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LOCALIZATION, TIMING AND SPECIFICITY OF GATING OF SOMATOSENSORY EVOKED POTENTIALS DURING ACTIVE MOVEMENT IN MAN

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SUMMARY

Short latency somatosensory evoked potentials (SEPs) to median nerve stimulation were recorded at different times simultaneously and after an order to move the ipsilateral thumb (0, 100, 200, 300, 400 and 780 ms). Brain potentials were derived from surface electrodes over the scalp at contralateral postcentral and precentral sites and muscle potentials were derived from surface electrodes over the thenar eminence. Ipsilateral thumb movement did not affect the early lemniscal P14 or the postcentral cortical N20 but was accompanied by attenuation of the cortical postcentral P27 and cortical precentral P22, N30 and P45. These results suggest that the gating induced by voluntary movement in man occurs at thalamocortical level and that the differential effect on postcentral N20 and precentral P22 may represent the activity of different generators.

Gating of SEPs during voluntary movement begins approximately 80-100 ms before EMG onset, is maximal at the time the EMG is maximal, and returns to control values after the EMG is concluded. These results suggest that the gating that precedes the onset of EMG activity is related to premotor events in the cerebral cortex and not to centripetal influences. Gating of SEPs to median nerve stimulation was localized to movements of areas innervated by the median nerve, that is, the ipsilateral thumb, index or middle finger, but not to movements of ipsilateral little finger or contralateral thumb.

INTRODUCTION

Several studies have shown an attenuation of somatosensory input to the cerebral cortex during voluntary movement (Coquery *et al.*, 1972; Lee and White, 1974; Papakostopoulos *et al.*, 1975; Jones, 1981; Rushton *et al.*, 1981). This phenomenon, termed gating, has been discussed with respect to its possible central or peripheral origins (Ghez and Pisa, 1972; Coulter, 1974; Rushton *et al.*, 1981; Angel *et al.*, 1984). Animal experiments suggest that the locus of this gating is within the central nervous system (CNS) at subcortical sites (Coquery *et al.*, 1972; Ghez and Pisa, 1972; Coulter, 1974), but the site of gating of human afferent somatosensory volleys during voluntary movement is not known.

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There are several electrophysiological events that occur in the cerebral cortex before the initiation of a voluntary movement. The contingent negative variation (CNV) appears during the period between a warning signal and the subsequent order to move (Walter *et al.*, 1964; Tecce, 1972). The Bereitschaftspotential is a slow negative wave occurring within one second before self-paced movements (Kornhuber and Deecke, 1965; Vaughan *et al.*, 1968; Shibasaki *et al.*, 1980). Finally, there is an increase in the firing rate of neurons in the motor cortex during the preparatory and precontraction periods of the movement (Schmidt *et al.*, 1974; Tanji and Evarts, 1976; Kubota and Hamada, 1979). The occurrence of these electrophysiological events suggest the possibility that gating of somatosensory input is initiated before the onset of movement as occurs in the visual system when visual thresholds are affected prior to the initiation of saccades (Latour, 1962; Zuber and Stark, 1966; Starr *et al.*, 1969). Contradictory results were reported in two papers regarding the modification of somatosensory afferent volleys prior to movement in humans. Coquery *et al.* (1972) found an increase in somatosensory evoked potentials (SEPs) before movement while Hazemann *et al.* (1975) reported a significant attenuation of SEPs beginning 440 ms before a voluntary movement. The results to be presented in this paper show that gating of somatosensory input in humans occurs *before* the initiation of a voluntary movement with its locus of action occurring in the CNS at thalamic and/or cortical levels. Moreover, the gating of sensory input is particular for movements of portions of the body from which the somatosensory input originates.

METHODS

Subjects

Recordings were obtained from 10 healthy subjects (5 men and 5 women), aged 15 to 32 years. They were tested in a sound attenuating chamber while seated comfortably. The left hand and forearm were attached comfortably to the arm of the chair allowing free movement of the fingers.

