P300 generation by novel somatosensory stimuli

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Summary

Event-related potentials (ERPs) to task-relevant target and task-irrelevant novel stimuli were recorded in a somatosensory discrimination task. Subjects pressed a button to mechanical taps of the fifth finger (targets, P = 0.12), randomly interposed in sequences of taps to the second finger (standards, P = 0.76). Two types of infrequent novel stimuli were delivered; one was a mechanical tap to the third or fourth finger (tactile novels, P = 0.06), another was an electric shock at the wrist (shock novels, P = 0.06).

Correctly detected targets generated a parietal maximal P300 (P3b, latency 335 msec). Shock novels generated a central maximal P300 with a shorter peak latency (298 msec) than the P3b. Tactile novels generated a P300 with a scalp distribution comparable to the shock novels. Unlike the P3b, P300 amplitude to both the shock and tactile novel stimuli habituated by 20–30% across the first several stimulus presentations.

These results indicate that, similar to the auditory and visual modality, task-irrelevant novel somatosensory stimuli generate a novelty P300 ERP. Differences in scalp distribution, latency and habituation characteristics suggest that the novelty P300 may have contributions from intracranial generators independent from target P300 sources.

Key words: novelty P300; target P300; somatosensory stimulation

Task-relevant correctly detected stimuli generate a parietal maximal P300. Non-target, deviant stimuli requiring no behavioral response generate an earlier latency, fronto-central P300. The target P300 has been operationally labeled the P3b and the novelty P300 the P3a. Target and novelty P300 event-related potentials (ERPs) have been reported in the visual (Courchesne et al. 1975; Beck et al. 1980) and auditory modalities (Knight 1984). Differences in scalp topography, latency and psychological variables necessary for novelty and target P300 generation suggest that multiple intracranial sources contribute to ERP positivity in the 300–600 msec range.

Controversy exists regarding the neural sources of both the target and novelty P300 ERPs. Intracranial and neuromagnetic recordings have suggested target P300 sources in limbic (Halgren et al. 1980; Wood et al. 1980; Okada et al. 1983), diencephalic (Yingling and Hosobuchi 1984; Katayama et al. 1985) and prefrontal (Wood and McCarthy 1985) regions. Scalp topographic analysis (Vaughan et al. 1983) and intracranial recordings (Smith et al. 1988) support a role of temporal-parietal junction in auditory and visual target P300 generation. Study of patients with focal cortical lesions (Knight et al. 1989) indicates that temporal-parietal junction is critical for auditory target P300 generation.

Less data are available on novelty P300 sources. Lesion studies indicate that prefrontal (Knight 1984; Scabini et al. 1989) and temporal-parietal cortex (Knight et al. 1989) contribute to auditory novelty P300 generation. However, patients with temporal-parietal lesions and absent visual novelty P300 response have partial preservation of their visual target P300 ERPs (Knight 1990). These results indicate that modality-specific neural circuits may be engaged during novelty and target P300 generation. Thus, study of ERP modality effects may provide insight into the neural mechanisms underlying P300 generation.

Target P300 ERPs have been reported in the somatosensory modality (Desmedt and Robertson 1977; Barrett et al. 1979; Snyder et al. 1980), although there are no reports of somatosensory novelty P300 ERPs. The current study was designed to assess whether task-irrelevant novel stimuli generate somatosensory novelty P300 responses.

Methods

Subjects

Twelve right-handed normal adults (6 males, 6 females; mean age 26.6 ± 8.3 years) served as subjects. Eleven of the subjects were naive to the purpose of the experiment and one was a laboratory person.
**Stimuli**

The stimuli consisted of mechanical taps to the digits and electric shocks to the wrist. Mechanical taps were delivered separately to the second (index), third (middle), fourth (ring) and fifth (little) finger tips. A solenoid was activated by a 50 msec square wave electric pulse, resulting in a upward 5 mm movement of a rod (diameter 2 mm) situated in the center of the solenoid. Four solenoids were attached to the position of each finger tip on a plastic hand brace. The hand brace fixed the hand in a natural position and limited movement of the fingers after contact with the rod. For the electric shock stimuli, electrical square pulses of 0.20 msec duration were delivered by a stimulator (Grass Instrument Co.) through an isolating transformer with electrodes placed over the median nerve at the wrist. The intensity of the shock stimulation was adjusted to the threshold for eliciting a visible twitch of the thenar muscle. The twitch threshold was determined by delivery of a few stimuli prior to ERP recording. There was no significant difference in intensity of delivered current between the right and left wrist.

