N2 and P3 components of event-related potential in first-episode schizophrenic patients: scalp topography, medication, and latency effects

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Abstract

Auditory N2 and P3 components of event-related potentials were assessed in first-episode schizophrenic and normal control subjects (n = 12/group). P3 amplitude was decreased in the patients most prominently over the frontal areas in contrast to a widespread P3 amplitude decrease reported in chronic schizophrenia. Moreover, frontal attenuation of P3 amplitude was greater in the non-medicated compared with medicated patients, a finding that suggests frontal areas are primarily affected at the onset of the first schizophrenic episode. Prolongation of N2 and P3 latencies was also observed in the patients, which indicates that stimulus classification and memory updating processes were slowed even in early stages of schizophrenia. These findings indicate that first-episode schizophrenic patients produce N2 and P3 abnormalities that are distinct from those in chronic patients, and that psychotropic medication can attenuate event-related potential effects in specific ways.

Keywords: First-episode schizophrenia; Event-related potentials; Auditory oddball paradigm; N2; P3; Frontal lobe

1. Introduction

The P300 or P3(b) component of the event-related potential (ERP) has been widely investigated in schizophrenic patients (Pritchard, 1986; Duncan et al., 1987; McCarley et al., 1991; Ford et al., 1992), with the main finding that auditory P3 amplitude is consistently smaller in patients relative to controls (e.g. McCarley et al., 1991; Ford, 1999; Jeon and Polich, submitted). The P3 is thought to be composed of an earlier and frontal/central maximum P3a potential, which is followed by a later and posterior maximal P3b waveform—the canonical P3(00) (cf. Picton,
1992; Polich, 1998). The P3a subcomponent reflects initial processing when a novel or distracting stimulus is detected, whereas P3b reflects the subsequent attentional resources when a target stimulus engages memory operations during task performance (Donchin and Coles, 1988; Katayama and Polich, 1998; Comerchero and Polich, 1999; Demiralp et al., 2001). Although the P3a has been little studied in these patients (Grillon et al., 1990; Merrin and Floyd, 1994), the P3b amplitude deficits observed in patients with schizophrenia are interpreted as indexing decreased attention capabilities (Baribeau-Braun et al., 1983; Barrett et al., 1986; McCarley et al., 1991; Ogura et al., 1991; Salisbury et al., 1994b). The effects of schizophrenia on P3 latency are not as consistent since some studies find prolonged latency (Muir et al., 1991; Glabus et al., 1994) and others report no reliable peak latency differences between patients and controls (Ogura et al., 1991), although a recent review reports significant P3 latency prolongation across studies (Jeon and Polich, submitted).

In addition, N2 amplitude is attenuated (O’Donnell et al., 1993, 1994; Ford et al., 1994; Salisbury et al., 1994a; Kasai et al., 1999; Laurent et al., 1999), and its peak latency prolonged in schizophrenia. These results suggest a disturbance and prolongation of the stimulus classification process (Brecher et al., 1987; Ogura et al., 1991; Hirayasu et al., 1998). Several studies of chronic schizophrenia showed that both N2 and P3 amplitude changes are correlated with illness duration (O’Donnell et al., 1993, 1995; Laurent et al., 1999). However, P3 amplitude deficits have been reported for early onset patients but not for the late onset form of the disorder, indicating that ERP changes reflect longer duration of illness and more severe information-processing deficits (Olichney et al., 1998). Few first-episode patients have been assessed using neuroelectric measures (Sponheim et al., 1994; Katsanis et al., 1996). A major ERP study did find P3 amplitude was abnormally small in medicated first-episode schizophrenia relative to first-episode affective psychosis patients and normal controls (Salisbury et al., 1998). Thus, as abnormal ERPs in first-episode schizophrenia cannot be due to chronic morbidity, long-term neuroleptic medication, or hospitalization, P3 component deficits may reflect disturbances of specific neurocognitive systems present at illness manifestation.

In this context it is important to note also that for chronic schizophrenic patients, medication does not increase reduced P3 amplitude to normal levels (Pfefferbaum et al., 1989; Anderson et al., 1991; Ford et al., 1994; Mintz et al., 1995; Mathalon et al., 2000)—a result that supports the suggestion that auditory P3 amplitude may be a trait marker for schizophrenia. However, a study of neuroleptic medication effects on patients with acute psychotic relapse demonstrated a return of P3 amplitude to normal level over the frontal regions but not over the posterior recording sites (Coburn et al., 1998). This finding suggests that in chronic patients medication may affect only the frontal aspects of the P3 ERP.

