### Title

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### Authors

Tapia, Maria C
Cohen, Leo G
Starr, Arnold



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# Attenuation of Auditory-Evoked Potentials during Voluntary Movement in Man[1]

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

Department of Neurology, University of California, Irvine, Calif., USA

**Key Words.** Auditory-evoked potentials · Movement · Sensory information · Vigilance

**Abstract.** Long-latency auditory evoked potential (AEP) amplitudes were reduced during voluntary thumb movement. The cerebral component $N_{100}$ did not change during the movement. By contrast, the cerebral potential $P_{200}$ and $N_{100}$–$P_{200}$ amplitudes were attenuated during the motor task compared to control. This report cannot clearly distinguish if the attenuation obtained in the AEP during the performance of the movement is related to motor events in the cerebral cortex causing 'gating' or to other factors such as habituation or vigilance changes.

## Introduction

The transmission of sensory information to the cerebral cortex is modulated during voluntary movement in man and animals. This phenomenon named 'gating', has been described for the visual [Volkman, 1962], auditory [Starr, 1964; Hazemann et al., 1975] and somatosensory [Coulter, 1973; Lee and White, 1974; Cohen and Starr, 1987] systems. We have studied changes in long-latency auditory-evoked potentials (AEP) in man during movement of the fingers. Our study utilized a distal movement of the thumb in response to a command whereas in the study by Hazemann et al. [1975] the movements were self-paced and involved the entire hand.

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## Methods

Long-latency AEP were analyzed in 6 young, healthy, right-handed students, engaged in moving the thumb towards the little finger on command.

1-kHz tone (3 ms rise/fall; 35 ms plateau at 75 dB SL and 0.7/s rate) were presented to the left ear through Telephonics TDH-39 earphones. The resulting cerebral potentials were recorded with scalp electrodes from the

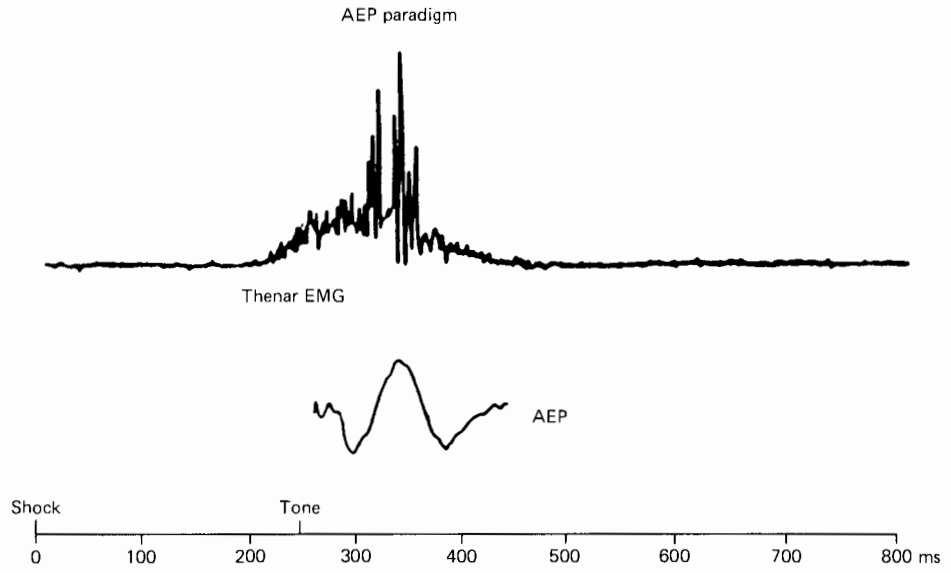


**Fig. 1.** AEP paradigm: Low-intensity shocks, 0.7/s applied to the close up wrist without eliciting any twitch of the muscles were the signal to move the thumb. Tones were presented 250 ms after each shock, to ensure that motor activity was present.

vertex $C_z$ referenced to inion. The EEG was amplified approximately 100000 times with a bandpass of 1–100 Hz. The AEP was averaged over a 400-ms analysis time for 150 trials.

*Paradigms*

AEP during movement: low-intensity shocks, 0.7/s applied to the median nerve at the wrist just below threshold for eliciting a twitch of the thenar muscles were the signal to move the thumb. The 1-kHz tone was presented 250 ms after each shock, when the muscles were active in moving the digit (fig. 1).

