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Altered electrophysiological pattern of target detection in schizophrenia in the Continuous Attention Test

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Abstract. Visual evoked potentials were examined in 50 male schizophrenic patients and 50 age-matched healthy male volunteers during performance of the Continuous Attention Test (CAT). In the patients emerged an evident deterioration of attentional processes: much higher index of errors and longer reaction time than in the healthy subjects. On the other hand, in patients the following alterations in evoked potentials were found: (1) in the non-target condition lower amplitudes of components: left-sided P1, bilateral N1 as well as frontal P3a; (2) a distinct electrophysiological pattern of target detection, consisting of an increase in amplitudes of either N1 or P3a as compared to the response to the non-target stimulus. Electrophysiological features were related to behavioural data. An increase in the amplitude of N1 during detection of the target, as well as a lower non-target amplitude of P3a, correlated with poor CAT performance, while an increase in the amplitude of P3a during detection of the target correlated with relatively shorter reaction time, and showed no correlation with an index of errors. The results suggest an underlying primary hyperarousability at lower stages of visual data processing (up to N1 latency range) accompanied by several secondary pathological and compensatory mechanisms at the higher stages.

Key words: schizophrenia, attention, event-related potentials

INTRODUCTION

Despite the unequivocal evidence confirming the presence of attentional disturbances in schizophrenia it has yet to be settled whether these are the cause or the effect of other symptoms of the disease. There is a considerable bulk of evidence showing that not only are conscious processes in schizophrenic patients less efficient, but also that this inefficiency is accompanied by hyperactivity at earlier, automatic stages of signal processing. One group of theories views disturbances of attention as a primary symptom, responsible for compensatory feed-back mechanisms which affect lower levels as increased sensory sensitivity (Baribeau-Braun et al. 1983, Mirsky and Duncan 1986). According to another group of theories, primary sensory hypersensitivity or disturbed signal filtering, which lead to distorted and non-selective perception, are responsible for informational "flooding" as well as secondary overload and diffusion of attention (Venables 1964, Shagass 1976, Freedman et al. 1983, Judd et al. 1992). Actually, in order to hypothesise about pathology of data processing, a link between neurophysiological and behavioural studies is necessary.

In numerous neurophysiological studies on consecutive stages of stimulus processing in schizophrenia, a number of changes related to event-related potentials (ERP) were described. However, the results were inconsistent, which in part was due to unestablished methodology: various paradigms, applied not always to the point, as well as various analyser systems examined. Furthermore, in the majority of those studies, psychometric tests used as generators of processed stimuli were applied only in order to provoke event-related potentials, and the assessment of relationships between task performance and neurophysiological signs of altered signal processing was restricted to a rather narrow range or neglected completely. Among the few exceptions is a series of studies by Strandburg (Strandburg et al. 1994a,b), concerning visual potentials evoked during work on the Span of Apprehension Task (SPAN) or Continuous Performance Test (CPT), where numerous ERP components were thoroughly taken into account and correlations between their parameters and behavioural data were analysed. However, despite numerous ERP findings observed in schizophrenic adults and children, there was a lack of direct correlations between those changes and test scores.

Among numerous tests of attention, the Continuous Performance Test has been proved to be the most efficient in studies on schizophrenia (for review see Cornblatt and Keilp 1994). Its easier versions, including the classical "X/AX" one, require the detection of a single symbol, i.e. "X" without or with priming by letter "A", in the random sequence of characters. Difficult variants, in particular the Identical Pairs version (IP-CPT) which requires detection of direct repetition of any symbol in the sequence, are shown to be sensitive for attentional deficits typical for schizophrenia, as revealing numerous errors of omission, independent of current clinical state. Moreover, IP-CPT is efficient not only in remitted patients but even in subjects with schizotypia (Grove et al. 1991) and appears to play a role as a predictor of a genetic predisposition to schizophrenia, with 91% specificity (Cornblatt and Erlenmeier-Kimling 1985, Winters et al. 1991), regardless of phenotypical expression. For these reasons the CPT-IP seems to be the best tool in studies on attentional disturbances in schizophrenia.

The purpose of the presented experiment was to assess visual potentials evoked by stimuli making up the Continuous Attention Test (CAT) (Tiplady 1992), which is a modified variant of IP-CPT, with non-verbal, abstractive stimuli. This test, previously applied in our laboratory to several diagnostic groups of psychiatric patients (Kasperska et al. 1996), yielded significantly inferior results in schizophrenic patients in comparison with those in patients with affective disorders and in healthy controls and that difference was not dependent on doses nor type of medication. The goal of the present study was an identification of electrophysiological correlates of attentional deficit in schizophrenia. In order to find them, the CAT scores were compared with alterations of the electrophysiological response for CAT stimuli.

