Pre-movement parietal lobe input to human sensorimotor cortex

Robert T. Knight, Jaswinder Singh and David L. Woods

Department of Neurology, University of California, Davis, Veterans Medical Center, Martinez, CA 94553 (U.S.A.)

(Accepted 13 June 1989)

Key words: Motor potential; Parietal lobe; Movement-related cortical potential; Movement; Sensorimotor

Movement-related cortical potentials (MRPs) were recorded in an auditory dichotic selective attention experiment in patients with focal lesions centered in either posterior superior temporal gyrus (temporal) or in lateral parietal cortex (parietal). Controls and temporal patients generated comparable pre-movement negative shifts (NSs) and motor potentials (MPs), onsetting about 400 ms prior to movement and maximal in amplitude over scalp sites contralateral to button press. Unilateral parietal cortex lesions markedly reduced the NSs but preserved the MP component of the MRP. The results indicate that human parietal association cortex exerts modulatory input to sensorimotor cortex, beginning at least 400 ms prior to movement. The differential effect on the NSs and MPs by parietal lesions suggests that these MRP components may have independent infracranial generators.

Animal studies and topographical, intracranial and magnetic recordings in humans indicate that pre-movement-related cortical potentials (MRPs) are generated predominantly in pre- and postcentral gyrus with possible additional contributions from supplementary and premotor cortex. These areas receive dense projections from parietal areas which have a documented critical role in control of complex goal-directed movements. In the present study we assessed the role of parietal cortex in generation of movement potentials by recording MRPs from patients with focal lesions in subregions of posterior association cortex.

Patient groups with lesions in either posterior superior temporal gyrus (temporal) or in lateral parietal cortex (parietal) were studied. The temporal lesions (n = 6, 5L and 1R, mean age 56.1 ± 9 years, mean lesion volume 48.3 cc) centered in Brodmann area 22 and included portions of auditory cortex (areas 41 and 42) and inferior angular (area 39) and supramarginal gyrus (area 40). Parietal lesions (n = 6, mean age 52.9 ± 11 years, mean lesion volume 40.3 cc) included rostral areas 39 and 40 and inferior area 7 (see Fig. 1a and 1b). Patient results were compared to age-matched controls (n = 9, mean age 53.3 ± 10 years). Details of patient selection criteria, clinical descriptions and CT reconstruction methods are presented elsewhere.

The experiment consisted of an auditory selective attention experiment with stimuli presented at intervals ranging from 200 to 400 ms. Tone bursts of 700 Hz in one ear and 1300 Hz in the other were presented dichotically with probabilities of 42% in each ear. The subjects attended to either right or left ear tones and pressed a button to random target tone bursts in that ear occurring on 5% of the trials. The targets were identical in frequency to the standard but longer in duration (75 vs 25 ms, 50 dB SL). Novel stimuli requiring no response occurred randomly on 3% of the trials in each ear. Four 8–10 min blocks of data were obtained.

EEG was recorded from 13 scalp sites in the international 10–20 system (FPz, F3, Fz, F4, T3, C3, Cz, C4, T4, P3, Pz, P4, Oz and below eye) referenced to a balanced two-vector non-cephalic electrode. The EEG was amplified (20 k), filtered (0.1–100 Hz), and continuously digitized (256 Hz/channel) on a general purpose computer (PDP 11/73).

The study was designed to record attention-
related activity to target and novel stimuli in these patients and the results are presented elsewhere. The patient groups had comparable reaction times (RTs) to correctly detected targets and percent correct detected targets (RTs: controls 774 ± 74 ms, parietal 867 ± 147 ms, temporal 853 ± 96 ms).

MRPs were extracted from epochs of raw EEG beginning 800 ms prior to correct button presses and continuing for 200 ms post-press. Trials contaminated by eye blinks, excessive EMG activity or amplifier blocking were rejected prior to averaging. All responses were made with the thumb of the right hand and sums of 120–180 trials were obtained from each subject.

Inspection of pre-movement epochs from −1200 to −500 ms revealed no MRP activity (i.e. readiness potential, RP) in any of the subjects. Mean amplitude measurements of the negative shift (NS) and motor potential (MP) were computed with reference to a baseline of activity −800 to −600 ms prior to button press. Measurement windows were −400 to −100 for the NS and −100 to 0 (button press) for the

---

Fig. 2. Overlapped group grand-averaged waveforms from controls, temporal, and parietal subjects. The MRPs were back-averaged from correctly detected targets in an auditory selective attention experiment. The NS is reduced in the parietal group with relative preservation of the MP.
CONTROL TEMPORAL PARIETAL

NS MP

Fig. 3. Scalp topographical maps of the NS and MP components of the MRP. The data are shown for control, temporal, and parietal subjects. The maps reveal the contralateral preponderance of the NS and MP in controls, temporals and parietals. The MP is seen at F3 and C3 in the parietal group.

MP. Onset latency of the NS was measured at the point of sustained (>20 ms) MRP activity over 1.5 μV.

