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## SHORT LATENCY MECHANICALLY EVOKED SOMATOSENSORY POTENTIALS IN HUMANS

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### Introduction

Somatosensory potentials evoked by electrical stimulation of peripheral nerves can be recorded by surface electrodes at various locations along the somatosensory pathway. The potentials correspond to activity in peripheral nerves (Dawson and Scott 1949; Gilliatt and Sears 1958; Buchthal and Rosenfalck 1966), spinal cord (Cracco 1973; Matthews et al. 1974; Jones 1977; Sances et al. 1978), brain stem (Cracco and Cracco 1976; Jones 1977; Kritchewsky and Wiederholt 1978), and somatosensory cortex (Dawson 1947; Goff et al. 1962; Allison et al. 1974).

While electrical stimulation of the peripheral nerves ensures their synchronous activation, its disadvantages are: (1) a lack of specificity with regard to the types of fibers activated, (2) a failure to activate nerve endings resulting in functional changes confined to them going undetected, and (3) a sensation of discomfort, and even pain that subjects may experience.

Reports on the potentials evoked by mechanical cutaneous stimuli (e.g. tapping on body surfaces) have been limited to the peripheral nerves (Sears 1959; Bannister and Sears 1962; McLeod 1966) and to components whose peak latencies and scalp topography indicate their origin to be in the cerebral cortex (Kjellman et al. 1967; Franzen and Offenloch 1969; Larsson and Prevec 1970; Nakanishi et al. 1973). The potentials evoked by mechanical stimuli which originate

in the spinal cord and brain stem levels have not yet been described in humans.

The purpose of this study was to examine in humans the potentials evoked at several levels of the somatosensory pathway by mechanical cutaneous stimulation, and to try and define components in addition to the ones previously described.

### Methods

The subjects were 23 adults ranging in age from 18 to 68 years, without neurological complaints or symptoms suggestive of abnormalities of somatosensory function. They rested on a bed in a sound-attenuating chamber, with their left hand supported on a warmed plastic mold and their left index finger strapped to its surface. The skin temperature of the index finger was monitored continuously, and maintained between 33 and 36°C. Evoked potentials to mechanical stimulation on the fingernail delivered at a rate of 4/sec were obtained from each subject in a single session.

The mechanical stimulus was produced by activating a moving coil vibrator with a 50 msec duration square electric pulse. The sound produced by the vibrator's movement was masked by white noise from a speaker placed near the subject. The vibrator's spindle was attached to a rod with a hemispheric tip, 7 mm in diameter, and adjusted to be perpendicular to and just in contact with the center

of the nail of the index finger. Activation of the vibrator resulted in a downward movement of the rod. In two of the subjects the displacement of the fingernail was recorded with a Linear Variable Differential Transformer (LVDT), with its core connected to a thin rod glued to the surface of the finger. The displacements measured from one of the

subjects are included in Fig. 1a. The latency of movement was 1.8 msec and peak displacement occurred by 4.8 msec.

Digital potentials were recorded from ring electrodes around the middle and proximal phalanges of the index finger (the middle phalanx electrode channelled to grid 1 of the differential amplifier). The other recording