METHODS

Subjects

Fifty-eight adult right-handed males (mean age 34.9 ± 9.5 years) with a diagnosis of schizophrenia according to DSM-IV (paranoid or undifferentiated type) and free of any accompanying diagnoses were examined. The patients were in a stable phase of illness (partial remission) and were taking supportive dozes of neuroleptic drugs. Mean intensity of symptoms according to PANSS scale (Kay et al. 1992) was 34 ± 18. Mean daily dose of neuroleptic drugs, converted to chlorpromazine equivalents, was 250 ± 165 mg. None of the patients received anticholinergic medication. Prior to the study an informed

consent from each subject was obtained. Eight patients were excluded from the final analysis: four because additional diagnoses emerged, and two due to a very poor test score in comparison with the other patients, which suggested they did not follow the instruction. In two cases the mean evoked potentials remained unanalysed for technical reasons. All in all, 50 patients were qualified for the psychometric and the evoked potential analysis.

Their results were compared to the data from 50 adult right-handed healthy male volunteers (mean age 33.0 ± 9.6 years) with no personal or family history of neurological or mental disturbances.

Procedure

THE CONTINUOUS ATTENTION TEST

The procedure was performed in a darkened, partially sound-proof room. Visual CAT stimuli were presented on a 14" monochromatic computer display with luminance of 10 cd/m^2, at a distance of 1 m from the eyes of the subject. The battery of randomly presented stimuli consisted of 40 target, 160 non-target and 40 atypical stimuli. The latter type of stimuli (digits), planned as feedback information, was introduced in our laboratory as a modification of the original CAT. However, in the present study subjects were not aware of their meaning and were asked to ignore them. Reactions for atypical stimuli are not analysed in this paper. Each of the 200 typical CAT stimuli was a geometrical pattern - a 3 x 3 matrix (10 x 10 cm) formed by random combinations of 5 bright and 4 dark squares. A target stimulus was defined as a direct repetition of any pattern and occurred with a probability of 0.16. The subjects were instructed to confirm the detection of a target stimulus by immediately pressing the button kept in the dominant hand. Stimulus exposure lasted 50 ms, and the interval between two successive stimuli varied randomly within the range of 1 to 2.5 s. Duration of a CAT session was approximately 8 min. In order to yield a larger number of hits, which was necessary for electrophysiological analysis (averaging), 3 sessions of CAT (separated by short, five-minute breaks) were performed, and their results were assessed jointly.

ELECTROPHYSIOLOGICAL DATA RECORDING

EEG signal was recorded from 21 derivations, according to the 10-20 system with additional Fpz and Oz electrodes. The movements of the left eye were recorded in a separate channel to allow subsequent artifact removal. The impedance of all electrodes was maintained below 5 kΩ. The EEG signal, referenced to linked mastoids, was amplified and filtered in the bandpass 0.15-30 Hz and digitized at 500 Hz sampling frequency and 12 bit resolution (Brainscope, MandI). Data recording computer procedure (EASYS2, Neuroscience) had an input from the other, stimulus generating computer, which enabled on-line registration of markers of CAT stimuli and reactions of patients. Evoked potentials were averaged off-line, after rejection of artifacts. They were related to pre-stimulus average EEG baseline and divided into separate classes dependent on kind of stimulus and reaction.

Data analysis

PSYCHOMETRIC RESULTS

In the CAT test we assessed the number of omissions (unidentified target stimuli) and commissions (button presses in response to non-target signals). These data were converted into standardized indices (Pigache 1976):

the omission index, $I_O = O/T$,

the commission index, $I_C = C/N$, and

the detection index, $I_D = 1 - O/T - C/N$ (with C/N as a correction for accidental hits),

where O = number of omissions, C = number of commissions (false detections), T = total number of target stimuli, N - total number of non-target ones.

Those indices were calculated in order to obtain a final total index of errors:

$I_E = (1 - I_D) + I_C = O/T + 2C/N$.

Mean reaction time was also measured.

Correlations of all the psychometric data with age, duration of illness, PANSS score and dose of drugs were examined.

ERP COMPONENT ANALYSIS

Two separate classes of averaged potentials were assessed: for correctly identified target stimuli and correctly ignored non-target ones. Cases, in which the number of units within any class available for analysis after artifact removal was lower than 30, were excluded from the averaging procedure. Evoked responses were analysed in the 1,000 ms post-stimulus time window.

The component measurements were carried out for those leads in which the highest amplitude values in the group mean were observed (Strandburg et al. 1994a,b, Shelley et al. 1996), i.e.:

P1 - in occipital (O1 and O2) and posterotemporal (T5, T6) leads, as the highest positive potential between 60 and 160 ms, preceding monotonic decrease of potential toward maximal negative values (descending slope of N1);

N1 - in the same set of leads, as the most negative potential between 130 and 220 ms, following P1 component;

P2 - in the same set of leads (T5, T6, O1, O2) and in central parietal one (Pz), as the positive component directly following ascending slope of N1 (between 180 and 280 ms);

N2 - in midline frontal lead (Fz), as the maximum negative deflection following component P2 in the 230-330 ms latency range;

P3 - two P3 components were observed in most cases: the earlier and shorter (i.e. more narrow as a graphical element) one, within a 300-480 ms latency range (P3a) and the later and longer one (P3b), within a 450-700 ms latency range, usually following the target stimuli. Since the locations of the maximal amplitudes of those two components varied, both were measured at three midline leads: Fz, Cz and Pz. Because of its long duration, the latency of P3b component was not assessed.