Stimulation Procedures

Percutaneous electric stimulation with bipolar electrodes was delivered to the left median nerve at the wrist. The intensity of the stimuli was adjusted to elicit a just visible twitch of the thenar muscles without causing pain. The number of stimuli delivered during control and movement condition was that necessary to evoke short latency somatosensory cerebral components (< 50 ms) resulting in a 20% or less amplitude difference on repeated control measures in the same subject and at the same session. This varied among subjects from 400 to 1400 stimuli depending on the relationship of the evoked potentials to the background EEG, etc. (i.e., signal/noise ratio). Clicks were presented through earphones as the order to move. The clicks were generated by passing 100 μ s duration pulses through the earphones at an intensity that was low (from 10 to 25 dB SL) but still clearly detectable.

Recording Procedures

SEPs were recorded with surface electrodes placed unilaterally or bilaterally at all or some of the following positions (fig. 1): (1) a postcentral electrode 3 cm behind the vertex Cz and 7 cm from the midline; (2) a precentral electrode 2 cm in front of Cz and 5 cm from the midline; (3) a frontal electrode 7 cm in front of Cz and 5 cm from the midline; and (4) a prefrontal electrode 9 cm in front of Cz and

FIG. 1. Scalp distribution of left median nerve SEPs. The components are labelled by their polarity and their approximate latencies (P14, N20, P22, N30 and P45). Vertical lines have been placed at the peaks of the subcortical P14, postcentral N20 and precentral P22 components. The scale over the scalp refers to the interelectrode distances. An asterisk (*) is placed before the averages obtained from precentral recording sites. Reference electrode at A2.

6 cm from the midline. Additional electrodes were placed 2 cm lateral to postcentral, precentral and frontal locations. In every case the reference was the earlobe ipsilateral to the recording electrodes, chosen to differentiate more effectively the postcentral and precentral components of median nerve SEP (N20 and P22) (Desmedt and Cheron, 1981; Mauguière *et al.*, 1983).

Electrode impedances were below 5 k Ω . The potentials were amplified with a gain of 500,000 using a bandpass of 30–3000 Hz (6 dB/octave slope) and averaged over a 51 ms time period with a dwell time of 0.1 ms and 512 points per channel. The EMG was recorded by a pair of surface electrodes placed over the thenar muscles and the resulting activity amplified and full wave rectified. EMG onset was signalled by a Schmitt trigger that was capable of triggering the averager when so desired. Movement related evoked potentials accompanying thumb movement were recorded by backaveraging the EEG (bandpass of 0.1–3000 Hz) time-locked with the EMG onset.

In 3 subjects we recorded simultaneously EEG activity at bilateral postcentral and precentral locations, EMG activity from the thenar eminence, and trigger signals from the clicks (order to move) and electric pulses (median nerve stimulation) on magnetic tape. Off-line analysis allowed us to analyse the trial by trial latencies of EMG from the thenar eminence, short-latency SEPs to median nerve stimulation, and movement-related EPs.

Experimental Paradigms

Effects of moving the thumb ipsilateral to median nerve stimulation. Pairs of click-median nerve stimuli separated by 250 ms were regularly delivered at 0.6/s in 10 subjects. They were instructed to respond to the clicks by flexing the thumb as quickly as possible after the click or, as a control, to relax. Control recordings were interspersed with those obtained during the different movement conditions. The interval of 250 ms was chosen to ensure that motor activity was already present when somatosensory stimuli were applied. Consistency of movements along each session was stimulated by the promise of financial reward and ensured by the experimenter's visual control of the behaviour.

Effects of varying time intervals between click and median nerve stimulation. Three of the subjects were also tested when median nerve stimulation was delivered simultaneously with the clicks (0 condition) or at 100, 200, 300, 400 and 780 ms after the clicks to encompass the precontraction, contraction and postcontraction periods. The order in which the intervals were selected was randomized. Subjects were instructed to respond to the clicks by flexing the thumb as in the first condition (Starr and Cohen, 1985).