The data were subjected to repeated measures ANOVA with corrected t-tests used to make specific electrode comparisons. Scalp topographical maps were generated from the normalized voltages for each subject. The contribution of each electrode was inversely weighed as a function of the cube of the inter-electrode distance.

The NS had comparable onset times in control and patient groups (control 425 ± 60 ms, temporal 458 ± 43 ms, parietal 428 ± 59 ms, P = n.s.). The NS was maximal over fronto-central regions contralateral to movement in all groups (control: difference between electrodes, $F_{12,96} = 4.43$, $P < 0.001$, C3 vs C4, $F_{1,8} = 16.42$, $P < 0.004$; temporal: difference between electrodes, $F_{12,60} = 3.76$, $P < 0.001$, C3 vs C4, $F_{1,8} = 8.51$, $P < 0.033$; parietal: difference between electrodes, $F_{12,60} = 2.35$, $P < 0.015$, P3 vs P4, $F_{1,5} = 13.38$, $P < 0.015$; see Figs. 2 and 3).

The MP, defined as negativity onsetting 100 ms prior to and extending to the button press, was also maximal over fronto-central and contralateral sites in all groups (control: difference between electrodes, $F_{12,96} = 5.97$, $P < 0.001$, C3 vs C4, $F_{1,8} = 11.32$, $P < 0.01$; temporal: difference between electrodes, $F_{12,60} = 5.27$, $P < 0.001$, C3 vs C4, $F_{1,5} = 7.17$, $P < 0.043$; parietal: difference between electrodes, $F_{12,60} = 7.71$, $P < 0.001$, C3,F3 vs C4,F4, $F_{1,11} = 7.34$, $P < 0.019$; see Figs. 2 and 3).

There was no difference in NS amplitude between control and temporal patients (see Figs. 2 and 3). In contrast, unilateral parietal lesions markedly reduced the NS over both hemispheres (mean amplitude over electrodes for controls vs parietals: $F_{1,13} = 5.50$, $P < 0.033$; at Cz, $F_{1,13} = 5.26$, $P < 0.037$; for temporal vs parietal at Cz, $F_{1,13} = 2.05$, $P < 0.05$). The NS reduction was comparable in both left and right lesioned subjects.

MP activity onsetting at about 100 ms was recorded from the parietal subjects at C3, but was reduced at all other central scalp sites (for mean amplitude at Cz, control vs parietal; $F_{1,13} = 2.13$, $P < 0.04$, parietal vs temporal; $F_{1,10} = 6.59$, $P < 0.027$; at C3, $P = n.s.$ for both comparisons). Inspection of the overlapped waveforms in Fig. 2 and the topographical maps in Fig. 3 shows MP activity in the parietal group over the scalp sites
contralateral to right hand press (i.e. C3, F3).

MRP research usually employs self-paced movement paradigms and D.C. or long time constant recording. Despite the rapid signal detection paradigm employed in the current study and the relatively short 1-s time constant, reliable NS and MP potentials with typical onset and scalp distributions were recorded in both control and temporal patients. However, the readiness potential which onsets about 1000 ms prior to movement was not observable, likely due to the bandpass constraints and high speed responding required in the study.

Unilateral parietal lesions reduced the NS at all electrode sites beginning at 400 to 300 ms premovement. The temporal profile of the lesion effect on the NS coupled with intracranial and magnetic data on NS sources in somatomotor cortex suggests that parietal association cortex provides a critical modulatory input to human pre- and postcentral gyri onsetting 400 to 300 ms prior to movement. Since the temporal and parietal patients had comparable lesion volumes, RTs and late negative waves to the targets, it is unlikely that the NS reduction in the parietal subjects is due to a general lesion effect, an inability or slowness in pressing the button or to a difference in overlapping cognitive potentials generated by the targets.

The NS was bilaterally reduced by unilateral lesions of either hemisphere. This is compatible with clinical observations that unilateral parietal lesions in humans can result in apraxia in both limbs. Intracallosal connections provide pathways for unilateral parietal cortex to bilaterally influence somatomotor cortices, either by direct connections or through relays in prefrontal, premotor or supplementary cortex.

The parietal lesions reduced the NS but preserved the MP over sensorimotor cortex contralateral to movement. These results support the notion that the NS and MP have different generator sources. The preserved MP in the parietal subjects was likely generated by pyramidal cell discharges in the precentral gyrus controlling thumb movement during the button press.

The functional significance of the NS suppression is not apparent from the present study. The current findings coupled with reports of parietal single units discharging 300 ms prior to movement cannot be readily explained by reafferentation theories of parietal participation in movement. The comparable RTs in parietal and temporal subjects further indicate that motor initiation can occur without NS generation. The role of parietal cortex in movements in 3-dimensional space suggests that the NS component of the MRP may index parietal input to sensorimotor cortex involved in visuomotor control.

Supported by NIH Grant NS21135 and the VA Research Service. Special thanks to Clay Clayworth for technical assistance in all phases of the project. All aspects of the research were carefully explained to both controls and patients who signed informed consent statements. The research was approved by the Institutional Review Boards of the Martinez Veterans Medical Center and the University of California, Davis.

---


