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Auditory event-related potential (ERP) and difference-wave topography in schizophrenic patients with/without active hallucinations and delusions: a comparison with young obsessive-compulsive disorder (OCD) and healthy subjects

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Abstract

Event-related potentials (ERPS) in schizophrenics have been reported to show a reduced P3 on the left and less frontal mismatch negativity (MMN). But the specificity of such findings to component, its locus, the type of eliciting event and patient group remains uncertain. Hence, we examined ERP topography for P3, N2 and 3 precursor peaks according to stimulus (3-tone oddball), attention condition (diffuse/focused) and four types of difference-waves. We contrasted 24 healthy and 13 OCD subjects with schizophrenic patients with high versus low ratings of active delusions and hallucinations (12 paranoid-hallucinatory, PH; 12 nonparanoid, NP). P3 peaks were delayed and reduced in NP and PH groups. Midline peaks were usual in focused attention and a right bias in diffuse attention. P3 responses to irrelevant standards remained lateral in NP and small in OCD patients. All showed a small left and anterior bias in the P3-like peak in difference-waves. Mismatch negativity waveform (MMN) peaks shifted to the right in OCD, to both sides in PH and posteriorly in NP patients. Frontal processing negativity was biased to the left (early) in NP and to the right (late) in PH groups. Early peak topography reflected some later changes (e.g. PH and NP groups; P1-like peak, right bias absent; N1-like peak depressed and widely distributed: NP group, P2-like peak smaller on the left). In OCD patients, peak latencies were topographically undifferentiated (P1, P2) or delayed (N2). The OCD group showed an unusual regional allocation of processing effort. Before 200 ms frontocentral activity was more widespread in PH and NP groups. Lateralization of negativity in target- and nontarget-derived difference-waves may reflect differential disruption of the frontal-temporal dialogue in registering important vs unimportant features. NP patients, in particular, treated irrelevant stimuli anomalously.

Keywords: Event-related potential; Topography; Attention; Mismatch negativity; Negative difference; Paranoia; Schizophrenia; Obsessive-compulsive disorder

1. Introduction

An essential step in relating the nature of the impairment of the information processing strategies of schizophrenic patients to the underlying substrate involves the generation of topographic maps of

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event-related potentials (ERPs) elicited by different types of stimuli. To date, few have analysed ERPs to different types of stimuli or to the features important to stimulus processing derived by subtracting ERPs in difference-waves away from the midline (but see work from two groups, e.g. Morstyn et al., 1983; Pfefferbaum et al., 1989). This report relates perceptual and attentional function reflected by ERP component amplitudes in raw data and their topography in normalized data in four subject groups.

The 3-tone oddball paradigm was used to compare regional activation between subject groups to stimuli differing in pitch and rarity (Pfefferbaum et al., 1984; Oades et al., 1988). These features are confounded in the 2-tone version (Breton et al., 1988). Recordings were systematically compared after a standard and two deviant tones, differing in pitch, to test for perceptual effects on ERP components. These were presented in a passive nontask and an active discrimination to examine the influence of diffuse vs focused attention. Five components were examined in the P1 to P3 latency range to assess illness-specific effects on earlier vs later and inhibitory (positivity) vs facilitatory (negativity) aspects of information processing (Schupp et al., 1994).

Four types of difference-waves were calculated to contrast different comparator processes. The main distinction was between waves derived from nontarget stimuli (i.e. deviant minus common standard, D/C; the perceptual trace of the stimulus difference) and the rest derived from the target-elicited response. The most important of these is the T/t wave (i.e. target-ERP minus the ERP elicited by the same stimulus in an earlier passive condition; the attentional trace arising from conditioning). Two other widely used difference-waves representing similar functions are presented for comparison: the T/D (target-ERP minus nontarget-ERP, here another deviant in the same session) and the Goodin-wave (target minus common tone-ERP in active less the same wave calculated in the passive condition) attributed to Goodin (Goodin et al., 1978; Faux et al., 1988). They illustrate the problems of confounding the stimulus features of rarity and pitch although the Goodin-wave attempts to control for the state of 'focused-attention'.

Our main questions were do impairments of information processing show up more in ERPs recorded in focused vs diffuse attentional conditions? Do they implicate one comparator process more than the others? Does topographic recording assist differentiation of the process and its locus?

Neuropsychological evidence suggests that schizophrenic patients are particularly impaired where controlled focused attentional processing is required but is more equivocal on the efficacy of their automatic processing capabilities (Straube and Oades, 1992). Certainly tomographic evidence has attributed anomalous frontal lobe function to schizophrenic patients with negative symptoms and unusual temporal lobe function to those with positive symptoms (e.g. cerebral blood flow, Liddle et al., 1992; EEG spectrum analysis of ERPs, Gruzelier et al., 1993; Gerez and Tello, 1995) yet evidence from ERP components remains sparse (see below).

A large literature reports on the prevalence of depressed auditory N1, P2 and P3 amplitudes in schizophrenia (Pritchard, 1986; Shenton et al., 1989a,b; Ford et al., 1992). A smaller literature implicates P3 depression in OCD (Towey et al., 1993). But the notorious ERP variability within the schizophrenias (Roemer and Shagass, 1990) has rarely been examined in separate subgroups or for a symptom-related topographic differentiation.

To be sure, there is evidence that reduced P3 amplitudes correlate with the severity of psychosis and with negative symptoms (Pfefferbaum et al., 1989; Strik et al., 1993). The degree of thought disorder and allusive thinking also influences P3 amplitude (McConaghy et al., 1993). These negative and positive, disorganized symptom clusters have been tentatively related to left and right hemispheric dysfunction expressed in the P3, respectively (Shenton et al., 1989b). However, on the basis of psychophysiological and neuropsychological measures, Gruzelier, Raine and colleagues (Gruzelier, 1984, 1994; Gruzelier and Raine, 1994; Gruzelier et al., 1995) have argued for a lateral imbalance model such that positive symptom clusters may reflect over-activity of the left hemisphere and under-activity of the right hemisphere and negative clusters the opposite state of imbalance. This we investigate here contrasting schizophrenic patients with and without hallucinations and delusions.

Three laboratories have also reported on the relationships of difference-waves with symptoms. A re-

duction of mismatch negativity (MMN, latency range 140 to 220 ms in the D/C wave) has been correlated with the severity of negative symptoms (Oades et al., 1993; Catts et al., 1995), although there may be both state and trait contributions (Oades, 1995; Javitt et al., 1995a). In contrast, a reduction of processing negativity in schizophrenic patients (latency range 180 to 240 ms in T/D and T/t waves) may not depend on illness severity and hence may be more trait related (Oades et al., 1994b). Nonetheless, improvement has been reported after acute illness (McPherson et al., 1991). But most studies to date have used the correlative approach. Here we contrast subgroups of patients with active symptoms of delusions and hallucinations (PH) and those without, the nonparanoid patients (NP) with predominant negative and few positive symptoms. From the above discussion we would expect late ERP components to be decreased in the NP group, particularly on the left.

Considering that the efficacy of early processing operations is likely to influence directly or indirectly later operations, we also examine early components in the ERP and difference-waves. Further, if attention influences stimulus-elicited P1, N1 and P2 (Hackley et al., 1987; Luck and Hillyard, 1994), then similar peaks should be, and indeed are, visible in difference-waves (Novak et al., 1990; Woldorff et al., 1991; Woods and Alain, 1993; Alain et al., 1993; Oades et al., 1995b; Javitt et al., 1995b,c). They may not only differ between subject groups but more importantly show up as precursors of later larger effects on N2 and P3 expression. On the assumption that negative and positive deflections in the ERP reflect facilitatory and inhibitory processes, respectively (Schupp et al., 1994) the expression of N1- and P2-like components may be especially affected in target-derived difference-waves. These require controlled information-processing and would be predicted to be impaired in schizophrenic patients with negative symptoms (Straube and Oades, 1992).

Patients with OCD were studied as a psychiatric comparison group to help delimit the specificity of findings with schizophrenic patients. They were chosen because of the attribution of a related monoaminergic (serotonin vs dopamine; Oades et al., 1994a) and anatomical dysfunction (ventral vs dorsolateral frontal cortex; Baxter et al., 1990). From a psychological viewpoint both schizophrenic and OCD patients may be said to process information anomalously (Reed, 1985) and from an etiological viewpoint OCD symptoms are not infrequently observed before the onset of psychosis in teenagers (Eggers, 1968; Thomsen, 1992).

In summary, by examining the ERP profile we expected to replicate well-known ERP anomalies in schizophrenia (e.g. MMN and P3 decreases), and to show that they have antecedents/consequences in earlier/later components. We also hypothesized that these components would show lateralized (e.g. P3) or posteriorly shifted maxima in the topographic analysis (e.g. MMN), specific to psychosis (vs OCD patients) and the NP subgroup (vs PH). With respect to the topography of earlier components the analysis is exploratory.

2. Patients and methods

2.1. Sample selection

ERPs were recorded from 31 newly admitted patients diagnosed by a clinical psychiatrist (DSM IIIR; ICD 9) with a schizophreniform psychosis (confirmed by a clinician and three psychologists who rated a semistructured clinical interview; Spearman rho 0.64–0.75 for all 64 questions). From these, seven were excluded; two with reactive psychosis, two who could not discriminate the tones and three who did not provide enough artifact-free data. The remaining 24 patients received a diagnosis of schizophrenia or schizoaffective psychosis.

They were divided into those with active symptoms of delusions and hallucinations (the paranoid-hallucinatory group, PH) and those without such active symptoms (the nonparanoid group, NP). These groups were determined by a median split for these symptom clusters rated on the scale for assessment of positive/negative symptoms (Andreasen, 1983, 1984; see scores in Table 1). There was no significant group difference on negative cluster ratings (NP scores were 5% (affect) to 30% (anhedonia) lower but 'attention' scores were 5% higher). Thus the NP group exhibited predominantly negative symptoms but without significant differences in severity (BPRS: Table 1). The groups included the following diagnostic types: PH, 9 paranoid, 2 schizo-affective and 1

disorganized: NP; 4 disorganized, 4 schizoaffective, 2 paranoid and 2 undifferentiated.

At the same time, 13 OCD patients who had never received psychotropic medication were rated on the Yale-Brown Obsessive-Compulsive scale and tested. Young healthy case-controls were selected from a pool of 40 tested to match the schizophrenic patients for gender and age within 6 months (one younger one was included as a control for the one young OCD patient). They reported that they were free of psychiatric illness requiring consultation (past or present), a history of organic disorder or substance abuse (urine samples were taken) and medication, except contraceptive substances. This design along with our reports on ERP development from 8 to 22 years (Oades et al., 1996a) preclude explanations of differences in terms of age.

