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'Gating' of somatosensory evoked potentials begins before the onset of voluntary movement in man

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The inflow of somatosensory information to the cerebral cortex is modified before and during active movement in animals. This phenomenon has been termed 'gating' and occurs at several levels of the sensory pathway. We studied somatosensory evoked potentials (SEPs) to stimulation of the median nerve at the wrist during voluntary movement of the ipsilateral thumb in man. Results indicate that SEPs are attenuated shortly after a command to move (approximately 100 ms before the onset of the electromyogram (EMG)), become maximally attenuated with maximum EMG and return to normal size when movement is finished.

The inflow of somatosensory information to the cerebral cortex is modified during active movements in man and animals[4,12]. This phenomenon has been termed 'gating' and experiments in animals suggest that transmission of sensory information is affected at several levels of the sensory pathway (i.e. dorsal columns nuclei, medial lemniscus, thalamus)[5,10,14] both during and even before the initiation of movement[5]. We have studied electrical activity of the somatosensory pathway in man using scalp electrodes (short latency somatosensory evoked potentials, SEPs) to stimulation of a mixed nerve of the hand during voluntary movement of the ipsilateral thumb. The results indicate that SEPs are attenuated shortly after a command to move (approximately 100 ms before the onset of the electromyogram (EMG)), become maximally attenuated when the EMG becomes maximal and return to normal size when the movement is finished.

Subjects were 3 healthy students aged 15, 18 and 19 years who were comfortably seated in a sound attenuating chamber. *Stimuli.* Percutaneous electrical stimuli were delivered to the left median nerve at the wrist. The intensity was adjusted to just elicit a visible twitch of muscles in the thenar eminence without causing pain. The number of stimuli delivered during control and movement conditions was that necessary to evoke short latency SEPs having a 20% or less amplitude difference on repeated control measures in the same subject during the same session. From 400 to 1400 stimuli were delivered depending on the relationship of the amplitude of the SEPs to the background electroencephalogram (EEG) (i.e. signal/noise ratio). *Recording procedures.* The SEPs were recorded with scalp electrodes over the contralateral postcentral site (3 cm behind the vertex Cz and 7 cm from the midline) referenced to the contralateral earlobe. Electrode impedances were below 5 kΩ. Electrical activity was amplified (5×10^5) using a bandpass of 30–3000 Hz and SEPs were averaged over a 51-ms time period (dwell time of 0.1 ms). The EMG was recorded from the thenar muscles, amplified and full wave rectified. *Experimental plan.* Low intensity auditory clicks (10–25 dB sensation level), generated by passing 100-μs pulses through earphones, were the signal to move the left thumb. The clicks were delivered every 3 s. The mean latency of EMG movement-related onset was 193.8 ± 42.0 ms (223.6 ± 64.0 ms, 144.8 ± 36.7 ms and 213.0 ± 72.7 ms in the 3 subjects tested). Median nerve stimuli were delivered at different times after the clicks: 0 (simultaneous), 10, 20, 50, 70, 90, 100, 200, 300, 400 and 780

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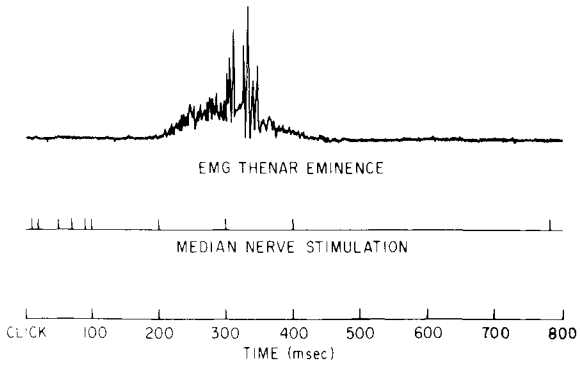

Fig. 1. Shows the experimental plan. Median nerve stimuli were delivered at different times after a signal to move the thumb (click) to encompass premovement, movement and postmovement periods. The top trace shows the averaged full wave rectified EMG from the thenar eminence of one of our subjects with the onset latency adjusted to the mean in the 3 subjects tested.

ms (Fig. 1). Control SEPs were recorded when the subject was instructed to ignore the clicks and not move. Approximately 6 h of recording time was required to complete the experimental plan in each subject. In two of the subjects this was accomplished during one day. These subjects were selected because their performance (reaction times) remained remarkably stable throughout the long recording period. *Data analysis.* Sensory- and movement-related evoked potentials were analyzed off-line. Amplitudes of the SEPs components were measured from a computer display with a cursor. The component latencies were measured from the onset of the electric shocks activating the median nerve. Amplitudes of SEPs recorded during movement are expressed as a percentage of controls. Amplitudes of median nerve SEPs preceded by clicks did not differ from those without clicks if subjects were not moving. *Statistics.* Repeated measures analysis of variance (ANOVA) was used to test amplitude differences. Post-test analysis was conducted using Duncan's multiple range test[1] to compare the control with the other movement conditions.

