Ann.Bull.RILP(1984)No.18, 267-294

SELECTIVE ATTENTIONAL FUNCTIONING AS REFLECTED IN ERPs DURING SYLLABLE DISCRIMINATION TASKS (II)
Correlations between Cognitive Deficits and Abnormal ERP Findings in Schizophrenics*

Tomomichi Kameyama**, Ken-Ichi Hiramatsu**, Shin-Ichi Niwa**, Osamu Saitoh**, Karen Rymar*** and Kenji Itoh

Summary

In order to investigate schizophrenic deficits in information processing, Event-Related Potentials (ERPs) recorded during syllable discrimination tasks from 22 schizophrenics (unmedicated 10; medicated 12) as well as 20 normal controls, were analyzed. In the normal controls, an increase in the amplitudes of the N100 component due to channel selective attention, as well as an augmentation of the late positive component upon detection of targets were observed. On the other hand, in the schizophrenics, neither an effect of channel selection nor one of target detection was observed in the ERPs. The unmedicated group of schizophrenics demonstrated shorter latencies for the N100 and P200 components derived from the T3 region as compared to the other two groups. These results seem to suggest that schizophrenics exhibit deficits in 'stimulus set', as well as 'response set', as defined by Broadbent. Furthermore, it was suggested that disturbances of selective attention in schizophrenics correlate with disturbances in the left hemisphere.

I Introduction

Ever since Cameron (1938)[1] proposed the overinclusion theory, many hypothetical models concerning schizophrenic attentional deficits have been proposed. It is possible to classify many of these hypothetical models into several groups employing Broadbent's theory of information processing as a basis. For example, the defective filter hypothesis proposed by McGhie and Chapman (1961)[2] is thought to be related to a disturbance in 'stimulus set' as defined by Broadbent (1971)[3]. On the other hand, the collapsed response hierarchy hypothesis proposed by Broen and Storms (1966)[4] is thought to be relevant to a dysfunction of 'response set' (Broadbent, 1971[3]).

Recently, Event-Related Potentials (ERPs) have been frequently used as physiological indices which represent information processing in the brain. Particularly, the component with a latency of approximately 100-200 msec in the Auditory Evoked Potentials (AEPs) of normals, which is known to be enhanced by

* Submitted to 'Biological Psychology'

** Department of Neuropsychiatry, Faculty of Medicine, University of Tokyo
*** Temple University Japan

increased attention, has been suggested as being related to the process of attenuation of irrelevant stimuli (that is, Broadbent's proposed 'stimulus set') (Hillyard, Hink, Schwent and Picton, 1973[5]; Hillyard and Picton, 1979[6]). It has also been suggested that the P300 component of AEP is related to the stimulus evaluation and decision-making process (that is, Broadbent's proposed 'response set') (Hillyard et al., 1973[5]; Hillyard and Picton, 1979[6]; Donchin, 1979[7]). Accordingly, ERP studies of schizophrenics can be expected to provide further information concerning schizophrenic attentional deficits, particularly in terms of Broadbent's "two-sets model" for attentional functioning.

It has been reported by some authors that schizophrenics demonstrate smaller amplitudes of the N100, P200 and P300 components of ERPs (Jones and Callaway, 1970[8]; Levit, Sutton and Zubin, 1973[9]; Roth and Canon, 1972[10]; Saletu, Itil, and Saletu, 1971[11]; Shagass, Straumanis, Roemer and Amadeo, 1977[12]). However, there are few investigations in which schizophrenic deficits in attentional functioning have been examined through ERP recordings during relatively complex psychological tasks requiring subjects to activate both sets of attentional functioning. Roth, Horvath, Pfefferbaum and Kopell (1980a)[13], Roth, Pfefferbaum, Horvath, and Kopell (1980b)[14] and Roth, Pfefferbaum, Kelly, Berger and Kopell (1981)[15] have reported that schizophrenics demonstrate a disturbance in the stimulus selection process, but at the same time they report that their experimental paradigm was not appropriate for examining 'stimulus set' and 'response set' separately. Baribeau-Braun, Picton and Gosselin (1983)[16] recorded ERPs in schizophrenics during dichotic listening tasks. They reported that schizophrenics showed an abnormal late stage, indicating inefficiency in processing information from detected targets. They concluded that the schizophrenic attentional deficit consists in control and maintenance of a selective processing strategy. Saitoh, Hiramatsu, Niwa, Kameyama, Itoh (1983)[17] recorded ERPs in schizophrenics during dichotic detection tasks. They reported that schizophrenics demonstrated disturbances in both 'stimulus set' and 'response set'. Their method had a minor flaw, however, in that they were unable to investigate a significant effect of 'stimulus set', channel selective attention, through comparison of ERPs elicited by the stimuli presented to the attended ear with those for stimuli presented to the non-attended ear, because the stimuli were presented to both ears simultaneously. In investigating schizophrenic deficits in terms of the two-sets model, syllable discrimination tasks such as those employed by Hink, Hillyard and Benson (1978)[18] seem pertinent, since these particular tasks activate the two sets successively. Therefore, ERPs were recorded during syllable discrimination tasks in this study.

In addition, many recent studies utilizing neuropsychological tests have been carried out on schizophrenics. These studies have suggested that 1) schizophrenics demonstrate a left hemispheric dysfunction (Corburn and Lishman, 1979[19]; Flor-

Henry, 1976[20]; Gur, 1978[21]; Hammond and Gruzelier, 1978[22]); or that 2) schizophrenics display a disturbance in the integration of both hemispheres (Beaumont and Dimond, 1973[23]; Carr, 1980[24]; Green, 1978[25]). At the same time, several authors have reported that hemispheric functions underlie attentional functioning (Dimond and Beaumont, 1973[26]; Dimond, 1976[27]; Ellenberg and Sperry, 1979[28], 1980[29]). Although attentional deficits are thought to play an important role in producing poor performance on neuropsychological tests in schizophrenics, few studies have been conducted to clarify the relationship between attentional deficits and hemispheric functioning in schizophrenics. Hence, this study also aimed at investigating this relationship through an analysis of abnormalities in ERPs recorded in the bi-temporal regions during syllable discrimination tasks.

II Method

II-1 Subjects

Subjects consisted of schizophrenic patients (11 males, 11 females) randomly selected from a group of schizophrenic patients under treatment at the Neuropsychiatric Outpatient Clinic, Tokyo University Hospital. All patients met the diagnostic criteria of DSM-III (American Psychiatric Association, 1980[30]) for schizophrenic disorders. The ages of the patients ranged from 19 to 39 years (mean age: 29.1 years). The duration of illness ranged from 1 to 19 years (mean: 7.0 years). Their academic background ranged from 9 to 16 years (mean: 13.6 years). Ten out of the 22 schizophrenics had not received any drugs for at least 4 weeks prior to the experiments (unmedicated group), while the remaining 12 schizophrenics were under medication at the time of the experiments (medicated group). Further details concerning the patients are presented in Table 1.

The unmedicated group consisted of 5 males and 5 females: mean age, 28.2 years; mean duration of illness, 5.2 years; mean academic level, 13.4 years. According to the DSM-III, the subtypes of the schizoprenic disorders of these subjects were as follows: disorganized type, 3; residual type, 3; paranoid type, 2; undifferentiated type, 2. Clinical symptoms for each schizophrenic patient were measured using the BPRS (Brief Psychiatric Rating Scale). The total BPRS scores of the unmedicated group ranged from 29 to 68 (mean: 43.4).

The medicated group consisted of 6 males amd 6 females: mean age, 29.8 years; mean duration of illness, 8.4 years; mean academic level, 13.8 years. The subtypes of the schizophrenic disorders for this group were as follows: residual type, 11; disorganized type, 1. Their total BPRS scores ranged from 25 to 51 (mean: 39.3). The medicated group had received various neuroleptics. The dosages were converted into equivalent dosages of chlorpromazine according to Lehmann's conversion table (1975)[31]. Their dosages ranged from 50 to 600 mg (mean: 344.5mg/day).

There were no significant differences between the two groups

in terms of age or academic level. There was also no significant difference between the two groups concerning total BPRS scores. However, the mean scores on item 5 (guilt feelings), item 11 (suspiciousness), and item 12 (hallucinatory behavior) were significantly higher in the unmedicated group ($t(20)=2.18$, 2.12, 2.30, respectively; $p<0.05$).

The twenty normal controls employed in the study by Hiramatsu, Kameyama, Niwa, Saitoh, Rymar and Itoh (1983a)[32] served as the control group. These normal controls consisted of 10 males and 10 females, with ages ranging from 20 to 38 years (mean age: 29.1 years); mean academic level: 16.5 years.

All of the patients and normal controls were right-handed and were found to be free from any hearing disabilities.

II-2 Procedure

The auditory stimuli, procedure, recording system, and data analysis were the same as reported in Hiramatsu et al. (1983a)[32]. Brief explanations for the above items are given below. Four CV-syllables (/ba/, /da/, /ga/, /za/) uttered by a male voice were presented to one ear, with the same four CV-syllables by a female voice being presented to the other ear through headphones monaurally. Each of the eight stimuli was presented randomly with an equal a priori probability of 0.125. The duration of the stimuli was 150 msec. The intensity of the stimuli was approximately 60 dBSL. The interstimulus intervals ranged from 800-1000 msec.

The subjects were seated in an anechoic room with eyes closed. First, they performed 2 runs of the control condition in which they simply listened to the stimuli, with the sidedness of the stimulus presentation being changed. Then, they were informed that a male voice would be presented to one ear, with a female voice being presented to the other, and were required to count silently the number of occurrences of a particular syllable in a given ear in each run (task condition). All of the subjects performed 16 runs of the task condition; that is, four target syllables X two voices (male or female) X two attended channels (left or right ear). The number of the target syllables for each run was set within the range of 18-26. After the conclusion of each run, the subjects were asked to give the number of the target stimuli detected.