PH and NP groups were well matched for age, gender, IQ and education but NP received nonsignificantly more neuroleptic medication (Table 1). OCD patients were slightly younger but were matched for gender and they paralleled the controls for IQ. All subjects preferred to use their right hand on tests of writing and pointing. Testing followed approval of the protocol by the clinic management, agreement of the therapists and nursing staff to the tests in principle and the time-point in each case, and the cooperation, understanding and consent of the patient and the legally responsible adult.

2.2. Recording procedure

A 3-tone oddball was presented between 9.00 and 11.00 a.m in successive passive and active discrimination sessions (diffuse vs focused attention: details in Oades et al., 1995a,b). Tones, from an exact gate-function-generator were played over DT48 earphones (Bayer Dynamics). If left-right hearing thresholds which were measured just before recording (Audio-Med BCA3) varied by 5 dB or more, levels were adjusted. One hundred 0.8, 1.4 and 2.0 kHz tones (65 dBSL) per trial-block were presented

Table 1
Characteristics of four subject groups (means and range for data)

	Gender		Age (yrs)	BPRS/Q	Symptoms		CPZ equiv.	Biperidene	IQ (SPM)	Educ (yrs)
	f	m			H	D				
PH	4	8	18.5 14–24	3.5 2.9–4.5 ($n=9$)	11.6 1.3–30.3	17.6 1.8–39.7	705 * 56–1630	3.7 * * 2–4	90 75–120 ($n=11$)	11.4 8–16
NP	6	6	18.9 14–24	2.8 1.8–4.1 ($n=9$)	1.7 0–6.3	2.8 0–9.3	1246 217–3000	6.4 4–8	91 77–104 ($n=10$)	10.0 7–13
				YBOCS						
OCD	4	9	16.3 11–19	23.4 14–40 ($n=8$)	–	–	–	–	115 75–144	9.8 5–13
				MMPI 2-7-8						
CON	10	14	18.5 11–24	49.1 40–68 ($n=14$)	–	–	–	–	116 95–144	12.0 6–16

* Excluding 1 PH and 2 NP medication-free patients; * * excluding 5 PH and 7 NP patients not receiving biperidene.
H and D = hallucinations and delusions (ratings after Andreasen, 1984); BPRS = brief psychiatric rating scale: severity as summed scores/18 questions; YBOCS = Yale-Brown scale for assessment of obsessive-compulsive symptoms; MMPI = Minnesota multiphasic personality inventory, schizotypal scales 2-7-8 (40–60 = normal, subjects > 16 years of age); CPZ = chlorpromazine equivalents (Rey et al., 1989); IQ (performance) = Raven's standard progressive matrices; educ = years of primary and secondary schooling, as apprentice or student.
Groups: PH = paranoid-hallucinatory psychosis; NP = nonparanoid psychosis; OCD = obsessive-compulsive disorder; CON = healthy controls.

in a Bernoulli sequence ($p = 70$, 15 and 15%, respectively, 50 ms duration, rise/fall time = 10 ms, SOA 1.2–1.7 s).

Subjects sat in an air-conditioned Faraday-room and were asked to fixate a small cross 1.5 m away on the wall to help reduce eye movement during recordings. They were told there would be a baseline recording over 2–3 trial blocks. After the passive session most had identified three tones, which were then demonstrated. They were then asked to discriminate between the tones by raising a finger from a metal contact as fast but as correctly as possible after the 1.4 kHz tone. Mean hit/false alarm rates (SD) were for healthy control subjects (CON) 88/1.6 (12/1), OCD 77/1.8 (20/2), PH 51/6.4 (17/9), NP 64/7.0 (24/7). The performance of PH and NP groups was worse than OCD and CON scores on both measures ($F(6,130) = 7$, $p < 0.0001$): but reaction times did not differ significantly (CON 545 (95), OCD 612 (110), PH 603 (116), NP 613 ms (108)).

Recordings were made from 19 sites (10:20, Electrocap) using linked ear reference, Fpz and Oz as separate ground electrodes with impedance < 2 kOhm at all sites. Data were recorded on a Siemens EEG 21 and Compaq pc, sampled at 250 Hz for 1024 ms with a 50 ms pretrigger time and amplified by 12 k using a band pass 0.3 to 70 Hz (6 dB/octave). EOG artifact (> 65 μV) recorded above the eye was eliminated. Data were evaluated offline with gross EMG interference reduced by the low pass filters (including a digital 25 Hz low-pass, rectangular filter). The sampling rate and the normalization of latency data reduced problems of aliassing and hardware induced delays in the topographic analysis of latencies (Pivik et al., 1993). The mean proportion of trials (SD) removed for artifact was CON 22% (10), OCD 35% (19), PH 38% (21), NP 29% (14). Only artifact- and error-free trials were evaluated; accepted standard/deviant tones per group averaged CON 205/67, OCD 199/67, PH 256/57, NP 210/57.

2.3. ERP measures and statistical methods

ERPs were evaluated at F7, F3, Fz, F4, F8; C3, Cz, C4; P3, Pz, P4; T3, T4, T5, T6. Data from these sites (rows 2–4 of a 5×5 grid, with Fp1/2 and O1/2 in rows 1 and 5) were used to construct topographic maps using a horizontal and vertical linear interpolation (Crout algorithm) to establish a matrix of 180×180 points. Amplitudes and latencies for 5 peaks were evaluated after the stimuli; the common standard (C, 0.8 kHz), the deviant (D, 2.0 kHz) and the 1.4 kHz tone (passive = t, active = T). Then peaks were evaluated similarly in four difference-waves, calculated by subtraction as follows: D/C = D − C, T/D = T − D, T/t = T − t and the Goodin-wave = (T − C) − (t − C).

P1 was the largest positive deflection prior to the N1 (30–100 ms, difference-wave 20–120 ms). N1 was the earliest large negative component (80–140 ms, difference-wave 60–180 ms). P2 was the largest positive post-N1 ERP preceding N2 (120–240 ms in all waveforms). N2 was the largest negative peak between N1 and P3 (150–300 ms). The P3 was the largest post-P2 positive component (240–540 ms). Data for each tone and condition were normalized separately by vector analysis (i.e. for each subject the mean of each component measure at each site was divided by the square root of the sum of the squared mean measures for the 15 recording sites). This tests for the potential independent effect of each tone and condition, as carried out in studies of modality effects (Naumann et al., 1992). It also corrects for inhomogeneity in the raw data, multiplicative effects on ERPs of changes of source strength in ANOVA (McCarthy and Wood, 1985) and anomalies arising in other methods by taking account of the variance at all sites (Naumann et al., 1992).

Our objective centred on the group-related topography of the normalized peak measures under the influence of condition (attention) and tone-type in the stimulus-elicited ERP and in the four difference-waves. A multivariate analysis with site as a within-subjects factor was used as a precaution against between subject variability. For the evaluation of stimulus-elicited ERPs, four-way MANOVAs were run for four subject groups, in two conditions (passive/active), with three tones at 15 electrode-sites with repeated measures. After applying Hotelling's T2 test as an assessment of potential between-measure differences, as recommended for profile analysis by Faux and McCarley (1990), main site effects and site interactions with group, condition and tone were recorded following Greenhouse-Geisser epsilon cor-

Table 2
ANOVA/MANOVA summary: amplitudes and latencies of five ERP components in passive/active conditions of the 3-tone oddball: raw data at Cz for effects of groups, tone and condition (condn) followed by normalized data for topography (site)

Amplitudes: (ERP) Component	P1	N1	P2	N2	P3
Factor:					
group (Cz)	* *	* * *	–	* * *	a
tone (Cz)	* * *	*	–	* *	* * *
condn (Cz)	–	–	–	–	* * *
group × tone	–	–	–	–	*
group × condn	–	–	–	–	–
tone × condn	–	–	–	–	* * *
site	* * *	* * *	* * *	* * *	* * *
group × site	* * *	* * *	* * *	* * *	* * *
tone × site	?	–	–	* * *	* * *
condn × site	?	–	–	* *	* * *
group × tone × site	–	–	–	?	* * *
group × condn × site	–	–	–	–	–
tone × condn × site	–	–	–	?	–
Latencies: (EPR)					
group (Cz)	* * *	* * *	–	* * *	* * *
tone (Cz)	–	–	–	* *	* *
condn (Cz)	–	–	a	* *	–
group × tone	–	–	–	–	–
group × condn	–	–	–	–	*
tone × condn	–	–	–	–	–
Site	* * *	* * *	* * *	* * *	* * *
group × site	* * *	* * *	* * *	*	* * *
tone × site	–	–	?	–	* * *
condn × site	–	–	* *	a	*
group × tone × site	–	?	?	–	?
group × condn × site	–	–	?	?	*?
tone × condn × site	–	–	–	–	?
Summary of ANOV/MANOVA for five ERP components in four difference-waves					
Amplitudes: (difference-wave)					
group (Cz)	a	–	–	–	–
wave (Cz)	–	–	a	–	* * *
group × wave	–	–	–	–	–
site	* * *	* * *	* * *	* * *	* * *
group × site	*	*	?	?	*?
wave × site	–	–	–	–	*?
group × wave × site	?	–	–	–	–
Latencies: (difference-wave)					
group (Cz)	–	–	*	* * *	* * *
wave (Cz)	–	–	–	* *	* * *
group × wave	–	–	–	–	–
site	* *	–	–	* *?	* * *
group × site	*	* *	*?	* *	–
wave × site	?	–	–	?	–
group × wave × site	–	*	?	* *	–

a = $0.1 > p > 0.5$, * $p < 0.05$, * * $p < 0.01$, * * * $p < 0.001$. ? = Conflict over decimal place for significance in Averaged vs Hotellings tests. An appendix of the detailed results of the MANOVA and ANOVA tests is available from the authors on request.

rection for SPSSX site-averaged test results (Greenhouse and Geisser, 1959; Iragui et al., 1993; Table 2). Interpretation was set in the context of the main effects for peak amplitude in a three-way ANOVA for group, condition and tone that can only be performed on the raw data and were thus restricted to data from Cz. This result is briefly described first under the sections on each component. Insignificant interactions ($p > 0.1$) are not reported. The terms 'tendency, trend and tend' refer to statistical significance of 5–10% or where Hotellings and averaged tests conflicted on the level of significance.

For difference-waves, three-way MANOVAs were run for four subject groups in four waveforms at 15 sites with repeated measures, separately for each component measure. After referring to the main effects at the vertex (raw data), we report the topography of peak measures (main effects of site and site-group/waveform interactions). Statistical results are reported following corrections, as above (see Table 2).

To determine where differences lay, significant site interactions were followed by paired Student *t*-tests for six selected comparisons within groups between anterior-posterior (F7/T5, Fz/Pz, F8/T6) and left-right sites (F7/F8, T3/T4, T5/T6). Paired comparisons were selected to guard against an excessive number of comparisons, but the Bonferroni procedure cautions alpha correction as follows: 1% after main site effects, 0.5/0.2% for age, condition or tone interactions and 0.1% after three-way interactions). Significant interactions with group were followed by conservative Scheffe tests (alpha 5%). Post-hoc tests are cited in the text.

