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Authors

Tapia, Maria C
Cohen, Leo G
Starr, Arnold

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Short communication

Selectivity of attenuation (i.e., gating) of somatosensory potentials during voluntary movement in humans

Maria C. Tapia, Leo G. Cohen and Arnold Starr

Department of Neurology, University of California Irvine Irvine, CA 92717 (U S A)

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Summary Attenuation of somatosensory evoked potentials (SEPS) recorded from the scalp during voluntary movement occurs for specific combinations of the finger moved and the peripheral nerve stimulated. The cerebral potential component occurring at a latency of 27 msec (P27) evoked either by stimulation of median nerve at the wrist or by stimulation of 1st and 2nd digit nerves in the fingers were selectively attenuated during movement of 1st digit but were not altered during movement of 5th digit. By contrast, the cerebral P27 component evoked by stimulation of ulnar nerve at the wrist or by stimulation of 5th digital nerve were attenuated during movement of that digit but were not altered during movement of 1st digit. Gating of somatosensory activity is a selective phenomenon occurring when movement involves the areas being stimulated.

Key words Somatosensory evoked potentials, Gating, Movement

Modification of central sensory processes during motor activity is referred to as 'gating' and has been described for somatosensory (Papakostopoulos et al 1975, Rushton et al 1981), auditory (Starr 1964, Hazemann et al 1975) and visual inputs (Volkman 1962, Adey and Noda 1973). In the somatosensory system 'gating' begins even before movement onset, during the precontraction period (Coulter 1974, Starr and Cohen 1985) implicating the action of central efferent systems in the modulation of sensory information. Asanuma (1981) suggested that activity of the pyramidal tract may influence the transmission of afferent impulses to higher centers including the motor cortex. However, details as to the relationship between the types of movement and the selectivity of afferent input modification are not known. This report utilizes scalp recordings of somatosensory evoked potentials in humans to demonstrate that somatosensory evoked potentials derived from stimulating a particular peripheral nerve are modified specifically for movements involving the body parts innervated by that particular nerve.

Methods

We studied somatosensory evoked potentials (SEPs) in 6 young, healthy, right-handed students, engaged in moving par-

ticular digits. Electrical stimuli were applied through surface electrodes to either mixed nerves at the wrist or to cutaneous nerves in the digits. The amplitudes of SEP to stimulation of digital nerves were smaller than to stimulation of mixed nerves at the wrist, requiring a larger number of trials in the former (up to 1500) to obtain reproducible averages. The averaged potentials evoked by these stimuli were recorded from surface electrodes placed over the peripheral nerve (PN) and the scalp in a quiet resting state or during movement. Peripheral nerve (PN) potentials to stimulation of the nerves at the wrist were recorded from a surface electrode over Erb's point referenced to (Fpz) during median and ulnar nerve stimulation. When stimulating digital nerves the peripheral nerve activity was recorded from two surface electrodes placed over the median or ulnar nerves at the wrist. Peripheral nerve potentials were monitored and did not change during the movements required in the experimental session. Somatosensory potentials were recorded from a scalp electrode located post-centrally 3 cm behind the vertex (Cz) and 7 cm from the midline contralateral to the limb stimulated, referenced to the ipsilateral earlobe (A1). The potentials were amplified with a gain of 500,000 using a bandpass of 30–3000 Hz (6 dB/octave slope). The amplitudes of the SEPs were measured between baseline and positive (P) or negative (N) peaks while peripheral nerve potentials (PN) were measured between baseline and the negative peak. The number of stimuli used to elicit SEPs varied from 150 to 500 for the different subjects and depended on achieving a 20% or less amplitude difference of the N20

Correspondence to Arnold Starr, MD, Department of Neurology, University of California Irvine, Irvine, CA 92717 (U S A)

component during repeated control measures in the same subject during the same session

The potentials were averaged over 51 msec using a dwell time of 0.1 msec. The averaged potentials were stored on the computer for further analysis.