Effects of moving different fingers. Five subjects were tested with an interval of 250 ms between clicks (order to move) and median nerve stimulation as in the first paradigm. They were instructed to respond to the clicks by flexing thumb, index, middle, little finger or the contralateral thumb. When asked to move the little finger, 3 of the subjects moved the little and ring fingers together.

Data Analysis

Sensory and movement related evoked potentials were analysed off-line. Amplitude and latencies of the various components of the recorded potentials were measured from a computer display with a cursor. SEP latencies to median nerve stimulation were measured from the onset of the electric shocks activating the median nerve. As the peak latencies of the SEP components showed small differences between averages we systematized the measurements of amplitude by choosing the maximal peak within a 5 ms range (± 2.5 ms) of the latencies of the control peaks. Absolute amplitudes of the SEP were measured between baseline and positive or negative peaks. Baseline was established by the computer defining the mean value across the entire waveform. This value was then subtracted from the peaks and troughs comprising the components. The amplitudes of SEPs recorded during movement are expressed as a percentage of controls recorded during the relaxed conditions in the same subject and session. We found that the clicks evoked inconstant evoked potentials because of their low intensity level (10–25 dB SL) and thus the amplitude of median nerve SEPs preceded by clicks did not differ from those without clicks if subjects were not moving.

Movement-related evoked potentials to thumb movement were back averaged from EMG onset obtained from the thenar eminence.

Statistical Analysis

Repeated measures analysis of variance (ANOVA) was used to obtain overall F values for the different groups of control and movement conditions. When justified by a significant overall F ($P < 0.05$), Duncan's multiple range test for nearly equal numbers was used to provide more detailed comparisons between the attenuation of different components of median nerve SEPs, the effects of different finger movement and the different time intervals between clicks and shocks. The relationship between attenuation of median nerve SEPs and time intervals between click and shocks was evaluated by the Pearson r correlation coefficient.

RESULTS

Electric stimulation of the median nerve evoked reproducible cerebral potentials with configurations that differed according to the location of the recording electrodes (fig. 1). A major difference was found between postcentral and precentral electrodes.

At the postcentral sites (postcentral and posterior temporal in fig. 1) there was an initial positive component peaking at 13.5 ± 1.0 ms (P14), followed by negative and positive components at 18.0 ± 1.0 ms (N20) and 23.5 ± 2.7 ms (P27), respectively (Table 1, fig. 1).

TABLE 1. COMPONENTS OF MEDIAN NERVE SEPs*

Subjects	Postcentral			Precentral			
	P14	N20	P27	P14	P22	N30	P45
1	12.4	18.5	24.5	12.6	19.6	26.0	37.0
2	13.5	19.3	24.0	13.8	20.5	26.2	33.2
3	14.8	18.4	22.0	13.2	19.3	22.3	37.2
4	12.7	17.1	21.2	12.8	18.4	21.4	35.8
5	12.5	18.6	22.0	12.7	18.8	25.0	40.0
6	13.0	19.0	29.1	13.1	20.0	28.9	40.0
7	14.1	17.7	23.2	14.1	18.8	22.6	40.0
8	13.0	17.2	26.8	13.2	18.6	27.7	34.7
9	15.3	18.7	22.0	15.2	20.3	25.2	38.6
10	14.0	16.0	20.0	13.8	18.0	27.4	36.0
Mean	13.5	18.0	23.5	13.4	19.2	25.3	37.2
SD	1.0	1.0	2.7	0.8	0.8	2.5	2.4

* Latencies of cerebral potentials to median nerve stimulation (ms).