3. Results

3.1. P1 Components

3.1.1. ERP amplitude and latency

Peaks at Cz were larger in the PH patients than in the other groups (Scheffe $p < 0.05$, Table 2) and were twice as large as those of the controls. Unlike the other groups, the P1 in PH patients increased in amplitude with increases of tone-frequency (Fig. 1). However, latencies in the NP patients were shorter than in the other groups (Scheffe $p < 0.05$; Cz group means (+SD) CON 47 (13), OCD 50 (18), PH 51 (17), NP 39 (14) ms).

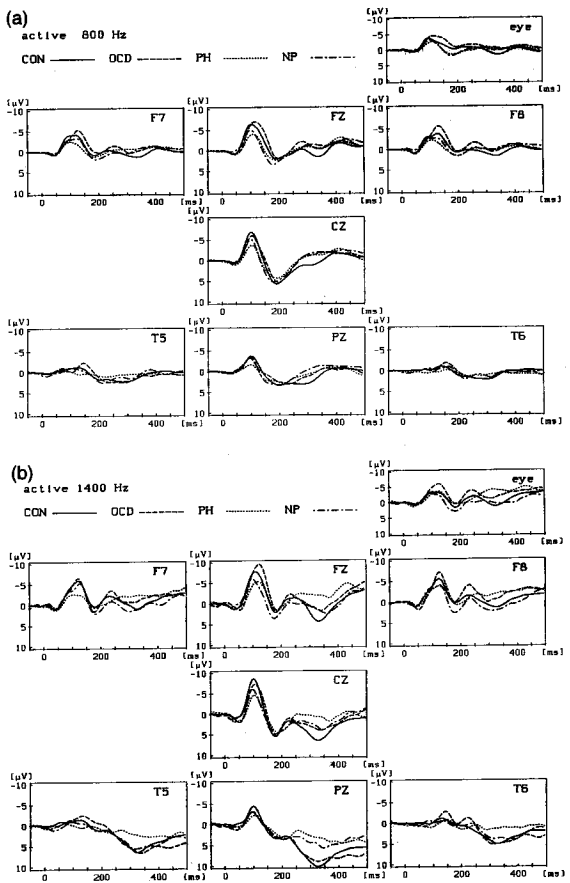
Group differences in the topographic distribution of P1 were significant (Table 2, normed data not shown). Peaks were larger at frontal vs posterior sites in CON, OCD and PH subjects (Fz/Pz, t +2.6 to +4.0, $p = 0.01$–0.0001) but not in the NP group. A lateral asymmetry was evident in the comparison groups with peaks on the right being larger than on the left (CON and OCD, t −3 to −5.3, $p = 0.003$–0.0001). Maxima at lateral sites were usually larger in the comparison groups than in the PH or NP groups (Scheffe $p < 0.05$). NP patients showed the reverse pattern with peaks increasing posteriorly (t −2.3 to −4.5, $p = 0.03$ to 0.0001), especially on the left side (T5, NP > CON, Scheffe $p < 0.05$). Across groups latencies were shorter on the left than the right (t −7 to −8.7, $p < 0.0001$) and shorter at lateral frontal vs posterior sites (t −9.4 to −13.7, $p < 0.0001$).

3.1.2. Difference-waves

At Cz, OCD subjects tended to show a larger P1-like deflection across waveforms than the other groups (Table 2), but there were no latency differences (Fig. 2).

The group × site effect indicated clear parietal maxima in the two comparison groups (t −2.4, $p = 0.016$) although only the OCD group showed this in all waveforms (t −3.1, $p = 0.003$, Fig. 2). In both schizophrenic groups frontal maxima were larger than posterior in the D/C wave comparing nontargets (t +4.9 to +5.5, $p < 0.001$, Fig. 2, trend group × wave × site interaction Table 2). As in the simple ERP, controls showed a right-sided bias (F8, T4; t −2.4 to −2.6, $p = 0.01$–0.02), but the PH group had larger peaks on the left (F7, T3; t +2.3 $p = 0.024$, Fig. 2).

A group × site interaction indicated that latencies tended to be shorter on the right than the left (e.g. F7/F8, PH and CON, t 2.0, $p = 0.05$) and were shorter over right frontal than posterior areas (PH and CON, t −2.5 to −5.2, $p = 0.014$–0.0001). Indeed, latencies were shorter in the PH than the OCD group on the right at F8, while the reverse was seen at T6 (Scheffe $p < 0.05$, Fig. 2 right).


(a)
active 800 Hz
CON
OCD
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NP
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F7
FZ
F8
CZ
T5
PZ
T6
(b)
active 1400 Hz
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T6
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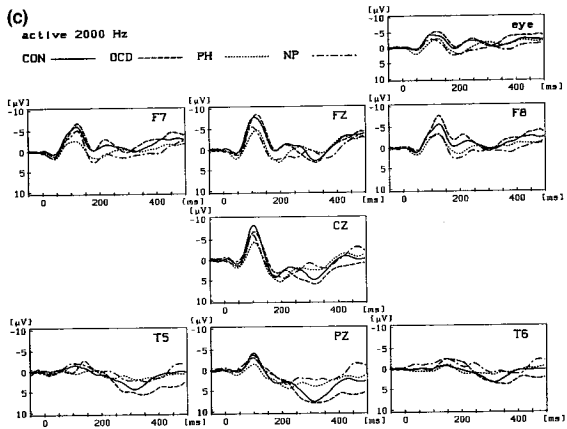


Fig. 1. Grand mean ERP waveforms (μV) for four subject groups at anterior (F7, Fz, F8), at the vertex (Cz) and at posterior recording sites (T5, Pz, T6) for 500 ms after the three tones in the active, discrimination condition: (a) 0.8 kHz (standard tone); (b) 1.4 kHz (target); (c) 2.0 kHz (deviant): insets show EOG representations of eye movements.

3.1.3. Summary (P1 component)

P1 ERP maxima were frontocentral and largest in the PH group, in whom the responses increased with pitch. But the NP group had a paradoxical centroparietal maxima without the usual marked anteroposterior potential gradient. In contrast, in the difference-wave the P1-like deflection was largest in OCD subjects and had centroparietal maxima like the controls.

Neither schizophrenic group showed the normal right-sided topographical bias for normed P1 ERP data. The similar right-sided bias for P1-like peaks in the difference-waves for the comparison subjects was not evident in the PH group. Both schizophrenic groups were unusual in showing clear frontal contributions in the nontarget comparison (D/C).

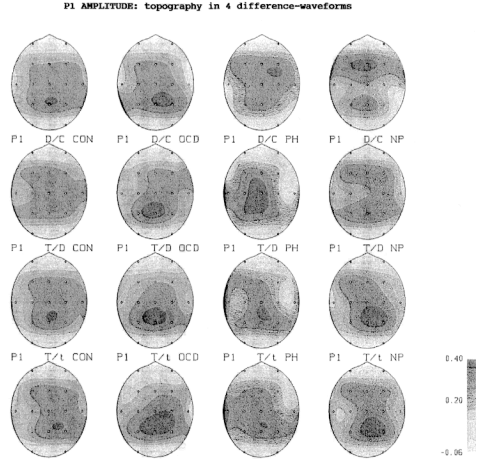
P1 ERP-latencies were generally shorter on the left and at frontal sites. P1-like latencies in the difference-waves tended to show the opposite lateral pattern but an even more marked short to long anteroposterior latency pattern. The OCD group was noticeable for a lack of such differentiation (Fig. 2 right).

3.2. N1 components

3.2.1. ERP amplitude and latency

At Cz, deviant tones elicited larger peaks than standards in all groups (Fig. 1). In the psychotic groups maxima were a third less than in the controls (OCD > CON > NP > PH, with OCD > PH, Scheffe $p < 0.05$). Response latencies at Cz were later in OCD and PH patients with respect to CON and NP subjects (Scheffe $p < 0.05$; Cz group means (+ SD) CON 105 (11), OCD 111 (17), PH 114 (23), NP 103 (14) ms, Table 2).

In the topographic analysis, N1 peaks were larger over all frontal vs posterior sites in each group (t -9.7 to -19.6, $p = 0.0001$; normed data not



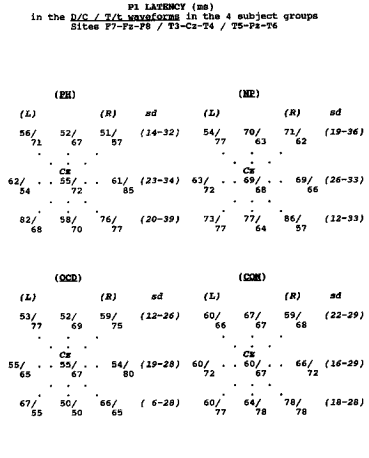


Fig. 2. Left: Topography of normalized mean P1-like peak amplitudes over the skull (anterior up). Horizontal rows show the four difference-waves; nontarget-derived D/C, and target-derived T/D, T/t, G-wv = Goodin-wave: vertical columns show the four subject groups; CON = controls, OCD = Obsessive-Compulsive Disorder, PH = Paranoid-Hallucinatory and NP = Nonparanoid Schizophrenia. P1-like peaks usually showed centroparietal maxima and positivity biased to the right: but PH and NP groups showed frontal maxima in the nontarget stimulus comparison (D/C). Right: A similar topographic representation of mean P1-like latencies (ms) over the skull (anterior up). These are shown from left (L) to right (R), with the vertex in the middle, for the nontarget- and target-derived waveforms (D/C, upper; T/t, lower). At the top are schemata for PH and NP patient groups and below are schemata for OCD and CON subject groups. The standard deviation range is shown in italics on the right of each row of recording sites. The PH group showed particularly short right frontal latencies.

shown). The only significant interaction of site was with group (Table 2). Scheffe tests showed for OCD patients a larger negative shift at frontal and temporal sites compared to other groups (e.g. OCD > PH and CON, but NP and CON > OCD at centroparietal sites).

With regard to the lateralization of peak amplitude, the OCD group tended to show a larger shift on the right than left at frontal sites while the NP group showed the opposite ($t - 4.8$, $p = 0.000$). But the left bias in NP patients did not extend posteriorly where a larger negative shift over T5 vs T6 was seen in PH, OCD and CON subjects ($t - 2.5$ to -3.9, $p = 0.017$ to 0.0001; compare raw data in Fig. 1).

N1 latencies were usually shorter on the left than right ($t - 2$ to -4.5, $p = 0.05–0.0001$). But OCD patients tended to show the opposite pattern, matching higher N1 amplitudes on the right. In the comparison groups, midline latencies were shorter at parietal than at frontal sites reflecting smaller peaks (Scheffe $p < 0.05$). But at lateral frontal sites, latencies were usually shorter than posteriorly ($t - 4.5$ to -9.3, $p = 0.001$). Indeed, the PH group had the shortest latencies at lateral frontal sites but the longest ones posterotemporally (Scheffe $p < 0.05$).