The present study was designed to address these issues by using auditory ERPs to assess a homogeneous group of first-episode, relatively young patients with schizophrenia whose medication status was well controlled. Patients with schizophrenia who were unmedicated or medicated were compared with well-matched unaffected controls. Given the findings outlined above, the major goals were: (1) to obtain a systematic analysis of ERP changes at schizophrenia onset; (2) to evaluate the effects of medication status in first-episode patients; and (3) to assay P3 amplitude scalp topography variation as well as the relative peak latency changes for both the N2 and P3 components.

2. Methods

2.1. Subjects

Patients were sampled from an ongoing prospective study of first-episode schizophrenia in the Psychotic Disorders Research Program, Department of Psychiatry, Istanbul Medical Faculty. A total of 12 right-handed in-patients (7 F, 5 M) were diagnosed as having schizophrenia by DSM-III-R criteria based on the SCID-I (Structured Clinical Interview for DSM-III-R, patient form) (Spitzer et al., 1992). The presence of the first psychotic episode was defined as no past diagnosis
of psychosis, an absence of previous in-patient care, and no previous antipsychotic medication. Other inclusion criteria were admission to in-patient care for at least 1 week and absence of organic disease with etiological significance. Mean duration of hospitalization was 38.6 days (18–57 day range). All patients were drug-naive when admitted to the hospital. At the time of ERP testing, six patients were not receiving any medication, and six were receiving zuclopenthixol to control the psychiatric symptoms of the acute phase (a thioxantane derivative, mean dose was equivalent to 315 mg/day chlorpromazine). Table 1 presents the mean scores on the Brief Psychiatric Rating Scale, the Scale for the Assessment of Positive Symptoms, and the Scale for the Assessment of Negative Symptoms. No significant score differences were obtained between the unmedicated and medicated patients.

2.2. Data acquisition

The subjects sat in an electrically shielded, sound-attenuated room that was dimly illuminated. The data were recorded from Ag–AgCl electrodes affixed with paste and tape at the midline (Fz, Cz, Pz) and lateral parietal (P3 and P4) locations, referenced to electrically balanced linked earlobes, with a forehead ground. The EOG was registered in horizontal and vertical directions for eye movement artifacts with bipolar recordings made from electrodes placed at the outer canthi of each eye and above/below the right eye. All electrode impedances were less than 5 kOhms. The bandpass filter was set at 0.1–70 Hz, with the data digitized at 250 Hz and stored on the hard disk of a PC for off-line analysis.

2.3. Stimuli and procedure

The stimuli were 1000 (standard) and 2000 (target) Hz tones of 900 ms duration (10 ms rise/fall), presented at 80 dB (SPL), with an inter-stimulus interval (ISI) of 2 s. The relatively long-duration stimuli were employed to avoid the superimposition of on- and off-potentials. The two different tones were presented in a random order, such that the target occurred on 20% and the standard occurred on 80% of the 150 total trials. The subject was instructed to keep a mental count of the target tone.

2.4. Data analysis

Single trials with EEG or EOG amplitudes exceeding ±90 μV were rejected automatically as artifact. The remaining trials were inspected visually for additional artifacts such as smaller EOG excursions and muscle activity. Target and standard stimulus trials were equated for the number included in the average by randomly selecting a subset of standard trials, with a mean of 26 for each trial type. P3 amplitude was measured relative to the mean of the prestimulus baseline, and peak latency was assessed as the time from stimulus onset to maximum peak amplitude within a 250–550 ms latency window. N2 component measures were obtained after subtracting the standards from the target waveforms and identified as the most negative point between 120 and 320 ms. This procedure was adopted in order to obtain N2 values relatively unaffected by the larger P3 component.

2.5. Statistical analyses

ERP measures were assessed in three ways: First, a two group (controls vs. patients) × three
midline electrodes (Fz, Cz, Pz) analysis of variance was performed on the amplitude and latency measures from each subject in each group. Second, a two group (controls vs. patients) × two parietal electrodes (P3, P4) analysis of variance was performed to assess lateralization effects. Third, medication status was assayed with similar analyses applied to three groups (controls, unmedicated, and medicated patients). Post-hoc comparisons employed the Scheffé procedure. Greenhouse-Geisser correction procedures were applied to the degrees of freedom when the repeated measure factor contained more than two levels, with only the corrected probability values reported.