### MECHANICALLY EVOKED DIGITAL POTENTIALS CONTROL EXPERIMENTS

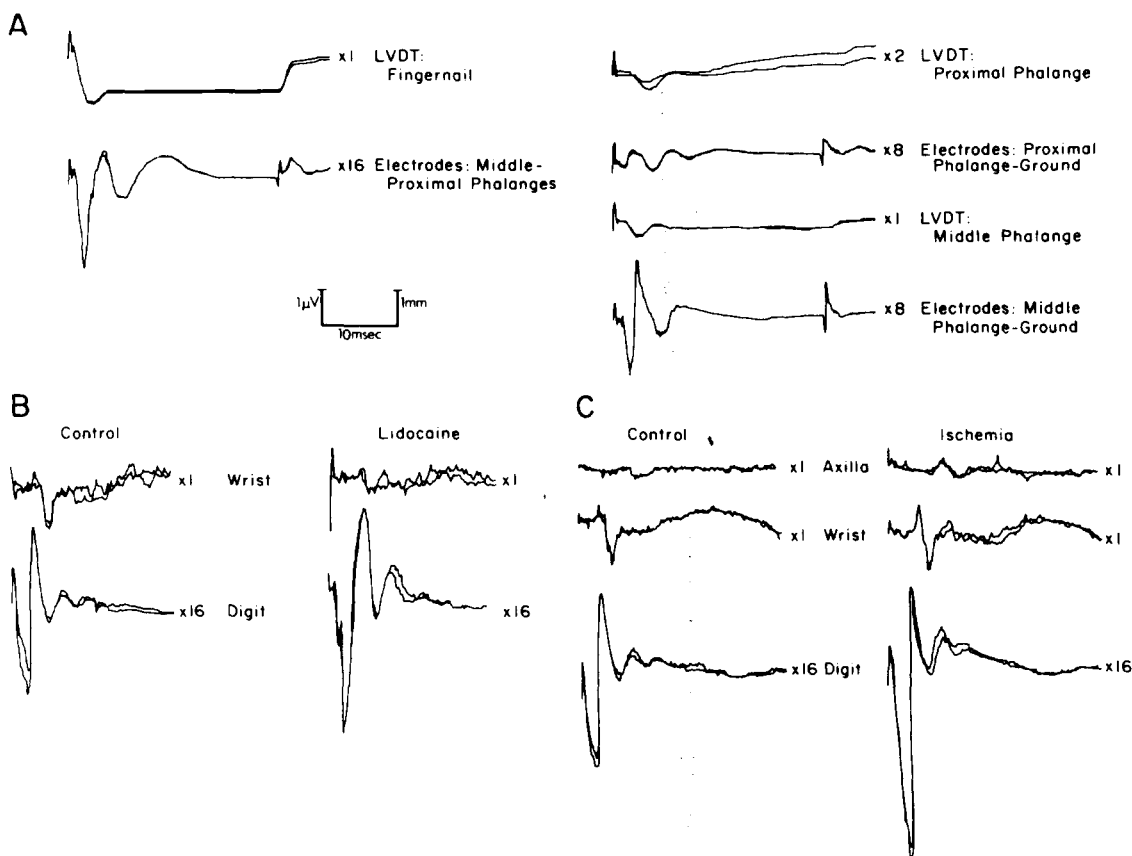


Fig. 1. Control experiments to assess the mechanically evoked potentials recorded by the digital electrodes. (A) Simultaneous recordings from electrodes and from an LVDT along the finger. Stimulus duration was 40 msec, producing an offset artifact in the electrode recordings. Note the identical onset and offset latencies, as well as the comparable wave forms of the mechanical displacements and the respective electric potentials recorded by the electrodes. (B) Recordings before (Control) and following (Lidocaine) nerve block at the distal phalange of the index finger. Note that although the potentials at the wrist were practically absent following the injection, the potentials recorded at the digit were actually increased in amplitude. (C) Recordings before (Control) and following 12 min of ischemia. Note that the latency of the wrist potential increased as a result of ischemia, as did the latency difference between the axillary and wrist potentials. In contrast, the latency of the potentials recorded from the digit remained unchanged, and their amplitude actually increased. Note the different display gains of the traces. The number next to each trace replaces the 1  $\mu$ V or 1 mm of the vertical calibration bar.

electrodes were 9 mm diameter silver cups attached to the skin by collodion glue and filled with conducting jelly. Electrode resistances were kept below 3 k $\Omega$ . Subjects were grounded by a metal plate attached to the dorsum of the left hand. Median nerve potentials at the wrist were recorded from two electrodes placed 3–4 cm apart and parallel to the nerve (the distal electrode channelled to grid 1 of the differential amplifier). Proximal median nerve potentials were recorded by an electrode located over the brachial artery near the axilla, referenced to an electrode placed over the insertion of the deltoid muscle. Brachial plexus activity was recorded by an electrode placed at Erb's point, which was referenced to the deltoid electrode. Upper neck activity was recorded from an electrode placed over the inion (Oz) referenced to an electrode placed over the second cervical vertebra (CII), or from CII referenced to an electrode over the middle of the forehead (Fpz). Somatosensory cortical activity was recorded from an electrode on the scalp at C4 (according to the 10-20 system) referenced to Fpz. An electrode configuration of C4-Erb's point was used as well.