3.2.2. Difference-waves

As with simple ERPs, maxima were frontocentral. Anteroposterior differences were least marked on the left (i.e. $t - 2.2$ to -3.0, $p = 0.03–0.003$; with PH and OCD not significant; Fig. 3). There was no lateralization in the controls. But negativity was distributed marginally more to the right in the schizophrenic groups (F8 for PH and T6 for NP groups, $t + 2$ to $+4$, $p = 0.05–0.006$). The effect is evident for the NP group in Fig. 3 with low negativity over left temporal sites in the target-derived waveforms (T5, CON and OCD > NP, Scheffe $p < 0.05$).

On latency measures, site interacted with group and waveform. The OCD group alone showed longer frontal vs posterior (t 2.8, $p = 0.007$) and longer right vs left latencies in target- and nontarget-derived waves, recalling the pattern in the simple ERP ($t - 2.4$ to -5.4, $p = 0.018–0.000$: but also CON T5 < T6, $t - 2.4$, $p = 0.006$). Here long latencies reflected the larger peaks. But, in contrast, NP patients had longer left vs right frontal and temporal latencies in the T/t target-derived wave ($t + 2.4$ to $+2.5$, $p = 0.01–0.02$; Fig. 3). Further, in the PH group, unlike the comparison groups, posterior latencies remained long (Fig. 3 on the right) and these reflected the smaller peaks. (The three-way interaction in Table 2 also reflected that NP patients had longer right than left posterior latencies in the D/C nontarget wave ($t - 3.9$, $p = 0.003$.)

3.2.3. Summary (N1 components)

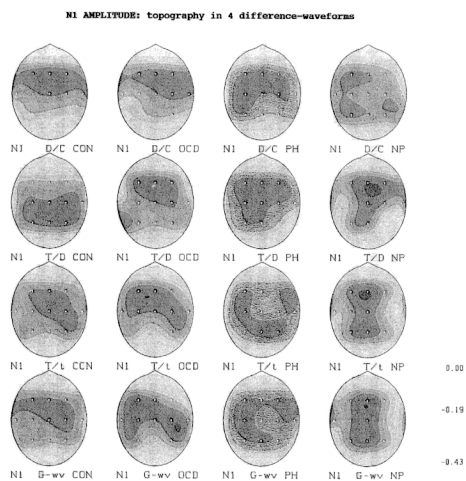
Frontocentral N1 amplitude in the ERP, was larger after deviant tones in all subjects; but by comparison with other groups it was reduced in schizophrenic patients. OCD patients had unusually large frontotemporal and small centroparietal peaks. N1 showed no asymmetries in the controls, but tended to be larger on the left in the NP and on the right in the OCD group (compare N2 below).

In difference-waves lateral shifts of negativity in patients contrasted with a symmetric distribution in the controls. Striking is the posterior spread of activity in the NP group, especially at temporal sites in the D/C nontarget-derived waveform (cf. possible precursor of the MMN). But in the T/t target-derived waveform NP showed little left temporal negativity, contrasting with the right-frontal/left temporal pattern in the PH group (i.e. possible precursors of the Negative-Difference wave; Fig. 3). That is to say there were early topographic shifts in schizophrenic patients during the processing of features of rarity or relevance at a latency of about 120 ms anticipating changes peaking at 240 ms. N1-like latencies were also already showing delays laterally for target-derived (e.g. T/t, Negative Difference in OCD, PH) and nontarget-derived comparisons (e.g. D/C or MMN in NP).

3.3. P2 components

3.3.1. ERP amplitude and latency

There were no main effects or interactions for the raw data at Cz. In the normed data, maxima were usually posterior (vs frontal sites, $t - 5.5$ to -6.9, $p = 0.0001$) but this was not confirmed for NP patients who showed more positivity at right frontal sites than the comparison groups (Scheffe $p < 0.05$; see raw data in Fig. 1). At the same time, peaks in OCD patients were smaller than in the others, after a



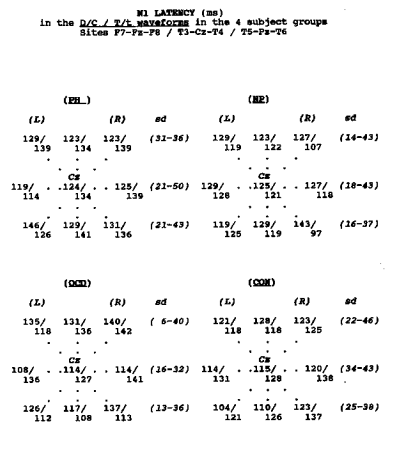


Fig. 3. Left: Topography of normalized mean N1-like peak amplitudes over the skull (anterior up). Horizontal rows show the four difference-waves; nontarget-derived D/C, and target-derived T/D, T/t, G-wv = Goodin-wave: vertical columns show the four subject groups; CON = controls, OCD = Obsessive-Compulsive Disorder, PH = Paranoid-Hallucinatory and NP = Nonparanoid Schizophrenia. N1-like peaks usually showed frontocentral maxima but anteroposterior differences were less marked in patient groups, especially on the left. Right: A similar topographic representation of mean N1-like latencies (ms) over the skull (anterior up). These are shown from left (L) to right (R), with the vertex in the middle, for the nontarget- and target-derived waveforms (D/C, upper; T/t, lower). At the top are schemata for PH and NP patient groups and below are schemata for OCD and CON subject groups. The standard deviation range is shown in italics on the right of each row of recording sites. In OCD patients N1-like latencies were usually shorter at left vs right sites but in NP patients latencies were usually shorter on the right.

large N1 (Scheffe $p < 0.05$ at T6), and contrasted with NP patients by being larger on the left than the right in all comparisons (t 2.5 to 3.4, $p = 0.013$–0.001; e.g. see rare tones in Fig. 1).

In all groups, latencies at Cz tended to be shorter during focused attention [Table 2; passive/active condition means (SD) CON 193/189 (23), OCD 200/192 (28), PH 199/190 (26), NP 192/190 (30) ms]. Latencies were usually shorter at lateral frontal than temporal sites (t -4.1, $p = 0.0001$). But, as with N1 (above), the opposite held for the NP group (t 2.4, $p = 0.004$). Of all groups, NP patients showed the longest latencies at F8 and the shortest ones at T5 (vs CON and PH; Scheffe $p < 0.05$). This asymmetry in the speed of processing may be pertinent to an understanding of the developing negative difference (T/t) asymmetry.

3.3.2. Difference-waves

The tendency for all subjects to have larger peaks at Cz in the T/t vs other difference-waves was not confirmed posthoc (Table 2). In the topographic analysis posterior maxima were more (CON and PH, t -2.1 to -3.2, $p = 0.05$–0.002) or less clearly expressed (NP and OCD, both t -1.7, $p = 0.09$; Fig. 4). At frontal sites positivity was biased more to the right in the CON and NP groups (t -2.7 to -3.7, $p = 0.001$–0.009) and to the left in the PH group, (t 2.5, $p = 0.015$). The left bias in PH patients extended to temporal sites (t 3.0 to 3.6, $p = 0.001$–0.005) and was emphasized by their showing less positivity than the NP patients at F8 (Scheffe $p < 0.05$; Fig. 4).

For latencies, site interacted clearly with group and modestly with group and waveform. At Cz, latencies were shorter in controls than in all other patient groups (Scheffe $p < 0.05$; Fig. 4 right): particularly on the right and at posterior sites in the D/C and Goodin-waves (t $+2.5$ to $+2.8$/ -2.3 to -2.5, $p = 0.01$–0.03). OCD group latencies only differentiated across site in the T/D waveform. Here, like controls, posterior latencies were shorter than frontal ones (t 3.0 to 4.3, $p = 0.01$–0.001), but unlike controls they were shorter on the left side (t -2.2 to -2.7, $p = 0.05$).

PH and NP patients tended to show the opposite antero-posterior difference. In nontarget-derived waves, the PH group had shorter latencies at frontal than posterior sites (t -4.5 to -5.4, $p < 0.001$; Fig. 4 right). NP patients showed this pattern for target-derived difference-waves (t -2.1 to -3.3, $p = 0.0.05$–0.007). (The figure also shows particularly long latencies at T5; PH vs CON, Scheffe $p < 0.05$.)

3.3.3. Summary (P2 components)

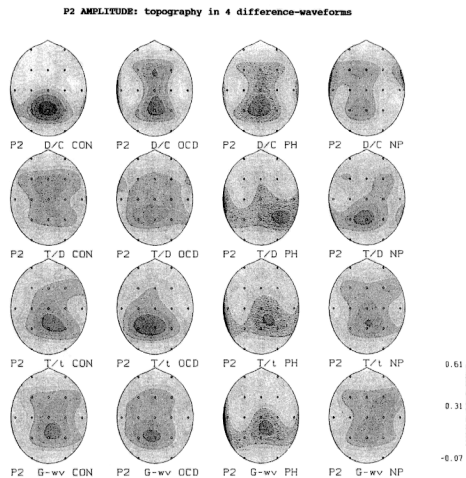
There was no decrease of amplitude in the schizophrenic groups as reported elsewhere (e.g. Shenton et al., 1989a). Centroparietal P2 maxima were the rule but positivity extended anteriorly to the right in the NP group and to the left in the OCD group. Similarly, P2-like maxima in the difference-waves had posterior loci with positivity extending frontally to the right in NP and to the left in PH patients. This pattern mirrors and anticipates the frontal asymmetry reported for the Negative-Difference wave (Oades et al., 1994b).

P2 ERP latencies decreased with focused attention and were often shorter on the left; but schizophrenic patients were less sensitive to condition. This is reminiscent of a report of slowed ERP responses in schizophrenics to standards relative to targets (Pfefferbaum et al., 1989). P2-like latencies in the difference-wave showed that speeds of processing were differentiating more since the N1, both according to group (longer in schizophrenic than comparison groups) and to the type of stimuli compared (shorter in nontarget- than target-derived waveforms at posterior sites in comparison groups). The OCD group continued not to show much topographic differentiation, but the relatively slow response at posterior sites in the schizophrenic groups could be a forerunner to slow P3 processing.

3.4. N2 components

3.4.1. ERP amplitude and latency

At Cz, the amplitude decreased with increases of tone-frequency, but at lateral frontal and temporal sites it increased (Scheffe $p < 0.05$; Table 2). In comparison with the diffuse attention condition negativity spread more to posterior sites during focused attention (Scheffe $F(1,364) = 9.0$, $p < 0.003$; data not shown for both conditions). At Cz, and all temporo-parietal sites, patients showed larger peaks than controls (PH and OCD at the more anterior and NP at posterior sites, Scheffe $p < 0.05$, Fig. 1).



