"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 movements 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. 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) both during and even before the initiation of movement. 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 × 10⁵) 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...
EMG THENAR EMINENCE

MEDIAN NERVE STIMULATION

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 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 (P14) representing activity in the medial lemniscus, followed by negative and positive components at 18.7 ± 0.5 ms (N20) and 23.9 ± 2.6 ms (P27) (Fig. 2). The N20 component of the SEPs is thought to represent thalamocortical activity while the P27 component is of cortical origin. The latencies of these components did not change during movement. The P27 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 P27 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 P27 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 (P27 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.

| 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 |
| 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| 7.0 | 17.1| 25.8| 2.5 |
| P <          | 0.05| 0.05| 0.01| 0.01| 0.01| 0.01| 0.01| 0.01| 0.01| 0.01| 0.01|

move (Fig. 2). Finally, the amplitude of P27 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 P27 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. The P27 amplitude was substantially diminished in every subject (to 53.8 ± 20.3%, P < 0.01). In contrast, P14 was variously affected, smaller in 3 subjects, larger in 5 subjects and N20 was also variously affected, smaller in 4 subjects, larger in 3 subjects. Thus, the grand average of P14 and N20 components showed no change with movement (114.0 ± 49.0%, P < 0.01) suggesting that subcortical (P14) and thalamocortical (N20) 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.

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. 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.

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) could affect the central transmission of other somatosensory inputs. In fact, activity in afferents from the periphery are postulated as the origin for attenuation of SEPs during passive movements. 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. 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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