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Brain event-related potential correlates of overfocused attention in obsessive-compulsive disorder

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Abstract

A hypothesis of overfocused attention in obsessive-compulsive disorder was investigated by measuring auditory event-related potentials (ERPs) during a selective attention task. Unmedicated patients ($n = 18$) with this disorder showed significantly larger attention-related processing negativity (PN), with earlier onset and longer duration, than did normal controls ($n = 15$). In the N200 region (160–250 ms), PN was larger in patients with fewer nonspecific neurological soft signs. This task, however, did not yield any group differences in mismatch negativity (N2a) or classical N200 (N2b). P300 amplitudes for attended targets were smaller for patient than normal groups, but the reverse was true for P300 and positive slow wave amplitudes for unattended nontargets. Collectively, these ERP abnormalities suggest a misallocation of cognitive resources. Because of the importance of the frontal lobe in the control of selective attention, PN enhancement in patients with obsessive-compulsive disorder may reflect hyperactivation of this region. This conceptualization is consistent with recent functional neuroimaging findings.

Descriptors: Obsessive-compulsive disorder, Event-related potentials, Processing negativity, N200, P300, Overfocused attention

Although much has been learned about obsessive-compulsive disorder (OCD) in recent years, mystery still surrounds this psychiatric disorder, especially concerning its cognitive dysfunction (Insel, 1992). Intrusive thoughts (obsessions) and/or repetitive behaviors (compulsions) that characterize this disorder are also present in other mental disorders, such as schizophrenia. OCD symptoms, however, are unique for being both egodystonic and associated with relatively intact reality testing. Understandably, this can intensify the displeasure associated with unwanted symptoms in these patients, who typically describe their obsessions or compulsions as "crazy" and "shameful" in nature. From a clinical perspective, these patients tend to exhibit heightened arousal and chronic vigilance concerning their symptoms and other salient stimuli. Heightened arousal and overfocused attention have also been key areas of interest in the investigation of the psychobiological basis of cognitive dysfunction in this disorder (e.g., Baxter et al., 1987; Turner, Beidel, & Nathan, 1985).

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Endogenous event-related potentials (ERPs) provide a means for the joint study of psychological measures of cognitive operations and electrophysiological measures of brain functioning (Donchin, Ritter, & McCallum, 1978; Sutton, Braren, Zubin, & John, 1965). This capability for illuminating both psychological and brain processes renders ERPs ideal for studying the physiological correlates of attentional or other cognitive dysfunction in psychopathology. Although relatively few ERP studies of OCD have appeared in the literature over the past 10–15 years, reports of ERP abnormalities in this clinical group have been relatively consistent. For example, studies in somatosensory (Shagass, Josiassen, & Roemer, 1988; Shagass, Roemer, Straumanis, & Josiassen, 1984a, 1984b), visual (Beech, Ciesielski, & Gordon, 1983; Ciesielski, Beech, & Gordon, 1981), and auditory modalities (Towey et al., 1990) have found evidence of distinctive ERP features in this disorder. Compared with ERPs of normal controls or other psychiatric groups, ERPs of OCD patients were distinguished by enhanced negativities, reduced latencies, or both.

Shagass et al. (1984a, 1984b, 1988) focused on early and middle latency evoked potentials for somatosensory, auditory, and visual stimuli. Although the evoked potentials of OCD patients differed from those of normal controls in several respects, one finding stood out as being specific to this disorder; namely, the negativity in the N60 region produced by somatosensory stimuli was larger in this patient group than in any other patient

group (including chronic schizophrenics, depressives, or other neurotics). After summarizing neurological, ERP, and electroencephalographic (EEG) findings, Shagass et al. (1984a) concluded that converging evidence does not fit any one theory of cerebral dysfunction proposed for OCD but instead provides partial support for each of three major theories: (a) increased local cortical excitability (Williamson, Allison, Goff, & Mattson, 1977), as associated with lesions in epileptics; (b) left frontal dysfunction (Flor-Henry, Yeudall, Koles, & Howarth, 1979), as associated with a loss of inhibition in these regions; and (c) increased arousal to minimal stimulation (Beech et al., 1983), as associated with cortical hyperactivation.

Ciesielski et al. (1981) and Beech et al. (1983) found distinctive features in the late ERP components of obsessional neurotics. These patients showed shorter latencies and smaller amplitudes of N200 and P300 than did matched normals during visuospatial tasks. This group difference was maximal when the complexity of the information processing requirements was greater. The findings of reduced latencies of N200 and P300 components in OCD are noteworthy because investigators have typically reported that other groups of psychiatric patients, for example, those having schizophrenic or depressive disorders (Blackwood et al., 1987; Bruder et al., 1991; Josiassen, Roemer, Shagass, & Straumanis, 1986; Levitt, Sutton, & Zubin, 1973; Roth, Duncan, Pfefferbaum, & Timsit-Berthier, 1986; Zubin, Sutton, & Steinhauer, 1986), show either no difference from or longer latencies than normal controls (as well as smaller amplitudes).