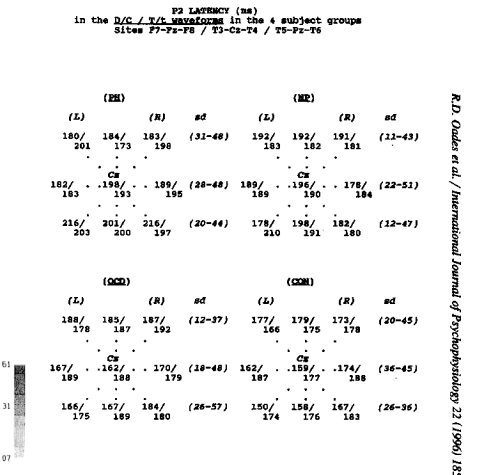


Fig. 4. Left: Topography of normalized mean P2-like peak amplitudes over the skull (anterior up). Horizontal rows show the four difference-waves; nontarget-derived D/C, and target-derived T/D, T/t, G-wv = Goodin-wave: vertical columns show the four subject groups; CON = controls, OCD = Obsessive-Compulsive Disorder, PH = Paranoid-Hallucinatory and NP = Nonparanoid Schizophrenia. P2-like positivity usually peaked posteriorly, but spread frontally in patients in the D/C waveform. Right: A similar topographic representation of mean P2-like latencies (ms) over the skull (anterior up). These are shown from left (L) to right (R), with the vertex in the middle, for the nontarget- and target-derived waveforms (D/C, upper; T/t, lower). At the top are schemata for PH and NP patient groups and below are schemata for OCD and CON subject groups. The standard deviation range is shown in italics on the right of each row of recording sites. Latencies at the vertex were longer in all patient groups and were topographically less differentiated in the D C wave in OCD patients.

The topographic analysis with normalized data confirmed the presence of frontal N2 maxima for most comparisons (t −11.8 to −13.8, $p = 0.0001$), but for the NP group this only held in the midline, for the lateral peaks were smaller than in the other groups (vs OCD, Scheffe $p < 0.05$). Peaks were symmetrically distributed in CON and OCD groups, but larger over left frontal areas in NP (t −2.2, $p = 0.03$) and left temporal sites in PH, compared to the right (t −2.7 to −4.3, $p = 0.009$–0.0001). For these areas, especially after the two irrelevant tones, group differences were significant (anterotemporal areas CON < PH, postero-temporal and parietal sites CON < NP, Scheffe $p < 0.05$, Table 2). In contrast NP patients at frontal sites showed least negativity (e.g. F8).

N2 latencies at Cz tended to be shorter after deviant than standard tones and were shorter in the active condition for the comparison but not the schizophrenic groups. Peaks were delayed by about 10% in the three patient groups (Table 3). Only the NP group showed a topographical latency difference (lateral frontal > temporal sites, t 2.5 to 3.6, $p = 0.01$–0.001) with latencies especially long at F8 in the passive condition (vs OCD and CON, Scheffe $p < 0.05$; cf. T/t asymmetry below).

3.4.2. *Difference-waves*

As with stimulus-elicited ERPs the amplitude of the N2-like component was larger over all frontal sites (t −4.0 to −7.7, $p = 0.0001$) except for the NP group. Fig. 5 shows the shift of D/C maxima (MMN) in the NP group to posterior temporal sites (vs frontal sites; Scheffe, $p < 0.05$). Amplitudes were not significantly lateralized in the comparison groups, but Fig. 5 shows that for the PH group MMN amplitude was 'bilateralized' to the sides, away from the midline (at Cz, PH < CON, $p < 0.05$ Scheffe) and reached more posteriorly on the right (T5 < T6, t +3.8, $p = 0.001$; Fig. 5). Normed amplitude data did not show a significant topographical asymmetry in target-derived waveforms.

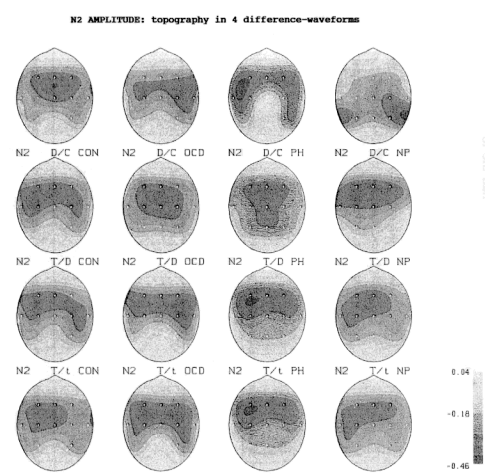
At Cz, significant main effects of group and waveform indicated that latencies were shorter in the comparison than in the schizophrenic groups and shorter in the MMN (D/C) than in the negative-difference wave (T/t). For topographic data there was a highly significant group × waveform × site interaction (Table 2). Longer frontal vs posterior latencies were only seen in CON and OCD groups (t 3.5 to 4.8, $p = 0.001$–0.0001). At postero-temporal sites the PH group had the longest latencies of any group on both sides in the D/C waveform (Scheffe $p < 0.05$; Fig. 5, right).

In target-derived waveforms (e.g. negative-difference, T/t) CON and NP showed little topographic differentiation. However, the PH group had longer latencies than the others (Scheffe $p < 0.05$), especially in the right-frontal/left-temporal diagonal axis. Here the latencies were longer than contralaterally (t +3.4 to +6.1, $p = 0.006$–0.0001) and, at left pos-

Table 3
N2 latencies (ms) at Cz for three tones in two attention conditions (SD in parentheses) for four subject groups

Group	Passive (diffuse attention)				Active (focused attention)			
	0.8 kHz standard	1.4 kHz deviant	2.0 kHz deviant		0.8 kHz standard	1.4 kHz target	2.0 kHz deviant	
CON	273	279	270	(38)	276	239	253	(47)
OCD	310	304	279	(65)	304	260	236	(51)
PH *	316	299	296	(62)	311	294	306	(67)
NP *	304	282	295	(67)	302	256	296	(70)

* = $p < 0.05$ (Scheffe) latencies slower than in controls: latencies shorter in active than passive condition for comparison not schizophrenic groups (normalized data, site × group $F(21,2442) = 1.9$ and site × condition × group $F(21,2442) = 1.7$, 5% significance after epsilon correction, see text).



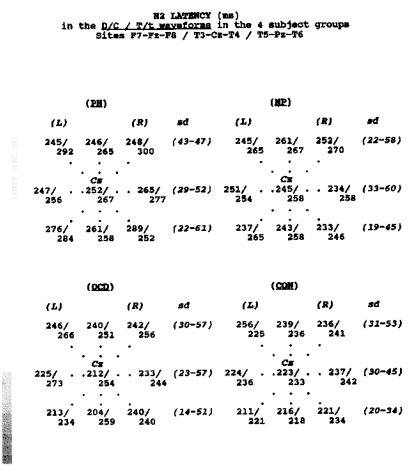


Fig. 5. Left: Topography of normalized mean N2-like peak amplitudes over the skull (anterior up). Horizontal rows show the four difference-waves; nontarget-derived D/C, and target-derived T/D, T/t, G-wv = Goodin-wave: vertical columns show the four subject groups; CON = controls, OCD = Obsessive-Compulsive Disorder, PH = Paranoid-Hallucinatory and NP = Nonparanoid Schizophrenia. Frontocentral N2-like maxima in CON, shifted in OCD patients to the right (D/C and T/t). In the D/C wave, peaks were bilateral in the PH and posterotemporal in the NP group. Right: A similar topographic representation of mean N2-like latencies (ms) over the skull (anterior up). These are shown from left (L) to right (R), with the vertex in the middle, for the nontarget- and target-derived waveforms (D/C, upper; T/t, lower). At the top are schemata for PH and NP patient groups and below are schemata for OCD and CON subject groups. The standard deviation range is shown in italics on the right of each row of recording sites. Latencies were longer in target-vs nontarget-based waveforms. PH and NP patients showed less of an antero-posterior decrease than comparison groups (cf. D/C wave).

tero-temporal sites they were longer than in any other group (Scheffe $p < 0.05$; Fig. 5 right).

3.4.3. Summary (N2 components)

N2 ERP peaks had frontal maxima as reported elsewhere (Simson et al., 1977). Negativity increased in the midline and decreased laterally with increases of tone frequency. Patients showed more negativity at Cz, with a left parietal bias in the schizophrenic groups. This spread was consistent with that seen normally with the change from diffuse to focused attention.

The posterior negative shift in our schizophrenic groups was similar to that seen with condition and may reflect an involvement of more cortical areas in effortful categorization processes. While this function is said to be indexed by N2 in healthy subjects, this was based on a decrease of amplitude with increasing difficulty (Novak et al., 1990). Others have reported an N2 decrease with the ease of discrimination (Fitzgerald and Picton, 1983; Alain et al., 1993). Our finding of similar increasing and decreasing functions with tone-type at different sites suggests that, (a) both of the reported findings could be correct depending on the stimulus features used and site recorded, and (b) 'categorization' may be a distributed process.

The interaction of tone type and attention condition for the schizophrenic groups is important for the interpretation of the reversed anteroposterior pattern for MMN amplitude in NP, and the processing negativity asymmetry of PH patients (discussed below, and in detail in Oades et al., 1994a,1996c).

3.5. P3 components

3.5.1. ERP amplitude and latency

Main effects of tone and condition at Cz indicated that, with respect to standards, deviant tones elicited larger peaks and targets even larger peaks in all groups. The main group effect and group × tone interaction referred both to the smaller increases of deviant tone responses in schizophrenic patients and to the small response to standards by the OCD group (vs CON, Scheffe $p < 0.05$; Table 2, Fig. 1).

The normalized data showed that P3 maxima were posterior in all cases (t −13.7 to −18.5, $p = 0.0001$; Fig. 6). The main differences for group, condition and tone (Table 2) were:

1 / Right bias: Across tones and conditions controls showed a small amplitude bias (T3 < T4, t −2.7, $p = 0.007$). This extended to right frontal and posterior positivity measured in both PH and NP groups (t −2 to −3.4, $p = 0.04$–0.001). At these lateral frontal sites there was more positivity in CON or NP than PH groups but at the postero-temporal sites NP and OCD groups showed more positivity than the others (Scheffe $p < 0.05$). OCD patients showed no lateralized pattern.

2 / Irrelevant tones: Deviants (including the 1.4 kHz tone) had a larger lateral frontal representation than the target (F7, F8; Scheffe $p < 0.05$) but at temporal sites the size of response was inversely related to tone-frequency (i.e. the smallest representation for 2.0 kHz; Scheffe $p < 0.05$).