Control recordings were made during the same median nerve stimulation but when the subject was instructed not to move. The control recordings were made both immediately before and after the movement period.

*Measurements*

The amplitudes of AEP recorded during movement are expressed as a percentage of the last control period without movement. Amplitudes of the evoked potentials were measured between the baseline and the initial negative ($N_{100}$) and subsequent positive ($P_{200}$) peaks, as well as peak to peak ($N_{100}$–$P_{200}$). We did not have controls for factors such as attention and habituation a contributing to this movement-related AEP attenuation.

*Statistical Analysis*

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 ns was used to provide detailed comparisons between the amplitudes of AEP recorded during the control periods and during thumb movement.

## Results

### *Attenuation of AEP during Thumb Movement*

The grand average of all of the subjects' long-latency AEP during control and movement conditions is shown in figure 2. There

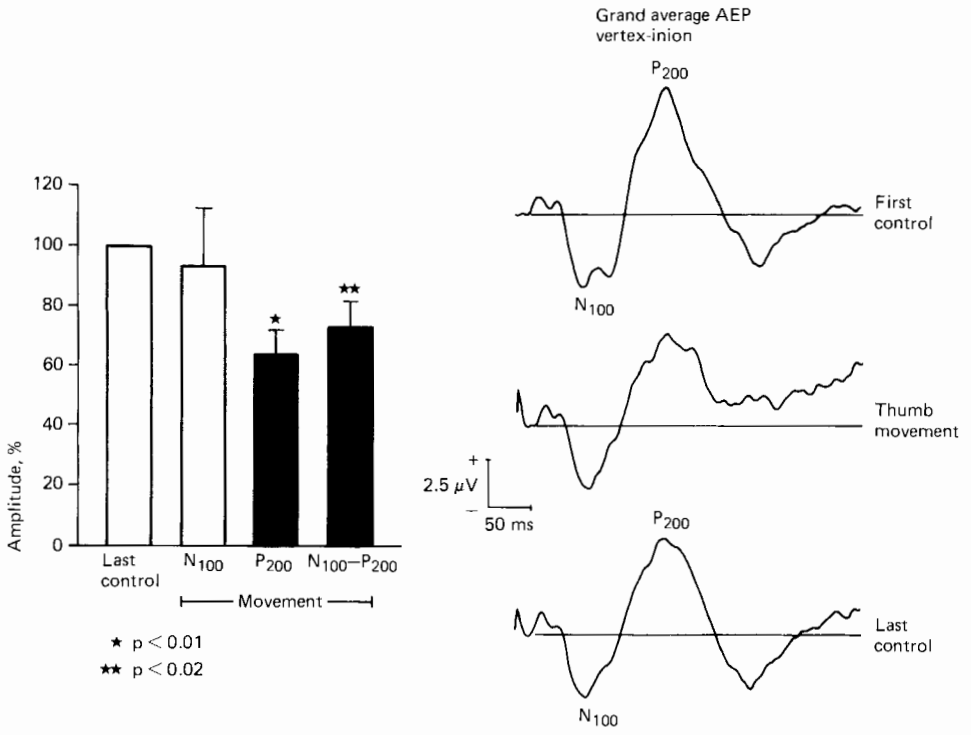


**Fig. 2.** To show the effects of thumb movement on long-latency AEP (see traces). The bar graphs represents the extent of attenuation of $N_{100}$, $P_{200}$ and $N_{100}$–$P_{200}$ components during the movement in relation to last control.

is a negative peak at 80.3 ± 9.2 ms ($N_{100}$) and a subsequent positive peak at 170.0 ± 16.9 ms ($P_{200}$) (see control traces). Controls were obtained when the subject was instructed to ignore the stimulus both before (first control) and immediately after the condition with thumb movement (last control). Since the amplitude of the last control was smaller than that of the first control, we used the last control to compare the effects of thumb movement. The latencies of the components did not change during movement of the thumb. The bar graphs contains the mean amplitudes and standard errors of the individual measurements for the different components during thumb movement relative to last control (100%). The $P_{200}$ and $N_{100}$–$P_{200}$ amplitudes were significantly attenuated ($p < 0.01$ and $p < 0.02$, respectively) during the movement compared to control periods.