In our initial study (Towey et al., 1990), OCD patients showed abnormal latencies and amplitudes of endogenous ERPs during an auditory oddball discrimination. Subjects were instructed to depress a button as soon as possible after hearing the rarer and louder of two alternating clicks, which differed from frequent nontarget clicks by 12 dB (easy task) or 8 dB (difficult task). Patients had larger negative amplitudes than did normal controls in N200 and slow wave regions during both easy and difficult tasks. Also, normal controls had longer N200 and P300 latencies during the difficult than during the easy task, but the patients did not. Towey et al. (1993) confirmed these findings with larger sample sizes and found that the enhanced negativity in the N200 region was correlated with less severe obsessions, better response to subsequent treatment with serotonin reuptake blockers, and fewer neurological soft signs. Moreover, N200 amplitude was larger over left than right hemispheres of OCD patients but not of normal controls.

At least three ERP components could have been responsible for the increased negativity in the region of N200: (a) mismatch negativity (MMN or N2a), (b) classical N200 (N2b), and (c) processing negativity (PN). Mismatch negativity appears to reflect the brain's automatic response to changes in repetitive auditory input and to reveal that the physical features of auditory stimuli are fully processed whether or not they are attended (Näätänen, 1990). Because MMN is thought to be part of the brain's orienting response to a stimulus, which is primarily sensitive to the physical characteristics of the stimulus and relatively insensitive to cognitive/task factors (Näätänen, 1990), MMN contrasts sharply with the other two negativities in question. Although N2b is less well understood than MMN, it seems to be sensitive to the significance of the stimulus, that is, to reflect cognitive/task factors rather than physical parameters of the stimulus. When the subject is told to ignore oddball tones, for example, N2b is not observed. Thus, in contrast to MMN, N2b is believed to reflect controlled processing (e.g., target detection) and requires attentional resources for its generation (Cammann, 1990). During directed listening tasks, PN appears to reflect a mechanism for selectively attending to stimuli defined by certain features; it is thought to be generated by a cerebral comparison or matching process by which the brain selects relevant stimuli from among irrelevant stimuli for further processing, when they differ in some physical feature such as spatial position or pitch (Näätänen, 1990).

The present study used an auditory selective attention task to evaluate whether PN is altered in OCD, especially in ways that could have contributed to the enhanced and extended negativities seen in our previous study of this clinical group (Towey et al., 1990). Given that patients with this psychiatric disorder have been characterized as exhibiting chronic overaroused and overfocused attention to their symptoms and other salient stimuli, it is logical to hypothesize that PN, as an ERP correlate of focal attention, might be exaggerated in this clinical syndrome. Because a larger PN is elicited by stimuli in the attended channel (both deviant targets and standard nontargets) than in the unattended channel, PN is normally measured as a negative difference waveform (Nd = attend − unattend nontarget waveforms). Differences between OCD patient and normal groups in Nd, therefore, might result from PN differences between groups in the attended channel, the unattended channel, or both. This source of ambiguity was dealt with in the present study by including both *attend* and *unattend* conditions in the statistical analyses, such that group differences in level of negativity to each condition could be examined along with group differences in Nd.

Although not ruling out the possible contribution of MMN or N2b, PN was considered the most plausible explanation because it could account not only for enhanced negativity in the region of N200, but also later slow wave activity. PN is known to develop over time during selective attention to auditory stimuli and to be characterized by early (100–300 ms) and late (300–500 ms) phases (Näätänen, 1982), although these latencies can vary as a function of task requirements. The early and late PN phases overlap the N200 and slow wave regions, respectively. It was hypothesized that cerebral overactivation in OCD is associated with overfocused attention, which results in exaggerated attention-related PN in its early phase and probably also its late phase. In addition to evaluating this hypothesis, the present study also measured neurological soft signs so as to extend our initial finding of a relationship between this measure of nonspecific neurological impairment and N200 amplitudes in OCD patients (Towey et al., 1993).

Method

Subjects

Patients were recruited from the Anxiety Disorders Clinic at New York State Psychiatric Institute. All clinical aspects of this study (i.e., screening, recruitment, clinical ratings, neurological soft sign examination, and drug treatment) were performed by the Anxiety Clinic staff under the supervision of Dr. Eric Hollander. Normal controls were recruited by local advertisement or by referral from a screening service for volunteers, recently implemented at New York State Psychiatric Institute.

There were 18 patients (14 men, 4 women; age: $M = 30.0$ years, $SD = 9.1$ years) and 15 normal controls (10 men, 5 women; age: $M = 32.5$ years, $SD = 9.6$ years). Each participant was screened

for normal hearing (i.e., hearing loss <30 dB or an asymmetry <10 dB between ears at 500, 1000, 2000, and 4000 Hz), right handed, between 20 and 60 years of age, and without any major medical problem. Patients were drug free for at least 2 weeks prior to ERP testing, met DSM-III-R (American Psychiatric Association, 1987) criteria for OCD, had no focal lesion on neurological examination, and had Hamilton Depression Scale scores below 16. Neurological soft signs were examined using a method described elsewhere (Hollander et al., 1990). Normal controls did not meet criteria for any major psychiatric disorder by semi-structured psychiatric interview. Diagnosis was confirmed independently by a trained clinician using a modified Schedule of Affective Disorders and Schizophrenia—Lifetime Anxiety version (Manuzza, Fyer, Klein, & Endicott, 1986). Each participant was instructed not to discuss anything with the ERP staff that might reveal their diagnostic status and received $10/hr for participating in the ERP testing.