3 / Focused attention: During the discrimination, most peaks became centred on the midline: in general positivity decreased on the right (T4, T6; $F(1,364) = 10.6$–26.7, $p = 0.001$–0.0001). This can be seen in Fig. 6 for each group and tone except for the OCD response to the standard (not significant) and strikingly, in the NP group, the maxima remained at lateral sites (Table 4).

In general, at Cz, latencies were shorter after deviant than standard tones. However, the PH patients showed longer latencies than the controls (Scheffe $p < 0.05$), especially after deviants and targets in the focused attention condition (vs OCD, CON; Scheffe $p < 0.05$: means for 1.4 kHz tone in passive active conditions, 346/344 CON, 421/354 OCD, 379/417 PH, 367/335 ms).

The latencies showed three topographical features: here interpretation is assisted by pointing out that at F8 latencies were typically longer in the least salient situation, after the standard (vs target, Scheffe $p < 0.05$) and in the passive vs active condition ($F(1,364) = 6.1$, $p = 0.01$). (1) All groups had shorter left vs right posterior temporal latencies (t −3.1 to −6.5, $p = 0.003$–0.0001), especially the OCD group (vs CON, Scheffe $p < 0.05$). (2) But on the left, frontal latencies were longer in all patient groups (t +2.7 to +4.0, $p = 0.009$–0.000). (3) On the right anteroposterior differences reversed: laten-

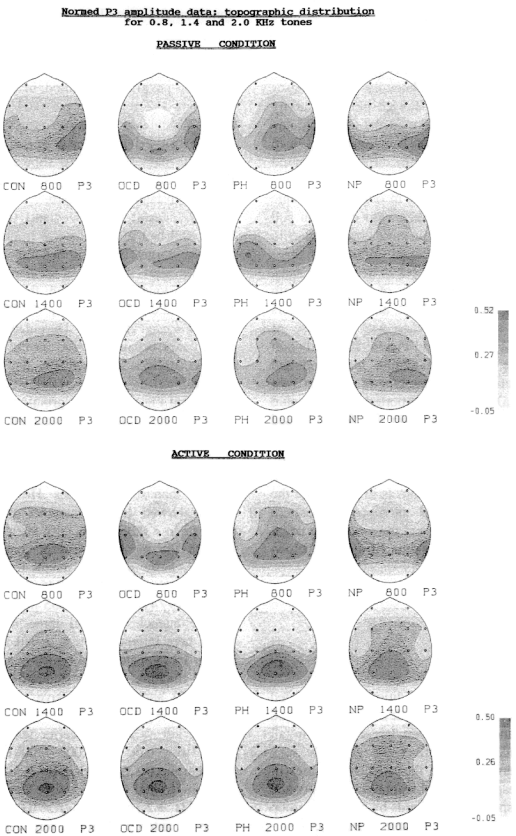

Normed P3 amplitude data: topographic distribution
for 0.8, 1.4 and 2.0 KHz tones
PASSIVE CONDITION
CON 800 P3
OCD 800 P3
PH 800 P3
NP 800 P3
CON 1400 P3
OCD 1400 P3
PH 1400 P3
NP 1400 P3
CON 2000 P3
OCD 2000 P3
PH 2000 P3
NP 2000 P3
0.52
0.27
-0.05
ACTIVE CONDITION
CON 800 P3
OCD 800 P3
PH 800 P3
NP 800 P3
CON 1400 P3
OCD 1400 P3
PH 1400 P3
NP 1400 P3
CON 2000 P3
OCD 2000 P3
PH 2000 P3
NP 2000 P3
0.50
0.26
-0.05

Table 4
Normalized P3 amplitude responses to the irrelevant common standard tone at posterior sites (T5, Pz, T6) in four subject groups under diffuse- and focused-attention (passive vs active) conditions

Group	Passive condition site			Active condition site		
	T5	Pz	T6	T5	Pz	T6
CON	0.284	0.303	0.343	0.275	0.379 [3]	0.235
OCD	0.449	0.330	0.473	0.378	0.369	0.349
PH	0.209	0.369	0.347	0.231	0.358 [1]	0.277
NP	0.336	0.313	0.520 [2]	0.416	0.264	0.450 [2]

In passive diffuse-attention conditions values tended to be higher over temporal (right, T6) than midline sites; under focused attention maxima shifted to the midline, except for the NP group: *t*-test for Pz vs T6 values; [1] trend $p = 0.1$, [2] $p = 0.05$–0.01 (t -2.1 to -2.9), [3] $p < 0.0001$ (t $+5.5$).

cies were longer at posterior than frontal sites in all groups except the PH (t -2.7 to -3.7, $p = 0.009$–0.0001).

3.5.2. *Difference-waves*

At Cz, the largest P3 occurred in the D/C waveform. But in the topographic analysis, posterior maxima were seen for all groups (t -3.3 to -11.0, $p = 0.002$–0.0001) and in all waveforms (t -4.4 to -9.2, $p = 0.0001$). All groups showed more (CON and NP t $+3.1$ to $+4.8$, $p = 0.003$–0.0001) or less of a left posterior temporal bias (PH and OCD, t $+1.8$ to $+2.4$, $p = 0.02$–0.08) in all waveforms (t $+2.2$ to $+4.2$, $p = 0.03$–0.001). This resulted in least positivity in the D/C waveform on the right at T4 and T6 (Scheffe $p < 0.05$). The NP group showed more of a lateral and anterior spread of positivity away from the peak maximum than the other groups (F7, F8; Scheffe $p < 0.05$).

Latencies were shorter at frontal vs posterior sites (t -4.0 to -7.7, $p = 0.0001$), at left vs right frontal (t 4.8, $p = 0.0001$) and at right vs left anterotemporal sites (t 2.5, $p = 0.01$). Across all sites, latencies were shorter in the D/C than all target-derived waveforms (Scheffe $p < 0.05$). Across all waveforms and sites latencies were shorter in the controls than the PH group and vs the NP group on the left (both effects, Scheffe $p < 0.05$).

3.5.3. *Summary (P3 components)*

We emphasize the P3 data in the ERP as, surprisingly, data for the similar late positive component in the difference-wave revealed little more than those for the stimulus-elicited waveform. Apart from the commonly found reduced amplitude in both schizophrenic groups the interest centres on the leftward topographic shift of P3 maxima with focused attention and the treatment of the irrelevant stimuli.

The size of the OCD response to the 0.8 kHz tone was less than in the controls and resembled that in the schizophrenic groups (Fig. 1). Further, for OCD patients, the topography at posterior sites was not differentiated in either of the attentional conditions as in controls. Only the NP group maintained the temporal maxima for the response to the standard in diffuse- and focused-attention conditions. (The standard tone produced a more widespread, if small, response than deviant tones in most subjects, particularly the patients.) However, deviant and target tones produced midline maxima in all groups. Deviant and target tones produced smaller P3 responses in the PH group at most sites, except at the Pz maximum. But both schizophrenic groups showed a right-sided P3 bias.

These results were reflected in a left temporal bias for the late positive component in the difference-wave. Otherwise, group differences were not manifest in difference-waves, including the Goodin-wave which might have reflected diffuse-focused attention differences.

Latencies were relatively shorter to deviant tones or during focused attention; but they were unexpectedly longer for the small peaks of the PH group and usually shorter on the left, the side opposite to peak maxima (especially in the OCD group).

Fig. 6. Topography of normalized mean P3 peak amplitudes over the skull (anterior up) in passive and active conditions. Top: Passive diffuse attention condition: Horizontal rows show the three tones (0.8, 1.4 and 2.0 kHz); vertical columns show the four subject groups; CON = controls, OCD = Obsessive Compulsive-Disorder, PH = Paranoid-Hallucinatory and NP = Nonparanoid Schizophrenia. Bottom: Active focused attention condition: similar representation as above. Across tones and groups P3 tended to peak at right temporoparietal sites during diffuse attention (upper figure). With focused attention P3 peaks shifted to the midline, except for the NP response to the irrelevant standard 0.8 kHz tone (lower figure and Table 4).

4. Comment

This study has been concerned with the principal effects of three types of tones, under conditions of diffuse and focused attention on the size and topographic distribution of amplitude and latency of five components in the simple, stimulus-elicited ERP of four groups of subjects. In parallel we analysed for five components in four types of difference-waves reflecting nontarget- and target-derived stimulus comparisons.

The results have been presented in an atlas-like manner in the previous section in the temporal sequence in which the components (P1, N1, P2, N2 and P3) appear after the eliciting stimulus. A preliminary summary and interpretation has been presented for each component in turn. The main results are now re-grouped in terms of the statistical model and the questions raised in the introduction. Statistically speaking, site interacted with psychopathology (group) from the earliest peak (P1), but tone or condition, apart from minor early effects, interacted later from P2 (latency) and N2 (amplitude) onwards.

This discussion therefore considers first the influence of the type of tone (perception). Second, there is the selective aspect of perception (attention). Here the influence of diffuse attention conditions are contrasted with focused attention and nontarget-derived difference-wave forms are contrasted with those derived from comparisons with targets. Lastly, there were group differences in processing that are not uniquely associated to one or the other forms of stimulus processing. As emphasized in the Introduction, the principal contrast is that between schizophrenics with or without symptoms of hallucinations or delusions and the left/right, anterior/posterior bias of the ERP responses.

4.1. Perception

The pitch of the tones modulated the size of the N2-P3 complex while the frequency of presentation influenced only N1 outright. However, tone deviance did interact with attention condition to influence the latency and amplitude of the N2-P3 complex (below).

4.1.1. N2 peak (pitch)

Increases of tone pitch were associated with decreases of N2 amplitude in the midline, but at lateral sites increases of pitch were associated with increased amplitude. Similarly, at lateral sites P3 amplitude increased with tone pitch. As suggested in the Results section on N2, the topographic analysis may be differentiating between lateral parts of auditory association cortex engaged in categorization in terms of stimulus features, while parts closer to the midline may be involved in a functional categorization in terms of 'target-like' features. This latter type of process is likely to require more effort and to involve input from frontal association areas (Nasman and Dorio, 1993). Hence, recording methods (e.g. type of tone, task and lack of topographic coverage) could explain reports of both increases and decreases of N2 amplitude with discrimination difficulty (Novak et al., 1990; Fitzgerald and Picton, 1983, respectively).

4.1.2. P1 peak (pitch)

Unexpectedly, P1 amplitude in PH patients was found not just to be larger than in other groups but to increase with tone pitch. While neither we (here) nor others (Mathew et al., 1993) have found schizophrenic subgroup differences in hearing thresholds, the latter group did report that hallucinators tended to be poorer at perceiving higher pitched tones, depending on their clinical state and time of day. Our result seems to conflict with this report but as it is derived from multitrial ERP recording, it may reflect a disinhibition or positive feed back deriving from (dys)function in higher cortical centres rather than poor ascending perceptual processing.