The P14, N20 and P27 components were present in each of the 10 subjects tested. After P27 only 4 of the 10 subjects showed subsequent negative and positive components (N, P) which we chose not to include in this study. At the precentral sites (the traces with an asterisk, i.e., prefrontal, frontal, precentral, anterior and middle temporal in fig. 1) the only similarity to the postcentral sites was an initial positive component peaking at 13.4 ± 0.8 ms (P14). The subsequent positive, negative and positive components at 19.2 ± 0.8 ms (P22), 25.3 ± 2.5 ms (N30) and 37.2 ± 2.4 ms (P45), respectively, were particular to the precentral sites (Table 1, fig. 1). The P14, N30 and P45 precentral components were present in each of the 10 subjects tested. However, there was some variability in the P22 in all subjects (very clear in subjects 1, 2, 3, 4, 5, 6, 7 and 10, less well defined in subjects 8 and 9 of fig. 2). The grand average of the 10 subjects also shows a very clear P22 (fig. 2B).

Effects of Thumb Movement on Median Nerve Somatosensory Afferent Volley

Ipsilateral thumb movement substantially diminished the postcentral P27 in all 10 subjects tested to $53.8 \pm 20.3\%$ ($P < 0.01$). In contrast, the P14 was variously affected, being smaller in 3 subjects (nos 6, 7, 9) and larger in 5 (2, 3, 4, 8, 10). The N20 was also variously affected, being smaller in 4 (2, 5, 6, 8) and larger in 3 (1, 9, 10). Thus the grand average of the P14 and N20 components showed no change with

FIG. 2. Effects of thumb movement on ipsilateral median nerve SEPs at postcentral and precentral positions. A, individual averages of the 10 subjects; B, grand average of these 10 subjects. In this and all subsequent figures the thin lines represent the control averages while the thick lines represent the averages obtained during movement. The amplitude changes of the different components accompanying movement are plotted in C for the postcentral site and D for the precentral site. Amplitude values are expressed as percentages of each component compared with control values (means $\pm$ SEM). In the text, reference is made to each subject by numbering in sequence from the topmost trace. Reference electrode on ipsilateral earlobe.

movement ($114.0 \pm 49.0\%$ and $103.2 \pm 29.0\%$, respectively, n.s.) (see Table 2 and bottom row of traces in fig. 2).

Thumb movement was also associated with a reduction in the precentral P22, N30 and P45 in the 10 subjects tested (to $19.5 \pm 26.3\%$, $29.0 \pm 18.9\%$ and $53.8 \pm 18.1\%$, respectively, $P < 0.01$) while the P14 was variously affected with the grand average showing no change ($107.0 \pm 55.4\%$, n.s.) (see Table 2 and fig. 2). The attenuation observed in contralateral postcentral P27 and precentral P22, N30 and P45 was also significant when compared with the amplitudes of the postcentral and precentral

P14 ($P < 0.01$ for all components). The peripheral afferent input was unchanged during the different experimental conditions. This is supported by the lack of changes in subcortical P14 and cortical N20 in the postcentral position and P14 in the precentral position. Moreover, in subsequent experiments (Tapia *et al.*, 1985, 1986), we also measured the amplitude of peripheral nerve evoked potentials at Erb's point and these also did not change during movement compared with control conditions. The effects of movement on attenuating median nerve somatosensory evoked potentials were not limited to the contralateral scalp since the precentral N30 was also attenuated in the precentral area *ipsilateral* to the limb being moved in the 3 subjects tested (see fig. 3).

TABLE 2. EFFECTS OF THUMB MOVEMENT ON THE AMPLITUDE OF SEPs*

Subjects	Postcentral			Precentral			
	P14	N20	P27	P14	P22	N30	P45
1	100	150	61	250	4	27	47
2	132	88	69	100	14	17	83
3	150	100	59	117	27	64	50
4	150	102	58	65	43	61	61
5	103	86	64	104	0**	18	54
6	86	67	43	115	0**	6	32
7	70	100	38	102	26	25	83
8	200	72	31	101	81	29	44
9	24	112	92	44	0**	26	30
10	125	155	23	72	0**	17	54
Mean	114.0	103.2	53.8	107.0	19.5	29.0	53.8
SD	49.0	29.0	20.3	55.4	26.3	18.9	18.1
P	n.s.	n.s.	< 0.01	n.s.	< 0.01	< 0.01	< 0.01

* Amplitudes of cerebral potentials to median nerve stimulation expressed as percentage of control values (100%). ** The component could not be distinguished from baseline. n.s. = not significant.