ERP/EEG Recording

EEG was recorded from 16 standard 10-20 system leads (standard electrode position nomenclature, 1990, American EEG Society) with an Electrocap (Neuroscience Model), all referenced to nose tip. Left mastoid was used for ground. The following scalp placements were used for recording sites: (a) frontal: Fz, F3, F4, F7, F8; (b) central: Cz, C3, C4; (c) temporal: T3, T4; (d) parietal: Pz, P3, P4; and (e) occipital: Oz. Electrooculographic artifact was monitored from electrodes at supra- and infraorbital sites of the right eye for vertical eye movements and at outer canthi of both eyes for horizontal eye movements (both bipolar). EEG data were recorded using a 16-channel Grass Neurodata system at 10 K gain, with a 0.01-30-Hz bandpass. Data were acquired at 100 samples/s. The acquisition system used two IBM-compatible computers with a continuous data digitization system. One computer was used for stimulus delivery and/or the timing and coding of events, and the second one was dedicated to the acquisition of data and stimulus-response timing information.

Directed Attention Task

The task was a modification of one used by Baribeau-Braun, Picton, and Gosselin (1983) in a study of attention in schizophrenia. Subjects received two concurrent randomized sequences of tone pips (73 dB and 250 ms duration, with 25 ms rise/fall times), one to the right ear and the other to the left ear, with equal probability of occurrence in either ear. The interstimulus intervals varied randomly from 1,200 to 1,600 ms (M = 1,400 ms), and the order of presentation of tones to the right or left ear was also random. The standard nontarget tones in the sequence were 1000 Hz in the right ear and 500 Hz in the left ear. The deviant target tones were 750 Hz and occurred randomly in each ear with a probability of 0.10. There were no constraints on consecutively occurring targets. An independent sequence of 100 tones was presented to each ear in each block. The *attend right* condition consisted of three consecutive blocks of trials, during which subjects were instructed to attend only to the right ear and press the response button with their dominant hand as soon as they heard the infrequent target tone in that ear. The *attend left* condition consisted of three consecutive blocks during which subjects were instructed to attend and respond only to targets in the left ear. The order of the attend left and attend right conditions was alternated across subjects in each group. Before experiencing either selective attention condition (attend left or attend right), each subject received a monaural, practice block (left oddball or right oddball), so as to familiarize subjects with the task demands, as well as the standard tones (nontargets) and the infrequent tones (targets) used in each condition.

ERP/EEG Analyses

Continuous EEG data were segmented so that each record extended from 200 ms prior to stimulus onset to 1,080 ms after it. ERP data segments were then averaged offline according to whether the stimulus occurred in the attended ear or unattended ear and whether it was a target or a nontarget. Trials contaminated by blinks or large-amplitude eye movements were excluded from analysis offline whenever the vertical eye channel exceeded 100 μV or the horizontal eye channel exceeded 25 μV. EEG channels used a cutoff of 75 μV. Eye movements below the threshold for rejection were corrected on a trial-by-trial basis using a standard linear correction procedure (Semlitsch, Anderer, Schuster, & Presslich, 1986).

Averaged ERP waveforms were computed for each event type (correct trials only) at each electrode for each subject and across subjects. Averaged voltages (relative to prestimulus baseline) were used to measure amplitudes of ERP components visible in averaged waveforms for target stimuli in four latency windows: N100 (70–150 ms); N200 (160–250 ms); P300 (260–550 ms); and slow wave (500–1,000 ms). These same latency windows were used to evaluate possible group differences in PN, which were evident in the averaged waveforms for nontarget stimuli. Use of these latency windows permitted a comparison of findings for ERPs observed in the target waveforms (e.g., N200 and P300) and ERPs typically measured in the nontarget waveforms (PN).

This decision to utilize the same windows to evaluate both target and nontarget waveforms was appropriate based on empirical grounds. Specifically, the N200 window bracketed the peak PN for both groups, and the early window allowed for an evaluation of group differences in PN onset, whereas the late window permitted an evaluation of group differences in PN duration. Thus, the following four latency windows were used for PN analyses: early (70–150 ms), peak (160–250 ms), middle (260–550 ms), late (500–1,000 ms).

In the interest of clarity, group differences in PN in each window were evaluated in an analysis of variance (ANOVA) of average voltages for nontargets for attend and unattend conditions (i.e., the two components of the Nd measure of PN). In this repeated-measures ANOVA (which used Greenhouse-Geisser adjusted degrees of freedom when appropriate), the between-group variable was group (patient, control) and the within-group variables were condition (attend, unattend), ear (left, right), hemisphere (left, right), and electrode (F7–F8, F3–F4, T3–T4, C3–C4, P3–P4). Statistical analysis for these lateral pairs of electrodes was performed to examine the possibility of hemispheric asymmetry for PN. The same ANOVAs as outlined above were performed on amplitudes for both target and nontarget stimuli (i.e., stimulus was included as a variable) to permit the evaluation of group differences in other ERP components of interest, such as N200 and P300.