During the runs, EEGs were recorded monopolarly with Ag-AgCl electrodes placed at the Cz, T3 and T4 regions, utilizing linked earlobe electrodes as references. The EEGs without artifacts were passed through a bandpass filter set at 1.5-25 Hz (-6 dB/oct) and averaged separately for the left and right ears into four categories. The four categories were as follows: 1) target syllables in the attended ear (N=128); 2) non-target syllables in the attended ear (N=384); 3) target syllables in the non-attended ear (N=128); 4) non-target syllables in the non-attended ear (N=384). The sampling periods were 20 msec before and 600 msec

after the stimulus onset.

Each of the ERP components was defined as follows: N100 was the most negative peak in the 56-156 msec period after the stimulus onset; P200 was the most positive peak in the 40-120 msec period after the N100 peak; the late positive component was the positive deflection in the 50-330 msec period after the P200 peak. The peak amplitudes and latencies of the N100 and P200 components, as well as the averaged amplitude of the late positive component, were measured. Each amplitude of the ERP components was measured with respect to a zero level, which was defined as the mean amplitude during a 20 msec pre- and post-stimulus period.

II-3 Statistical analysis

The data was analyzed utilizing the ANOVA program of the SPSS (Statistical Package for the Social Sciences). The ERPs derived from the Cz region were analyzed (Cz-ANOVA), with the amplitudes and latencies of the N100 and P200 components, as well as the averaged amplitudes of the late positive components, being employed as dependent variables. Age was employed as a covariant, with the following as independent variables:

1) In the control condition:
   i) normal controls: SEX (Male/Female); EAR (the sidedness of the stimulus presentation: Left/Right)
   ii) schizophrenics: SEX; EAR; MED(Medicated/Non-medicated)
2) In the task condition:
   i) normal controls: SEX; EAR; CHANNEL (Attend/Non-attend); SYLLABLE (Target/Non-target)
   ii) schizophrenics: SEX; EAR; CHANNEL; SYLLABLE; MED
   iii) normal controls plus schizophrenics: EAR; CHANNEL; SYLLABLE; DIAG (Normal/Schizophrenia)

The data of the ERPs derived from the T3 and T4 regions were combined and analyzed, adding the factor REGION (T3/T4) to the above factors (T3,T4-ANOVA).

To examine the influence of neuroleptics on the amplitudes and latencies of ERPs, Pearson's correlations between drug dosages and ERPs were calculated for the medicated group. To examine the relationship between psychiatric symptoms and ERPs, Pearson's correlations between total BPRS scores and ERPs were calculated for the unmedicated group, as well as the medicated group.

To examine the relationship between performance levels and ERPs, we calculated EIs (Error Indexes) according to the following formula:

$$EI = \frac{|\text{number of targets} - \text{subject's answer}|}{\text{number of targets}} \times 100.$$

Pearson's correlations between these EIs and ERPs were calculated for the schizophrenics as well as for the normal controls.

Furthermore, Pearson's correlations between drug dosages and total BPRS scores as well as between drug dosages and EIs, were calculated for the medicated group. Pearson's correlations between total BPRS scores and EIs were calculated for the unmedicated group, as well as the medicated group.

III Results

Figure 1 illustrates the averaged ERP patterns of the schizophrenic and normal control subjects. The amplitudes of the N100 and P200 components of the schizophrenics were smaller than those of the normal controls. The waveform of the P300 component, which was clearly elicited by the target stimuli in the normal controls, could not be clearly identified in the schizophrenics.

The amplitudes and latencies of the N100 and P200 components and the averaged amplitudes of the late positive components derived from the Cz region are presented in Table 2, and the corresponding values for the T3 and T4 regions in Table 3. The averaged amplitudes of the late positive components derived from the Cz, T3, and T4 regions under the task condition are presented in Table 4.

III-1 The Effect of SEX on ERPs

The ANOVA revealed that the interaction of SEX X REGION was significant for the following four ERP components in the normal controls under the task condition: 1) the amplitudes of T3-N100 and T4-N100; 2) the latencies of T3-N100 and T4-N100; 3) the amplitudes of T3-P200 and T4-P200; and 4) the latencies of T3-P200 and T4-P200 ($F(1,293)=5.62$, 12.72, 13.45, 9.67, respectively, $p<0.05$).

The ANOVA revealed that SEX did not interact significantly with any other factor in the normal controls or in the schizophrenics. Since this study aims mainly at the effects of CHANNEL and SYLLABLE on ERPs, the factor SEX is excluded from the following discussion.

III-2 ERPs in the control condition

The effect of EAR X REGION on the amplitudes of T3-N100 and T4-N100 was significant in the normal controls ($F(1,75)= 8.28$, $p<0.01$), but not in the schizophrenics. Furthermore, in the normal controls, the effect of EAR on the amplitude of T4-N100 was significant ($F(1,37)=15.95$, $p<0.01$), but its effect was not significant on the amplitude of T3-N100. That is, in the normal controls, the amplitude of T4-N100 elicited by the stimuli presented to the left ear was larger than that for the right ear. However, this effect was not observed in the schizophrenics.

The effect of REGION on the latencies of T3-N100 and T4-N100 was significant in the schizophrenics, as well as in the normal controls (normals: $F(1,75)=9.37$, $p<0.01$; schizophrenics: $F(1,79)=$

Table 1 Subjects

case	age	sex	DSM-III subtype	duration of illness (years)	neuroleptic dosages (mg/day)	total BPRS scores	education (years)
Medicated schizophrenics							
1	35	M	residual	19	375	44	16
2	30	M	residual	4	188	42	16
3	30	F	residual	10	50	45	12
4	39	M	residual	4	225	51	16
5	24	F	residual	4	213	44	14
6	34	M	residual	11	600	34	16
7	28	M	residual	9	375	25	12
8	32	M	residual	12	95	41	12
9	23	F	residual	4	600	34	12
10	33	F	residual	7	558	32	16
11	19	F	residual	4	268	38	12
12	31	F	disorganized	13	600	42	12
Unmedicated schizophrenics							
13	25	F	disorganized	8	0	52	12
14	30	F	undifferentiated	3	0	45	16
15	32	F	paranoid	5	0	39	9
16	27	F	undifferentiated	3	0	59	12
17	24	M	disorganized	4	0	68	13
18	34	M	residual	10	0	31	12
19	28	M	paranoid	1	0	29	16
20	28	M	disorganized	6	0	46	16
21	26	M	residual	5	0	34	12
22	28	F	residual	7	0	31	16

Table 2 Mean amplitudes (μV) and latencies (msec) of the N100 and P200 components and mean LPC (μV) derived from the Cz region.

			Control	Task	
				Attend	Non-attend
N100	Amplitude	Normal	3.61±1.66	4.07±1.88	3.53±1.40
		Schiz.	3.39±1.91	3.52±1.94	3.27±1.97
N100	Latency	Normal	94.7±11.4	97.4±10.2	97.2±11.3
		Schiz.	94.5±10.5	96.0±11.6	96.3±11.3
P200	Amplitude	Normal	4.00±2.09	3.05±1.53	4.17±1.68
		Schiz.	4.09±1.63	3.33±1.57	3.77±1.80
P200	Latency	Normal	190.6±14.4	180.3±14.5	184.9±11.3
		Schiz.	173.8±15.2	175.8±14.0	175.9±16.1
LPC		Normal	-0.45±0.55	0.12±0.92	-0.65±0.70
		Schiz.	-0.49±0.74	-0.42±0.68	-0.50±0.58

Table 3 Mean amplitudes (μV) and latencies (msec) of the N100 and P200 components and mean LPC (μV) derived from the T3 and T4 regions elicited by left ear and right ear stimuli.

			Control Condition		Task Condition	
			Left ear	Right ear	Left ear	Right ear
T3	N100 Amplitude	Normal	1.85±0.98	1.92±0.97	1.67±0.94	1.99±1.05
		Schiz.	1.39±1.17	1.73±0.97	1.55±0.91	1.50±1.07
	N100 Latency	Normal	90.2±15.7	95.8±26.8	94.2±19.1	101.7±21.0
		Schiz.	86.6±19.2	89.5±22.6	92.0±17.0	96.3±26.2
	P200 Amplitude	Normal	1.82±1.54	1.23±1.22	1.38±1.25	1.36±1.20
		Schiz.	1.73±0.66	1.29±1.02	1.30±1.07	1.18±0.84
	P200 Latency	Normal	185.8±23.6	188.2±31.2	176.4±23.6	193.1±25.4
		Schiz.	173.8±24.5	177.1±22.5	175.4±23.3	184.5±25.9
	LPC	Normal	0.07±0.43	0.01±0.48	2.10±1.07	1.78±0.96
		Schiz.	0.06±0.38	0.03±0.56	1.38±0.94	1.29±0.96
T4	N100 Amplitude	Normal	2.56±0.99	1.41±0.88	2.10±1.07	1.78±0.96
		Schiz.	1.47±0.86	1.23±0.66	1.38±0.94	1.29±0.96
	N100 Latency	Normal	109.2±20.4	104.2±15.7	111.3±17.0	105.3±18.4
		Schiz.	109.3±21.3	98.9±18.6	108.0±19.8	102.5±19.0
	P200 Amplitude	Normal	1.96±1.64	2.13±1.17	2.12±1.29	1.67±1.15
		Schiz.	1.40±0.89	1.56±0.89	1.40±1.04	1.15±1.11
	P200 Latency	Normal	199.6±29.1	196.6±22.9	197.4±27.6	189.3±23.1
		Schiz.	191.8±182.	180.7±24.9	190.1±23.1	185.0±22.0
	LPC	Normal	0.03±0.56	0.34±0.46	0.40±0.48	0.04±0.47
		Schiz.	-0.10±0.43	-0.08±0.37	0.11±0.36	-0.13±0.43

Table 4 Mean LPC (μV) derived from the Cz, T3 and T4 regions in the task condition.