We attempted to assess the physiological significance of the attenuation accompanying movement by relating it to the change in amplitude of SEP with changes in stimulus intensity. The extent of the attenuation of the SEP components that accompanied movement (between 30 and 60%) is equivalent to the attenuation that accompanies a decrease in the intensity of median nerve stimulation from one third to two thirds of the motor threshold. However, such a decrease in stimulus intensity was accompanied by attenuation of *all* subcortical and cortical components, whereas attenuation during movement was component-specific (compare figs 2 and 4).

To control for the effects of the regularity of stimulus presentations on reaction times and SEP changes we tested 2 subjects at both the regular rate of 0.6/s and at an irregular rate of approximately 0.3/s. This allowed us to analyse the EMG onset on a

trial by trial basis and define the contribution of anticipation in regular versus irregular presentations since such differences in stimulus rate, per se, do not modify reaction times greatly (Klemmer, 1967; Niemi and Näätänen, 1981). EMG onset occurred on the average 62.5 ms earlier for regular presentations (latencies in the

FIG. 3. Effects of right thumb movement on SEPs to bilateral median nerve stimulation at the wrist. Note that N20 and P27 and P22 and P45 are attenuated only over the left scalp, whereas N30 is attenuated bilaterally. The P14 is also unaffected bilaterally. Reference electrode on ipsilateral earlobe.

FIG. 4. Effects of median nerve stimulus intensity on the amplitude of the postcentral P27. The stimulus necessary to evoke a slight thenar twitch in this subject was 9 mA (100%); 3 mA (33%) represented the sensory threshold, and 6 mA (66%) was twice sensory threshold.

FIG. 5. Scalp distribution over the right scalp of the effects of left thumb movement on SEPs to left median nerve stimulation at the wrist. Note that postcentral P27 is slightly attenuated at both postcentral and posterior temporal sites and precentral P22, N30 and P45 are attenuated at prefrontal, frontal, anterior temporal and middle temporal sites. The P14 and N20 are also unaffected.

2 subjects were 213 ± 72 and 144 ± 36 ms) than for irregular presentations (latencies in the 2 subjects were 276 ± 58 and 206 ± 85 ms) but the attenuation of SEP during movement was comparable at the two presentation rates. The variability of the reaction times observed in these experiments might be a factor in 'smoothing' the curves of amplitude attenuations as a function of the click-shock intervals.

The scalp distribution of the median nerve SEPs during control and movement conditions is shown in fig. 5 in 1 subject. Postcentral P27 and precentral P22, N30 and P45 were attenuated at the different electrode sites while the P14 and N20 were not affected. The latencies of the different components of the median nerve SEPs did not change during these experiments.

FIG. 6. Effects on SEPs of stimulating median nerve simultaneously and at different times after the command to move the thumb for 3 subjects. The abscissa refers to the time interval in ms between the command to move (the click) and the shock to the median nerve. The evoked potentials from one of the subjects is shown in A and the amplitudes of P27 from the 3 subjects are shown in B. The time course of the rectified EMG from 1 subject is shown at the bottom of B with the EMG onset adjusted to the mean latency of EMG onset in the 3 subjects. Note that P27 is attenuated at 100 ms, before the occurrence of muscle activity, and remained attenuated until 400 ms, returning to control values at 780 ms when EMG activity was completed.

These results indicate that the effects of thumb movement on diminishing the amplitude of median nerve sensory volleys recorded over the scalp occur central to the generation of P14 and N20.