As a confirmatory adjunct to the above analyses, ANOVAs were also computed for the full array of electrodes so as to include the midline sites (Fz, Cz, Pz, Oz). In addition, an analysis evaluated possible group differences in onset latency of Nd at Cz. The Nd waveforms for this analysis were constructed from the attend minus the unattend waveforms (using wave-

forms derived from left and right stimuli, weighted equally). After applying a digital lowpass filter (7 Hz cutoff; 96 dB/octave), the quarter peak latencies of the Nd were measured for each subject and compared across groups using a *t* test. The filter was used to attenuate alpha band noise to facilitate scoring in a few subjects, although it had negligible effect otherwise.

Results

Behavioral Data

Accuracy scores during the directed attention task did not differ statistically for clinical patient and normal control groups. Target detection was near perfect for both groups. Total hits were 97.4% ($SD = 3.3$) for normal controls and 96.4% ($SD = 3.1$) for patients; hits were operationally defined as a button press occurring from onset of a target stimulus to just prior to the next stimulus. There were very few false alarms (0.3% for controls, 0.5% for patients). Groups also had virtually equivalent response times for correct detections: mean response time of 552.1 ($SD = 93.2$) ms for normal controls and 549.4 ($SD = 94.6$) ms for clinical patients.

ERP Data

Processing negativity (PN) was the attention-related ERP of primary interest in this study. Thus, figures and ANOVAs focus on answering empirical questions regarding this component, for example, whether PN is enhanced, extended, and/or lateralized in OCD. Other endogenous ERPs of interest (i.e., MMN, classic N200, P300, and slow wave) were examined within the same framework for comparison purposes and for consistency and clarity of the ERP data presentation.

PN. Figure 1 shows the average waveforms for nontarget stimuli recorded at Cz and collapsed across left/right ear presentation. Waveforms for attend and unattend conditions are shown in the top panel for the normal controls and the middle panel for the patient group. For both groups, attend and unattend condition waveforms begin to diverge 100–200 ms after stimulus onset and are maximally different in the 200–350-ms region, with some differences extending into the subsequent slow wave region. In general, the divergence between attend and unattend condition waveforms appears to be larger and more extensive for patient than normal groups.

The bottom panel of Figure 1 shows the corresponding attend minus unattend waveforms, that is, the negative difference waveforms (Nd), for the patient and normal groups. As expected, this waveform has an early and late phase. The early phase, peaking at about 200 ms, is prominent for both groups, and the late phase is more distinct for patients. Most strikingly, patients produced an Nd waveform that starts earlier, remains larger, and ends later than the Nd waveform for normal controls. Although the bottom panel of Figure 1 depicts Nd from Cz only, that is, where PN appears to be most prominent, Figure 2 shows that the same group differences in Nd were also evident at the other scalp sites. As expected, PN appears to be larger at anterior (frontal and central) than posterior (parietal and occipital) electrode sites (Näätänen, 1990). However, patient − normal differences in Nd waveforms are clearly visible over the full array of scalp electrodes.

Group differences in PN (as measured by Nd) emerged in the ANOVAs for nontargets as interactions between group and condition (attend, unattend). Simple effects analyses were computed

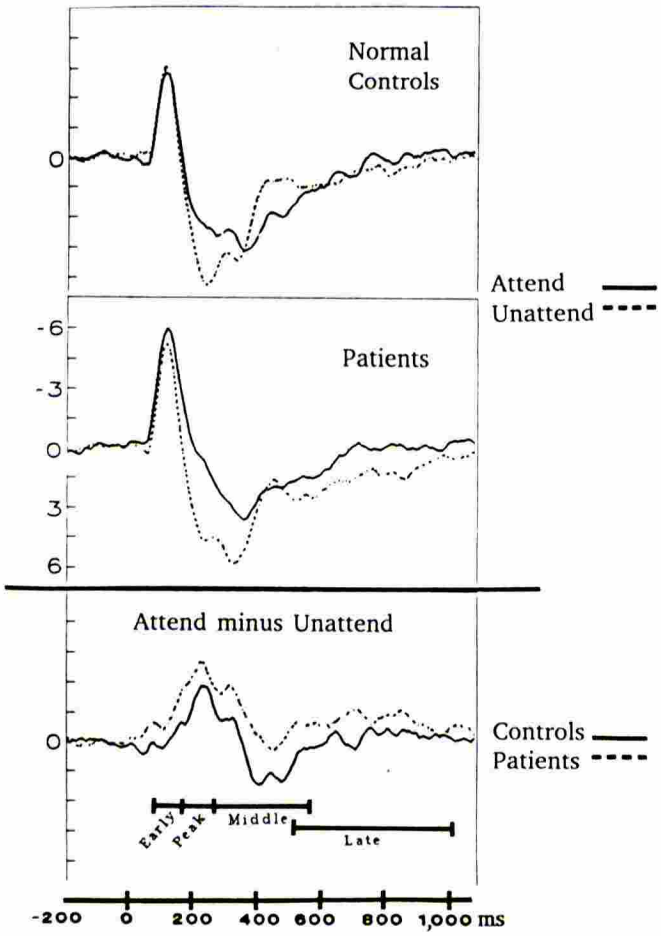

Figure 1. Nontarget waveforms (Cz) for each condition (attend and unattend) for normal (top panel) and patient (middle) groups and the difference waveforms (Nd, attend minus unattend) (bottom).

for each group to help interpret these interactions. Figure 3 illustrates the effect of directing attention for each group, which can be clearly seen by comparing attend and unattend condition waveforms. A decrease in positive activity is equivalent to increased negativity on the positive side of baseline. Nd can be visualized by inspection of the slope of the lines—the steeper the slope between attend and unattend (where attend is more negative or less positive), the greater the magnitude of Nd.