			Left ear	Right ear
Cz	Attend Target	Normal Schiz.	0.51±0.94	0.29±1.01
			-0.34±0.65	-0.41±0.92
	Attend Non-Target	Normal Schiz.	-0.21±0.86	-0.10±0.72
			-0.54±0.57	-0.38±0.57
	Non-Attend Target	Normal Schiz.	-0.69±0.91	-0.77±0.78
			-0.56±0.54	-0.29±0.65
	Non-Attend Non-Target	Normal Schiz.	-0.59±0.46	-0.55±0.60
			-0.61±0.62	-0.55±0.48
T3	Attend Target	Normal Schiz.	0.59±0.53	1.11±0.92
			0.25±0.65	0.38±0.77
	Attend Non-Target	Normal Schiz.	0.20±0.63	0.51±0.55
			0.08±0.37	0.27±0.40
	Non-Attend Target	Normal Schiz.	-0.26±0.45	0.12±0.61
			-0.05±0.42	0.18±0.37
	Non-Attend Non-Target	Normal Schiz.	-0.19±0.30	0.17±0.44
			-0.14±0.33	0.15±0.26
T4	Attend Target	Normal Schiz.	0.65±0.57	0.23±0.61
			0.22±0.54	-0.16±0.49
	Attend Non-Target	Normal Schiz.	0.40±0.48	0.15±0.46
			0.07±0.25	0.02±0.21
	Non-Attend Target	Normal Schiz.	0.32±0.43	-0.10±0.41
			0.08±0.34	-0.18±0.57
	Non-Attend Non-Target	Normal Schiz.	0.24±0.33	-0.12±0.26
			0.05±0.23	-0.20±0.34

15.00, $p<0.01$). That is, the latency of T3-N100 was shorter than that of T4-N100 in both groups.

III-3 ERPs in the task condition

III-3-1 The amplitudes of N100

Fig. 2 illustrates the mean amplitudes of Cz-N100 in the normal controls as well as the schizophrenics. Judging from the results of the ANOVA, the main effect of DIAG on the amplitudes of Cz-N100 was significant ($F(1,319)=4.12$, $p<0.05$). That is, the amplitudes of Cz-N100 in the normal controls were larger than those of the schizophrenics. In the normal controls, the main effect of CHANNEL was significant ($F(1,151)=4.59$, $p<0.05$); however, in the schizophrenics, this CHANNEL effect was not significant ($F(1,167)=0.72$, n.s.). Thus, only in the normal controls, the amplitudes of Cz-N100 elicited by the stimuli presented to the attended ear were larger than those for the the non-attended ear.

As in the case of Cz-N100, the effect of DIAG on the amplitudes of T3-N100 and T4-N100 was significant ($F(1,639)= 35.56$, $p<0.01$). That is, the amplitudes of T3-N100 and T4-N100 in the normal controls were larger than those of the schizophrenics. Furthermore, in the normal controls, the interactive effect of REGION X EAR on the amplitudes of T3-N100 and T4-N100 was significant ($F(1,303)=8.78$, $p<0.01$). This interaction indicates that the amplitudes of T3-N100 and T4-N100 were larger when the stimuli were presented to the ear contralateral to the EEG-deriving site than when the stimuli were presented to the ipsilateral ear (Fig. 3). However, in the schizophrenics, this interaction was not significant. Instead, the main effect of REGION tended to be significant in the schizophrenics ($F(1,335)=3.47$, $p<0.1$). That is, T4 produced smaller amplitudes of the N100 component than did T3.

III-3-2 The latencies of N100

The ANOVA revealed that no factors or interactions had a significant effect on the latencies of Cz-N100 in the schizophrenics or in the normal controls. The main effect of DIAG on the latencies of T3-N100 and T4-N100 was significant ($F(1,639)=4.93$, $p<0.05$). That is, the latencies of T3-N100 and T4-N100 in the schizophrenics were shorter than those of the normal controls. The main effect of REGION was significant in both groups (normals: $F(1,303)=23.90$, $p<0.01$; schizophrenics: $F(1,335)=26.74$, $p<0.01$). That is, the latencies of T3-N100 were shorter than those of T4-N100. Furthermore, the interaction of REGION X EAR was also significant in both groups (normals: $F(1,303)=10.14$, $p<0.01$; schizophrenics: $F(1,335)=5.26$, $p<0.05$). This interaction indicates that the latencies of T3-N100 and T4-N100 were shorter when the stimuli were presented to the ear ipsilateral to the EEG-deriving site than when presented to the contralateral ear for both groups of subjects (Fig. 4).

III-3-3 The amplitudes of P200

The effect of DIAG on the amplitudes of Cz-P200 was not significant ($F(1,319)=0.10$, n.s.). However, a CHANNEL effect was observed in both normal controls and schizophrenics ($F(1,151)=19.93$, $p<0.01$; $F(1,167)=2.91$, $p<0.1$, respectively). Contrary to the results for Cz-N100, the amplitudes of Cz-P200 elicited by the stimuli presented to the non-attended ear were larger than those of the attended ear for both groups (Fig. 5). This CHANNEL effect was less prominent in the schizophrenics than in the normal controls (DIAG X CHANNEL: $F(1,319)=3.53$, $p<0.1$).

The effect of DIAG on the amplitudes of T3-P200 and T4-P200 was significant ($F(1,639)=19.27$, $p<0.01$). That is, the amplitudes of T3-P200 and T4-P200 in the normal controls were larger than those of the schizophrenics. The effect of REGION X EAR on the amplitudes of T3-P200 and T4-P200 was not significant in the normal controls or in the schizophrenics. In the normal controls, the effect of REGION was significant on the amplitudes of T3-P200 and T4-P200 ($F(1,303)=15.87$, $p<0.01$). That is, the amplitudes of T4-P200 were always larger than those of T3-P200 irrespective of the sidedness of the stimulus presentation (left or right ear). However, in the schizophrenics, this REGION effect was not observed, and the amplitudes of T4-P200 were nearly equal to those of T3-P200 (Fig. 6).

III-3-4 The latencies of P200

The ANOVA revealed that the effect of DIAG on the latencies of Cz-P200 was significant ($F(1,319)=19.52$, $p<0.01$). That is, the latencies of Cz-P200 in the schizophrenics were shorter than those of the normal controls. In the normal controls, the effect of CHANNEL was significant ($F1,151)= 4.95$, $p<0.05$). That is, the latencies of Cz-P200 elicited by the stimuli presented to the attended ear were shorter than those of the non-attended ear. However, in the schizophrenics, this CHANNEL effect was not observed (Fig. 7).

As in the case of Cz-P200, the effect of DIAG on the latencies of T3-P200 and T4-P200 was significant ($F(1,639)=7.83$, $p<0.01$). That is, the latencies of T3-P200 and T4-P200 in the schizophrenics were shorter than those of the normal controls. The effect of REGION was significant in both groups (normals: $F(1,303)=9.25$, $p<0.01$; schizophrenics: $F(1,335)=9.34$, $p<0.01$). That is, the latencies of T3-P200 were shorter than those of T4-P200. Furthermore, the interaction of REGION X EAR was also significant in both groups (normal controls: $F(1,303)=19.06$, $p<0.01$; schizophrenics: $F(1.335)=8.15$, $p<0.01$). This interaction indicates that the latencies of T3-P200 and T4-P200 were shorter when the stimuli were presented to the ear ipsilateral to the EEG-deriving site than when the stimuli were presented to the contralateral ear for both groups (Fig. 8).

III-3-5 The averaged amplitudes of the late positive components (LPCs)

The ANOVA revealed that the effect of DIAG on the Cz-LPCs was significant ($F(1,319)=6.38$, $p<0.05$). That is, the Cz-LPCs in the normal controls were larger than those in the schizophrenics. In the normal controls, the effect of CHANNEL on the Cz-LPCs was significant ($F(1,151)=37.11$, $p<0.01$). That is, the Cz-LPCs elicited by the stimuli presented to the attended ear were larger than those of the non-attended ear. In the normal controls, the effect of CHANNEL X SYLLABLE on the Cz-LPCs was significant ($F(1,151)=7.85$, $p<0.01$). Furthermore, in the normal controls the effect of SYLLABLE on the Cz-LPCs elicited by the stimuli presented to the attended ear was significant ($F(1,75)=7.66$, $p<0.01$). However, this SYLLABLE effect was not observed for the non-attended ear. These results indicate that the target syllables in the attended ear (that is, the target stimuli) produced the largest Cz-LPC values in the normal controls. However, in the schizophrenics, neither a CHANNEL effect nor an interaction for CHANNEL X SYLLABLE was observed (Fig. 9).

Similar results were obtained through the ANOVA employing EI as a covariate for the Cz-LPCs elicited by the stimuli presented to the attended ear (DIAG X TARGET: $F(1,158)= 3.916$, $p<0.05$).

The effect of DIAG on the T3-LPCs and T4-LPCs was also significant ($F(1,639)=25.49$, $p<0.01$). That is, the T3-LPCs and T4-LPCs in the normal controls were larger than those of the schizophrenics. The effect of CHANNEL on the T3-LPCs and T4-LPCs was significant in both groups (normals: $F(1,303)=61.18$, $p<0.01$; schizophrenics: $F(1,335)=11.19$, $p<0.01$). That is, the T3-LPCs and T4-LPCs elicited by the stimuli presented to the attended ear were larger than those of the non-attended ear. This CHANNEL effect was less prominent in the schizophrenics than in the normal controls (DIAG X CHANNEL: $F(1,639)=16.44$, $p<0.01$). In the normal controls, the interaction of CHANNEL X SYLLABLE was significant ($F(1,303)= 8.37$, $p<0.01$). As in the case of the Cz-LPCs, the effect of SYLLABLE on the T3-LPCs and T4-LPCs elicited by the stimuli presented to the attended ear was significant ($F(1,151)= 11.76$, $p<0.01$). However, this SYLLABLE effect was not observed for the non-attended ear in the normal controls. These results indicate that the T3-LPCs and T4-LPCs elicited by the target stimuli were larger than those elicited by the non-target stimuli. On the other hand, in the schizophrenics an interaction for CHANNEL X SYLLABLE was not observed. Furthermore, the T3-ANOVA and T4-ANOVA revealed that the effect of SYLLABLE on the LPCs elicited by the stimuli presented to the attended ear was sig-nificant for the T3-LPCs, but not for the T4-LPCs, in the normal controls. In the schizophrenics, the effect of REGION on the T3-LPCs and T4-LPCs was significant ($F(1,335)=10.54$, $p<0.01$). That is, the T3-LPCs were larger than the T4-LPCs in the schizophrenics. However, this REGION effect was not observed in the normal controls. On the other hand, when the LPCs elicited by the target stimuli alone were analyzed, the effect of REGION was significant in the normal controls as well as in the schizophrenics (normals: $F(1,75)=7.13$, $p<0.01$; schizophrenics: $F(1,79)=4.71$, $p<0.05$). That is, the target stimuli produced larger LPCs at the

T3 region as compared to the T4 region in both groups (Fig. 10).