Results. Electrical stimulation of the median nerve evoked reproducible SEPs beginning with an initial positive component at 13.4 ± 0.6 ms (P_{14}) representing activity in the medial lemniscus[7], followed by negative and positive components at 18.7 ± 0.5 ms (N_{20}) and 23.9 ± 2.6 ms (P_{27}) (Fig. 2). The N_{20} component of the SEPs is thought to represent thalamocortical

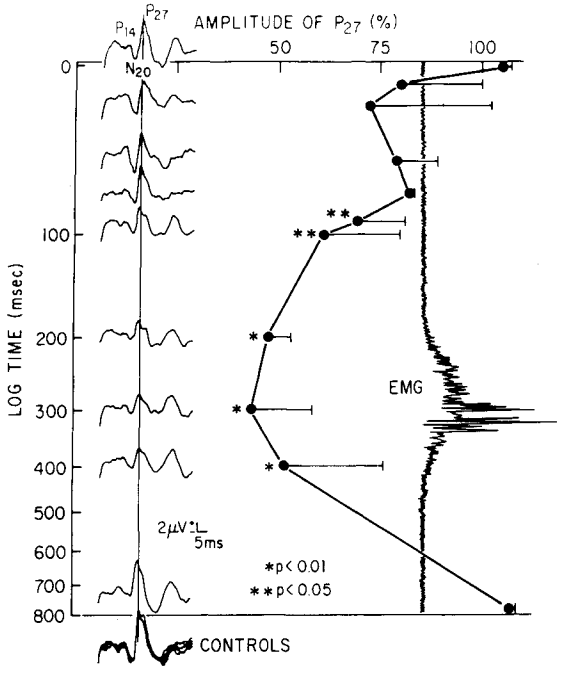

Fig. 2. Shows the effects of thumb movement on the amplitude of SEP to median nerve stimulation at varying time intervals (ordinate) between the signal to move (click) and the delivery of the median nerve stimulus. The time course of the rectified EMG from one subject is shown to the right side of the figure with the EMG onset adjusted to the mean latency of the 3 subjects. SEP from that same subject is on the left side of the figure. Control recordings shown at the bottom of the figure were obtained when the subject was instructed to ignore the click and not move. These controls were taken interspersed between experimental recordings and are the 100% values against which the experimental conditions were compared. Note the small amplitude variability of the control SEPs. In this subject the amplitude of the P_{27} component decreased as early as 20 ms (the second trace) and remained attenuated through 400 ms. The mean amplitude of P_{27} from the 3 subjects is shown in the graph. Note the progressive attenuation of the P_{27} amplitude in the period before the EMG, reaching maximal attenuation during the peak of the EMG and returning to control values after the movement terminated (780 ms).

activity while the P_{27} component is of cortical origin[8]. The latencies of these components did not change during movement. The P_{27} amplitude was significantly attenuated 90 ms after the signal to move, approximately 100 ms before EMG onset (Fig. 2, Table I). Moreover, in the subject depicted in the left hand side of Fig. 2, the attenuation of P_{27} is evident as early as 20 ms after the signal to move, 120 ms before EMG onset (EMG in this subject began 144.6 ± 36.7 ms after the clicks). The attenuation of the P_{27} component continued during movement becoming maximal between 200 and 400 ms after the command to

TABLE I

Effects of thumb movement on the amplitude of SEP

Amplitudes of cerebral potentials to median nerve stimulation (P_{27} component) expressed as percentages of control values (100%). Interval refers to the time between the clicks (order to move) and the delivery of the median nerve stimulation.

	Control	*Interval (ms)*										
		0	*10*	*20*	*50*	*70*	*90*	*100*	*200*	*300*	*400*	*780*
Jeremy	100	101	100	42	76	81	83	82	54	62	80	105
Lisa	100	105	83	71	70	82	61	58	48	42	48	108
Dana	100	110	59	108	91	84	65	44	40	28	29	110
$\bar{X}$	100	105.3	80.7	73.7	79.0	82.3	69.7	61.3	47.3	44.0	52.3	107.7
S.D.	0	4.5	20.6	33.0	10.8	1.5	11.7	19.2	7.0	17.1	25.8	2.5
$P <$							0.05	0.05	0.01	0.01	0.01	

move (Fig. 2). Finally, the amplitude of P_{27} returned to control values (107.7 ± 2.5%) after the movement terminated. Thus, both during and prior to movement, there is an attenuation of the P_{27} cortical component of SEPs. In a separate study in 10 subjects we reported changes in SEPs amplitudes when the median nerve was stimulated 250 ms after the order to move the thumb[3]. The P_{27} amplitude was substantially diminished in every subject (to 53.8 ± 20.3%, $P <$ 0.01). In contrast, P_{14} was variously affected, smaller in 3 subjects, larger in 5 subjects and N_{20} was also variously affected, smaller in 4 subjects, larger in 3 subjects. Thus, the grand average of P_{14} and N_{20} components showed no change with movement (114.0 ± 49.0% and 103.2 ± 29.0%, respectively, not significant) suggesting that subcortical (P_{14}) and thalamocortical (N_{20}) activity are both unaffected. Attenuation of SEPs is not due to changes in subject vigilance and attention since the earliest task-related change in SEPs affects those components with latencies longer than 55 ms[6].