Table 1 summarizes the significant results for the data in Figure 3. The normal controls showed a significant difference between attend and unattend conditions in the peak window, as seen in the simple effects analysis, but showed no evidence of Nd in the other windows. In contrast, patients showed significant Nd in all four latency windows. The difference in Nd effects between patient and normal groups was reflected in significant Group × Condition interactions for each latency window. There were no significant hemisphere effects or interactions involving group and hemisphere. The significant Group × Condition interaction for the early epoch suggests that Nd was present at an earlier latency in patients than in controls. To further examine this possibility, quarter peak latencies of the Nd waveform at Cz were analyzed for each subject. Quarter peak latency of the Nd waveform at Cz was significantly shorter ($t[31] = 2.49$, $p = .018$) for patients ($M = 80.0$ ms, $SE = 15.2$ ms) than for nor-

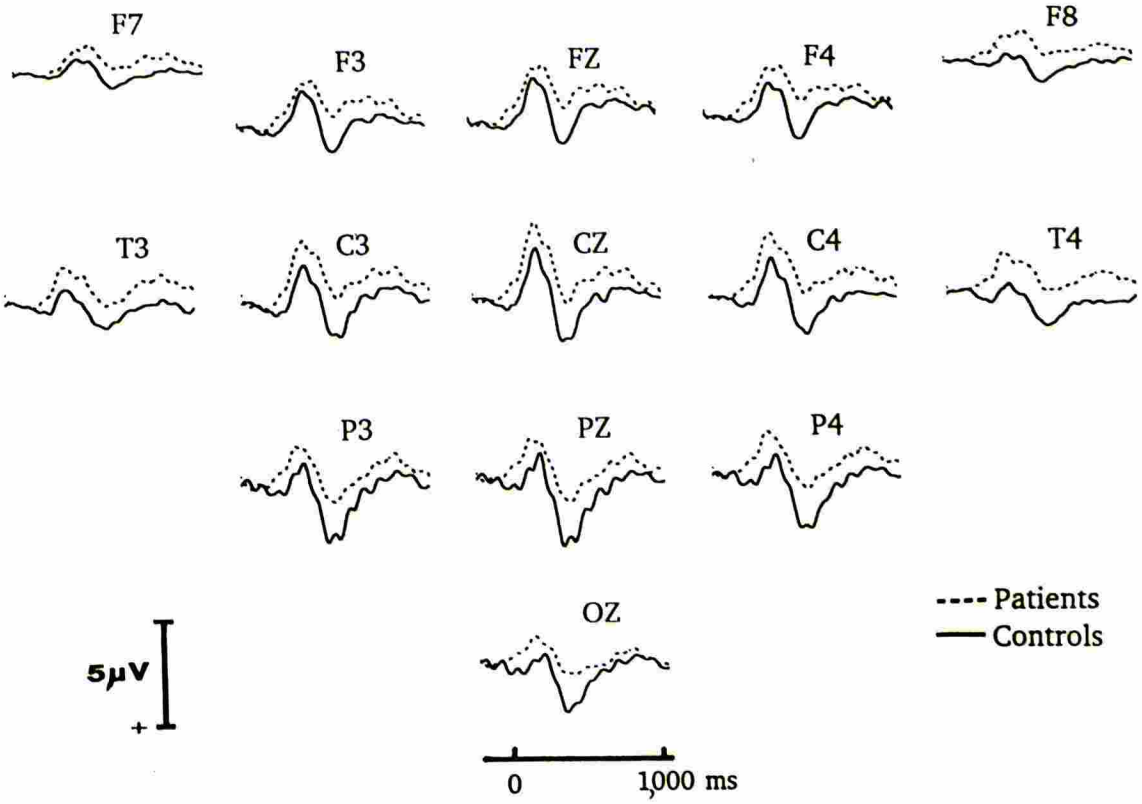

Figure 2. Topographical distribution of Nd.

mal controls ($M = 131.2$ ms, $SE = 13.2$ ms). This difference confirms that Nd had a shorter latency for patients than for controls.

In addition to these group differences in Nd, patient and normal groups also differed significantly in averaged voltage in the peak window (160–250 ms) (group: $F[1,31] = 6.95$, $p = .013$). As can be seen in Figure 3, patients showed overall greater negativity (less positivity) to nontarget stimuli than did normal controls during both attend and unattend conditions. Figure 3 also illustrates the origin and time course of the Nd differences between patients and controls. Group differences for the early epoch are due to enhanced negativity elicited by the attended nontargets in patients, whereas those for peak and middle epochs reflect differences in both attended and unattended nontargets. The effect for the late epoch appears mainly due to group differences in the ERP amplitude for unattended nontargets.