The auditory P1 amplitude is reported to increase with sound intensity and from about 70 dB be smaller in schizophrenics (Griffith et al., 1995). It would be possible to suggest that the 'augmenting' tendency was confirmed in our PH sample should higher tones be appreciated subjectively as louder. But our finding of larger peaks in these patients suggests rather that subgroup differences have been overlooked in previous studies. Thus, for example, Strandburg et al. (1994) found no group effects in a visual task, and Landau et al. (1975) reported flatter peaks in schizophrenic patients; yet Pritchard (1986) noted opposing tendencies between acute and chronic paranoid schizophrenics on P1-N1 measures.

4.1.3. N1 peak (rarity)

Across groups and condition, N1 amplitude was larger after the deviants than the standards. The

effect was most marked in the OCD group and least marked in the PH group. Despite this OCD and PH, patients had longer N1 latencies than the other groups.

N1 amplitude suppression in schizophrenia is not unusual (Pfefferbaum et al., 1989; O'Donnell et al., 1994). But that this effect was least marked in the PH group is consistent with the increased P1 noted for this group, given that the deviants in the current task were higher pitched than the standards.

Interpretation of the effects on the N1 component have been diverse. At one extreme, Clifford and Williston (1993) argue from manipulations of stimulus context that perceptual rather than attentional functions are indexed here. At the other extreme, O'Donnell et al. (1994) argue from the effects of psychopathology that changes result from different levels of arousal. As our results show that deviants still influence the schizophrenics' responses we would argue in parallel with Hansen and Hillyard (1988) that N1 marks both the perceptual registration in the auditory cortex of a stimulus and the start of further (selective) processing, for which in schizophrenics' resources are limited.

The sensitivity of the OCD group is consistent with the report of a larger frontal target-N1 in OCD patients (Towey et al., 1993): our use of a 3-tone oddball leads us to suggest that the effect may relate more to the effect of stimulus rarity than its association with a target. While the longer latency of the response in OCD patients and a subgroup of schizophrenics is not easy to interpret, it does throw doubt on an early claim that neuroleptics cause a blanket increase of latency in early ERPs (Saletu, 1978).

4.2. Attention-releated effects in the ERP

4.2.1. P2 peak

The first significant effect of attention condition on an ERP component was the decrease of the P2 latency with the focusing of attention. In comparison groups this was particularly evident on the left side. P2 latencies in schizophrenic patients were less sensitive to condition.

In a 2-tone oddball paradigm, Javitt et al. (1995c) reported that P2 latencies increased in the active condition for healthy but not schizophrenic subjects. In fact, the combined data for their medicated and unmedicated groups and our NP and PH patients are very similar, which suggests that whatever the constraints on processing in healthy subjects were, the schizophrenics' responses were impervious to them. The differences in the controls' responses probably reflects the increased demands on processing in our 3-tone task.

4.2.2. N2 peak

The attentional condition also exerted a differential influence on the subjects' N2 amplitudes and latencies. N2 peak negativity spread more posteriorly as attention became focused. This effect was more pronounced in patients. After irrelevant tones a left frontal bias was seen in NP and a left temporal bias in PH patients. Although deviants elicited responses with shorter latencies in all subjects, the focusing of attention decreased latencies only in the comparison groups. In NP patients latencies remained long, especially at right frontal sites.

In contrast, schizophrenic target-N2 peaks have been reported to be smaller than in controls at C3/4 and T3/4 sites (O'Donnell et al., 1993). However, their patients, who had prominent PH symptoms, showed a positive potential shift from N1 to N2, that may have been an exaggeration of the similar PH vs NP trend seen here (Fig. 1) and thus may have masked the spread of negativity we observed.

The latency changes are likely to reflect the effort required and difficulty experienced in the discrimination. Increases of latency with increased discrimination difficulty are consistent with reports from healthy subjects (Novak et al., 1990). The problem in schizophrenics was attributed by Michie et al. (1990), in particular, to their relatively prolonged response to the nontarget stimulus. This is one of several examples showing that it is the unusual processing here of irrelevant stimuli by schizophrenics that is remarkable, especially if the situation demands effort.

4.2.3. P3 peak

The widespread finding that P3 amplitude was larger to targets than to deviants, and larger to deviants than to standards was replicated. This pattern was repeated in the smaller peaks shown by both schizophrenic groups. The putative P3a response to deviants was larger than that to targets

only at lateral frontal sites. For most subjects, under diffuse attention conditions, the small response to standards was localized at temporal rather than parietal sites. If deviants were considered then there was a right-sided bias. However, under focused attention conditions these maxima moved to the midline. As a further example of the anomalous treatment of irrelevant stimuli by NP patients, their standard P3 response remained at temporal sites during focused attention.

In comparison subjects, P3 latencies were shorter after deviant stimuli. This was not evident in PH patients and the difference was exaggerated in the focused attention condition. For comparison subjects, the more irrelevant the stimulus (i.e. diffuse vs focused, standard vs deviant) the longer was the latency at right frontal sites. This differentiation was less marked in patients, particularly the PH group. This is the mirror-image of effects seen with the N2 peak and is another example of unusual treatment of irrelevant stimuli in schizophrenic patients.

Several laboratories have recently reported a relative decrease of P3 amplitude on the left in schizophrenic patients (Koga et al., 1987; Kraft et al., 1991; Sieg et al., 1991; McCarley et al., 1993; Strik et al., 1994). But others have not found this (Moore et al., 1992) or indeed report a decrease on the right in paranoid patients (Maurer and Dierks, 1987). Like Pfefferbaum and colleagues (1989) we found a right bias at frontal sites in schizophrenic groups and a leftward shift with the introduction of the discrimination in all subjects. This shift across all tones (except for the 0.8 kHz response in the NP group) argues against the possibility that our finding of a right bias reflected movement artifact. Rather it supports our contention that NP patients in particular are impaired in processes needed to actively ignore and suppress responses to irrelevant material – activation is still abnormally picked up from temporal recording sites, as with the MMN (below).

OCD patients have been reported to show decreased P3 amplitude in a 2-tone oddball (Towey et al., 1993). But Fig. 1 shows that responses were more differentiated with 3 tones: peak size increased with pitch from being abnormally low to a bit higher than normal. One may also note the apparent long duration of the positive shift (Fig. 1, not evaluated here). Thus, stimulus saliency seems likely to dominate the OCD information processing strategy. But importantly the topographic analysis showed no major deviation from the controls.

In the context that P3 ERP latencies were normally shorter after deviant than standard tones but were longer if the tone became the target, PH latencies were (a) longer overall, (b) longer in the focused attention condition and (c) did not show the usual pattern of being shorter over frontal and longer over posterior sites. These results are consistent with a topographically widespread increase in effort required in PH subjects for processing leading up to the putatively P3 indexed function of updating (Donchin and Coles, 1988). In contrast, in NP patients the problems in terms of increased latency were found more at left frontal sites. Indirectly supporting this is a reported association between increased P3 latency in schizophrenia and increased technetium uptake measured by SPECT in the left frontal area (Blackwood et al., 1994).

4.3. Attention-related effects in the difference-waves: nontarget- and target-comparisons

4.3.1. P1-N1-P2-like components

Already at P1-like latencies PH patients show an anterior shift of the normal parietal locus of a positive component which showed shorter latencies on the right. These are the same patients characterized by hallucinations and delusions who showed a marked P1 in the simple ERP and the topographic change has elements in common with the later P3-like late positive component. However the OCD group showed the largest P1-like component suggesting that selective comparative processing at this level is least marked in these patients.

The early negative, N1-like component, symmetrically frontocentral in comparison subjects, was biased to the right with a shorter latency on the right in NP patients, but only for target-derived waveforms. The opposite pattern was evident in nontarget-derived waveforms. Thus, the two types of comparative processing are already differentiating from normal at this early stage, but as the pattern is not the same as later MMN or processing negativity, we must have a look at the ways in which it may be regarded as a precursor.

For nontarget comparisons in both schizophrenic groups, maxima shifted left (like MMN in the PH group). But there was also a postero-lateral spread of negativity extending further on the right than in comparison groups. Latencies were increased in both schizophrenic groups but not differentiated laterally as in MMN. For target-derived comparisons, PH patients show some lateralization of negativity away from the midline to both sides, reminiscent of MMN, while NP patients show midline maxima (anterior and posterior) not resembling the left frontal bias in processing negativity (here and Oades et al., 1994b). Thus, the idea that the N1-like component in target-derived waveforms represents an early 'negative difference' type of processing negativity (Alain et al., 1993; Woods and Alain, 1993) rests with the similarity to the target-elicited ERP which was larger at left- and smaller at right-frontal sites. Target-derived difference-waves here indicated that N1-like components still reflected stimulus feature comparisons.

P2-like deflections with parietal maxima in nontarget-derived waveforms in most subjects had shorter latencies than in target-derived waveforms. In controls, maxima in target-derived waves shifted to the right and spread anteriorly to the right. However, the anterior spread was more right-biased in NP and left-biased in PH patients. In comparison groups, latencies were uniformly shorter at posterior loci. However, PH patients showed shorter frontal latencies in nontarget and NP patients shorter frontal latencies for target-derived comparisons. This already suggests that the sequence of activation of brain areas in making stimulus comparisons is different in these two groups and anticipates developments in the next components.

4.3.2. N2-like components (mismatch and processing negativity)

MMN in the D/C waveform became lateralized in PH patients (left maxima but more right-sided spread) and more exclusively lateral and posterior in NP patients (right-sided maximum). In the raw data, frontal MMN was halved in PH and virtually absent in NP patients (Oades, 1995). OCD patients merely showed a right frontal bias. Curiously, longer frontal than posterior latencies were recorded for NP, OCD and controls but the reverse was seen in PH patients.

From our data it is easy to see that the frontal N2-like peak in the D/C wave (MMN; Näätänen, 1990) is conventionally reported as absent in schizophrenics (Catts et al., 1995; Javitt et al., 1993, 1995b,c; Oades et al., 1993; Shelley et al., 1991). But, while it was absent in NP patients, it was merely reduced in amplitude in our patients with active PH symptoms (Oades, 1995). Thus, the question of the type of patient selected could explain why Kathmann et al. (1995) could not find a reduction of MMN in their sample. Also pertinent is the state of attention. Both here and in the study of O'Donnell et al. (1994) the apparent decrease is more evident during focused attention, when controls show an increase of MMN amplitude (Oades and Dittmann-Balcar, 1995). It is noteworthy that Catts et al. (1995) and Kathmann et al. (1995) both used an accessory visual task while recording MMN, but only the former study found a reduction of MMN in the schizophrenic sample. However, evidence of task-performance to substantiate the focusing of attention, was not provided for the visual task.