Effects of Varying Time Intervals between Click and Shocks

In 3 subjects, median nerve stimulation was delivered at 100, 200, 300, 400 and 780 ms after the clicks. The results are depicted graphically together with one of the subject's evoked potentials and rectified EMG in fig. 6. There was an attenuation of the P27 component *before* the initiation of the EMG. At a latency of 100 ms after the command to move (click) during the precontraction period the P27 diminished to $61.3 \pm 19.2\%$ ($P < 0.01$). At 200, 300 and 400 ms, during the contraction period, the

P27 component diminished to $47.3 \pm 7.0\%$ ($P < 0.01$), $44.0 \pm 17.1\%$ ($P < 0.01$) and to $52.3 \pm 25.8\%$ ($P < 0.01$), respectively. Finally, at 780 ms during the postcontraction period the P27 component returned to control values to $107.5 \pm 2.5\%$ (n.s.).

Movement-related Potentials

In an attempt to differentiate the median nerve SEPs from thumb movement-related evoked potentials we back averaged from EMG onset and recorded from F3 and P3 referenced to the ipsilateral ear when only the command to move (the click) was presented without an associated median nerve stimulus. We were unable to define a consistent movement-related evoked potential using this montage and paradigm making it unlikely that the algebraic summation of such movement-related potentials with the SEPs were responsible for the SEP amplitude attenuation.

TABLE 3. EFFECTS OF THE MOVEMENT OF DIFFERENT FINGERS ON SEPs*

Subjects	Ipsilateral				Contralateral
	Thumb	Index	Middle	Little	Thumb
1	37	50	91	64	82
2	94	55	55	76	132
3	12	5	12	48	90
4	68	21	66	103	95
5	16	52	24	72	80
Mean	45.4	36.6	49.6	72.6	95.8
SD	35.1	22.3	31.9	20.1	21.1
P	< 0.01	< 0.01	< 0.01	n.s.	n.s.

* Amplitudes of cerebral potentials to median nerve stimulation expressed as percentage of control values (100%).

Effects of Moving Different Fingers

The attenuation of median nerve somatosensory afferent input that accompanied movement was localized to the hand stimulated. In 5 subjects the postcentral component P27 diminished to $45.4 \pm 35.1\%$ ($P < 0.01$) during ipsilateral thumb movement but was not affected by movements of the contralateral thumb ($95.8 \pm 21.1\%$, n.s.) (fig. 3). Moreover, the attenuation of P27 was not similar for all ipsilateral finger movements. P27 diminished to $36.6 \pm 22.3\%$ ($P < 0.01$) during ipsilateral index finger movement, to $49.6 \pm 31.9\%$ ($P < 0.01$) during ipsilateral middle finger movement and to $72.6 \pm 20.1\%$ (n.s.) during ipsilateral little finger movement (fig. 7, Table 3).

In 1 subject we stimulated simultaneously both median nerves and recorded bilaterally (fig. 3) while the subject was moving the right thumb. The attenuation of postcentral N20 and P27 and precentral P22 is clearly localized to the hemisphere contralateral to the hand being moved, whereas N30 in precentral sites was

FIG. 7. Effects of moving different digits on median nerve SEPs. Results from a representative subject are shown in A and the mean of the amplitude (+ SEM) of P27 in 5 subjects in B. Note that attenuation of P27 occurred during movement of those digits innervated by the stimulated median nerve (i.e., thumb, index and middle fingers) whereas contralateral thumb or ipsilateral little finger movements were without effect.

attenuated bilaterally during this task. These experiments showed that the gating of afferent somatosensory input arising from stimulation of the median nerve accompanied movements of those structures innervated by that nerve.