Nd and neurological soft signs. A correlation was computed between Nd amplitude in the peak window (i.e., attend minus unattend difference in 160–250-ms region) and total number of neurological soft signs. Patients with larger Nd tended to have fewer neurological soft signs ($r = .56$, $p < .05$). This result is consistent with a prior finding of larger N200 amplitude associated with fewer neurological soft signs in OCD patients (Towey et al., 1993).

Other negative ERPs. Figure 4 shows the ERP waveforms for rare and frequent stimuli in the attended and unattended channels. Only midline sites are given because findings were essentially the same for midline and lateral sites. The difference

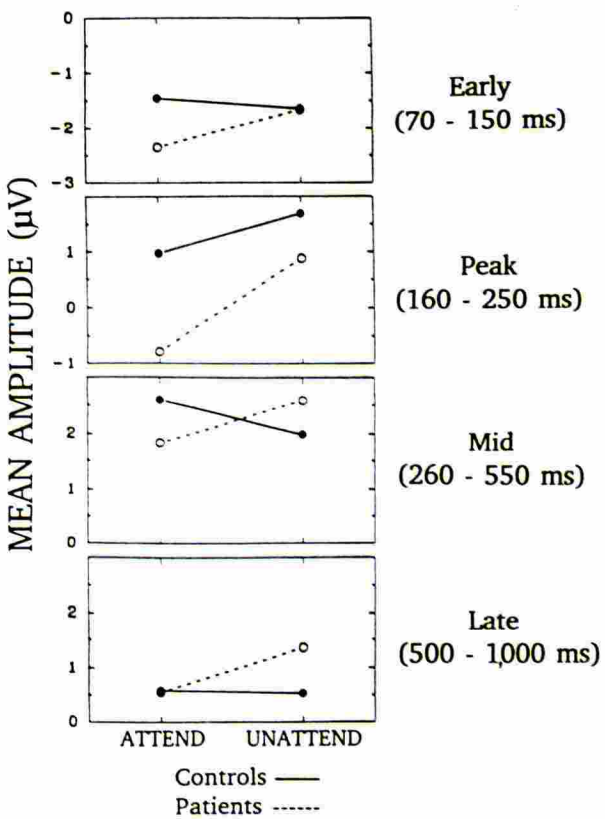

Figure 3. Mean ERP amplitudes for nontargets during attend and unattend conditions in early, peak, middle, and late windows, averaged over 10 lateral electrodes.

Table 1. *Significant Results for Nontarget Attend and Unattend Waveforms (Figure 3)*

Window	ANOVA	*F*	*df*	*p*	Simple effect	*F*	*df*	*p*
Early (70–150 ms)	Group × Condition	12.10	1,31	.0015	Patient	17.20	1,17	.0007
Peak (160–250 ms)	Group	6.95	1,31	.0130	Patient	51.91	1,17	.0001
	Group × Condition	6.88	1,31	.0134	Normal	12.32	1,14	.0213
Middle (250–550 ms)	Group × Condition	17.15	1,31	.0002	Patient	12.32	1,17	.0027
Late (500–1,000 ms)	Group × Condition	5.39	1,31	.0270	Patient	11.08	1,17	.0040

waveforms obtained by subtracting rare minus frequent waveforms are shown on the bottom. These difference waveforms permit group comparison of other negative ERPs, that is, MMN and N2b. MMN is known to reflect an automatic response to deviant stimuli and is seen most clearly in waveforms for unattended stimuli (Näätänen, 1990), where overlap with N2b is attenuated. The difference waveforms for the unattend condition (bottom left panel of Figure 4) show clear evidence of MMN, peaking at about 200 ms. An ANOVA of average voltage in the 160–250-ms (peak) window for unattended stimuli indicated that the groups did not differ significantly in MMN (Group × Stimulus interaction: $F[1,31] = 1.09$, $p > .25$). Classical N200, which is thought to reflect controlled processing during attended conditions (Näätänen, 1990), is most clearly seen in the difference waveforms for the attend condition (bottom right panel of Figure 4). Like MMN, N200 amplitudes did not differ for patient and control groups (Group × Stimulus interaction: $F[1,31] = 0.52$, $p > .25$). These analyses also showed no hemisphere or Group × Hemisphere interactions. The same findings held in ANOVAs of MMN and N200 amplitudes at only the lateral frontal sites (F3, F4). Separate ANOVAs of MMN and N200 amplitudes for the full array of scalp electrodes (i.e., including midline sites) also failed to detect a group difference. Thus, these findings suggest that the enhanced negativity in OCD patients was not reflected in either mismatch negativity or N200 components recorded during a selective attention task.