The interactive effect of REGION X EAR on the T3-LPCs and T4-LPCs was significant in both groups (normals: $F(1,305)=41,81$, $p<0.01$; schizophrenics: $F(1,335)=23.48$, $p<0.01$). This interaction indicates that both T3-LPCs and T4-LPCs were larger when the stimuli were presented to the ear contralateral to the EEG-deriving site than when the stimuli were presented to the ipsilateral ear for both groups (Fig. 11).

III-3-6 Summary of the results in the task condition

1) The amplitudes of Cz-N100, T3-N100 and T4-N100 in the schizophrenics were smaller than those of the normal controls. In the normal controls, the amplitudes of Cz-N100 elicited by the stimuli presented to the attended ear were larger than those of the non-attended ear. However, this CHANNEL effect was not observed in the schizophrenics. In the normal controls, both the amplitudes of T3-N100 and T4-N100 were larger when the stimuli were presented to the ear contralateral to the EEG-deriving site than when the stimuli were presented to the ipsilateral ear. However, in the schizophrenics, this crossover effect was not observed; instead, the amplitudes of T3-N100 were larger than those of T4-N100.

2) The latencies of T3-N100 and T4-N100 in the schizophrenics were shorter than those in the normal controls. The latencies of T3-N100 were shorter than those of T4-N100 in both groups. The latencies of T3-N100 and T4-N100 were shorter when the stimuli were presented to the ear ipsilateral to the EEG-deriving site than when the stimuli were presented to the contralateral ear for both groups.

3) The amplitudes of Cz-P200 elicited by the stimuli presented to the non-attended ear were larger than those of the attended ear for both groups. This CHANNEL effect was less prominent in the schizophrenics than in the normal controls. The amplitudes of T3-P200 and T4-P200 in the schizophrenics were smaller than those in the normal controls. In the normal controls, the amplitudes of T4-P200 were always larger than those of T3-P200; however, this REGION effect was not observed in the schizophrenics.

4) The latencies of Cz-P200, T3-P200 and T4-P200 were shorter in the schizophrenics than in the normal controls. In the normal controls, the latencies of Cz-P200 elicited by the stimuli presented to the attended ear were shorter than those of the non-attended ear; however, in the schizophrenics, this CHANNEL effect was not observed. The latencies of T3-P200 were shorter than those of T4-P200 in both groups. The latencies of T3-P200 and T4-P200 were shorter when the stimuli were presented to the ear ipsilateral to the EEG-deriving site than when the stimuli were presented to the contralateral ear in both groups.

5) The Cz-LPCs, T3-LPCs and T4-LPCs in the schizophrenics

were smaller than those of the normal controls. In the normal controls, the Cz-LPCs, T3-LPCs and T4-LPCs elicited by the stimuli presented to the attended ear were larger than those of the non-attended ear. However, in the schizophrenics, this CHANNEL effect was not observed for the Cz-LPCs. The CHANNEL effect was observed for the T3-LPCs and T4-LPCs in the schizophrenics as well, however, this effect was less prominent in the schizophrenics compared to the normal controls. The Cz-LPCs and T3-LPCs elicited by the target stimuli were larger than those elicited by the non-target stimuli in the normal controls. However, in the schizophrenics, this result was not observed. In the normal controls, the T3-LPCs were larger than the T4-LPCs for the target stimuli alone. On the other hand, in the schizophrenics, the T3-LPCs were always larger than the T4-LPCs. The T3-LPCs and T4-LPCs were larger when the stimuli were presented to the ear contralateral to the EEG-deriving site than when the stimuli were presented to the ipsilateral ear for both groups.

III-4 The influence of neuroleptics on ERPs

III-4-1 Comparison of the unmedicated group and the medicated group of schizophrenics

The ANOVA revealed that in the schizophrenics the effect of MED on the latencies of T3-N100 and T4-N100 was significant in the control condition, as well as the task condition ($F(1,79)=15.00$, $p<0.01$; $F(1,335)=22.24$, $p<0.01$, respectively). The interactive effect of MED X REGION on the latencies of T3-N100 and T4-N100 was also significant in both the control and the task conditions ($F(1,79)=3.89$, $p<0.05$; $F(1,335)=7.13$, $p<0.01$, respectively). As shown in Fig. 12, the latencies of T3-N100 in the unmedicated group were shorter than those of the medicated group, while the latencies of T4-N100 were nearly equal in both groups.

As in the case of N100, the effect of MED on the latencies of T3-P200 and T4-P200 was significant in the control and the task conditions ($F(1,79)=7.98$, $p<0.01$; $F(1,335)=6.45$, $p<0.05$, respectively). The interactive effect of MED X REGION on the latencies of T3-P200 and T4-P200 was also significant in both the control and the task conditions ($F(1,79)=4.72$, $p<0.05$; $F(1,335)=9.67$, $p<0.01$, respectively). As shown in Fig.13, the latencies of T3-P200 in the unmedicated group were shorter than those of the medicated group, while the latencies of T4-P200 were nearly equal in both groups. In addition, the latencies of N100, as well as the P200 components derived from the T3 and T4 regions in the medicated group, were found to be nearly equal to those of the corresponding components in the normal controls.

The effect of MED on the latencies of Cz-P200 in the task condition was significant ($F(1,167)=4.85$, $p<0.05$). That is, the latencies of Cz-P200 in the unmedicaed group were shorter than those in the medicated group.

Furthermore, in the task condition the interaction of MED X

CHANNEL X SYLLABLE on the T3-LPCs and T4-LPCs was significant ($F(1,335)=5.49$, $p<0.05$). The effect of MED and the interactive effect of MED X SYLLABLE on the T3-LPCs and T4-LPCs elicited by the stimuli presented to the attended ear were significant ($F(1,159)=4.84$, 4.21, $p<0.05$, respectively). As can be seen in Fig. 14, the T3-LPCs and T4-LPCs for the target stimuli in the unmedicated group were larger than those of the medicated group but were smaller than those of the normal controls.

III-4-2 The relationship between ERPs and neuroleptic dosages in the medicated group

To determine what, if any, effect neuroleptics have on ERPs, Pearson's correlations between the ERPs and the neuroleptic dosages (i.e. chlorpromazine equivalent dosages) were calculated. In the control condition, the amplitudes of T4-N100 and Cz-P200 correlated positively, and the latencies of Cz-N100 correlated inversely with the neuroleptic dosages ($r=0.48$, 0.45, -0.35, $p<0.05$, respectively). In the task condition, the amplitudes of T4-N100 and T4-P200 elicited by the target stimuli correlated positively, and the Cz-LPCs and the T4-LPCs elicited by the target stimuli correlated inversely with the neuroleptic dosages ($r=0.37$, 0.36, -0.68, -0.36, $p<0.05$, respectively).

III-5 The relationship between ERPs and psychotic symptoms

In order to determine if psychotic symptoms have any effect on ERPs, Pearson's correlations between the ERPs and the total BPRS scores were calculated in the unmedicated group. In the control condition, the latencies of T3-N100 and T3-P200, and the amplitudes of T4-P200, the Cz-LPCs, and T4-LPCs correlated inversely with the total BPRS scores in the unmedicated group ($r=-0.58$, -0.50, -0.45, -0.60, -0.48, $p<0.05$, respectively). In the task condition, the amplitudes of Cz-N100, and the latencies of T3-N100 and T4-P200, as well as the Cz-LPCs, T3-LPCs and T4-LPCs elicited by the target stimuli, correlated inversely with the total BPRS scores in the unmedicated group ($r=-0.44$, -0.55, -0.46, -0.40, -0.47, -0.49, $p<0.05$, respectively).

III-6 The relationship between ERPs and performance levels

The performance levels were represented in terms of EIs. The mean EI was 5.3% (left ear stimuli: 4.0%; right ear stimuli: 6.6%) in the normal controls, with the mean EI of the schizophrenics being 25.4% (left ear stimuli: 24.3%; right ear stimuli: 26.5%).

Pearson's correlations between the ERPs and the EIs were calculated, and it was found that in the normal controls, the Cz-LPCs and T4-LPCs elicited by the target stimuli presented to the left ear, as well as the T3-LPCs elicited by the target stimuli presented to the right ear, correlated inversely with the EIs ($r=-0.41$, -0.44, -0.59, $p<0.05$, respectively). It was also found that in the schizophrenics, the amplitudes of Cz-N100, the latencies of Cz-N100, and the T4-LPCs elicited by the target

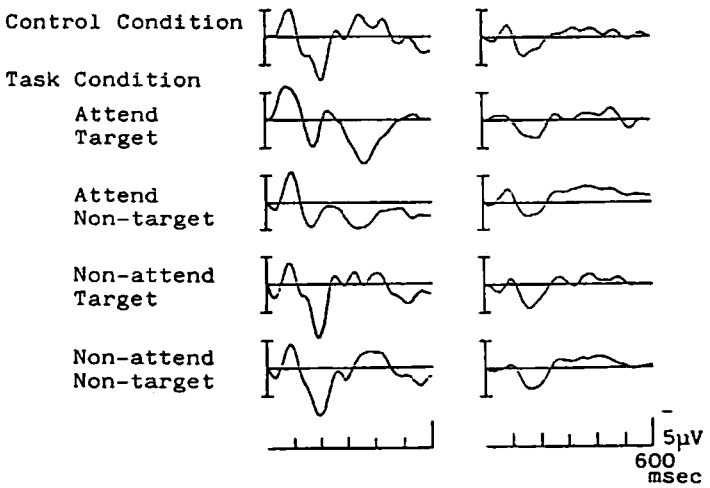

Fig. 1. Representative ERP waveforms derived from the Cz region. Left: normal control subject (30 years old, male). Right: unmedicated schizophrenic patient (24 years old, male). "Attend Target": target stimuli presented to the attended ear; "Attend Non-target": non-target stimuli presented to the attended ear; "Non-attend Target": the same syllables as the target stimuli presented to the non-attended ear; "Non-attend Non-target": syllables different from the target stimuli presented to the non-attended ear.