## Discussion

The results of this study in humans show an attenuation of long-latency AEP during voluntary movement of the thumb. A similar phenomenon was reported in cats by Starr [1964] who studied the influence of motor activity on auditory-evoked responses recorded from the auditory pathways

within the brain. The potentials were attenuated when the animals were engaged in orienting movements. However, if the cats were sitting still, click-evoked responses were of control amplitudes. The middle ear muscles were active during such orienting movements and Starr concluded that a dual regulatory system consisting of both peripheral neuromuscular as well as of central mechanisms was active in modifying click-evoked responses during movement. Brugge and Merzenich [1973] reported in monkeys that the activity of a large proportion of neurons in the auditory cortex were markedly reduced during periods of bodily movements. In studies in humans, Hazemann et al. [1975] showed that voluntary movement in man induces a generalized central modulation responsible for the attenuation on $N_{100}$ and $P_{200}$ AEP components. In this regard, our findings are similar, showing a reduction of $P_{200}$ and $N_{100}$–$P_{200}$ components to tone stimulation during thumb movement.

Many reports have been written concerning the changes in the vertex potential secondary to attention. According to Picton and Hillyard [1974] the $N_{100}$ and $N_{100}$–$P_{200}$ components in man increase during selective attention. In our experiments there was a significant attenuation of $N_{100}$ but not $P_{200}$ between the first and second control recording sessions. These results suggest that $P_{200}$ attenuation observed during movement is more likely related to motor task and not to attention or vigilance changes.

Though the most important representation of auditory cortical fields lie in the superior temporal lobe, the works of Nelson and Bignall [1973] and Newman and Lindsley [1976] have established that acoustic responses are also observed in frontal and parietal polysensory areas in monkeys. Perrault and Picton [1984] found that the generator of the vertex potential $N_{100}$ involves two or more sources. Moreover, there is a general agreement that the cerebral cortex has multiple interrelations between the different areas [Porter, 1981]. Cortical motor neurons engaged in thumb movement might suppress other cortical neurons responsible for the generation of long-latency AEP. Thus, the attenuation of the $P_{200}$ and $N_{100}$–$P_{200}$ components could result from cortico-cortical inhibitory connections [Porter, 1981].

To study the gating phenomenon in the auditory system with more precision requires the measurement of those components resistant to habituation, i.e. such as the short-latency auditory brain stem responses or even the middle-latency responses. However, the number of trials required to obtain the latter averages is beyond the capacity of most subjects (i.e. 1 000 trials) for making repetitive movements to command in a stereotyped manner.

Gating of sensory input has been reported in other systems: Volkman [1962] demonstrated suppression of visual sensitivities during voluntary eye movements. Starr and Cohen [1985] have shown that gating of somatosensory-evoked potentials from stimulation of the median nerve at the wrist occurs at a cortical level. The lemniscal component P14 and the thalamocortical $N_{20}$ component were unchanged, whereas the postcentral cortical $P_{27}$ component was attenuated during voluntary movement. This gating of somatosensory input occurs before the initiation of EMG activity in the premovement period [Starr and Cohen, 1985]. It is likely that this mechanism for gating are related to premotor events

in the cerebral cortex and not to peripheral factors.

## Conclusions

This report demonstrates that long-latency AEP were attenuated during voluntary movement in man, $P_{200}$ and $N_{100}$–$P_{200}$ components decreased in amplitude compared to control recordings. It was difficult to be certain if the attenuation observed in the long-latency AEP during the performance of the movement was related to the performance of the motor task or to other mechanisms such as habituation or vigilance changes.

## Potentiels évoqués auditifs corticaux pendant le mouvement volontaire du sujet

Les potentiels évoqués auditifs corticaux ont présenté une atténuation de leur amplitude pendant le mouvement volontaire du premier doigt de la main droite par comparaison aux potentiels évoqués auditifs corticaux des mêmes sujets pendant les périodes d'immobilité (contrôle). La composante cérébrale $N_{100}$, ne diminua pas d'amplitude lors du mouvement du premier doigt; au contraire, les amplitudes des potentiels cérébraux $P_{200}$ et $N_{100}$–$P_{200}$ présentèrent une atténuation. Cette étude recherche si lors de l'exécution de mouvements l'atténuation obtenue des potentiels auditifs corticaux est provoquée dans le cortex cérébral par des événements moteurs ou par d'autres facteurs liés à l'habitation ou à des modifications de la vigilance.

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Arnold Starr
Department of Neurology
University of California
Irvine, CA 92717 (USA)