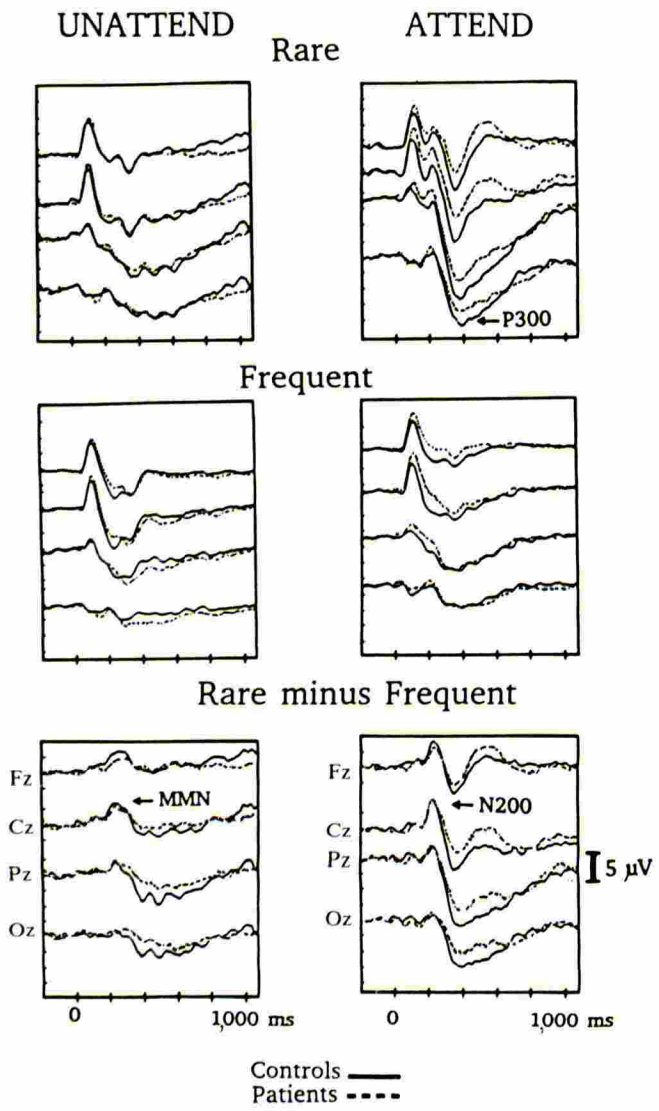

Figure 4. Group averages showing mismatch negativity (MMN), classical N200, and later positivity findings (P300).

Positive ERPs. Figure 4 also shows evidence of group differences in P300-like activity, that is, in the 260–550-ms window. P300 amplitude for attended targets (upper right panel of Figure 4) appears to be smaller in patient than in normal groups, but this group trend fell short of being statistically significant (Group: $F[1,31] = 3.91$, $p = .057$). P300 amplitude for unattended nontargets (middle left panel of Figure 4), however, was larger in patient than in normal groups at posterior electrodes (Group × Electrode: $F[2,61] = 4.03$, $p = .023$). Positive slow wave amplitude (in the 500–1,000-ms window) was also greater in patient than in control groups for the unattended nontarget condition (Group: $F[1,31] = 6.96$, $p = .013$). These group differences in P300 and slow wave were not dependent on hemispheric site, that is, there was no Group × Hemisphere interaction. Separate ANOVAs of the P300 and slow wave amplitudes for the full array of scalp electrodes (i.e., including midline sites) confirmed the above group differences at lateral electrodes.

Discussion

ERP findings of the present study are consistent with a hypothesis of overfocused attention in OCD patients. Most strikingly, unmedicated patients with this disorder showed enhanced and extended attention-related processing negativity (PN) when compared with that of normal controls. PN is thought to be related to a frontal lobe mechanism controlling directed attention (Näätänen & Picton, 1987). The enhanced PN in OCD patients

could therefore stem directly from hyperactivation of frontal lobe regions, which would also be consistent with evidence of hypermetabolism in the frontal lobe regions in this clinical group (for a review, see Insel, 1992). Baxter et al. (1987) suggested that the difficulty this clinical group has in diverting attention from their obsessions results from hyperactivity in frontal lobe regions responsible for regulating and directing attention and inhibiting reactivity to external interfering stimuli. Given the recent caution that regional metabolic changes of the brain may not be linked to mental function or to brain activity (Insel, 1992), the finding of enhanced attention-related PN in OCD patients is important because it provides a possible physiological link between abnormal metabolic and cognitive findings in this disorder.

The greater PN in OCD patients contrasts sharply with findings of reduced PN in schizophrenic patients using similar auditory selective attention paradigms (Baribeau & Laurent, 1986; Michie, Fox, Ward, Catts, & McConaghy, 1990). Further study is, however, needed to determine whether this and other findings in the present study are specific to OCD or are shared by other types of anxiety disorders. A study that employs anxiety controls (social phobic and panic patients) is underway.

Compared with that in normal control waveforms, P300 amplitude in patient waveforms was smaller for attended targets but larger for unattended nontargets. Positive slow wave amplitude for unattended nontargets was also larger for patient than for normal groups. P300 amplitude reflects the allocation of attentional resources to task relevant stimuli (Donchin, Karis, Bashore, Coles, & Gratton, 1986; Pritchard, 1981). Larger slow wave amplitude is thought to reflect further processing and increased cognitive effort (Ruchkin & Sutton, 1983). These ERP abnormalities, therefore, suggest that this clinical group may be misallocating cognitive and/or attentional resources when processing salient stimuli. The conclusion that greater cognitive and/or attentional effort was directed at processing unattended nontargets by the patient than by normal groups is consistent with the view that OCD is more properly conceptualized as an attentional dysfunction disorder (Drake et al., 1992). Recently, conflicting neuropsychological findings in this disorder were reviewed and explained in terms of reduced cognitive shifting ability, especially concerning nonverbal material (Head, Bolton, & Hymas, 1989). This deficit in cognitive shifting ability could be related to the growing body of evidence of attentional abnormalities in OCD.