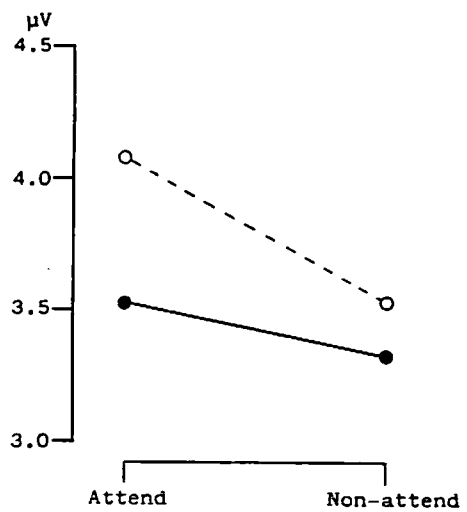

Fig. 2. Mean amplitudes of the N100 component at the Cz region. "Attend" and "Non-attend" designate conditions when the stimuli were presented to the attended ear and non-attended ear, respectively. O- - -O, Normals (n=20x4); ●——● , Schizophrenics (n=22x4).

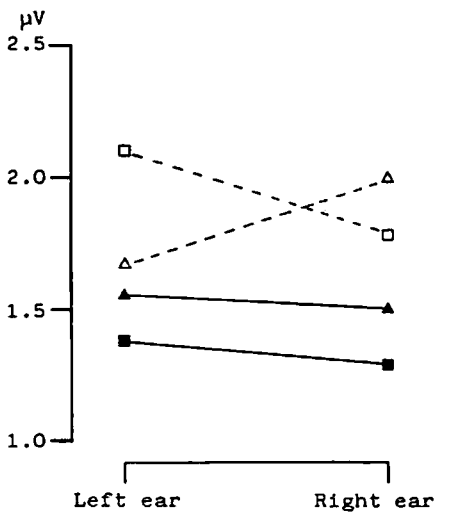

Fig. 3. Mean amplitudes of the N100 component at the T3 and T4 regions. "Left ear" and "Right ear" designate conditions when the stimuli were presented to the left ear and right ear, respectively. Normals (n=20x4): △- - -△,T3, □- - -□,T4; Schizophrenics (n=22x4): ▲——▲,T3, ■——■,T4.

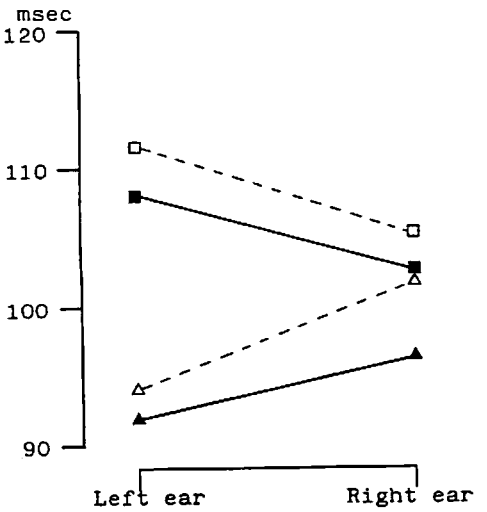

Fig. 4. Mean latencies of the N100 component at the T3 and T4 regions. Normals (n=20x4): △- - -△,T3, □- - -□, T4; Schizophrenics (n=22x4): ▲——▲, T3, ■——■,T4.

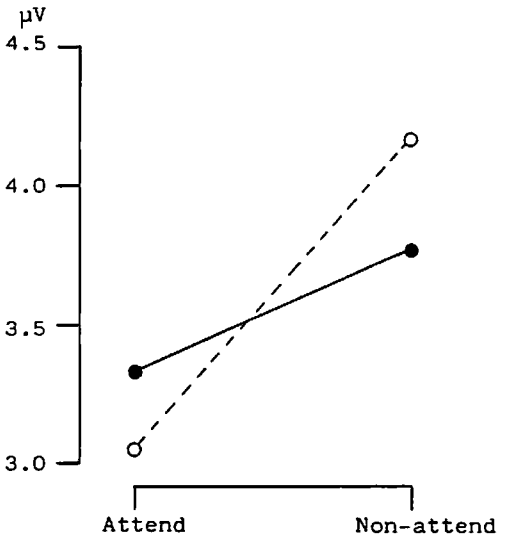

Fig. 5. Mean amplitudes of the P200 component at the Cz region. O- - -O, Normals (n=20x4); ●——●, Schizophrenics (n=22x4).

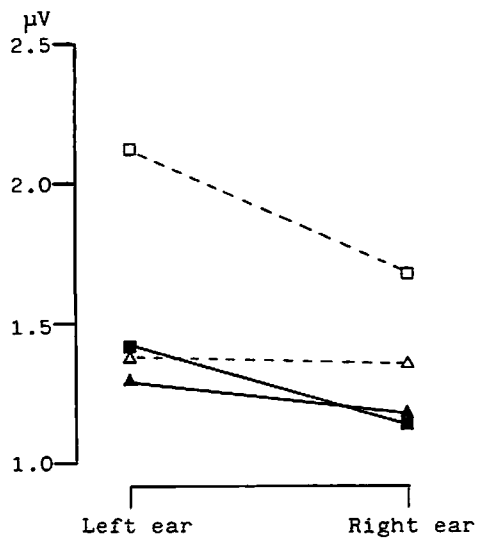

Fig. 6. Mean amplitudes of the P200 component at the T3 and T4 regions. Normals (n=20x4): △- - -△,T3, □- - -□, T4; Schizophrenics (n=22x4): ▲——▲, T3, ■——■,T4.

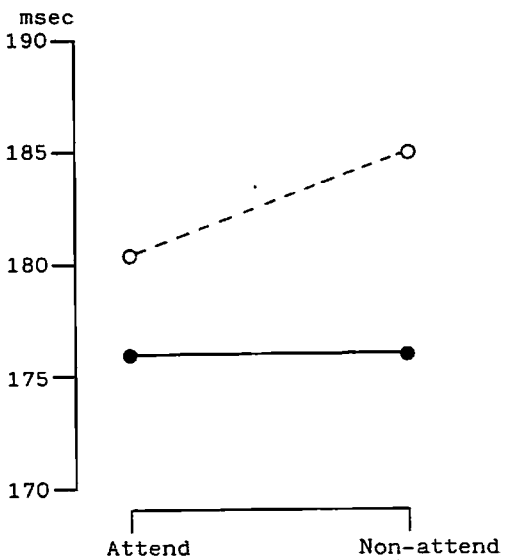

Fig. 7. Mean latencies of the P200 component at the Cz region. O- - -O, Normals (n=20x4); ●——●, Schizophrenics (n=22x4).

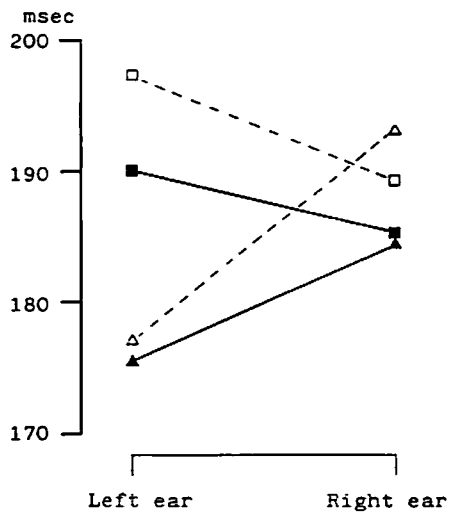

Fig. 8. Mean latencies of the P200 component at the T3 and T4 regions. Normals (n=20x4): Δ----Δ,T3, □----□, T4; Schizophrenics (n=22x4): ▲——▲, T3, ■——■,T4.

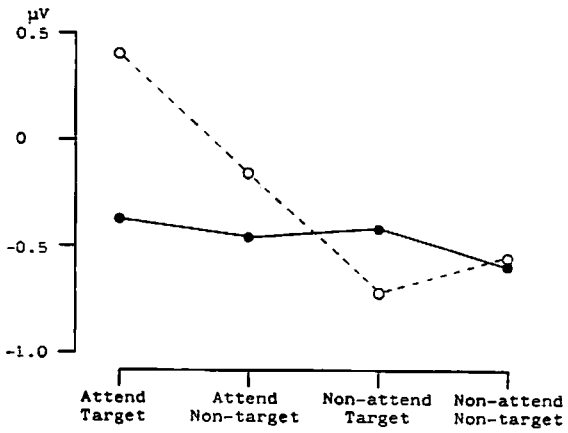

Fig. 9. Mean LPCs at the Cz region. "Attend Target": target stimuli in the attended ear; "Attend Non-target": non-target stimuli in the attended ear; "Non-attend Target": the same syllables as the target stimuli in the non-attended ear; "Non-attend Non-target":syllables different from the target stimuli in the non-attended ear. O----O,Normals (n=20x2); ●——●,Schizophrenics (n=22x2).