The amplitudes of components were measured from baseline to peak, where as a baseline the mean pre-stimulus EEG was adopted. Like CAT results, ERP data were correlated with age, duration of illness, PANSS score and dose of drugs.

STATISTICS

Because of non-normal distribution of CAT scores in the control group, for between-group comparisons of psychometric results the non-parametric Mann-Whitney tests were applied.

Points of measurement of electrophysiological parameters varied among components in number and locations. For each component its amplitudes (or latencies) from the whole array of points of its measurement were organized into a set of data and then put as dependent vector to two-way MANOVA by groups and condition (SPSS 4.0 software package, Norusis 1990). The results were supported by univariate two-way analyses of data from individual locations. In cases of significant interactions or main effects, lower-order analyses were performed.

Psychometric and electrophysiological data were correlated by the Spearman rank correlation test. Also correlations of both sorts of data with age, duration of illness, PANSS score and dose of drugs were examined with that test.

As a criterion of significance P=0.05 in two-tailed tests was adopted.

Cases with single missing values (with doubtful result of measurement of single peaks) were excluded from tests including that value.

RESULTS

Psychometric data

In the patient group the detection index was lower than in the control group ($I_D = 0.56 \pm 0.21$ vs. 0.93 ± 0.06, $U = 90.0$, $P<0.00005$). The error index was much higher ($I_E = 0.47 \pm 0.21$ vs. 0.08 ± 0.07, $U = 89.0$, $P<0.00005$) and was mainly due to omissions ($I_O = 0.41 \pm 0.21$ vs. 0.06 ± 0.06, $U = 86.0$, $P<0.00005$), whereas the commission indices in both groups were similar ($I_C = 0.03 \pm 0.05$ vs. 0.02 ± 0.03, NS).

Reaction time was significantly longer in the patient group (582 ms ± 70 vs. 518 ms ± 73, $U = 646.5$, $P = 0.0001$). In both groups there were no relationships between reaction time and test indices. There were also no significant correlations between CAT results and age, duration of illness, PANNS score or doses of drugs.

Electrophysiological data

AMPLITUDES

P1:the two-way MANOVA for P1 measured in four locations (O1, O2, T5, T6) revealed a significant group effect ($F_{4,171} = 3.35$, P=0.011), mainly due to left-sided amplitudes, i.e. P1 at O1 ($F_{1,174} = 12.26$, P=0.001) and at T5 ($F_{1,174} = 4.05$, P=0.046) as indicated by additional univariate two-way analyses (ANOVAs). Neither condition effects nor group by condition interactions were observed.

N1: two-way MANOVA for N1 at O1, O2, T5, T6 revealed a group effect ($F_{4,167} = 5.75$ $P<0.0005$), in which mainly posterotemporal (T5,T6) amplitudes contributed ($F_{1,170}$=14.03, $P<0.0005$ and $F_{1,170} = 11.25$, P=0.001, respectively). There were no significant condition ef-

TABLE I

Differences in potentials evoked by non-target stimuli in patients (S) and healthy subjects (C). Data concerning relevant components (i.e. indicated by preceding two-way MANOVAs) put to one-way MANOVAs are presented. Significant effects are followed by univariate analyses (UV) of data for individual locations significantly contributing in those effects, with corresponding mean values and standard deviations

Components and locations	D.F.	F	P	Mean values of amplitudes or latencies in S vs. C groups
amplitudes				mean values of amplitudes (μV)
P1 (O1, O2, T5, T6)	4,83	4.26	0.017	
UV: P1 (O1)	1.86	11.75	0.007	1.63 ± 1.50 vs. 2.82 ± 2.30
N1 (O1, O2, T5, T6)	4,83	3.58	0.010	
UV: N1 (T5)	1,86	8.08	0.006	-1.78 ± 2.11 vs. 3.30 ± 2.75
UV: N1 (T6)	1,86	11.75	0.001	-1.87 ± 1.62 vs. -3.56 ± 2.84
P3a (Fz, Cz, Pz)	3,86	7.58	0.001	
UV: P3a (Fz)	1,88	8.29	0.005	6.08 ± 4.11 vs. 8.73 ± 4.99
UV: P3a (Cz)	1,88	15.28	<0.0005	6.55 ± 3.36 vs. 9.93 ± 4.69
latencies				mean values of latencies (ms)
P2 (O1, O2, T5, T6, Pz)	5,72	4.93	0.001	
UV: P2 (O2)	1,76	9.85	0.002	238 ±1 8 vs. 228 ± 20
N2 (Fz)	1,88	7.41	0.008	295 ± 36 vs. 276 ± 34
P3a (Fz, Cz, Pz)	3,85	6.20	0.001	
UV: P3a (Fz)	1,88	6.83	0.011	401 ± 31 vs. 381 ±39

fects. However, a borderline group by condition interaction emerged ($F_{4,167}$ = 2.65, P=0.049), mainly due to group by condition interactions indicated by two-way ANOVAs for N1 at locations T6 and O1 ($F_{1,129}$ = 5.28, P=0.023 and $F_{1,129}$ = 4.39, P=0.038, respectively), which dictated cautious treatment of the group main effect until lower-order analysis could be performed (see below).