ERP differences between the patient and control groups in processing negativity, P300, and positive slow wave were not accompanied by any group differences in performance measures of response accuracy or response times. In terms of distinguishing such patients from normal controls, therefore, endogenous ERPs seem to be sensitive to different aspects of information processing than are behavioral measures. A lack of behavioral differences between groups may indicate that a compensatory mechanism in OCD was operable and/or that the task was not cognitively demanding enough to elicit performance differences. Both groups performed close to 100% accuracy. Because of this ceiling effect, there remained little room for any advantage of enhanced attention to be manifest as improved performance. However, when a more difficult auditory oddball task was used in a prior ERP study of this clinical group (Towey et al., 1990), there was still no difference in either accuracy or reaction time between patient and normal groups. It remains unclear why OCD and normal groups show such differences in endogenous ERPs (e.g., Nd, P300, and slow wave) without any group differences in task performance measures. A follow-up study about to begin may clarify this issue because it employs multiple tasks at different levels of cognitive difficulty (i.e., binaural oddball, selective attention, and dichotic listening tasks).

Although patients had overall greater processing negativity than did normal controls, those patients with larger early Nd (160–250 ms) tended to have fewer neurological soft signs. Similarly, a different sample of OCD patients had overall greater N200 than did normal controls in an auditory oddball task, and those patients with larger N200 tended to have fewer soft signs (Towey et al., 1993). These patients also tended to have fewer obsessions prior to treatment and better response to subsequent treatment with a serotonin reuptake blocker. These findings are of significance given the recent recognition that OCD is a heterogeneous diagnostic syndrome, whose subtypes can be distinguished by clinical features such as treatment response and neurological soft signs (Hollander, DeCaria, Saoud, Klein, & Liebowitz, 1991). Negativity in the region of N200 appeared to distinguish two subtypes (Towey et al., 1993). One subtype with enhanced ERP negativity was relatively unimpaired neurologically, had mild to moderate obsessions, and was more responsive to treatment with a serotonin reuptake blocker. A second subtype with less negativity was more neurologically impaired, had more severe obsessions prior to treatment, and was unresponsive to drug treatment. Further study is needed to evaluate the potential value of processing negativity effects for differentiating between these two subtypes.

Further study is also needed to clarify N200 findings. Our initial study used a binaural oddball task and found enhanced N200 and negative slow wave amplitudes in OCD (Towey et al., 1990). The present study used a selective attention task and found larger and longer duration PN in patient than in normal groups but no group differences in mismatch negativity for unattended stimuli or classic N200 for attended stimuli. These results reinforce the suspicion that attention-related processing negativity may have contributed to our prior findings of larger negativity in N200 and slow wave regions for patients. However, the finding of enhanced N200 for patients in our prior study was larger over left than right hemisphere, whereas the finding of enhanced PN for patients in the present study was not lateralized. Hence, not all of the N200 effects seen for patients in an auditory oddball task may have been due to attention-related PN. One possible explanation for these effects is that directing attention to right or left ear stimuli alters the allocation of attentional resources to target and nontarget stimuli. As a result, N200 findings may differ for binaural oddball and directed attention tasks. Our next study will test patients in both binaural oddball and directed attention tasks with the same stimuli to permit a direct comparison of N200 findings across these paradigms.

Although the present study provides support for hypotheses of overfocused attention and cerebral (frontal?) hyperactivation in OCD, the exact neurophysiological basis remains unknown. Despite major advances in brain imaging technology, a clearly localized neuroanatomical substrate for this disorder has not been found (Insel, 1992). As Insel pointed out, studies using magnetic resonance imaging have failed to find evidence of brain structural abnormalities in patient versus control groups, whereas functional abnormalities have been found in this disorder using other brain recording technologies, especially positron emission tomography (PET) and single-photon emission computed tomography (SPECT).

Overall, findings based on brain recording technologies have been neither consistent nor pathognomic for OCD (Hollander et al., 1990). Inconsistencies could be due to one or more sources of variability within samples, such as symptom severity, diagnostic heterogeneity, and subtle brain abnormalities which are difficult to localize (i.e., neurological soft signs). Sampling error with small samples could understandably obscure group effects and yield inconsistent findings across or within different recording techniques. One solution to this problem is to increase sample size, but this is often impractical for many brain recording technologies. ERPs are a notable exception, however; they are relatively inexpensive and noninvasive and do not involve exposure to radioactivity. ERP technology is best suited for answering questions about the temporal domain (e.g., stages of information processing), and therefore, it can complement other brain imaging technologies that are more sensitive to the spatial domain. It would be of potential value, for example, to measure both attention-related ERPs and regional brain metabolism (e.g., by using PET or SPECT) in OCD and related psychiatric disorders, such as schizophrenia (Enright & Beech, 1990). Thus, imaging findings of hyperfrontality in OCD patients might prove to be directly linked with ERP findings of enhanced attention-related PN in the same patients. Similarly, imaging findings of hypofrontality in schizophrenia (e.g., Andreasen et al., 1992; Buchsbaum et al., 1992) might prove to be linked with ERP findings of reduced PN (Barieau & Laurent, 1986; Michie et al., 1990).

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