The potentials were amplified with a gain of 200,000 using a band pass of 30–3,000 c/sec (3 dB down points). The digital potentials were recorded with a gain of 50,000. The potentials evoked in response to 1,000 stimuli were averaged over a 51 msec time period by a four-channel averager using a dwell time of 200  $\mu$ sec and 256 addresses per channel. A duplicate of each average was made to assess reproducibility. The averaged potentials were plotted by an X-Y plotter (positivity at grid 1, or negativity at grid 2, of the differential amplifier plotted as an upward deflection and labelled 'P'). The data were stored on magnetic tape for further analysis. Amplitudes and latencies of various components were determined from the computer CRT screen with a cursor. Latencies were measured from the onset of the electrical pulse delivered to the mechanical vibrator. Amplitudes were measured between positive and the following

negative peaks. Nerve conduction velocities between electrode placements along the median nerve were determined by measuring the distance between the initial electrode at each placement that recorded the propagated volley, and then dividing this distance by the latency difference between the negative peak of the potentials recorded at the two placements.

In one subject we determined the effects of ischemia and digital nerve block on the peripheral nerve potentials. Ischemia was produced by inflating a blood-pressure cuff around the upper arm above the systolic pressure for 12 min. The nerve blockade was produced by injecting 2 ml of 1% lidocaine around the distal phalange of the index finger.

## Results

Fig. 2 shows the potentials recorded from a subject with potentials of average amplitudes. The results will be described in terms of the potentials' presumed site of origin along the somatosensory pathway.

### *Median nerve*

The mechanically evoked potentials recorded by the digital electrodes (Fig. 1) were overwhelmed by an artifact of the movement interfering with the definition of neural events. This was indicated by (1) the potentials recorded from the digital electrodes corresponded in both wave form and latency to the movement of the finger itself (Fig. 1a), and (2) the mechanically evoked potentials recorded from the digit persisted, and were paradoxically even larger, following anesthetic blockade of the digital nerves (Fig. 1b) as well as during ischemia (Fig. 1c). If these potentials were of biological origin they should have diminished with ischemia and anesthesia. The increased amplitude probably represents changes in tissue impedance as a result of the techniques used to produce ischemia and nerve blockade. Thus, the

## MECHANICALLY EVOKED SOMATOSENSORY POTENTIALS

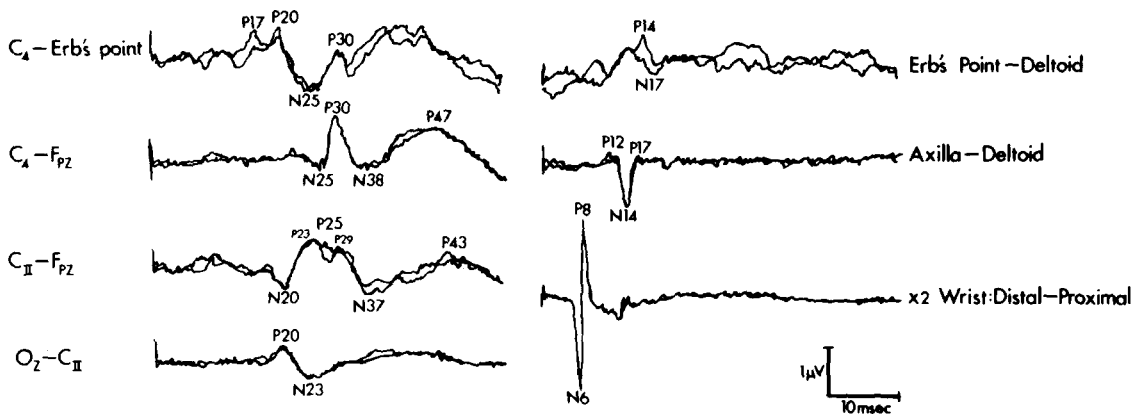


Fig. 2. Potentials recorded at various electrode configurations from a subject with potentials of average amplitudes. Note the different display gain of the potentials recorded at the wrist. The amplitude calibration bar would be  $2 \mu\text{V}$  for that trace. The components were labeled according to the convention of stating the polarity at grid 1 (P or N) followed by the component's average latency rounded to the nearest msec.

afferent activity evoked in the digital nerves by mechanical stimulation could not be distinguished by our recording techniques.