**Procedures**

The subject was seated in a reclining chair in a sound attenuated chamber and was instructed to minimize eye movements and blinks throughout the periods of stimulation. The experiment consisted of 4 blocks. Half of the subjects (3 males and 3 females) received right-hand stimulation on the first and fourth block and left-hand stimulation on the second and third block while another half of the subjects received the alternate sequence. Each block consisted of 500 stimuli delivered at a rate of 1/sec. Of these stimuli, 76% were mechanical tactile stimuli to the second finger (standards), 12% were mechanical tactile stimuli to the fifth finger (targets), 6% were mechanical tactile stimuli to the third or fourth finger (tactile novels), and 6% were electric shock stimuli to the median nerve (shock novels). Target and novel stimuli were interspersed at random in the sequence. Stimulus randomization and delivery were controlled by a PC personal computer. The subject was instructed to press a button with the thumb of the non-stimulated hand only upon detection of tactile stimuli to the fifth finger and not to respond to the other stimuli. Two or 3 min rest periods intervened between blocks. During the experiment white noise (35 dB) was presented through headphones to mask the sound produced by the rod movement.

**Recording system**

Brain electrical activity was recorded using Ag/AgCl electrodes placed at 15 scalp locations (Fpz, F3, Fz, F4, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6 and Oz) based on the 10–20 system and below the left eye, all referenced to linked earlobes. Electrode impedances were kept below 5 kΩ. The EEG was amplified (bandpass 0.1–100 c/sec), digitized (250 Hz/channel) and stored on magnetic tape for off-line analysis by a PDP 11/73 computer. The averaging epoch was 1024 msec, including 200 msec of prestimulus baseline. Individual trials with excessive muscle activities (greater than 80 μV peak-to-peak amplitude at T3 and T4) or eye blinks (greater than 100 μV peak-to-peak amplitude at Fpz and below eye channel) were excluded. An average of 12% of all stimulus trials were rejected. Component amplitude was measured relative to the 200 msec prestimulus baseline. The P300 component was defined as the most positive peak occurring in a post-stimulus window of 250–450 msec. The window was determined from inspection of individual averages and group superaverages.

**Statistical analysis**

The data were subjected to univariate and multivariate analysis of repeated measures (Vasey and Thayer 1987). Tukey test was used for post-hoc pairwise comparison. For the analysis of scalp distribution 3 midline scalp sites (i.e., Fz, Cz and Pz) were used. To analyze the effects of event repetition on P300 amplitude and obtain adequate signal to noise, the first 3 (1–3), second 3 (4–6), third 3 (7–9) and forth 3 (10–12) stimuli were averaged across blocks and subjects. Single electrode data at Cz for shock and tactile novels and at Pz for targets were used to habituation effects.

**Results**

Fig. 1 shows the evoked potentials to standard tactile stimuli, target tactile stimuli, and tactile and shock novel stimuli. Each trace represents the averaged response to right hand stimulation in 1 subject. Grand average ERPs over 12 subjects are presented in Fig. 2.

**Amplitude**

Frequent tactile standard stimuli did not generate late components following the P190 component. Correctly detected target stimuli generated a parietal maximal P300 with a mean latency of 335 msec at Pz (for scalp distribution, F (2, 10) = 42.3, P < 0.001; amplitude at Pz > Cz > Fz, P < 0.01). Infrequent tactile novels generated a P300 component maximal at centro-parietal electrodes (for scalp distribution, F (2, 10) = 5.95, P < 0.05; amplitude at Cz and Pz > Fz, P < 0.01). P300 amplitude to the tactile novel was smaller than that evoked by targets (over all electrodes, F (1, 11) = 8.95, P < 0.05). P300 amplitude to tactile novel stimulation to the fourth finger was larger than that evoked by stimuli to the third finger (the fourth finger = 13.6 μV, the third finger = 11.8 μV at Cz, F (1, 11) = 5.14, P < 0.05). Shock novels generated a larger P300 than the targets (179% increase at Fz, 174% at Cz and 127% at
Pz; over all electrodes, $F(1, 11) = 6.91, P < 0.05$). The P300 component generated by shock novels had largest amplitude at Cz (for scalp distribution, $F(2, 10) = 6.96$, $P < 0.05$; amplitude at Cz and Pz > Fz, $P < 0.01$; Table I).