P2: for P2 at O1, O2, T5, T6, Pz two-way MANOVA revealed no significant effects or interactions.

N2: two-way ANOVA for the only considered location (Fz) revealed no significant results.

P3a:for P3a at Fz, Cz and Pz the two-way MANOVA yielded a significant group effect ($F_{3,168}$ = 4.47, P=0.005) as well as a condition effect ($F_{3,168}$ = 5.49, P=0.001). Two-way ANOVAs indicated group effects for locations Fz and Cz ($F_{1,170}$ = 12.90, P<0.0005 and $F_{1,170}$ = 6.63, P=0.011, respectively) and condition effects for locations Fz and Cz ($F_{1,170}$ = 13.83, P<0.0005 and $F_{1,170}$ = 4.65, P=0.033, respectively).

P3b:two-way ANOVA testing P3b component at Pz location revealed both group ($F_{1,112}$ = 6.57, P=0.012) and condition effect ($F_{1,112}$) = 26.39, P<0.0005) and no significant group by condition interaction. However, difficulties in precise measurement of the component in the non-target condition appeared to be responsible for missing values in many cases.

In order to assess directions of those main effects and a mechanism of the interaction related to N1 component, additional lower-order analyses were performed.

TABLE II

Differences in potentials evoked by target stimuli in patients (S) and healthy subjects (C). Data concerning relevant components (i.e. indicated by preceding two-way MANOVAs) put to one-way MANOVAs are presented. Significant effects are followed by univariate analyses (UV) of data for individual locations significantly contributing in those effects, with corresponding mean values and standard deviations

Components and locations	D.F.	*F*	*P*	Mean values of amplitudes or latencies in S vs. C groups
amplitudes				mean values of amplitudes (μV)
P1 (O1, O2, T5, T6)	4,86	1.65	NS	
N1 (O1, O2, T5, T6)	4,81	2.71	0.036	
UV: N1 (T5)	1,84	6.02	0.016	-2.16 ± 2.22 vs. -3.46 ± 2.44
P3a (Fz, Cz, Pz)	3,85	1.67	NS	
P3b (Pz)	1,83	6.39	0.012	8.25 ± 4.48 vs. 11.19 ± 5.22
latencies				mean values of latencies (ms)
P3a (Fz, Cz, Pz)	3,71	2.76	0.048	
UV: P3a (Fz)	1,73	8.16	0.006	399 ± 37 vs. 374 ±30

Significant results of one-way MANOVAs exploring only group effects for each condition are presented in Tables I (for non-target) and II (for target stimuli). The averaged response for non-target stimulus (c.f. Table I) in patients, as compared to healthy subjects, was characterized by lower amplitudes of components: P1 at left-sided occipital (O1) derivation, N1 at posterotemporal leads bilaterally (T5,T6) and P3a at frontal and central midline locations.

After target stimulus (as shown in Table II) many of those between-group differences faded or disappeared, because of amplitude increases, present only in the patient group (see below). However, univariate analysis revealed a maintenance of low N1 amplitude at left posterotemporal (T5) location. The P3b component, excessive in the target condition at the midline parietal location, also had a lower amplitude in patients than in the control group.

One-way MANOVAs exploring condition effects for amplitudes in each diagnostic group revealed no significant results for healthy subjects. Only univariate analysis for amplitude of component P3b at Pz location exposed its significantly higher value after target (compared to non-target) stimulus (5.60 ± 3.22 vs. 11.60 ± 5.79, $F_{1,61} = 20.49$, $P<0.0005$).

Contrary to results in the healthy subjects, in the patient group (c.f. Table III) significant results emerged for N1 and P3a component amplitudes, revealing their significant increase in target (compared to non-target) condition. The most notable condition effect on N1 component occurred at derivations T6 and O1, while amplitude of the P3a increased mainly at midline frontal (Fz) location. Additionally, like in the control group, a significant increase of P3b amplitude in response to target was observed, but it was not so distinct as in the healthy subjects. Figures 1 and 2 illustrate differences between averaged responses to non-target and target stimuli in the control and in the patient group, respectively.

An increase of amplitude of P3a at Fz during detection of target in patients yielded a weak correlation with daily dose of neuroleptic medication ($\rho=+0.34$, $P=0.02$). No other correlations of ERP data with medication were found. There were no correlations of the data with age, duration of illness or PANSS score.

TABLE III

Differences in potentials evoked by non-target (N) and target (T) stimulus in the patient group. Data concerning relevant components (i.e. indicated by preceding two-way MANOVAs) put to one-way MANOVAs are presented. Significant effects are followed by univariate analyses (UV) of data for individual locations significantly contributing in those effects, with corresponding mean values and standard deviations

Components and locations	D.F.	*F*	*P*	Mean values of amplitudes in the patient group for conditions N and T
amplitudes				mean values of amplitudes (μV)
N1 (O1, O2, T5, T6)	7,77	3.05	0.010	
UV: N1 (T6)	1,80	4.58	0.035	-1.95 ± 1.64 vs. -2.68 ± 2.12
N1 (O1)	1,8	4.60	0.035	-2.58 ± 2.38 vs. -3.52 ± 3.12
P3a (Fz, Cz, Pz)	3,81	4.46	0.015	
UV: P3a (Fz)	1,83	14.49	0.0005	5.86 ± 3.91 vs. 9.50 ± 4.40
P3a (Cz)	1,83	10.14	0.002	6.75 ± 3.20 vs. 9.02 ± 3.90
P3b (Pz)	1,70	8.20	0.006	5.21 ± 2.57 vs. 8.21 ± 4.69

LATENCIES

P1 and N1: two-way MANOVA revealed no significant results.