Low intensity (10–25 dB sensation level) clicks produced by activating TDH-39 earphones with a 100 μ sec duration pulse at a rate of 0.7/sec were the signal to move. The mean latency of the EMG from the thenar muscles indicating the onset of the reaction time, measured in three of the subjects, was 193.8 ± 42.0 msec (223.6 ± 64.0 , 144.8 ± 36.7 and 213 ± 72.7 msec). All subjects performed with an accuracy of 100%. We did not record individual trials so that measures of the relationship between reaction time speeds and SEP amplitudes were not performed. Peripheral nerves were stimulated 250 msec after each click when movement had begun and the EMG was of high amplitude (Starr and Cohen 1985). The same rate of stimulation of the peripheral nerve was used in the control sessions when the subjects were instructed to be still. We studied the effects on SEP of two different movements mediated by muscles innervated by different peripheral nerves. First, opposition of the thumb (digit 1) towards digit 5 (primarily a median nerve innervated movement) and secondly, abduction of digit 5 (primarily an ulnar nerve innervated movement). The opposition of digit 1 is mainly mediated by the opponens pollicis muscle, innervated by the median nerve, while the abduction of digit 5 is mainly mediated by the abductor digiti minimi innervated by the ulnar nerve.

Four types of stimuli were used to evoke SEP during these movements: median nerve at the wrist (6 subjects), ulnar nerve at the wrist (5 subjects), digital nerves 1–2 (4 subjects), digital nerve 5 (4 subjects). These stimuli evoked a sequence of SEP components labeled by the convention of N or P (negative or positive) signifying their polarities on the scalp overlying the sensory receiving areas and their approximate latency in msec. The amplitudes of these components were measured and analysis of variance (ANOVA) was used to obtain overall *F* values for the different groups of control and movement conditions. 'Duncan's multiple range test for nearly equal *n*'s' was used to provide more detailed comparisons between the amplitudes of SEP recorded during control, digit 1 and digit 5 movement conditions.

Results

Electrical stimulation of the median and ulnar nerves at the wrist evoked similar latency SEP (Fig. 1). 'thalamocortical' N20 peaked at 18.4 ± 1.2 msec and 18.8 ± 1.2 msec respectively, cortical P27 peaked at 22.7 ± 1.8 msec and 22.4 ± 1.3 msec respectively (see Emerson and Pedley 1984 for a discussion of origins of these components). Electrical stimulation of digits 1–2 and 5 evoked the same sequence of components but at approximately 3 msec longer latencies, N20 peaked at 21.8 ± 0.8 msec and 21.7 ± 0.8 msec respectively, P27 peaked at 25.3 ± 1.7 msec and 25.2 ± 1.3 msec respectively. The peripheral nerve afferent volleys and N20 components to the different stimuli

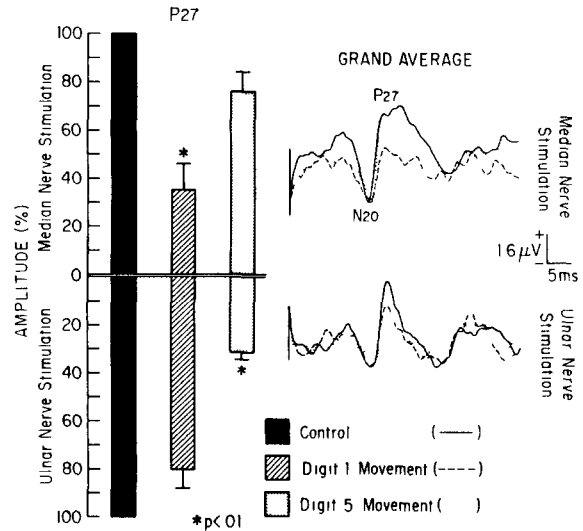


Fig. 1 To show the effects of digit 1 and 5 movement on the grand average SEP from 5 subjects to median (top half of figure) and ulnar (bottom half of figure) nerve stimulation. The thalamocortical N20 and cortical P27 components are labeled. Note that the P27 component of SEP to median nerve stimulation was attenuated during digit 1 movement (upper traces) whereas the P27 component of the SEP to ulnar nerve stimulation was most attenuated during digit 5 movement (lower traces). The peripheral nerve potentials evoked by these stimuli did not change during these movements. The bar graphs plot the attenuation of the P27 component during median nerve stimulation (upper portion) and ulnar nerve stimulation (lower portion) as a function of the finger that was moved.