DISCUSSION

Locus of the Gating

The results of this study in humans using scalp recording techniques show that gating of somatosensory input evoked by stimulating the median nerve occurs central to the generation of the scalp derived P14 and N20 components. P14 is preserved in patients with thalamic lesions (Mauguière and Courjon, 1981) and most probably originates from the medial lemniscus with its onset corresponding to activation of caudal lemniscal fibres and its peak representing arrival of the lemniscal

signal at the thalamus (Desmedt and Cheron, 1981). The subcortical P14 component was recorded at several different electrode positions and did not change during thumb movement, showing no evidence for gating of somatosensory input below cortical level. We also know from other control studies that the peripheral nerve volley recorded over Erb's point was also unaffected by the movement (Tapia, Cohen and Starr, unpublished observations). The postcentral N20 was unchanged during thumb movement whereas the precentral P22 was attenuated. Since N20 is generated most probably in the primary somatosensory cortex (Emerson and Pedley, 1984) while P22 is generated in the frontal cortex (Desmedt and Cheron, 1981; Mauguière *et al.*, 1983), there is a differential gating effect of movement on cortical somatosensory processes. We have considered two explanations to account for this phenomenon. (1) N20 and P22 may be generated by separate thalamocortical pathways to parietal and frontal lobes, respectively, as proposed by Desmedt and Cheron (1981) and Papakostopoulos and Crow (1984). A gating of the P22 component could occur at thalamic or cortical level without affecting the separate thalamocortical pathway responsible for N20 (Asanuma, 1981; Evarts, 1981; Jones, 1983). The subsequent attenuation of the postcentral P27 could result from corticocortical inhibitory connections. (2) Alternatively, P22 may be generated serially after N20 through corticocortical connections between parietal areas 2 and 5 and frontal areas 4 and 6 (Asanuma, 1981). If this were the case, gating would occur in the cerebral cortex somewhere between the generators of N20 and P22 but affecting selectively P22. In patients with lesions in the postcentral cortex, changes in N20 occur without modification of P22, giving less credence to this last possibility (Mauguière *et al.*, 1983).

If the locus of the gating involves the cerebral cortex as our results strongly suggest, two mechanisms may be responsible for the attenuation of SEPs. (1) Cortical motor neurons involved in thumb movement also take part in the generation of the cortical SEPs. Thus during movement, those neurons controlling thumb movement are unable to react to median nerve stimulation, resulting in a smaller amplitude SEP. This mechanism could also account for the selective attenuation of the precentral P22 which reflects activity of motor neurons without influencing the postcentral N20 originating from sensory neurons. (2) Cortical motor neurons involved in producing the requested movement might suppress other cortical neurons involved in the generation of the SEP, a form of corticocortical inhibition (Porter, 1981).

Timing of the Gating

The gating of somatosensory evoked potentials associated with movement begins before the initiation of EMG activity. In agreement with these results, sensory thresholds are elevated *before and during* voluntary movement in the stimulated limb (Papakostopoulos *et al.*, 1975; Coquery, 1978; Angel *et al.*, 1984).

There are several indices of cerebral cortical activity that *precede* movement. (1) The movement related potentials evoked by self-paced thumb movements (MP) show changes in the cerebral cortex before the initiation of the movement. The MP is composed at least of three main components: a slow negative shift (Bereitschaft-

potential) appearing 1 s before EMG onset, a positive-negative deflection between 50–150 ms before EMG onset and, finally, a positive deflection between 50–150 ms after EMG onset (Gilden *et al.*, 1966; Shibasaki *et al.*, 1980). The sequence of the three electrophysiological findings has been related physiologically to the facilitation of the motor cortex, activation of pyramidal tract and kinaesthetic feedback respectively (Schmidt *et al.*, 1974). (2) Approximately 12% of task-related cortical neurons in the motor cortex increase their discharge frequency at least 250 ms before EMG onset with the majority of the task-related neurons changing discharge rate between 50–150 ms before EMG onset (Schmidt *et al.*, 1974). Recall that it is in this same time period (100 ms) that the gating of SEPs becomes clear. (3) The CNV (contingent negative variation) is a heterogeneous electrical brain wave recorded over the scalp when a preparatory stimulus (S1) is followed by an imperative stimulus (S2) to which a motor response is made. It begins approximately 300 ms after S1 and has an abrupt decline about 120 ms after S2. It is bilaterally symmetric (Cant *et al.*, 1966) in contrast to the contralateral topography of movement-related potentials (Vaughan *et al.*, 1968). The CNV is most clearly related to the psychological processes of attention and arousal (Tecce, 1972). Our design did not allow study of events between S1 and S2, but only after S2 (order to move). The CNV amplitude decreases after S2 while the gating of SEP associated with the command to move increases making a relation between CNV and gating unlikely.