P2:two-way MANOVA for five locations of the component revealed significant group effect ($F_{5,142} = 3.39$, P=0.006), confirmed by two-way ANOVA for location O2 ($F_{1,146} = 9.86$, P=0.002). Neither a significant condition effect nor group by condition interaction were observed.

N2: the two-way ANOVA for the only point of measurement (Fz) revealed only a group effect ($F_{1,168} = 10.45$, P=0.002).

P3a: two-way MANOVA indicated a significant group effect ($F_{3,168} = 4.74$, P<0.002) in which contributed mainly latency at Fz.

P3b: as it was mentioned above, for this component measurement of latency was not performed.

Lower-order MANOVAs exploring only group effects on latencies for each condition are presented in Table I (for non-target) and II (for target stimuli). Latencies of components: P2 (especially at right occipital location O2) and N2 in patients as compared to those in the control group were significantly longer following non-target stimulus, while the latency of P3a at midline frontal lead Fz was significantly longer in patients in both conditions. Because of a lack of significant condition effects revealed by two-way MANOVAs for latencies, the one-way MANOVAs considering condition effects on latencies in each diagnostic group were not performed.

Correlations between CAT results and ERP data

Spearman rank correlation test revealed numerous correlations with indices of CAT performance in both groups. Many of these correlations, which are not directly linked with the current topic, particularly those referring to the healthy subjects, are not analysed here, but will be discussed in another paper (in preparation). Only correlations related to electrophysiological alterations observed in schizophrenic patients are presented here.

First, there were no significant correlations between lower (compared to those in the control group) amplitudes of P1 or N1 component in patients and their CAT results. However, an increase of N1 component amplitude at occipital locations in response to target stimulus correlated with an index of errors (ρ=+0.40, P=0.009 at O1 and ρ=+0.45, P=0.002 at O2). In the long-latency range, lower P3a component amplitude in non-target condition, measured at Fz location, correlated with an index of CAT errors (ρ=-0.46, P=0.002), but its increase in response to target at that location correlated inversely