Whether the MMN change reflects a disoriented dipole, a translocated source of activity or another aspect of the recording situation remains to be determined. As different brain pathways compete with each other to mediate a given function (Raichle, 1994), a small translocation of source in the cortical convolution in lateral temporal, secondary auditory cortex (Tiitinen et al., 1993), should not be overlooked in patients potentially suffering some atrophy after an insult on cerebral function (review, Straube and Oades, 1992). A small change of dipole orientation seems reasonable considering our finding that MMN appeared 'bi-lateralized' in PH patients, was dependent on active symptom state and thus may represent a transitional stage towards the situation in NP patients (see Oades et al., 1996c for a more detailed discussion).

In target-derived waveforms (processing negativity and its special case the negative difference, T/t) normalized data showed left frontal maxima in most subjects. This suggests that the substrate for target processing is intact. The asymmetry in the raw data (negativity right frontal left temporal in PH and vice versa in NP patients, Oades et al., 1994b) is reflected in the peak latencies. This suggests that it is the sequence of activation of different cortices that dif-

fers between the psychotic groups. Considering that the source of difference-wave negativity is reported to lie in (left) auditory association areas in temporal (e.g. MMN, Tiitinen et al., 1993) to inferior parietal cortices (e.g. processing negativity, Woods et al., 1993), and in view of evidence of lateral frontal asymmetries in other components already described, we propose a PH/NP distinction on the basis of frontal input to comparative target-derived stimulus processing in the temporal parietal lobe. The impairment may reside in the input from left frontal areas in PH and right frontal areas in NP patients.

Partial support for our result may be derived from O'Donnell et al. (1993) who studied a mixed group of schizophrenic patients with a 2-tone oddball. Processing negativity was found to be reduced at T3 and T4 sites. However, their subjects were quite different from ours, being chronically ill with tomographic signs of atrophy and symptoms of thought disorder. In a dichotic listening paradigm, decreased frontal processing negativity amplitude in acute and remitted schizophrenics has also been found (McPherson et al., 1991). As noted earlier, the reason we did not find a general decrease of processing negativity amplitude may reflect that these authors' use of the stimulus feature of location was more influential than our manipulation of pitch.

4.3.3. P3-like late positivity complex

In general, late positivity peaked left of the parietal midline which reflected much less positivity on the right in nontarget- vs target-derived waveforms. Latencies were shorter in nontarget- than target-derived waveforms, particularly at left frontal and right temporal sites. Apart from the longer latencies exhibited by the schizophrenic patients, topographic differences reflecting patient groupings were not marked.

There was a broader anterolateral positive shift in the NP, and, nonsignificantly, in the OCD group, but group differences evident in simple ERPs did not appear in the difference-waves. These results are difficult to reconcile with other reports of large decreases in late positive components in the difference-waves of schizophrenics (O'Donnell et al., 1994; Javitt et al., 1995b). However, claims of associations of amplitude decreases with cerebral atrophy in patients considerably older than our own (Egan et al., 1994; McCarley et al., 1993) could be taken to explain the absence of group differences in patients perhaps too young to show significant CNS degeneration.

Frontal P3a-like components from a T/D-like waveform with amplitudes similar to the present study, were reported for healthy and schizophrenic subjects (Michie et al., 1990), while posteriorly the P3b-like component, consistent with our own data, was smaller in the schizophrenics but did not show any asymmetry at T3 or T4. However, studies with the Goodin-wave (Morstyn et al., 1983; Faux et al., 1988) resemble more our stimulus-elicited P3 results. Their controls showed peaks left of the midline while schizophrenics showed a right anterior shift with significantly less left temporal positivity. Analysing this difference they noted that schizophrenics differed in the D/C-like subtraction wave more on the left and the controls more on the right. As a differential P3 response to deviant and standard tones would be expected, one could argue that the schizophrenic impairment was more on the right, even though by reference to controls there was a relative decrease on the left.

4.4. General group ERP topographic differences

Notable P1 features were an increased amplitude in PH and a decreased latency in NP patients; further the usual right-sided asymmetry was absent in the schizophrenic groups and maxima were more posterior in the NP group. Latencies in the OCD group were less topographically differentiated.

A shorter P1 latency has been noted before (Saletu, 1978) and in the context of Saletu's findings, its presence in our NP group may indicate that these patients were not responding to neuroleptic treatment.

However, with regard to topography, schizophrenics have been reported as not showing the normal pattern of right-sided asymmetries in the P1 latency range in visual and somatosensory modalities (Strandburg et al., 1990; Abe et al., 1991, contrast Connolly et al., 1983; Furlong et al., 1990), as we have now shown here for the auditory modality. This might reflect problems with the more automatic processing of information attributed to P1. For example, prepulse inhibition of the P1, typically reduced in

schizophrenics (e.g. Griffith et al., 1995), has been reported to be smaller on the left than the right (Schall et al., 1996). This implies a marked left-sided component that would contribute to P1 symmetry in the patients. The absence of a clear frontocentral maximum for the NP group may be an early sign of impaired frontal input (Shagass and Roemer, 1991), which attains its clearest expression later in the MMN.

Features of the N1 component were that peaks were smaller in schizophrenics and biased to the left (right impairment) in NP patients but were larger in OCD patients and biased to the right.

Considering that N1 amplitude was smallest in the PH group (and not left-biased), it is intriguing that left sided N1 peaks could be improved under amphetamine, known for its potential for inducing paranoid psychosis (Buchsbaum et al., 1981). This indirectly supports our findings of unusual processing reflected at left frontal sites in patients with hallucinations and delusions. Egan et al. (1994) also found modestly decreased N1 amplitude in their schizophrenic sample of largely undifferentiated patients and noted a correlation of amplitude with MRI measures of white and grey matter in the left prefrontal area. The positive nature of the correlation is consistent with the left bias in our NP sample.

Latencies were normally shorter at lateral frontal (vs posterior) and at posterior (vs frontal) midline sites. This finely tuned sequence of activation was not found in the OCD group where, despite the topographic pattern of peak amplitudes, latencies were less differentiated across sites than in the other groups. If N1 is viewed as reflecting the allocation of processing capacity (Hink et al., 1978), then this lack of differentiation may reflect poor selectivity, despite increased activation.

The main features of the P2 component were that it was a minor peak for OCD patients and its normal parietal locus shifted right frontally in NP patients.

With respect to the modest P2 size in the OCD group it is of interest that we recently reported 2-fold increases of P2 amplitude in two groups of impulsive patients with attention-deficit hyperactivity or complex-tic disorder (Oades et al., 1996b). As OCD patients are usually regarded as anything but impulsive, this supports our interpretation of the component as reflecting the inhibition of the competition of other channels for influencing or mediating the further processing of the stimulus.

P2 amplitude did not appear to differ in our schizophrenic sample in contrast to the report of a decrease from Merrin and Floyd (1994). But their effect may be regarded as nonspecific in two senses. Firstly, a decrease was also seen in patients with mood disorders (Merrin and Floyd, 1994), and secondly, an increase in schizophrenics was reported from a visual paradigm (Strandburg et al., 1994). Examining midline sites, O'Donnell et al. (1994) found P2 peaks to be larger frontally and to increase in the active oddball condition in healthy but not schizophrenic subjects. Our raw and normalized data differ in the localization of peak maxima, except for NP patients. But our data concur, albeit nonsignificantly, that amplitude tended to increase with focused attention, except in the PH patients. Two aspects of the studies could account for the topographical differences. Firstly, the tones we used were not so loud. This could be important for response localization considering that P2 often augments with stimulus intensity (Hegerl and Juckel, 1993). Secondly, our subjects were nearly 20 years younger on average.

The tendency for a left lateralized peak in the NP patients contrasts with a right bias that has been reported for schizophrenics (Shenton et al., 1989a). However, the patients in Shenton's report exhibited clinical features more like our PH than NP group. Pfefferbaum et al. (1989) found larger than normal peaks in medicated schizophrenics but smaller ones in non-medicated schizophrenics. The relative frontal increase in our largely medicated NP group is not inconsistent with this finding.

The N2 and P3 components have been extensively considered above under perceptual and attentional influences. N2 tended to be larger and later in patients vs controls and biased to the right in OCD and to left frontal/temporal sites in NP/PH patients, respectively. Over and above the association of P3 amplitude and locus with perception and attention-related processing, P3 was localized marginally right of the parietal midline. Positivity extended further anteriorly in the schizophrenic groups and laterally in OCD patients. Peaks in the raw data were notably smaller in schizophrenics after deviant and in OCD patients after standard tones. It is worthy of note that

damage to the temporo-parietal area 22 has been associated with decreases of P3a and P3b amplitude (Woods et al., 1993). This area has particularly rich connections with frontal and cingulate areas (Molnar, 1994), whose input was postulated above to be so important for the respective asymmetries of processing negativity in NP and PH patients.

Finally, although we did not specifically investigate the influence of medication, we believe it played a subordinate if not therapeutic role (Straube and Oades, 1992; Faux et al., 1993; cf. similarity of N1 and P3 loci between groups). If these major peaks showed similar localization trends then it would seem redundant to explain other differences between PH and NP groups in terms of nonsignificantly different levels of medication.

5. Conclusions

We have shown that the comparison of stimulus-elicited ERPs (diffuse and focused attention) and difference-waves (target- and nontarget-derived) shows up nonevident aspects of the topography of information processing (e.g. the relative symmetry of the N2 in the PH group contrasts with the lateral patterns in the MMN and 'negative-difference').

The comparison of target- and nontarget-derived difference-waves showed quite distinct frontal and temporal patterns of activity in NP patients (e.g. the contrast of the MMN with the 'negative-difference').

Study of the minor components in difference-waves (P1-P2) indicated the extent to which they were precursors of later N2- and P3-like processing. Thus, similarities were more evident for positive than negative deflections; but the symmetry of N1-like peaks in controls and their asymmetry in the NP and OCD groups were both indicative of later N2-like changes.

Analysis of these components illustrated the value of subdividing the schizophrenic group according to the predominance or near-absence of hallucinatory and delusory symptoms (e.g. MMN amplitude decreased according to the diagnostic trait of schizophrenia but the extent of the decrease depended on the symptom-state).

Asymmetries of latency or amplitude, repeated for successive components were opposed in these two schizophrenic groups and lead to the proposition that frontal inputs to temporo-parietal processing are more impaired on the right in NP and on the left in PH patients, supporting the neuropsychopsychological hypothesis of Gruzelier et al. (1993, 1995).

Lastly, we used an OCD comparison group and young patients, mostly acutely ill for the first time. The contrast with the more frequently used chronic schizophrenics vs mood-disordered comparison groups provided a useful extension to the specificity of the changes recorded. Further, in our sample the OCD responses depended more on stimulus- than task-related features. This implies that the reported similarity of target and nontarget P3 responses (Towey et al., 1994) may reflect more the adventitious and less the core symptoms. The youth of the psychotic patients may also explain differences in P3-like amplitudes and loci between schizophrenic samples.

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