did not significantly change during any movement condition (Tables I and II). In contrast, the P27 component was attenuated during certain combinations of movements and nerve stimulation. Opposition of the thumb towards digit 5 was accompanied by attenuation of potentials evoked by both median nerve and digit 1–2 stimulation (P27 diminished to $35.67 \pm 27.10\%$ and to $44.75 \pm 10.50\%$ respectively, $P < 0.01$, Fig. 1 top section, digit 1 movement, and Table I left-hand columns). In contrast, potentials evoked by both ulnar nerve and digit 5 stimulation were not significantly affected during this same digit 1 movement (Fig. 1 bottom section, Table I right-hand columns).

Abduction of the fifth finger was accompanied by attenuation of potentials evoked by both ulnar nerve and digit 5 stimulation (P27 diminished to $31.80 \pm 7.29\%$ and to $37.00 \pm 12.36\%$ respectively, $P < 0.01$, Fig. 1 bottom section, digit 5 movement, and Table II right-hand columns). In contrast, potentials evoked by both median nerve and digits 1–2 stimulation were not significantly affected during this fifth digit movement (Fig. 1 top section and Table II left columns).

Attenuation of SEP is not due to changes in subject vigi-

TABLE I

Effects of thumb opposition (digit 1 movement) on the amplitude of SEP *

Subjects	Stimulus											
	Median nerve			Digits 1 and 2			Ulnar nerve			Digit 5		
	Nerve	N20	P27	Nerve	N20	P27	Nerve	N20	P27	Nerve	N20	P27
1	90	86	65	105	58	57	120	58	57	92	31	108
2	93	89	18	92	79	36	153	111	83	124	69	87
3	101	67	37	112	110	36	80	94	94	94	67	72
4	-	101	69	105	76	50	-	135	68	-	100	75
5	-	54	0	-	-	-	-	100	100	-	-	-
6	-	77	25	-	-	-	-	-	-	-	-	-
X	94.67	79.00	35.67	103.50	80.75	44.75	117.67	99.60	80.40	103.33	66.75	85.50
S D	5.69	16.77	27.10	8.35	21.59	10.50	26.59	28.04	17.87	17.93	28.22	16.34
P <	NS	NS	0.01	NS	NS	0.01	NS	NS	NS	NS	NS	NS

- = not recorded NS = not significant

* Amplitudes of cerebral SEP recorded during movement are expressed as percentage of control in the same subject and session

TABLE II

Effects of small finger abduction (digit 5 movement) on the amplitude of SEP *

Subjects	Stimulus											
	Median nerve			Digits 1 and 2			Ulnar nerve			Digit 5		
	Nerve	N20	P27	Nerve	N20	P27	Nerve	N20	P27	Nerve	N20	P27
1	88	70	75	88	82	73	107	66	31	100	55	35
2	99	89	45	115	86	93	92	83	41	100	109	30
3	112	114	81	89	152	87	114	75	31	138	119	28
4	-	100	107	89	92	57	122	85	21	-	100	55
5	-	109	72	-	-	-	-	111	35	-	-	-
6	-	96	78	-	-	-	-	-	-	-	-	-
X	99.67	85.83	76.33	95.25	106.50	75.75	108.25	84.00	31.80	112.67	95.75	37.00
S D	12.69	39.65	19.84	13.18	31.21	14.03	12.50	16.85	7.29	21.94	28.25	12.36
P <	NS	NS	NS	NS	NS	NS	NS	NS	0.01	NS	NS	0.01

- = not recorded NS = not significant

* Amplitudes of cerebral SEP recorded during movement are expressed as percentage of control in the same subject and session

lance and attention since the alterations in SEP were specific only for certain combinations of nerve stimulation and movement conditions. Moreover, the earliest task-related change in SEP that has been reported affects those components with latencies longer than 55 msec (Desmedt and Robertson 1977).