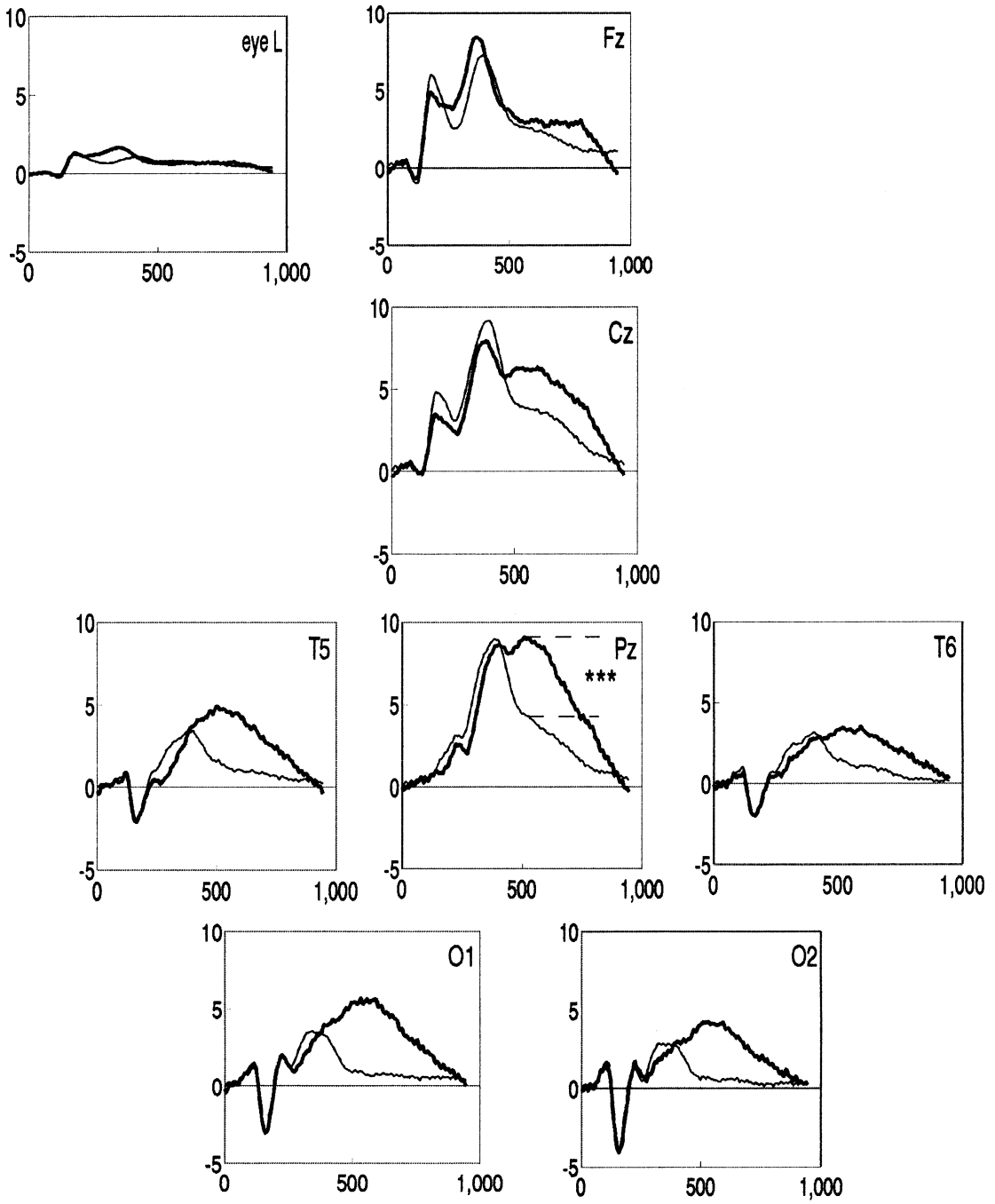

Fig. 1. Grand averages of potentials evoked by non-target (thin line) and target (thick line) stimulus in the control group (n = 50). An increase of the P3b in parietal location was the only change in amplitudes during detection of target. Significance of target effect is described by asterisks *** as P<0.0005.

with reaction time (ρ=-0.37, P=0.018). Correlations between longer latencies of later components and index of errors were also observed, reaching significance for N2 component in non-target condition and P3a component in target condition, both at Fz location (ρ=+0.37, P=0.016 and ρ=+0.41, P=0.006, respectively).