The electrodes overlying the median nerve at the wrist recorded (see Fig. 2) biphasic activity (N6-P8). This activity was not an artifact, as it diminished when the digital nerve was blocked (Fig. 1b). The relative amplitudes of the positive and negative components of this whole-nerve action potential were dependent on the relative proximity of the proximal and distal recording electrodes to the nerve trunk, making the measure of absolute amplitudes very variable.

The electrodes overlying the proximal portion of the median nerve near the axilla recorded activity that consisted of a major negative peak (N14) preceded and followed by smaller positive peaks (P12 and P17).

#### Brachial plexus

The potentials evoked at the brachial plexus by mechanical stimulation could not be identified clearly in the majority of the subjects, due to residual noise after averaging of 1,000 trials. When detected, the potentials

consisted of a positive-negative complex (P14-N17).

#### Upper neck

The potentials recorded over the neck at CII referenced to Fpz consisted of an initial negative peak (N20) followed by a positive peak (P25). This initial negative-positive complex reversed in direction when the Oz-CII configuration was used. In some subjects, P25 was actually double peaked (P23 and P29). P23 corresponded in latency to N23 recorded by the Oz-CII configuration, and P29 to P30 of the C4-Fpz recording.

#### Cortex

The cortical potentials recorded from the C4 electrode referenced to Fpz consisted of an initial negative component (N25), which was not clearly identifiable in all subjects, followed by two prominent positive peaks (P30 and P47). P30 and P47 were separated by a negative peak (N38). In the CII-Fpz recordings a negative-positive complex could be recorded (N37, P43) which was not identi-

fiable in the Oz-CII recordings, suggesting a cortical generator (see discussion).

### Far-field

The electrode configuration of C4-Erb's point has been used in clinical tests because of its ability to define a series of components, originating in structures from brachial plexus to cortex, in response to electrical stimulation of peripheral nerves. The potentials evoked by mechanical stimulation began with a component (P20) which occurred at the same latencies as the potentials recorded over the upper neck (compare with the Oz-CII recording). The subsequent complex was identical to the N25-P30 recorded using the C4-Fpz configuration. When only 1,000 trials were averaged, a poorly reproducible component (P17), corresponding in latency to the volley at Erb's point, was detected preceding P20.

The average values for amplitude and latency of various components labeled in Fig. 2 are listed in Table I.

The average value for conduction velocity along the wrist to axilla portion of the median nerves of the 23 subjects was found to be 56.55 m/sec with a standard deviation of 5.99. The effect of age on this conduction

TABLE I

Average values of latencies and amplitudes, with standard deviations, of some of the components of the mechanically evoked somatosensory potentials recorded at various locations as labeled in Fig. 2.

Recording site	Component	Latency (msec)	Amplitude ( $\mu$ V)
Wrist	N6	6.2 $\pm$ 0.7	1.5 $\pm$ 0.7
Upper-arm	N14	13.8 $\pm$ 1.4	0.7 $\pm$ 0.3
CII-Fpz	N20	20.4 $\pm$ 2.0	0.4 $\pm$ 0.2
	P25	25.4 $\pm$ 2.0	1.1 $\pm$ 0.3
	N37	36.6 $\pm$ 2.8	
	P43	42.8 $\pm$ 4.6	
C4-Fpz	P30	30.5 $\pm$ 3.4	1.3 $\pm$ 0.6
	N38	37.7 $\pm$ 3.8	
	P47	46.7 $\pm$ 2.2	