3. Results

3.1. Task performance

Control and patient subjects performed the counting task in a highly similar and accurate fashion, with 30.1 ± 0.3 and 30.0 ± 1.8 mean target tones detected, respectively (t < 1, d.f. = 22, P > 0.85). Additionally, no difference in the target
3.2. ERP results

Fig. 1 illustrates the ERP grand averages from the first-episode schizophrenic patients and normal controls. The primary effects are the decrease of the P3 amplitudes in first-episode patients compared with controls, especially over the frontal electrode, and that N2 and P3 latencies are appreciably shorter for the controls relative to the patients. Fig. 2a presents the mean P3 amplitudes from the midline electrodes of the control, medicated, and unmedicated first-episode patients with schizophrenia. Fig. 2b presents the mean P3 latency for each of the three groups. Fig. 2c illustrates the mean N2 latency for the three groups (N2 amplitudes did not differ among the groups and are not presented). Table 2 displays the means and standard deviations of the N2 and P3 amplitudes and latencies. Table 3 summarizes the statistical analyses applied to the N2 and P3 amplitudes and latencies including the antero-posterior distribution (Fz, Cz, Pz) and laterality (P3 vs. P4) effects. Both analyses were carried out first for control vs. patient comparisons and then for comparisons among control subjects, unmedicated patients, and medicated patients.

3.3. N2 and P3 amplitudes

No significant differences were found for N2 amplitude. P3 amplitude was significantly smaller for the patients compared to controls \( (P < 0.05) \). More important, the schizophrenic patients evinced greater amplitude decreases at the frontal electrode.

Table 2
Mean (± S.D.) of N2 (top) and P3 (bottom) amplitude and latency measures for the unaffected controls and all patients with schizophrenia \((n = 12/\text{group})\) from each recording electrode.

<table>
<thead>
<tr>
<th>Channel</th>
<th>Controls N2 amplitude (µV)</th>
<th>Controls N2 latency (ms)</th>
<th>Patients N2 amplitude (µV)</th>
<th>Patients N2 latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fz</td>
<td>9.6 ± 2.8</td>
<td>315.0 ± 36.3</td>
<td>393.0 ± 60.4</td>
<td></td>
</tr>
<tr>
<td>Cz</td>
<td>10.7 ± 3.8</td>
<td>308.0 ± 42.6</td>
<td>393.7 ± 59.1</td>
<td></td>
</tr>
<tr>
<td>Pz</td>
<td>10.8 ± 4.3</td>
<td>304.7 ± 30.9</td>
<td>393.0 ± 62.3</td>
<td></td>
</tr>
<tr>
<td>P3</td>
<td>8.9 ± 4.6</td>
<td>320.0 ± 33.3</td>
<td>388.0 ± 60.7</td>
<td></td>
</tr>
<tr>
<td>P4</td>
<td>10.1 ± 4.5</td>
<td>315.0 ± 38.7</td>
<td>392.7 ± 60.9</td>
<td></td>
</tr>
</tbody>
</table>
Table 3
Summary of analysis of variance

<table>
<thead>
<tr>
<th>Factor (d.f.)</th>
<th>N2 Amplitude</th>
<th>N2 Latency</th>
<th>P3 Amplitude</th>
<th>P3 Vector</th>
<th>P3 Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>P</td>
<td>F</td>
<td>P</td>
<td>F</td>
</tr>
<tr>
<td>Group (1,22)</td>
<td>&lt;1</td>
<td>–</td>
<td>17.7</td>
<td>0.001</td>
<td>5.1</td>
</tr>
<tr>
<td>Electrode (2,44)</td>
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<td>–</td>
<td>0.4</td>
<td>–</td>
<td>16.9</td>
</tr>
<tr>
<td>G×E (2,44)</td>
<td>&lt;1</td>
<td>–</td>
<td>0.2</td>
<td>–</td>
<td>3.6</td>
</tr>
<tr>
<td>Group (2,21)</td>
<td>&lt;1</td>
<td>–</td>
<td>8.8</td>
<td>0.002</td>
<td>2.6</td>
</tr>
<tr>
<td>Electrode (2,42)</td>
<td>1.6</td>
<td>–</td>
<td>&lt;1</td>
<td>–</td>
<td>20.6</td>
</tr>
<tr>
<td>G×E (4,42)</td>
<td>&lt;1</td>
<td>–</td>
<td>&lt;1</td>
<td>–</td>
<td>2.8</td>
</tr>
<tr>
<td>Group (1,22)</td>
<td>&lt;1</td>
<td>–</td>
<td>18.3</td>
<td>0.001</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Electrode (1,22)</td>
<td>&lt;1</td>
<td>–</td>
<td>&lt;1</td>
<td>–</td>
<td>5.2</td>
</tr>
<tr>
<td>G×E (1,22)</td>
<td>1.5</td>
<td>–</td>
<td>&lt;1</td>
<td>–</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Top: Summary of analysis of variance (2 control vs. patient groups×3 midline electrodes) for the N2 and P3 measures from unaffected controls and all patients (n=12/group). Middle: Summary of analysis of variance (3 control vs. unmedicated vs. medicated patient groups×3 midline electrodes) for the N2 and P3 measures from the two patient subgroups (n=6/subgroup). The results of the vector transformation analysis performed on just the P3 amplitude data are also presented (see text for details). Bottom: Summary of analysis of variance (2 control vs. patient groups×2 parietal electrodes) for the N2 and P3 measures from unaffected controls and all patients (n=12/group).