**Habituation**

P300 amplitude to shock novels decreased by 17% from events 1–3 to events 4–6 and by 24% to both events 7–9 and events 10–12 ($F(3, 9) = 5.30, P < 0.05$; events 1–3 > events 7–9 and events 10–12, $P < 0.05$). Target P300 did not show this habituation ($F(3, 9) = 0.69, P = n.s.,$ see Fig. 3). The shock novel P300 topographical distribution shifted slightly posteriorly over time although the effect was not significant (Fz/Pz amplitude ratio = 0.73 for events 1–3 and 0.68 for events 10–12, $F(1, 11) = 1.05, P = n.s.$). Novel P300 latency was not affected by event repetition.

**Latency**

The mean latency of the P300 evoked by shock novels (312 msec over all electrodes) was significantly shorter than that for targets (334 msec over all electrodes, $F(1, 11) = 6.07, P < 0.05$; at Pz, $F(1, 11) = 7.12, P < 0.05$) and tactile novels (345 msec over all electrodes, $F(1, 11) = 13.1, P < 0.01$, see Table I).

**Topography**

To compare the scalp distributions of the P300s generated by the 3 different stimuli, the amplitude of the P300 evoked by each stimulus was normalized within each subject (McCarthy and Wood 1985). The normal-

### Table I

Mean amplitude (µV) and latency (msec) of the P300 evoked by different stimuli.

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Targets</th>
<th>Tactile novels</th>
<th>Shock novels</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amplitude</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>9.24 ± 4.50</td>
<td>7.90 ± 2.65</td>
<td>16.51 ± 4.72</td>
</tr>
<tr>
<td>Cz</td>
<td>14.91 ± 6.67</td>
<td>11.85 ± 4.64</td>
<td>25.92 ± 9.50</td>
</tr>
<tr>
<td>Pz</td>
<td>18.80 ± 6.48</td>
<td>12.16 ± 5.46</td>
<td>23.83 ± 7.53</td>
</tr>
<tr>
<td><strong>Latency</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fz</td>
<td>320 ± 25</td>
<td>343 ± 25</td>
<td>323 ± 43</td>
</tr>
<tr>
<td>Cz</td>
<td>325 ± 36</td>
<td>349 ± 28</td>
<td>298 ± 38</td>
</tr>
<tr>
<td>Pz</td>
<td>335 ± 37</td>
<td>348 ± 31</td>
<td>299 ± 35</td>
</tr>
</tbody>
</table>

Values are for right-hand stimulation (mean ± S.D.), N = 12.

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Fig. 1. ERP wave forms at Fz, Cz and Pz to the standard, target, tactile novel or shock novel stimuli for right-hand stimulation. Each tracing represents the averaged ERP from a single subject.
Fig. 2. ERP grand average wave forms at Fz, Cz and Pz to the standard, target, tactile novel or shock novel for right-hand stimulation. The grand averages were made across 12 subjects.

ized potentials evoked by targets, tactile novels and shock novels are plotted in Fig. 4 as a function of scalp locus. Both tactile and shock novels generated P300 components maximal at Cz and Pz, while targets generated P300 ERPs maximal at Pz. This effect on distribution was significant (tactile novels vs. targets, $F (2, 10) = 4.89, P < 0.05$; shock novels vs. targets, $F (2, 10) = 9.43, P < 0.01$). Single electrode comparisons showed that P300 amplitude for tactile novels was larger at Fz and Cz than that for targets ($P < 0.05$). P300 amplitude for shock novels was larger at Fz and Cz ($P < 0.01$) and smaller at Pz ($P < 0.05$) than that for targets. The distribution of the P300s generated by the shock novels and the tactile novels were similar ($F (2, 10) = 0.81, P = n.s.$).

Fig. 3. P300 amplitude habituation with repetitions of shock novel, tactile novel and target events. P300 amplitudes on each set of 3 consecutive events were averaged across 4 blocks and 12 subjects. Note the rapid habituation of the novel P300 amplitude.

Fig. 4. Normalized mean amplitude of P300s evoked by targets, tactile novels and shock novels plotted as a function of electrode locations. P300 amplitudes were normalized within each subject and averaged across 12 subjects.
P300 amplitude and latency for target and novel stimuli were comparable for right- or left-hand stimulation at midline scalp sites. P300s evoked by both target and novel stimuli showed symmetrical scalp amplitude distributions for both left- and right-hand stimulation.

**Behavior**

Mean reaction times to targets were 456 msec for right-hand stimulation and 467 msec for left-hand stimulation ($P = \text{n.s.}$). The correct detection rate (correct hits/total number of targets) was 98% and false alarm rate [(total hits – correct hits)/total hits] was 3% for both hands of stimulation.