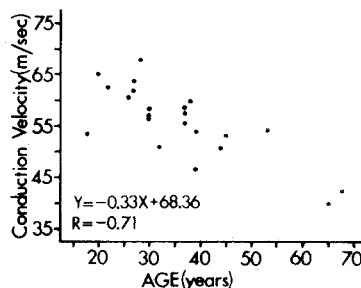


Fig. 3. Scatter diagram of nerve conduction velocity along the wrist to axilla portion of the median nerve, for each of the subjects, as a function of age. Regression constants and the correlation coefficient are also given.

velocity is demonstrated in the scatter diagram in Fig. 3, which also contains its regression constants and correlation coefficient. When a similar function of age was calculated for the digit to wrist portion, using the digital potentials, the slope was found to be 0.07 and the correlation coefficient was 0.18, indicating lack of effect.

### Discussion

The results of this study show that mechanically evoked potentials can be recorded along the somatosensory pathway. The potentials recorded from the electrodes overlying the digital nerve in response to mechanical stimulation could have provided objective evidence of the activity of receptors and terminal portions of the nerves. The failure of both ischemia and nerve blockade to attenuate these potentials, the lack of an effect of age on the conduction velocity along the distal peripheral nerve (as calculated using these potentials) coupled with the potentials' identical morphology to that of the finger displacement are strong evidence of their artifactual origin. Sears (1959) and McLeod (1966) probably attributed these mechanically evoked potentials to neural events because they did not have the benefit of transducers that recorded displacement of the finger adjacent to the electrodes and because of differ-

ent interpretations of control experiments. Nevertheless, we believe that the combination of evoked potentials recorded at the wrist in response to electrical stimulation of the digital nerves and mechanical stimulation of the finger could provide the information necessary for defining receptor and nerve-ending impairments in peripheral neuropathies.

The biphasic morphology of the potentials at the wrist is consistent with their being recorded by both electrodes of a differential recording pair placed along the nerve. The primarily negative morphology of the potentials recorded from the proximal peripheral nerve electrodes suggests that these potentials were primarily recorded by the axillary electrode and that the contribution from the deltoid electrode, which would give rise to an upward deflection, was overwhelmed by the downward deflection produced by the axillary potentials. The deltoid contribution was, however, evident in the recordings from Erb's point, where N17 was preceded by P14. The latency of P14 was identical to that of the proximal median nerve activity.

Both the inversion of direction of N20 recorded with the CII-Fpz electrode configuration, compared to P20 recorded using the Oz-CII configuration and the similar amplitude of P20 recorded by the C4-Erb's point configuration (Fig. 2), are consistent with this component being generated primarily at the upper cervical region and recorded as a far field potential. The double peaked morphology of P25 (P23 and P29) in some subjects, suggests that P25 has two distinct generators. The latency correspondence of P23 to the Oz-CII recorded N23 would suggest that its generator is in the upper cervical cord or medulla. The similar latencies of P29 and the C4-Fpz recorded P30 would indicate a cortical generator of P29, recorded by CII-Fpz. The latency of N20, relative to activity at the wrist (N6-P8), is comparable to that reported by others using electrical stimulation of nerves (Liberson and Kim 1963; Matthews et al. 1974; Jones 1977; Sances et al. 1978; Kritchevsky and Wiederholt 1978). These

authors localized the generator of this component to the upper spinal cord, possibly to the dorsal column nuclei. On the other hand, there has been a report that cortical mechanically evoked potentials were dependent on the ventrolateral tracts in the spinal cord (Nakanishi et al. 1974). Thus, additional evidence is necessary to determine conclusively the generator of these mechanically evoked potentials.

The positive peak in the potentials recorded with C4-Fpz (P30) corresponded in latency to the potentials recorded from the scalp overlying the somatosensory cortex as previously reported (Kjellman et al. 1967; Franzen and Offenloch 1969; Larsson and Prevec 1970; Nakanishi et al. 1973). The comparable latencies of N38 and P47, recorded by C4-Fpz, and of N37 and P43 recorded by CII-Fpz and their absence in the Oz-CII recordings, support the suggestion that this complex was also generated at the cortical level, most probably in the frontal region. Only careful scalp mapping correlated with recordings in patients with clearly demarcated lesions can resolve the generator sites for the various components of the evoked potentials.