relative to the controls as indicated by the significant interaction \( P<0.05 \). Post-hoc assessment demonstrated that this effect originated primarily from the frontal area (Fz vs. Cz and Pz: \( F=4.2, \text{d.f.}=1,22, P<0.05; \text{Cz vs. Pz: } F=3.0, P>0.09 \)). Thus, P3 amplitude was smaller in the first-episode patients compared with controls, with the frontal area specifically affected relative to the other electrodes.

To assess whether the group by electrode interaction was topographically robust, P3 amplitudes were re-analyzed after the data were normalized by vector length (McCarthy and Wood, 1985). The P300 component amplitude value from each subject was divided by the square root of the sum of the squared amplitudes over the three midline electrode locations from that subject’s group. This analysis normalized the group effect between the controls and patients so that only the scalp distribution information was compared between both groups (Johnson, 1993). The vector analysis on the P3 amplitudes increased the significance of the group×electrode interaction effect \( P<0.003 \), which substantiates the claim of the predominantly frontal amplitude decrease in the first-episode schizophrenic patients relative to the unaffected controls.

The second analysis found that P3 amplitudes from the lateral parietal line demonstrated no difference between groups. However, significantly larger P3 amplitudes for the right \( (P4) \) compared with the left \( (P3) \) electrode \( (P<0.05) \) were obtained.

The third analysis included the medication factor, but no reliable overall group difference was found. However, as suggested by Fig. 2a, the two patient groups and controls did demonstrate a significant group×electrode interaction \( (P<0.05) \). Post-hoc tests revealed that this topographic effect between the control group compared with the medicated and unmedicated patient groups stemmed from the significantly smaller frontal amplitudes of the unmedicated patients compared with the control subjects \( (P<0.01) \). This outcome was confirmed after vector normalization \( (P<0.001) \). However, no other significant differences were obtained between any two groups in any other electrode location, between the controls com-
pared with the medicated patients \((P>0.15)\), or between the unmedicated and medicated patients \((P>0.48)\) for the frontal site.

### 3.4. N2 and P3 latency

In the first analysis carried out on data from midline electrodes, N2 peak latency was longer for the schizophrenic patients compared with controls \((256\ vs.\ 197\ \text{ms},\ P<0.001)\), as was P3 latency \((393\ vs.\ 309\ \text{ms},\ P<0.001)\). The second analyses of the parietal electrodes also found overall significant patient group prolongation for both N2 and P3 latencies \((P<0.001\ and\ P<0.001,\ respectively)\).

Analyses including the medication group variable also revealed overall group differences for both N2 and P3 latencies \((P<0.002\ and\ P<0.001,\ respectively)\). Post-hoc assessments found significant latency differences between normal controls vs. unmedicated as well as normal controls vs. medicated patients at each electrode site for both the N2 \((\text{Fz}:\ P<0.01\ and\ P<0.03;\ \text{Cz}:\ P<0.02\ and\ P<0.03;\ \text{Pz}:\ P<0.005\ and\ P<0.05)\) and P3 \((\text{Fz}:\ P<0.05\ and\ P<0.005;\ \text{Cz}:\ P<0.05\ and\ P<0.005;\ \text{Pz}:\ P<0.002\ and\ P<0.02)\) components, but not between medicated and unmedicated patients \((P>0.6\ for\ all\ conditions)\). Thus, medication did not affect the N2 and P3 latencies.