There are electrical events taking place in the cerebral cortex prior to movement that might relate to the occurrence of gating. With self-paced voluntary movement in man, a negative potential shift begins approximately one second before movement onset, becoming maximal 50–150 ms before movement onset, precisely at the time period when attenuation of the SEPs became evident in our study[11]. When monkeys move in response to a signal (similar to our situation), pyramidal tract neurons in areas 4, 3a and junctional area 2/5 discharge in the precontraction period, beginning at 250 ms and peaking 50–150 ms before EMG onset, again at the time period when attenuation of the SEPs became evident in our study[9]. In addition, there are peripheral factors that could contribute to attenuation of SEPs during movement. For instance, during motor activity, afferent discharges from peripheral receptors (i.e. muscle spindles)[15] could affect the central transmission of other somatosensory inputs[2]. In fact, activity in afferents from the periphery are postulated as the origin for attenuation of SEPs during passive movements[12]. Thus, once movement is initiated, it is likely that there is a summation of both central and peripheral factors leading to the attenuation of SEPs. However, only central factors can be responsible for the attenuation of SEPs prior to movement onset.

The initiation of gating before movement onset has also been described for the visual system with changes in visual thresholds appearing before saccadic onset[13]. It may be that gating of different sensory inputs in man have common mechanisms and functions related to perfecting motor performance. This report provides objective data about subcortical and cortical processing of somatosensory afferent information in normal man during motor activity by using scalp-derived SEPs recording techniques. The application of SEPs measures during movements in patients with disturbed motor function (i.e. Parkinson's disease, Huntington's chorea, cortical lesions) may provide insights into the physiopathology of these diseases.

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1 Bruning, J.L. and Klintz, B.L., Duncan's multiple range test. In *Computational Handbook of Statistics,* Scott, Foresman, Glenview, IL, 1968, pp. 115–117.
2 Burk, D., Skuse, N.F. and Lethlean, A.K., Cutaneous and muscle afferent components of the cerebral potentials evoked by electrical stimulation of the human peripheral nerves, *Electroencephalogr. Clin. Neurophysiol.*, 51 (1981) 579–588.
3 Cohen, L.G. and Starr, A., Gating of somatosensory input occurs at the cortex before voluntary movement, *Neurology,* 35 Suppl. 1 (1985) 129.
4 Coquery, J.M., Role of active movement in control of afferent input from skin in cat and man. In G. Gordon (Eds.), *Active Touch,* Pergamon Press, New York, 1978, pp. 161–169.
5 Coulter, J.D., Sensory transmission through lemniscal pathway during voluntary movement in the cat, *J. Neurophysiol.*, 37 (1974) 831–845.
6 Desmedt, J.E. and Robertson, D., Differential enhancement of early and late components of the cerebral somatosensory evoked potentials during forced-paced cognitive tasks in man, *J. Neurophysiol.*, 271 (1977) 761–782.
7 Desmedt, J.E. and Cheron, G., Central somatosensory conduction in man: neural generators and interpeak latencies of the far-field components recorded from neck and right or left scalp and earlobes, *Electroencephalog. Clin. Neurophysiol.*, 50 (1980) 382–403.
8 Emerson, R.G. and Pedley, T.A., Generator sources of median somatosensory evoked potentials, *J. Clin. Neurophysiol.*, 1 (1984) 203–218.
9 Fromm, C., Contrasting properties of pyramidal tract neurons located in the precentral or postcentral areas of corticorubral neurons in the behaving monkey. In J.E. Desmedt (Ed.), *Motor Control Mechanisms in Health and Disease,* Raven Press, New York, 1983, pp. 329–345.
10 Ghez, C. and Pisa, M., Inhibition of afferent transmission in cuneate nucleus during voluntary movement in the cat, *Brain Research,* 40 (1972) 145–151.
11 Kornhuber, H.H. and Decke, L., Hirnpotentialänderungen bei Willkürbewegungen and passiven Bewegungen des Menschen: Bereitschaftspotential und reafferente Potentiale, *Pflügers Arch., Gesamte Physiol. Menschen Tiere,* 284 (1965) 1–17.
12 Rushton, D.N., Rothwell, J.C. and Craggs, M.D., Gating of somatosensory evoked potentials during different kind of movements in man, *Brain,* 104 (1981) 465–491.
13 Starr, A., Angel, R. and Yeates, H., Visual suppression during smooth following and saccadic eye movement, *Vision Res.*, 9 (1969) 195–197.
14 Tsumoto, T., Nakamura, S. and Iwama, K., Pyramidal tract control over cutaneous and kinesthetic sensory transmission in the cat thalamus, *Exp. Brain Res.*, 22 (1975) 281–294.
15 Vallbo, A.B., Muscle spindle afferent discharge from resting and contracting muscles in normal human subjects. In J.E. Desmedt (Ed.), *New Developments in electromyography and clinical Neurophysiology, Vol. 3,* Karger, Basel, 1973, pp. 251–262.