation of mixed and cutaneous sensory nerves, or to muscle stretch. The site of this inhibitory process is rostral to the lumbar cord, since cortical but not lumbar evoked potentials were affected.

Pyramidal tract fibers that project on the lemniscal pathway control or limit centripetal afferent input during movement.[8,19] This effect is exerted at the dorsal column nuclei through presynaptic and postsynaptic inhibition, and attenuates lemniscal responses during voluntary forelimb movements in cats.[7] Also, pyramidal tract stimulation can excite or inhibit neurons in the dorsal horn[20] or thalamus.[21] Therefore, active inhibitory mechanisms can modify afferent inputs transmitted through the dorsal column-medial lemniscus pathway. The dorsal column nuclei seem to be the site of interaction between neuronal systems that subserve active muscular contraction and afferent input, but the thalamus and cerebral cortex are other possible sites. Part of the gating effect of contraction could be peripheral,[9] because contraction evokes afferent volleys that travel in both cutaneous and muscular nerves and can alter other sensory-evoked activity in dorsal column nuclei.[22-25]

Attenuation of the initial positive-negative components of the cerebral SEP in our experiments during muscle contraction was 36%, similar to the 20% found by Ghez and Pisa[7] in cats during movement. The possible perceptual consequences of such attenuation was addressed by Coquery,[6] who found in humans a decrease in the perception of electric cuta-

neous stimulation before and during voluntary movement.[6]

We observed that attenuation of cerebral SEPs was proportional to the magnitude of the contractile force (figure 5). Coulter[8] also found in cats that the amount of depression of lemniscal responses was related to the magnitude of the corresponding muscle activity during movement.

In summary, vibration at 60 Hz inhibited spinal and cerebral potentials evoked by PTN or muscle stretch, without affecting cutaneous afferent processing. The mechanisms probably involve presynaptic inhibition of Ia afferents for the potentials evoked by PTN stimulation, and a combination of both presynaptic inhibition and muscle spindle receptor occupancy for muscle stretch-evoked potentials. In contrast, muscle contraction exerts an inhibitory effect on the potentials evoked by both cutaneous and Ia afferent volleys. The mechanisms involved are central to the lumbar spinal cord and could reflect modulation of the excitability of neurons in central parts of the pathway—ie, the gracilis, cuneatus, and thalamic nuclei or cerebral cortex.

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