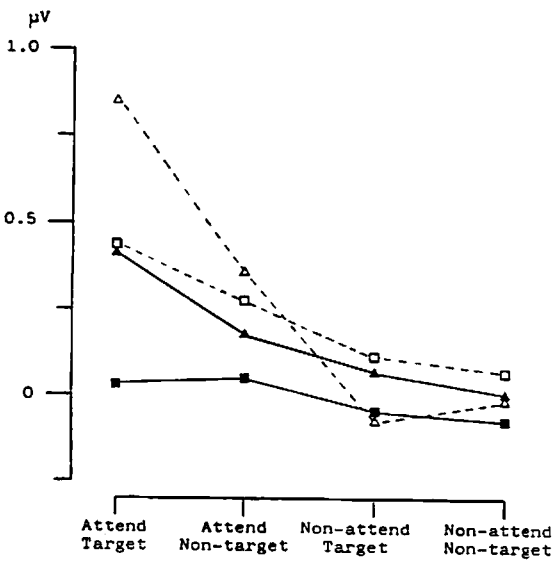

Fig. 10. Mean LPCs at the T3 and T4 regions. Normals(n=20x2): Δ----Δ,T3, □----□,T4; Schizophrenics (n=22x2): ▲——▲,T3, ■——■,T4.

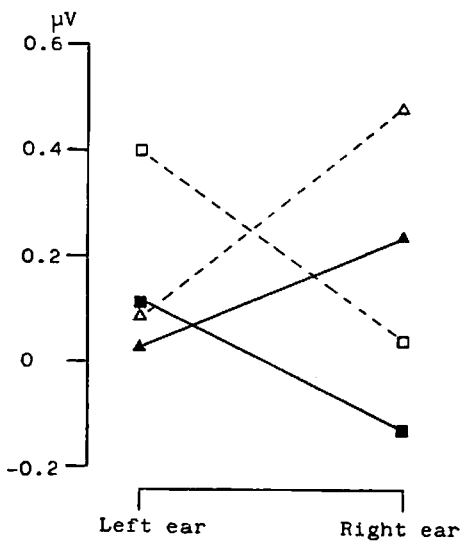

Fig. 11. Mean LPCs at the T3 and T4 regions elicited by the stimuli presented to the left and right ear. Normals (n=20x4): Δ----Δ,T3, □----□, T4; Schizophrenics (n=22x4): ▲——▲, T3, ■——■,T4.

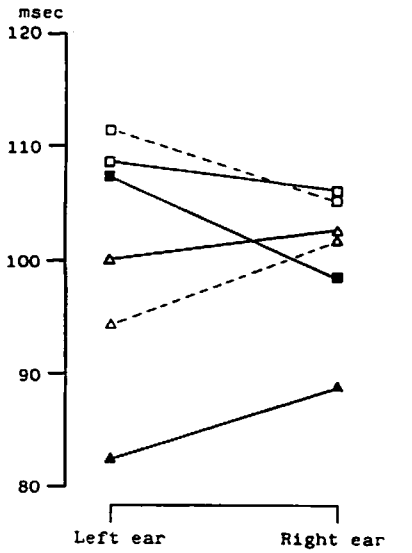

Fig. 12. Mean latencies of the N100 component at the T3 and T4 regions. Normals(n=20x4): Δ---Δ,T3,□----□,T4; Medicated schizophrenics (n=12x4): Δ——Δ ,T3, □——□,T4; Unmedicated schizophrenics (n=10x4): ▲——▲,T3, ■——■ ,T4.

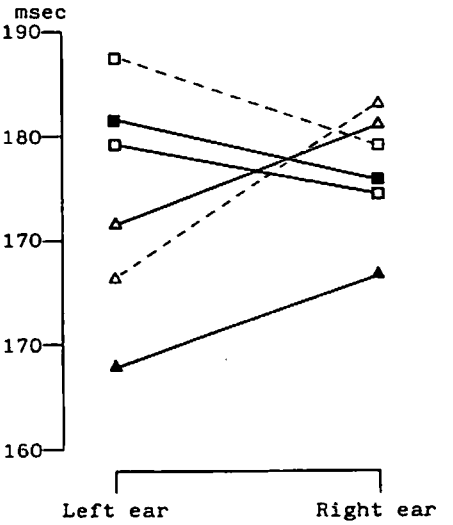

Fig. 13. Mean latencies of the P200 component at the T3 and T4 regions. Normals(n=20x4):Δ- - -Δ,T3,□- - -□,T4; Medicated schizophrenics (n=12x4): Δ——Δ ,T3, □——□,T4; Unmedicated schizophrenics (n=10x4): ▲——▲,T3, ■——■ ,T4.

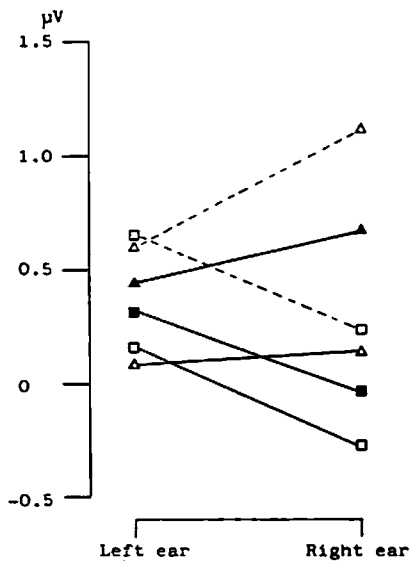

Fig. 14. Mean LPCs at the T3 and T4 regions elicited by the target stimuli. Normals (n=20x1): Δ----Δ, T3,□----□,T4; Medicated schizophrenics (n=12x1): Δ——Δ,T3, □——□,T4; Unmedicated schizophrenics (n=10x1): ▲——▲,T3, ■——■,T4.

stimuli presented to the left ear, as well as the latencies of Cz-N100, T3-N100 and T4-N100 elicited by the target stimuli presented to the right ear, correlated inversely with the EIs ($r=-0.36$, -0.48, -0.41, -0.70, -0.37, -0.41, $p<0.05$, respectively).

III-7 Interaction among total BPRS scores, neuroleptic dosages and EIs

The total BPRS scores correlated positively with the EIs in the unmedicated group ($r=0.61$, $p<0.05$). In the medicated group, the total BPRS scores correlated inversely with the neuroleptic dosages ($r=-0.52$, $p<0.05$). No significant correlations were seen between the total BPRS scores and the EIs or between the neuroleptic dosages and the EIs in the medicated group.

IV Discussion

IV-1 ERPs and selective attentional functioning in normal controls

The N100 amplitude was enhanced for stimuli in the attended ear, while the P200 amplitude was reduced for the same stimuli in the normal controls. It has been reported by other investigators that an endogenous prolonged negative component with a peak latency of 200 msec is observed when the ERPs elicited by stimuli in an unattended channel are subtracted from those elicited by stimuli in an attended channel (Naatanen, Gaillard and Mantysalo, 1978[33]; Hansen and Hillyard, 1980[34]). It can be speculated that this endogenous negative component overlapped with the exogenous N100 and P200 components and produced an increase in the N100 amplitude, along with a decrease in the P200 amplitude, for the attended ear in this study.

According to some investigators, P300 component reflects stimulus evaluation and target detection, i.e., decision making (Hillyard, Picton and Regan, 1978[35]; Donchin, 1979[7]). Fitzgerald and Picton (1983)[36] have reported that P300 components reflect the final stage in stimulus discrimination, which consists of higher order control processes. On the other hand, it has also been felt by some investigators that the P300 component reflects post decision 'closure' mechanisms (Desmedt, 1980[37]).

In the present study, the LPCs, which represented the P300 amplitudes, were enhanced for target stimuli in the attended ear and correlated positively with the performance levels. Based on these results it can be concluded that the LPC reflects the stimulus evaluation and target detection process. Thus, it can be said that potentials including the N100 and P200 components reflect channel selective attention ('stimulus set' as defined by Broadbent), and that late positive components reflect stimulus evaluation and response selection ('response set' as defined by Broadbent).

IV-2 ERPs and selective attentional functioning in schizophrenics

The mean EI, which was utilized as an index of performance levels, was 25.4% in the schizophrenics and 5.3% in the normal controls. These figures seem to indicate that the schizophrenics were rather motivated and concentrated fairly well on the required tasks, since the mean EI of the schizophrenics was far smaller than the EI value (300%) we would have expected if they had counted the number of target stimuli in a random manner.

The schizophrenics demonstrated smaller amplitudes for the N100 components and failed to demonstrate a channel effect for the N100 component. On the other hand, they displayed a channel effect for the P200 component, but less prominently compared to the normals. Assuming that the channel effect on the N100 and P200 components is reflected in a prolonged negative shift as described in IV-1, it can be speculated that the negative shift overlaps the P200 component, but not the N100 component, in the case of schizophrenics. In other words, the negative shift in schizophrenics can be assumed to appear after some delay with a low amplitude. Therefore, it can be speculated that attentional functioning for channel selection in schizophrenics is less effective and delayed as compared to normal controls. This delay may be relevant to previous reports that information processing in schizophrenics is slow (Yates, 1966[38]; Saccuzzo and Braff, 1981[39]).

The schizophrenics demonstrated smaller amplitudes for the LPCs and, moreover, failed to display enhanced LPCs to the target stimuli. These results indicate that schizophrenics have a disturbance in a certain stage of information processsing that is reflected in LPCs, that is, target detection or stimulus evaluation. It can be speculated that this disturbance is, at least in part, responsible for the low performance levels in the schizophrenics.