**Discussion**

In the current study both target and novel somatosensory stimuli generated P300 ERPs. P300 ERPs to novel stimuli were first reported in the visual modality (Courchesne et al. 1975). Novel visual stimuli evoked large (14–17 $\mu$V), fronto-central P300 ERPs with latencies of 360–380 msec. Correctly detected task-relevant visual stimuli evoked posteriorly distributed P300s (11–15 $\mu$V) with latencies of 380–430 msec in the same subjects. Non-attended auditory deviants were initially reported to generate an earlier latency (about 240 msec), fronto-central P3a response (Squires et al. 1975). In an auditory discrimination task, infrequent, unpredictable novel sounds generated a fronto-central P300 with mean latency of 367 msec, which was different in topography and latency from the parietal maximal P300 (mean latency 388 msec) evoked by the attended target sounds (Knight 1984).

The P300 amplitude evoked by the shock novels in the current study was larger than that evoked by the tactile novels. The shock novels were more deviant from the targets than the tactile novels. In the visual modality, greater contrast from the targets generated higher amplitude novelty P300s (Courchesne et al. 1978). Thus, the amplitude of the novelty P300 seems to depend on the degree of stimulus novelty or the magnitude of the deviation relative to the standard stimuli. Shock stimuli also produce some aversive feeling, which as in the auditory modality (Roth et al. 1984) may contribute to the amplitude of the shock novelty P300.

The amplitude of the somatosensory novelty P300, but not the target p300, habituated rapidly. The habituation of the novelty P300 was similar to that reported in experiments that used a fixed non-target deviant through the experiment (Ritter et al. 1968; Woods and Elmasian 1986). The degree of habituation observed in the current study was less than that reported in the visual (42%, Courchesne 1978) and auditory (27%, Knight 1984) modalities. Less somatosensory novelty P300 habituation may be due to the fact that in order to determine twitch threshold shock stimuli were delivered prior to the experiment. This may have contributed to pre-experiment habituation of the novelty P300.

The peak latency of the P300 to shock novels was significantly shorter than the P300 to tactile targets. Generation of the somatosensory novelty P300 with a shorter latency than the target P300 is consistent with findings in the visual and auditory modalities (Courchesne et al. 1975; Knight 1984). The P300 has been proposed to reflect a CNS component of the orienting response (Ritter et al. 1968; Squires et al. 1975; Ford et al. 1976; Snyder and Hillyard 1976). The shorter shock novelty P300 latency may reflect automatic orientation, whereas target P300 latency may be prolonged due to the interposed decision process necessary before target P300 generation.

The topographical scalp distribution of the novelty P300 was different from that of the target P300. However, the shock novel P300 had a comparable scalp distribution to the tactile novel P300 in spite of differences in latency and amplitude. This similarity in topography suggests that the two novelty P300s share common generator mechanisms. The somatosensory novelty P300 had a centro-parietal scalp distribution whereas the visual and auditory novelty P300s have a fronto-central distribution. The shock novel stimuli employed in this experiment were different in comparison to previous visual studies. Whereas the same shock stimulus was always administered, different visual novel stimuli were presented on each trial in previous visual studies. Courchesne presented the hypothesis that readily categorized events elicit parietal P300 waves and those which cannot be categorized elicit frontal P300 waves (Courchesne 1978). Prior categorization of the shock stimuli in the training session and repeated administration of the same shock stimuli may have contributed to the more central somatosensory novelty P300 distribution. The scalp distribution of the novelty P300 was symmetrical to either hand of stimulation, indicating that bilaterally synchronous hemispheric generators or deep midline sources are active during novelty P300 generation.

Pfefferbaum et al. (1985) reported that the P300 to stimuli that required a subject to inhibit a response (no-go) was different from the conventional target P300. The no-go P300 is smaller, later in latency and has equal amplitudes at central and parietal sites compared with the target P300. The P300 to tactile novel stimuli had characteristics similar to the no-go P300. Thus, novelty P300 responses may be related to the no-go aspects of the task.

The current data indicate that task-irrelevant somatosensory novel somatosensory stimuli generate a P300 ERP. The P300 to the novel stimuli appears to be equivalent to the novelty P300 reported in the visual

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and auditory modalities for several reasons. First, the somatosensory novelty P300 was generated by task-irrelevant novel stimuli during an active discrimination task; second, the somatosensory novelty P300 ERP showed rapid habituation with repeated stimulus presentation; third, latencies were 20–30 msec earlier than the P300 to targets stimuli; and fourth, novelty P300 amplitudes were maximal at central scalp sites. These data indicate that P300 responses are generated to relevant and deviant stimuli in all sensory modalities.

References