Given the consistent group differences for both N2 and P3 latencies, these data were further analyzed by assessing their correlational association independently for the controls and patients. Fig. 3 illustrates the scattergrams of N2 and P3 latencies (Pz electrode), with individual regression lines used to reflect the trends for each group. A significant linear association was found between component latencies for the control subjects \((r=0.46,\ P<0.01)\), with no reliable association obtained across all patients \((r=-0.09,\ P>0.75)\). Thus, the overall significant delays for N2 and P3 latencies obtained for the patients were accompanied by a striking latency dissociation between the peaks themselves for the patients relative to controls. The implications of this finding will be discussed below.
4. Discussion

4.1. P3 amplitude

First-episode schizophrenic patients demonstrated a frontal P3 amplitude decrease relative to controls. This effect is complementary to the P3 amplitude deficits observed in chronic schizophrenia (Ogura et al., 1991; Muir et al., 1991; Glabus et al., 1994) but is in contrast to the reported temporal-parietal P3 attenuation in first-episode schizophrenic patients discussed below (Salisbury et al., 1998). The general decrease of P3 amplitude in schizophrenia may reflect disturbed attentional resource allocation mechanisms, whereas the localized frontal decrease of the P3 amplitudes in first-episode schizophrenic patients in the present study may index frontal lobe dysfunction. Support for this view from previous studies is diverse but reasonably consistent: Frontal P3 amplitude from a selective attention task is smaller in chronic schizophrenia, which implies that component separation rather than structural deficit, whereas trait-dependent parietal P3 amplitude may be related to structural abnormalities in temporo-parietal regions (cf. McCarley et al., 1991; O'Donnell et al., 1993; Ford et al., 1994; Mintz et al., 1995; Mathalon et al., 2000).

In the only other ERP report on first-episode schizophrenic patients, Salisbury et al. (1998) found posterior midline and left temporal P3 amplitude reduction rather than the frontal ERP patient effects of the present study. The different outcomes may have originated from several factors. (1) The former study assessed patients who were all medicated whereas in the present study half of the patients were unmedicated, with the frontal P3 amplitude difference significant between normal controls and unmedicated patients but not between normal controls and medicated patients. Normal P3 amplitudes over frontal areas also have been reported in medicated schizophrenics (Morrin et al., 1983). (2) The counting task performance for patients was significantly worse than that for controls in the former study, but counting task performance was virtually perfect for both patients and controls in the present study. As counting task performance determines the number of correct trials that enter the ERP average, such effects could contribute to differences in amplitude topography patterns between the studies. (3) The stimulus characteristics of the two studies were quite different, with relatively long and loud auditory stimuli and a large frequency difference between the standard and target tones employed here. Variation in auditory stimulus characteristics have been found to produce differential ERP effects in patients with schizophrenia and Alzheimer’s disease, as well as normal and elderly young subjects (e.g. Polich, 1986; Vesco et al., 1993; Salisbury et al., 1994b; Sugg and Polich, 1995; Polich and Pitzer, 1999), so that the longer and loud stimuli in the present study may have influenced the relatively higher frontal amplitude in the control group that would enhance the frontal P3 deficit in the patients. In sum, appreciable inter-study differ-
ences could have readily contributed to the P3 frontal vs. temporo-parietal differences between the previous and present study.

4.2. The hypofrontality hypothesis

The present findings are consonant with the hypothesis of prefrontal hypofunctionality in schizophrenia. For example, during mental cognitive task activity schizophrenic patients demonstrate increases in parietal blood flow, whereas unaffected controls produce increases in frontal blood flow (Ingvar and Franzén, 1974). Structural imaging has indicated that many schizophrenic patients have prefrontal sulcal prominence (Pfefferbaum and Marsh, 1995), as well as prefrontal and frontal gray matter deficits (Breier et al., 1992; Nopoulos et al., 1995). Positron emission tomography (PET) has revealed hypometabolism in the frontal lobes particularly on the left side and changes in the temporal lobes (Buchsbaum, 1990; Biver et al., 1995; Schröder et al., 1996), with a recent report demonstrating hypofrontality in unmedicated schizophrenia patients during performance of a serial verbal learning task (Hazlett et al., 2000). Functional magnetic resonance imaging (fMRI) findings support the PET results, with decreased activation in the right mesial prefrontal cortex and cingulate and the left thalamus of schizophrenics compared with controls during a continuous performance task (Volz et al., 1999). Thus, a variety of imaging studies suggest that structural and metabolic hypofrontality occurs in patients with schizophrenia (Velakoulis and Pantelis, 1996).