We examined the relation between movement-related potentials (MP) and the attenuation of SEPs in the 3 subjects tested for the 'timing' experiments, all of them showing very clear movement-related potentials to 'self-paced' thumb movements. For this purpose we back-averaged from EMG onset and recorded from F3 and P3 referenced to the ipsilateral ear. We were unable to define a consistent movement-related evoked potential by this method making unlikely that such movement-related potentials influenced SEP's amplitudes. The failure to detect such movement-related potentials is troublesome but may represent the fact that the commanded movements occurred at a relatively fast rate (1 per 2 s) whereas during self-paced movements the clearest motor potentials were obtained at relatively slower rates (1 per 5–10 s).

Mechanisms of the Gating

There is some agreement that the *location* of gating during active movement occurs centrally but its mechanism has been unclear. Two hypotheses have been proposed. (1) Efferent impulses from upper motor neurons may be transmitted to sensory relay nuclei and other areas of the cerebral cortex leading to gating of afferent volleys as has been described in animals at the dorsal spinal cord (Fetz, 1968), the dorsal column nuclei (through presynaptic and postsynaptic inhibition) (Ghez and Pisa, 1972) and the thalamus (Tsumoto *et al.*, 1975; Yingling and Skinner, 1977). Our results in humans show a cortical or thalamocortical site for gating (i.e., subcortical P14 is unaffected, while cortical P22, P27, N30 and P45 are attenuated). (2) Ascending afferent volleys generated in the periphery by the movement may be

able to interact in the CNS to gate other 'less relevant' inputs (Angel *et al.*, 1984). This phenomenon is possible since somatosensory evoked potentials to stimulation of slowly conducting cutaneous afferents may be suppressed by synchronously elicited activity in IA afferents (Burke *et al.*, 1981). However, during a voluntary contraction the onset of EMG activity precedes the afferent discharge of muscle spindles (Vallbo, 1973), making it unlikely that a centripetal mechanism could account for gating occurring prior to the onset of movement.

Specificity of Gating

The gating of SEPs by voluntary motor activity is well localized. Median nerve SEPs were attenuated by moving those portions of the limb innervated by that nerve, that is, ipsilateral thumb, index or middle finger, but not ipsilateral little finger or contralateral thumb. Perhaps the portions of the motor cortex involved in these median nerve movements innervated become insensitive to input from the median nerve for a period of time both before and during the movement to account for the phenomenon of gating of SEP (Asanuma *et al.*, 1968; Rosén and Asanuma, 1972). The topographical selectivity of SEP attenuation in our experiments points to a very localized process in the gating of afferents to the cerebral cortex.

Gating of sensory input has also been shown to occur in the visual system where saccadic eye movements are associated with changes in discharge patterns in the lateral geniculate body (Malcolm *et al.*, 1970) and cerebral cortex (Adey and Noda, 1973). Moreover, the detection of a test flash diminishes both *before and during* the initiation of a saccade (Latour, 1962; Zuber and Stark, 1966; Starr *et al.*, 1969). The results presented in this paper show that gating in the somatosensory system in man also occurs both before and during motor activity. It may be that 'gating' of different sensory modalities in man have common mechanisms and function.

The interrelation between sensory input and motor activity defined in this study may have relevance for the investigation of clinical disorders of motor function. For instance, it is possible that patients with disturbed motor function due to various structural or neurochemical mechanisms (e.g., Huntington's chorea, Parkinson's disease, pyramidal tract lesions) may have alterations in both the extent and timing of such gating of somatosensory input. The methods and normative results presented in this paper will allow the study of such sensorimotor interactions in patients with neurological disorders.

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