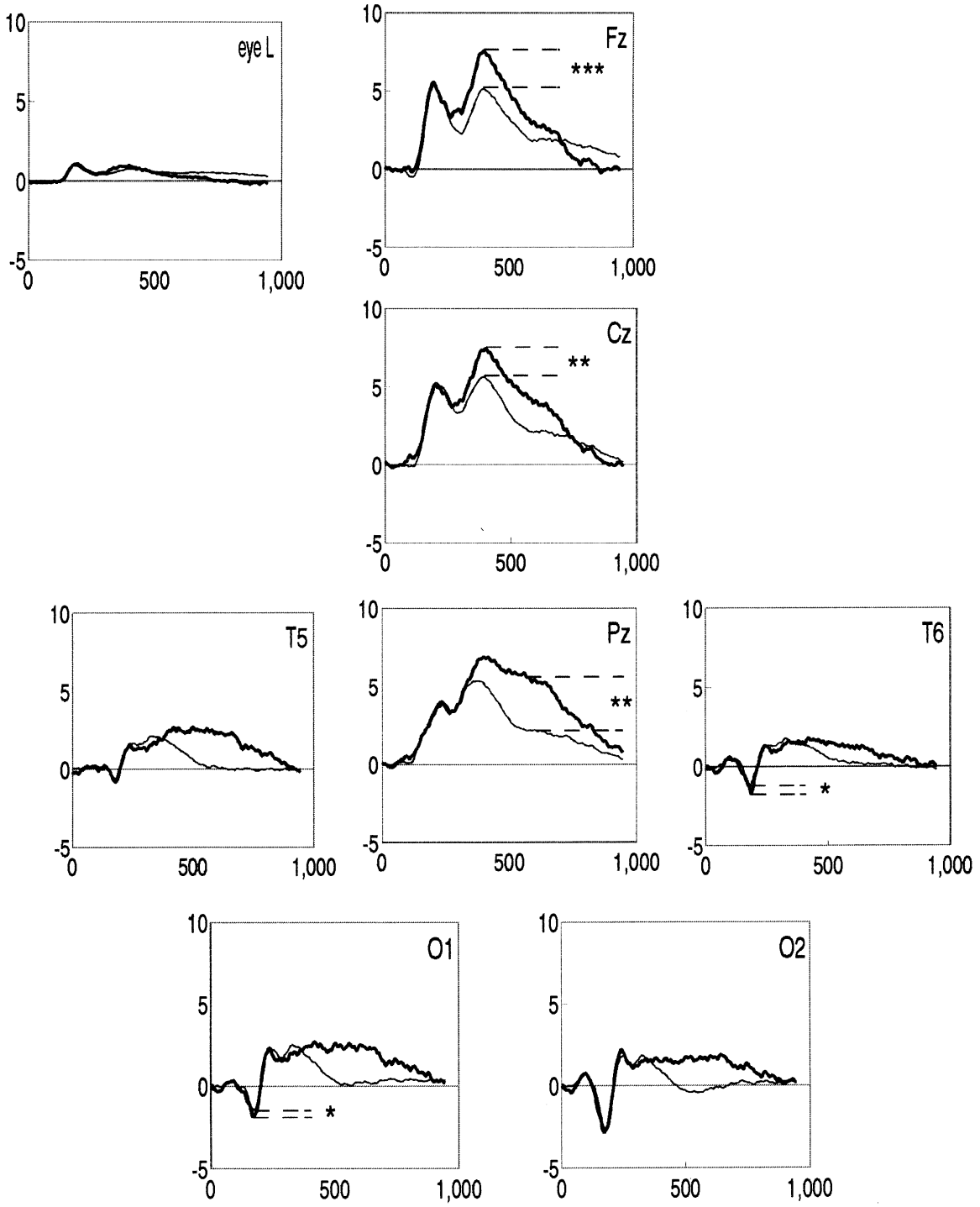

Fig. 2. Grand averages of potentials evoked by non-target (thin line) and target (thick line) stimulus in schizophrenic patient group (n = 50). Apart from an increase of the P3b at parietal location, the figure demonstrates increases of amplitudes of N1 and P3a components as reaction to target. Significance of the target effects is described by asterisks (* as $P<0.05$, ** as $P<0.01$, *** as $P<0.001$).

DISCUSSION

Apparently worse CAT results in the patient group, with a higher number of errors mainly contributed by omissions, are consistent with results obtained in numerous previous studies with the CPT (Wohlberg and Kornetzky 1973, Cornblatt et al. 1989, Nuechterlein 1991). Longer reaction time in patients has also been reported (i.e. Strandburg 1994b). Low index of commissions does not differentiate the groups and additionally confirms correct comprehension of the instruction by the patients as well as their good co-operation.

It is less probable, that inferior CAT results in the schizophrenic group are due to concurrent neuroleptic medication. First, similarly to our previous probes with CAT (Kasperska et al. 1996), there was no direct correlations between psychometric data and doses of drugs. Second, studies with the CPT revealed negligible detriment, or even an improving effect, of phenothiazines on the test performance (Spohn et al. 1977, Harvey et al. 1990). In the study by Kasperska et al. (1996) CAT distinguishes schizophrenic patients among the others, hence, despite the lack of a non-schizophrenic control group in the present study, it is also less likely that the results are influenced by general effects of a psychiatric illness. As described by Nuechterlein (1991) and Bergman et al. (1992), there is also no correlation between the test results and duration of illness. Independence of CAT results of PANSS score in clinically stable, remitted patients confirms a high sensitivity of the CAT for disturbances of attention in schizophrenia even at their subclinical expression.

As revealed by the material presented above, schizophrenic patients initially (i.e., in response to the non-target stimulus) have low amplitudes in exogenous (P1, N1) as well as endogenous (P3a) components as compared to healthy subjects. However, when the target stimulus is introduced, the majority of those between-group differences usually disappear due to the increase of amplitude of components N1 and P3a, which is observed only in the schizophrenic group.

The low left-sided P1 amplitude in the group of patients, following non-target stimuli, may be difficult for unequivocal interpretation because of lack of relevant correlations. Strandburg et al. (1994a) likewise reported asymmetry (right-side dominance) of P1 following visual stimuli. Romani et al. (1986) and Matsuoka et al. (1996) reported its lower amplitude bilaterally. Low amplitude of a component may be due either to unstable latency or to decreased arousability of relevant neurone groups. However, alternatively, it may be a result of their unsynchronized activity, caused by sensory hypersensitivity and chaotic, continual arousal by numerous background stimuli. A similar mechanism has been postulated by Freedman et al. (1983) for the auditory P50, which is also diminished in schizophrenia patients. On the other hand, there were the reports of an influence of attentional processes onto the baseline level of arousal of P1 component generators (Gomez Gonzalez et al. 1994, Heinze and Mangun 1995). In these studies, higher P1 over the hemisphere corresponding to attended visual area was observed. In the present study, where stimuli are presented in the centre of the visual field, bilateral influence of spatial attention is expected and an asymmetry of P1 component may also suggest diminished susceptibility of the left hemisphere for central regulation.

Support for the suggested hypersensitivity at the level of automatic stimulus analysis may be found in the increase of basically diminished N1 amplitude in response to the target, not found in the healthy subjects. Within the exogenous component range, after a stimulus which is a repetition of a previous one and with an interstimulus interval of 1-2.5s, gating or habituation would be more expected (Nagamoto et al. 1989, Freedman and Mirsky 1991). Although a facilitation effect during visual stimulation was already evidenced in healthy subjects, it was observed at an interstimulus interval of 100-120 ms (Shagass and Schwartz 1963, Nagamoto et al. 1989) and was diminished in schizophrenic patients (Szelenberger 1989). Quasi-increase of N1 amplitude, due to the overlap of processing negativity (Näätänen 1982) is less probable: processing negativity is a result of a signal selection based on its physical properties, which in the present study are the same for either non-target or target stimuli and each stimulus must be consciously processed. Moreover, processing negativity has been shown to be diminished in schizophrenia (Baribeau-Braun et al. 1983, Michie et al. 1990). Also the alternative possibility of an arousing effect of attention-related processes onto N1 (Jerger et al. 1992, Gomez Gonzales et al. 1994, Wagner et al. 1996) may be excluded, because the increase of N1 amplitudes during target detection correlates with the error index and worsening of detection. This seems to confirm a pathological mechanism of that feature. Although an influence of medication on ERP data cannot be excluded, the target effect onto N1 component does not correlate with doses of neuroleptic

drugs. Furthermore, as is mentioned below, the only ERP finding that correlates with doses of drugs was also the only finding that correlates with better CAT performance. This may lead to a conclusion analogous to those by Spohn et al. (1977) or Harvey et al. (1990), that medication has a negligible or even slightly improving effect on test results and is not responsible for ERP findings related to poorer detection.