Discussion

It has been amply demonstrated that there is an attenuation of SEP during movement of the stimulated limb in both animals and humans (Ghez and Pisa 1972, Papakostopoulos et al 1975, Rushton et al 1981, Cohen and Starr 1987). Moreover, Rushton et al (1981) showed in humans that SEPs to

stimulation of the digital nerve of the thumb were attenuated more with movements of that thumb than with movements of the index and little fingers. Our results extend these observations by showing that attenuation of SEPs during movement can be extremely selective, affecting only certain combinations of neural input and movements of particular portions of the same limb in a doubly dissociated manner. Attenuation of a median or 1st digital nerve evoked potential accompanied movement of the thumb (digit 1), but not the little finger (digit 5), whereas attenuation of an ulnar or 5th digital nerve evoked potential accompanied movement of the little finger but not the thumb. The attenuation of SEP during such focal voluntary movements must be at a cortical level since both the thalamo-cortical N20 and the peripheral nerve components were un-

changed, whereas the post-central cortical P27 component became attenuated. The attenuation was equally evident with cutaneous (digital nerve) as well as Ia afferent inputs (mixed nerve at the wrist), even though the Ia inputs project to cerebral somatosensory area 3a, whereas cutaneous inputs project to areas 1 and 3b (Phillips et al 1971).

The motor representation of the digits is large (Penfield and Rasmussen 1950) with extensive interconnections to other cortical regions (Jones et al 1978). Studies of the hand area of monkey motor cortex indicate that it receives afferent input from superficial as well as from deep receptors located in restricted parts of the distal forelimb (Rosén and Asanuma 1972). The columnar arrangement of the afferent inputs coincides with the columnar organization of efferent zones with each efferent column receiving sensory information from that portion of the limb to which it projects (Rosén and Asanuma 1972, Strick and Preston 1982). In this regard, Lemon (1979) demonstrated that the pyramidal neurons in motor cortex (area 4) have very restricted zones of sensory input, many were influenced only by movement of a single joint of the finger and only in one direction.

Thus the specificity of sensorimotor interconnections in motor cortex may account, in part, for the selectivity of attenuation of SEP demonstrated in this report. We can propose several mechanisms underlying this selective attenuation of SEPs. First, cortical motor neurons involved in voluntary movement of particular digits may contribute to the generation of the SEP. During movement of particular digits these neurons may be unable to respond to stimulation of inputs from this digit leading to a reduction in amplitude of the SEP. Secondly, motor neurons in the cerebral cortex may suppress activity in other cortical neurons responsible for the SEP (a form of cortico-cortical inhibition) requiring, in addition, a specificity of such inhibitory connections to only certain of these neurons. Finally, pyramidal tract neurons may modulate at subcortical sites the transmission of ascending somatosensory information (Ghez and PISA 1972). However, the SEP data in humans indicate that the subcortical transmission of afferent input is unchanged during movements (Cohen and Starr 1987).

The definition of specific topographical effects of motor activity on sensory processes as defined in this report may have a bearing on the study of 'plasticity' of sensory and motor functions in humans following lesions of both peripheral and central pathways. For instance, Merzenich et al (1984) have shown that the distribution of afferents from the digits in the cerebral cortex in monkey changes as a function of amputation of individual digits. The measurement of somatosensory evoked potentials in man both at rest and during movement, following naturally occurring damage to peripheral nerves, may provide comparative evidence of such changes in human cerebral cortex.

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