Despite the general consistency of the imaging results on hypofrontality, however, several state variables can affect patient measurement outcomes. For example, hypofrontality with rCBF is more readily observed during cognitive tasks relative to measurements during rest, although left anterior temporal rCBF levels at rest correlated positively with Positive Symptom total score during rest (cf. Parellada et al., 1998; Higashima et al., 2000). Hypofrontality in schizophrenia may be a dynamic phenomenon across time, possibly related to symptomatology levels (Spence et al., 1998). This hypothesis is supported by ERP findings that indicate a strong inverse relationship between frontal P3 amplitude (perhaps related to anterior cingulate activity) and increased severity of auditory hallucinations (Turetsky et al., 1998), as well as the medication effect of the present study. The sensitivity of ERP measures in this context is highlighted by fMRI findings of patients with schizophrenia during a working memory task, which found an increased activation in dorsolateral prefrontal cortex that was inversely correlated with task performance (Manoach et al., 1999). This result indicates that the neural circuitry involved in working memory may be relatively inefficient in the patients compared with controls. Finally, decreased frontal size may also be present at the onset of schizophrenia (Nopoulos et al., 1995), although HMPAO-SPECT evaluation has indicated hypofrontality was not present in the first episode of schizophrenic disorder but occurred after at least 2 years of illness (Ravizza et al., 1995). Taken together, neuroelectric measures of schizophrenia may therefore be more sensitive to frontal circuit dysfunction than to their overall hypofunction, as P3 amplitude in the frontal region may be an earlier indicator of hypofrontality than changes in rCBF patterns.

4.3. N2 and P3 latency

Previously, P3 latency prolongation in schizophrenia has generally been reported, although not consistently (Ogura et al., 1991; Muir et al., 1991; Jeon and Polich, submitted). However, the present findings indicate a clear prolongation of both N2 and P3 latencies in first-episode schizophrenia, which is consonant with prolonged reaction times in schizophrenia and implies a slowing of stimulus classification and memory updating operations (Roth et al., 1980; Pfefferbaum et al., 1984; Schwartz et al., 1989; Kemali et al., 1991; Anderson et al., 1995). N2 amplitudes from the difference waveforms did not discriminate between the first-episode schizophrenics and the controls, although decreased N2 amplitude has been reported for late-stage schizophrenics (Pfefferbaum et al., 1984; Ogura et al., 1991). Hence, it is reasonable to suggest that the N2 neural sources are not affected during the early stages of schizophrenia but deteriorate as the disease progresses. This
assumption is compatible with previous findings for chronic schizophrenics, where the decrease of the N2 amplitude was found to be associated with disease chronicity (O’Donnell et al., 1993).

The regression analyses on N2 and P3 latencies also support the notion that these ERP components are differentially affected by schizophrenia symptom onset. N2 and P3 latencies of the schizophrenics were not correlated, whereas the control subjects did show a strong relationship between peak latencies. Given the present results of unchanged N2 amplitudes and prolonged N2 latencies in conjunction with decreased P3 amplitude and prolonged latencies for schizophrenia patients, it is reasonable to suppose that the cognitive mechanisms reflected by the N2 and P3 components are differentially affected in the first-episode schizophrenics. Although the genesis of these effects is unclear, the normal N2 amplitudes and prolonged N2 latencies may imply that stimulus classification time is delayed in first-episode patients, whereas the smaller P3 amplitudes and delayed peak latencies would index deficits in attention allocation and memory updating processes that are affected by disease onset. However, because N2 and P3 latency are dissociated in the schizophrenia patients, the increased P3 latency cannot be attributed solely to the slowing of early information processing and disease chronicity (Jeon and Polich, submitted).

5. Conclusion

In contrast to the overall decrease of P3 amplitude in chronic schizophrenia and the temporo-parietal amplitude decrease reported in first-episode schizophrenics, the present findings indicate that the frontal P3 amplitude deficits are clearly evident in unmedicated first-episode patients. These outcomes suggest that medication effects must be taken into account with respect to overall and frontal P3 amplitudes in schizophrenic patients, because neuroleptic medication can reverse decreased component amplitude in frontal areas (Coburn et al., 1998). Finally, N2 and P3 latencies were independently prolonged in first-episode schizophrenics, such that stimulus classification and memory updating processes are separately affected in the early stages of schizophrenia. Taken together, these results contribute to the growing utility of ERPs as sensitive assays for this major psychiatric disorder.

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