Lower amplitude of P3a component in non-target condition in patients is also associated with inferior test scores. However, contrary to analogous target effect on N1, its increase in response to the target stimulus is accompanied by relatively better test performance. Hence, it may be a sign of compensation by an increased involvement of structures responsible for conscious information processing. Similar conclusions, though based on slightly different premises (higher P3a component in schizophrenic patients as compared to healthy controls), have been suggested by Rockstroh et al. (1994) and Strandburg et al. (1994a). That compensatory effect may be in part due to the medication, because of weak correlation of a change of an amplitude of P3a with doses of neuroleptic drugs. An increase of a P3a amplitude in response to visual target stimulus in schizophrenia patients was also reported by Shelley et al. (1996), but in CPT-AX version of the CPT.

The low P3b amplitude following target stimulus in schizophrenic patients has been widely reported (Brecher and Begleiter 1983, Pfefferbaum et al. 1984, Mirsky and Duncan 1986, Pritchard 1986, Pfefferbaum et al. 1989, Michie et al. 1990, Strandburg et al. 1990, Friedman 1991), but relatively rarely after visual stimuli (Pass et al. 1980, Szelenberger et al. 1991). However, that feature is not specific for schizophrenia, as reported in many psychiatric disorders associated with intellectual deterioration, only functional as well as those due to degenerative processes (Diner et al. 1985, Frank et al. 1994, Polich et al. 1994, Schroeder et al. 1994, Karayanidis et al. 1995, Kemner et al. 1995). Taking into account that in healthy subjects an inverse correlation between an amplitude of P3b component and difficulty of task was described (Polich 1987), lower P3b amplitude in schizophrenic patients may indicate relatively higher levels of CAT difficulty for them as compared to the healthy people.

Discussing inter-group differences in P3 latency range, in particular those related to detection of target, it is necessary to remember that ERPs following a correctly identified target must be influenced by the readiness potential (BP - Bereitschaftpotential) with further components preceding a motor reaction: pre-motion positivity (PMP) and motor potential (MP) (Deecke and Kornhuber 1977). Moreover, one might say that an altered electrophysiological pattern of target detection observed in patients, in part relating to the P3a, might be rather a result of alterations in those movement potentials. Hence, it is necessary to consider how the sequence: BP-PMP-MP, which typically reveals a slow (although not strictly monotonous) development towards negative values and reaches the peak with an onset at 60-50 ms before a movement, may influence the result on the P3a, which consists of an amplitude increase in patients and lack of amplitude changes in the control group. If we consider group mean values of reaction time, such an influence is possible under an assumption that an increase of P3a amplitude in response to target occurs in both groups, but in control subjects it is compensated by concurrently developing negative potential, while in patients is not. Two reasons are then to be suspected in patients: (1) lower amplitude of the BP or (2) weakened influence of the BP onto the P3a, caused by more distinct time-separation between their peaks, which results from longer reaction time. In the latter case, however, an increase of P3a amplitude should be linked with longer reaction time, while in the current study an inverse correlation occurred. This means that the more a separation between latency of P3a and time of reaction in patients is similar to the small separation observed in the healthy subjects, the more inter-group differences are visible. It is also doubtful that the first considered reason, i.e., diminished amplitude of the BP in patients, would give a benefit, which is shorter reaction time. Hence, the inter-group differences in electrophysiological pattern of target detection in range of P3a seem not to be an artifact due to interference with overlapping potentials related to a movement.

Discussing target effect onto P3a component in schizophrenic patients we must also consider the reports of positive shift of potential evoked by repeated visual stimuli in healthy subjects (Rugg et al. 1988). However, in their study not graphic but verbal stimuli were applied, and the authors conclude that repetition effect is a result of attenuation of endogenous negativity observed after unrepeated stimuli. Furthermore, in a study involving graphical stimuli (Zhang et al. 1997) an inverse effect of stimulus repetition onto amplitude of late positive components in healthy subjects was reported.

If we then return to the proposed resolution, that a target effect onto basically diminished amplitude of the P3a in the remitted patients is an expression of compensatory features emerging in the frontal region of the brain, we must state that such a compensation improves only the time of reaction. Lack of its influence on index of errors confirms that the high number of errors (in fact: omissions) during CAT performance might be a trait marker of schizophrenia. In contrast, the other ERP feature contributing an altered electrophysiological pattern of target detection in patients, i.e., an increase of amplitude of the N1 component, is apparently correlated with that unrecoverable index of errors. Hence, target effect onto an amplitude of the N1 component might be an ERP trait marker of primary underlying pathology, consisting in hypersensitivity on the earlier, automatic stage of visual signal processing. Such a conclusion resembles those inspired by the researchers on gating of an auditory P50 (Siegel et al. 1984) and may affirm the concept of primary sensory overload predisposing to and underlying schizophrenia (McGhie and Chapman 1961, Venables 1964).

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