The absence of clearly defined potentials at Erb's point in response to mechanical stimulation was due to a poor signal-to-noise ratio which resulted from the low amplitude of the neural signal evoked by mechanical stimulation coupled with high-amplitude myogenic noise. This explanation is supported by the better definition of mechanically evoked potentials recorded from Erb's point when the number of trials comprising the average was increased. The noisier recording obtained using the C4-Erb's point configuration, compared to recordings of the same components which avoided the brachial plexus electrode also supports this explanation.

Although slow adapting fibers may be activated by the prolonged mechanical stimulus used in this study, the potentials recorded were most probably initiated by fast adapting fibers. This was indicated by the single synchronized volley recorded over the

peripheral nerve, as well as by identical potentials recorded when the mechanical stimulus's duration was only 5 msec.

The nerve conduction velocity obtained by measuring peak latency differences reflects the modal velocity of conduction in the nerve trunk. The peripheral nerve conduction velocities computed in this study were comparable to previously reported values using electrical stimuli (Dawson and Scott 1949; Mayer 1963; Buchthal and Rosenfalck 1966; Liberson et al. 1966; Kemble and Peiris 1967). The decrease in peripheral nerve conduction velocity, as a function of age, that was found in this study is consistent with previously reported studies using electrical stimulation (Norris et al. 1953; Kemble 1967).

The nerve conduction velocities calculated indicate that the fibers generating the potentials were fast conducting nerve fibers. Although slower conducting fibers may also have been activated by the mechanical stimulus, the currents generated by them were probably too small to be recorded using surface electrodes.

Thus, we have shown that mechanical stimulation of the fingernail can evoke in humans a sequence of potentials from several levels of the sensory pathway. The potentials recorded were of comparable morphology to previously described electrically evoked somatosensory potentials. This recording method, which avoids some of the problems associated with electrical stimulation, may allow a detailed analysis of the function of the somatosensory pathway from receptor to cortex in clinical disorders leading to the definition of both the site and extent of deficits.

## Summary

Somatosensory potentials evoked by mechanical stimulation were recorded by surface electrodes over (1) the digital nerves in the index finger, (2) the median nerve at the wrist, (3) the median nerve near the axilla, (4) the brachial plexus, (5) the cervical cord at

CII, (6) the scalp overlying the somatosensory cortex. Nerve conduction velocities varied inversely with age and ranged from 43 to 68 m/sec. Mechanically evoked potentials recorded from the electrodes overlying the digital nerves were an artifact of the finger movement. All other electrode configurations recorded potentials comparable to those evoked by electrical stimulation of nerves. These mechanically evoked potentials could prove useful in the assessment of clinical disorders of somatosensory function from receptor to cortex in man.

## Résumé

### *Potentiels somato-sensitifs de courte latence évoqués mécaniquement chez l'homme*

Des potentiels somato-sensitifs évoqués par stimulations mécaniques ont été enregistrés par des électrodes de surface au niveau: (1) des nerfs digitaux de l'index; (2) du nerf médian au poignet; (3) du nerf médian près du creux axillaire; (4) du plexus brachial; (5) de la corde cervicale à CII; (6) du scalp au-dessus du cortex somato-sensitif. Les vitesses de conduction nerveuse varient de façon inverse avec l'âge et vont de 43 à 68 m/sec. Les potentiels évoqués de façon mécanique enregistrés au niveau d'électrodes qui recouvrent les nerfs digitaux constituent un artéfact du mouvement du doigt. Toutes les autres configurations d'électrodes enregistrent des potentiels comparables à ceux qui sont évoqués par stimulation électrique des nerfs. Ces potentiels évoqués de façon mécanique peuvent se révéler utiles dans l'appréciation, chez l'homme, de l'origine de troubles cliniques de la fonction somato-sensitive entre le récepteur et le cortex.

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