Next, the relationship among ERPs,psychotic symptoms and neuroleptic dosages will be discussed. The amplitudes of Cz-N100 elicited by the target stimuli correlated inversely with the total BPRS scores in the unmedicated group. In the medicated group, the amplitudes of T4-N100 elicited by the target stimuli correlated positively with the drug dosages, while the total BPRS scores correlated inversely with the drug dosages. These results indicate that, as clinical symptoms improve, the amplitudes of N100 components increase. The latencies of T3-N100 correlated inversely with the total BPRS scores in the unmedicated group. The latencies of T3-N100 and T3-P200 in the unmedicated group were shorter than those in the medicated group and shorter than those in the normal controls. Eleven out of the 12 medicated patients were schizophrenics of the residual type, while 7 out of the 10 unmedicated patients were in the acute phase of schizophrenia. The mean scores for the three items of the BPRS, that is, guilt feelings, suspiciousness, and hallucinatory behavior, were significantly higher in the unmedicated group than in the medicated group. Therefore, it can be speculated that schizo-

phrenics demonstrate shorter ERP latencies in the T3 region during the active phase. In addition, it is felt that neuroleptics normalize these shortened latencies. A lateralized effect for neuroleptics has been noted by other authors, too (Mysolobodsky and Weiner, 1976[40]; Waziri, 1980[41]; Mintz, Tower and Mislobodsky, 1982[42]). Mintz et al. have suggested that treatment utilizing neuroleptics restores hemispheric balance in schizophrenic patients by selectively suppressing the hyperactive left hemisphere.

Saletu et al. (1971)[11] have reported that schizophrenics exhibit shorter latencies than normal controls and that schizophrenics with a thought process disorder (TPD) show shorter latencies than do schizophrenics without a TPD. They have also reported that EEG analysis reveals that schizophrenics with a TPD have a greater amount of low voltage fast activity than schizophrenics without a TPD, and they suggest that schizophrenics with a TPD exhibit more hyperarousal than schizophrenics without a TPD. Roth, Krainz, Ford, Tipklenberg, Rothband and Kopell (1976)[43] and Roth et al. (1981)[15] have reported that P200 latency is shorter for schizophrenics than for normal controls, and that hallucinators exhibit shorter N100 and P200 latencies than non-hallucinators. Furthermore, Roth et al. (1976)[43] have reported that in normals, P200 latencies are shorter when subjects are engrossed in a book than when they must pay attention to auditory stimuli. Therefore, the shorter latencies for N100 and P200 exhibited by the unmedicated group in this study can be considered to reflect a disturbance in attentional focusing, that is, 'stimulus set'. According to Saletu, the shorter latencies of N100 and P200 in unmedicated subjects reflect a hyperarousal state. As reported by Broen and Storms (1966)[4], the response hierarchy is more severely disturbed during the hyperarousal state in schizophrenics, resulting in a lower performance level. In this study, the mean EI of the unmedicated group was 31.6%, that of the medicated group being 20.2%. Based on the findings described above, it can be postulated that the greater hyperarousal state of the unmedicated schizophrenics, in comparison to the medicated schizophrenics, interferes more markedly or widely with 'stimulus set', and this interference combined with a disturbance in 'response set' results in poorer performance levels. The T3-LPCs and T4-LPCs for the target stimuli in the medicated group were smaller than those in the unmedicated group. In the medicated group, the Cz-LPCs and T4-LPCs correlated inversely with the drug dosages, while drug dosages correlated inversely with the total BPRS scores. These results indicate that neuroleptics may decrease the amplitude of LPCs. Two hypotheses are possible in explaining the reduced amplitudes of LPCs in the medicated group: 1) Neuroleptics decreased the amplitudes of LPCs that could have increased through an improvement in clinical symptoms due to medication. 2) Negative schizophrenic symptoms which are relatively resistant to medication compared to positive symptoms are responsible for producing the reduced amplitudes of LPCs.

There seem to be relatively few investigations in which ERP

measurements are employed during complex psychological tasks to examine schizophrenic deficits in information processing. Roth et al. (1980a[13], b[14], 1981[15]) have reported that schizophrenics demonstrate a disturbance in selective attention which is related to the stimulus selection process. Saitoh et al. (1983)[17] recorded ERPs in schizophrenics during dichotic detection tasks and reported that schizophrenics demonstrate disturbances in both 'stimulus set' and 'response set'.

Hemsley (1975)[44] concluded that schizophrenics demonstrate disturbances in both 'stimulus set' and 'response set', following Broadbent's model. Furthermore, based upon the results of a Card Sorting Test and dichotic listening tests, he reported that disturbances in 'stimulus set' were observed in depressive patients, as well as schizophrenics, and concluded that disturbances in 'response set' were more prominent in schizophrenics (Hemsley and Zawada, 1976[45]; Hemsley and Richardson, 1980[46]). Levit et al. (1973)[9] recorded visual and auditory evoked potentials in schizophrenics, psychotic depressives, and normal controls. They reported that the amplitudes of P300 were smaller in schizophrenics than in depressives and normals; while among these three groups there was no significant difference in N100 amplitudes. Their results seem to support Hemsley's hypothesis mentioned above.

At this point, we may question whether abnormalities of ERPs in schizophrenics reflect a state or a trait of schizophrenia. In the above discussion, it was suggested that the amplitudes of LPCs do not increase in patients who exhibit negative schizophrenic symptoms, even if their positive schizophrenic symptoms have been improved by neuroleptics. In fact, the amplitudes of LPCs did not increase with the improvement of clinical symptoms in the medicated group. Hence, it can be speculated that LPCs reflect a trait of schizophrenia, not a state. Friedman, Vaughan and Erlenmeyer-Kimling (1982)[47] recorded AEPs from children at high risk for schizophrenia and normal control children. They reported that the high risk subjects showed significantly less late positive amplitudes (P350 and P400) than the normal control subjects. They speculated that this amplitude reduction seen in children with a genetic risk for schizophrenia may be a premorbid indicator for the development of the psychosis. The results of this study add credence to the possibility that the P300 components of ERPs reflect a trait of schizophrenia.

IV-3 Selective attention and hemispheric function in normal controls

In the normal controls, both the amplitudes of T3-N100 and T4-N100 in the task condition were larger when the stimuli were presented to the ear contralateral to the EEG-deriving site as compared to stimuli presented to the ipsilateral ear. In the control condition, this effect was observed only in the T4 region. The latencies of T3-N100 and T4-N100, as well as of T3-P200 and T4-P200 were longer when the stimuli were presented to the ear contralateral to the EEG-deriving site as compared to

stimuli presented to the ipsilateral ear in the task condition. These results indicate that some mechanism works in syllable discrimination tasks such as in the dichotic listening tests reported by Kimura (1967)[48], with this mechanism activating the transmission of the sound presented to the contralateral ear and inhibiting the sound presented to the ipsilateral ear.

Since there was no significant difference between the amplitudes of T3-N100 and those of T4-N100, it can be suggested that the left hemispheric dominance for verbal tasks is not observed in the stage of information processing which is reflected in the N100 component. On the other hand, the T3-LPCs elicited by the target stimuli were larger than the T4-LPCs. Accordingly, it can be suggested that left hemispheric dominance is observed in the stage which is reflected in the P300 component.

IV-4 Selective attention and hemispheric dysfunction in schizophrenics

The schizophrenics failed to demonstrate an enhancement in the amplitude of the N100 component at the electrode sites contralateral to the ear of the stimulus presentation. This result indicates that schizophrenics have some disturbance in the integration mechanism of both hemispheres which functions under the condition of dichotic listening in normal controls. Disturbances in the integration of both hemispheres in schizophrenics have been reported by other authors (Beaumont and Dimond, 1973[23]; Green, 1978[25]; Carr, 1980[24]). Saitoh et al. (1983)[17] have also reported that schizophrenics display a disturbance in the integration of both hemispheres based upon their results for ERPs recorded during dichotic detection tasks.

The unmedicated group of schizophrenics in our study demonstrated shorter latencies for the N100 and P200 components compared to the normal controls exclusively in the T3 region. The medicated group of schizophrenics displayed latencies for the N100 and P200 components nearly equal to those of the normal controls even in the T3 region. These results seem to indicate that schizophrenics have some disturbance in the functioning of the left hemisphere. Flor-Henry (1969)[49] have reported that schizophrenia-like symptoms which are observed in temporal lobe epileptics are more frequent in patients who have an epileptic focus in the left temporal region, as compared to right focus patients. Many other reports also suggest that schizophrenics demonstrate left hemispheric dysfunction (Gruzelier and Venables, 1974[50]; Gur, 1978[21]; Hammond and Gruzelier, 1978[22]; Roemer, Shagass, Straumanis and Amadeo, 1978[51], 1979[52]; Hiramatsu, Saitoh, Kameyama, Niwa and Itoh, 1983b[53]; Saitoh, et al., 1983[17]). However, hypotheses about hemispheric dysfunction in schizophrenia seem to be more or less confusing. For example, Schweitzer (1982)[54] has reported that schizophrenics may have a primary deficit in their right hemisphere, and that left hemispheric overactivation may be a compensatory mechanism for the primary failure of the schizophrenic's right hemisphere to maintain normal attention and vigilance.

The present study supports the left hemispheric dysfunction hypothesis. It is significant that in our study a left hemispheric dysfunction was observed exclusively in the unmedicated schizophrenics who exhibited positive schizophrenic symptoms.

Gruzelier (1981)[55] compared the clinical features of paranoid schizophrenia to those of nonparanoid patients and pointed out that positive symptoms are related to left hemispheric dysfunction; while negative symptoms are related to right hemispheric dysfunction. Magaro (1981)[56] also reported that there is evidence for left-hemisphere deficits or right-hemisphere dominance for nonparanoid schizophrenics, speculating left-hemispheric dominance (right hemisphere deficits) for paranoid schizophrenics. Based on these findings, it can be hypothesized that the left hemispheric dysfunction of schizophrenia is related to its positive symptoms.

References

1. Cameron, N. S. (1938). Reasoning, regression, and communication in schizophrenics. Psychological Monographs, 50, 1-33.
2. McGhie, A. and Chapman, J. (1961). Disorders of attention and perception in early schizophrenia. British Journal of Medical Psychology, 34, 103-116.
3. Broadbent, D. E. (1971). Decision and Stress. Academic Press, London.
4. Broen, W. E, Jr. and Storms, L. H.(1966). Lawful disorganization: The process underlying a schizophrenic syndrome. Psychological Review, 73, 265-279.
5. Hillyard, S. A., Hink, R. F., Schwent, V. L. and Picton, T.W. (1973). Electrical signs of selective attention in the human brain. Science, 182, 177-180.
6. Hillyard, S. A. and Picton, T. W. (1979). Event-related brain potentials and selective information processing in man. In Desmedt, J. E. (Ed.), Progress in Clinical Neurophysiology, Vol 6. Karger: Basel.
7. Donchin, E. (1979). Event-related brain potentials: A tool in the study of human information processing. In Begleiter, H. (Ed), Evoked potentials and Behavior. Plenum Press: New York.
8. Jones, R. T. and Callaway, E. (1970). Auditory evoked responses in schizophrenics. Archives of General Psychiatry, 2, 291-298.
9. Levit A. L., Sutton, S. and Zubin, J. (1973). Evoked potential correlates of information processing in psychiatric patients. Psychological Medicine, 3, 487-494.
10. Roth, W. T. and Cannon, E. H. (1972). Some features of the auditory evoked responses in schizophrenics. Archives of General Psychiatry, 27, 466-471.
11. Saletu, B., Itil, T. M. and Saletu, M. (1971). Auditory evoked responses, EEG, and thought process in schizophrenics. American Journal of Psychiatry, 128, 336-344.
12. Shagass, C., Straumanis, J. J., Roemer, R. A. and Amadeo, M. (1977). Evoked potentials of schizophrenics in several sensory modalities. Biological Psychiatry, 12, 221-235.
13. Roth, W. T., Horvath, T. B., Pfefferbaum, A. and Kopell, B.S.

(1980a). Event-related potentials in schizophrenics. Electroencephalography and Clinical Neurophysiology, 48, 127-139.
14. Roth, W. T., Pfefferbaum, A., Horvath, T. B. and Kopell, B.S. (1980b). P3 reductions in auditory evoked potentials of schizophrenics. Electroencephalography and Clinical Neurophysiology, 49, 497-505.
15. Roth, W. T., Pfefferbaum, A., Kelly, A. F., Berger, P. A. and Kopell, B. S. (1981). Auditory event-related potentials in schizophrenia and depression. Psychiatry Research, 4, 199-212.
16. Baribeau-Braun, J., Picton, T. W. and Gosselin, J-Y. (1983). Schizophrenia: A neurophysiological evaluation of abnormal information processing. Science, 219, 874-876.
17. Saitoh, O., Hiramatsu, K., Niwa, S., Kameyama, T. and Itoh, K. (1983). Abnormal ERP findings in schizophrenics with special regards to dichotic detection tasks. In Flor-Henry, P. and Gruzelier, J.(Eds.), Laterality and Psychopathology. Elsevier: Amsterdam, 379-394.
18. Hink, R. F., Hillyard, S. A. and Benson, P. J. (1978). Event-related brain potentials and selective attention to acoustic and phonetic cues. Biological Psychology, 6, 1-16.
19. Colburn, C. J. and Lishman, W. A. (1979). Lateralization of function and psychotic illness: A left hemisphere deficit? In Gruzelier, J. and Flor-Henry, P. (Eds.), Hemisphere Asymmetries of Function in Psychopathology. Elsevier: Amsterdam.
20. Flor-Henry, P.(1976). Lateralized temporal-limbic dysfunction and psychopathology. Annals of the New York Academy of Sciences. 280, 777-795.
21. Gur, R. E. (1978). Left hemisphere dysfunction and left hemisphere overactivation in schizophrenia. Journal of Abnormal Psychology, 87, 226-238.
22. Hammond, N. and Gruzelier, J. (1978). Laterality, attention, and rate effects in the auditory temporal discrimination of chronic schizophrenics: The effect of treatment with chlorpromazine. Quarterly Journal of Experimental Psychology, 30, 91-103.
23. Beaumont, J. G. and Dimond, S. J. (1973). Brain disconnection and schizophrenia. British Journal of Psychiatry, 123, 661-662.
24. Carr, S. A. (1980). Interhemispheric transfer of stereognostic information in chronic schizophrenics. British Journal of Psychiatry, 136, 53-58.
25. Green, P. (1978). Defective interhemispheric transfer in schizophrenia. Journal of Abnormal Psychology, 87, 472-480.
26. Dimond, S. J. and Beaumont, J. G. (1973). Difference in the vigilance performance of the right and left hemispheres. Cortex, 9, 256-265.
27. Dimond, S. J. (1976). Depletion of attentional capacity after total commissurotomy in man. Brain, 99, 347-356.
28. Ellenberg, L. and Sperry, R. W. (1979). Capacity for holding sustained attention following commissurotomy. Cortex, 15, 421-438.
29. Ellenberg, L. and Sperry, R. W. (1980). Lateralized division of attention in the commissurotomized and intact brain. Neuropsychologia, 18, 411-418.

30. American Psychiatric Association (1980). Diagnistic and Statistical Manual of Mental Disorders (DSM-III), APA, New York.
31. Lehmann, H. E. (1975). Psychopharmacological treatment of schizophrenia. Schizophrenia Bulletin, 13, 27-45.
32. Hiramatsu, K., Kameyama, T., Niwa, S., Saitoh, O., Rymar, K. and Itoh, K. (1983a). Schizophrenic deficits in information processing as reflected in event-related potential abnormalities during syllable discrimination tasks. In Mendlewicz, J. and Van Praag, H. M. (Eds.), Advances in Biological Psychiatry, Vol 13. Karger: Basel, 63-74.
33. Naatanen, R., Gaillard, A. W. K. and Mantysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. Acta Psychologica, 42, 313-329.
34. Hansen, J. C. and Hillyard, S. A.(1980). Endogenous brain potentials associated with selective auditory attention. Electroencephalography and Clinical Neurophysiology, 49, 277-290.
35. Hillyard, S. A., Picton, T. W. and Regan, D. (1978). Sensation, perception, and attention: Analysis using ERPs. In Callaway , E., Tueting, P. and Koslow, S. H. (Eds.), Event-related Brain Potentials in Man. Academic Press: New York.
36. Fitzgerald, P. G., and Picton, T. W. (1983). Event-related potentials recorded during the discrimination of improbable stimuli. Biological Psychology, 17, 241-276.
37. Desmedt, J. E. (1980). P300 in serial tasks: An essential post-decision closure mechanism. Progress in Brain Research, 54, 682-686.
38. Yates, A. J. (1966). Data processing levels and thought disorder in schizophrenia. Australian Journal of Psychology, 18, 103-117.
39. Saccuzzo, D. P. and Braff, D. L. (1981). Early information processing deficit in schizophrenia. Archives of General Psychiatry, 38, 175-179.
40. Mintz, M., Tower, R. and Myslobodsky, M. S. (1982). Neuroleptic-induced lateral asymmetry of visual evoked potentials in schizophrenia. Biological Psychiatry, 17, 815-828.
41. Waziri, R. (1980). Lateralization of neuroleptic-induced dyskinesia indicates pharmacologic asymmetry in the brain. Psychopharmacology, 68, 51-53.
42. Mislobodsky, M. and Weiner, M (1976). Pharmacological implications of hemispheric asymmetry. Life Sciences, 19, 1467-1478.
43. Roth, W. T., Krainz, P. L., Ford, J. M., Tinklenberg, J. R., Rothbard, R. M. and Kopell, B. S. (1976). Parameters of temporal recovery of the human auditory evoked potential. Electroencephalography and Clinical Neurophysiology, 40, 623-632.
44. Hemsley, D. R. (1975). A two-stage model of attention in schizophrenia research. British Journal of Social and Clinical Psychology, 14, 81-89.
45. Hemsley, D. R. and Zawada, S. L. (1976). 'Filtering' and cognitive deficit in schizophrenia. British Journal of Psychiatry, 128, 456-461.
46. Hemsley, D. R. and Richardson, P. H. (1980). Shadowing by

context in schizophrenia. Journal of Nervous and Mental Disease, 168, 141-145.
47. Friedman, D., Vaughan, H. G, Jr. and Erlenmeyer-Kimling, L. (1982). Cognitive brain potentials in children at risk for schizophrenia: Preliminary findings. Schizophrenia Bulletin, 8, 514-531.
48. Kimura, D. (1967). Functional asymmetry of the brain in dichotic listening. Cortex, 3, 163-178.
49. Flor-Henry, P. (1969). Psychosis and temporal epilepsy: A controlled investigation. Epilepsia, 10, 363-395.
50. Gruzelier, J. and Venables, P. H. (1974). Bimodality and lateral asymmetry of skin conductance orienting activity in schizophrenics: Replication and evidence of lateral asymmetry in patients with depression and disorders of personality. Biological Psychiatry. 8, 55-73.
51. Roemer, R. A., Shagass, C., Straumanis, J. J. and Amadeo, M. (1978). Pattern evoked potential measurements suggesting lateralized hemispheric dysfunction in chronic schizophrenics. Biological Psychiatry, 13, 185-202.
52. Roemer, R. A., Shagass, C., Straumanis, J. J. and Amadeo, M. (1979). Somatosensory and auditory evoked potential studies of functional differences between the cerebral hemispheres. Biological Psychiatry, 14, 357-373.
53. Hiramatsu, K., Saitoh, O., Kameyama, T., Niwa, S. and Itoh, K. (1983b). A lateralized abnormality of auditory evoked potentials in a case of schizophrenia. Biological Psychiatry, 18, 367-373.
54. Schweizer, L. (1982). Evidence of right cerebral hemisphere dysfunction in schizophrenic patients with left hemisphere overactivation. Biological Psychiatry, 17, 665-673
55. Gruzelier, J. (1981). Hemispheric imbalance masquerading as paranoid and nonparanoid syndrome? Schizophrenia Bulletin, 7, 662-673.
56. Magaro, P. A. (1981). The paranoid and schizophrenic: The case for distinct cognitive style. Schizophrenia Bulletin, 7, 632-661.