Ann. Bull. RILP (1983) No. 17, 277-288

# SCHIZOPHRENIC DEFICITS IN INFORMATION PROCESSING AS REFLECTED IN ERP ABNORMALITIES DURING SYLLABLE DISCRIMINATION TASKS*

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## Introduction

Recently, Event-Related Potentials (ERPs) have been utilized to investigate human sensory information processing. Hink et al. (1978) recorded ERPs during syllable discrimination tasks, and found that the N1 component in normals was enhanced for all stimuli in the attended ear, while the P3 component was enhanced only for 'target' stimuli in that ear. They concluded that the N1 and P3 waves provide converging physiological evidence for the existence of multiple levels of selective attention such as those proposed by Broadbent and Treisman. Broadbent (1971) proposed two sets of selective attention in information processing, one being the 'stimulus set' and the other the 'response set.' Hink et al. also concluded that the stimulus set corresponds to the N1 component, while the response set corresponds to the P3 component.

In this study, schizophrenic deficits in information processing, specifically disturbances in attentional functioning, were investigated in terms of ERP findings. More specifically, ERPs recorded during syllable discrimination tasks, similar to those employed by Hink et al., were utilized as indexes for estimating disturbances in attentional functioning.

## Method

### *Subjects*

Twenty-two schizophrenics (11 males and 11 females; ages 19-39, mean 29.1 years), as well as twenty normal controls (10 males and 10 females; ages 20-38, mean 29.1 years), were used in this study. All of the schizophrenics met the diagnostic criteria of DSM-III for schizophrenic disorders. Ten out of the 22 schizophrenics (5 males, 5 females) had not received any drugs for at least 4 weeks prior to the experiments, while the remaining 12 schizophrenics (6 males, 6 females) were under medication at the time of the experiments. All subjects were right-handed.

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* Submitted to "Advances in Biological Psychiatry" (Mendlewicz, J. and Van Praag, H.M. (eds.), Karger, Basel).

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*Auditory Stimuli*

Four CV-syllables (/ba/, /da/, /ga/, /za/) by a male voice were presented to one ear, with the same four CV-syllables by a female voice presented to the other ear through headphones monaurally. The four CV-syllables by the male voice and by the female voice were digitized and stored in a mini-computer (DEC, LSI 11/2) and were modified in waveshape into a duration of 150 msec. Each of the eight stimuli was presented randomly with an equal a priori probability of .125. The syllables by the male voice were placed in one channel (ear) and those by the female voice in another channel and presented through stereo-headphones (TEAC, HP102) monaurally, with an intensity of approximately 60 dBSL. The interstimulus intervals ranged between 800–1,000 msec.

*Procedure*

The subjects were seated in an anechoic room with eyes closed. They were informed that a male voice would be presented to one ear, and a female voice to the other ear, and were required to silently count the number of occurrences of a particular target syllable in a given ear in each run. All of the subjects performed 16 runs; that is, four target syllables X two voices (male or female) X two attended channels (left or right ear). The order of the 16 runs was randomized for all subjects, with the number of the target syllables for each run being set between 18–26. After the conclusion of each run, the subjects were asked to give the number of target stimuli detected.

*Recording system*

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 were then amplified using a DC-preamplifier (HP 8811A) with a bandpass filter set at 1.5–300 Hz (–6 dB/oct.) and recorded on FM analog tapes along with stimulus trigger pulses (TEAC 12–202). During the experiment, the EEGs were monitored by means of a polygraph.

*Data analysis*

The EEGs without artifacts were passed through a bandpass filter set at 1.5–25 Hz, off line, and then digitized at a sampling frequency of 250 Hz/CH. The EEGs derived from each region during the 16 runs for each subject, were compiled and averaged separately for the left and right ears into four categories using a mini-computer (DEC, LSI 11/2). 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); and 4) non-target syllables in the non-attended ear (N = 384). Thus, each of these averaged potentials contained an equal proportion of responses to each of the four syllables in each ear. The analysis periods were from 20 msec before to 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 and within 600 msec after stimulus onset. The amplitudes and latencies of the N100 and P200 components, as well as the averaged amplitude of the late positive component (LPC), were measured employing the mini-computer and printed out using a line-printer. 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.

The data obtained were analyzed utilizing the ANOVA program of the Statistical Package for the Social Sciences (SPSS).

## Results

1. The amplitude of the N100 component derived from the Cz region (Cz-N100)

Fig. 1 shows the mean amplitudes of Cz-N100 (the N100 component derived from the Cz region) in the normal controls and the schizophrenics. Judging from the results of the ANOVA, the main effect of the factor "DIAG (Normals/Schizophrenic)" 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 normals were larger than those in the schizophrenics. In the normals, the main effect of "CHANNEL (Attend/Non-attend)" 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, in the normals, the amplitudes of Cz-N100 elicited by the stimuli presented to the attended ear were larger than those for the non-attended ear. However, in the schizophrenics this result was not observed.

2. The amplitude of T3-N100 and T4-N100

As in the case of Cz-N100, an effect for "CHANNEL" on the amplitudes of T3-N100 and T4-N100 was observed in the normals [$F(1,303) = 7.28$, $p < 0.01$] but not in the schizophrenics [$F(1,335) = 0.36$, n.s.]. Furthermore, in the normals, the resulting interactive effect of "REGION (T3/T4)" × "EAR (Left/Right)" on the amplitudes of the N100 component was significant [$F(1,303) = 8.78$, $p < 0.01$]. This interaction indicates that both the amplitude of T3-N100 and T4-N100 was larger when the stimuli were presented to the ear contralateral to the EEG-source side than when the stimuli were presented to the ipsilateral ear (Fig. 2). However, in the schizophrenics, this interactive effect was not significant [$F(1,335) = 0.05$, n.s.]. 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 T3.

3. The amplitude of Cz-P200

The effect of the factor "DIAG" on the amplitudes of Cz-P200 was not significant [$F(1,319) = 0.10$, n.s.]. Both the normals and the schizophrenics displayed a channel effect [$F(1,151) = 19.93$, $p < 0.01$; $F(1,167) = 2.91$, $p < 0.01$, respectively]. However, contrary to Cz-N100, the amplitudes of Cz-P200 elicited by the stimuli presented to the non-attended ear were larger than those of the stimuli presented to the attended ear for both groups (Fig. 3). This channel effect was less

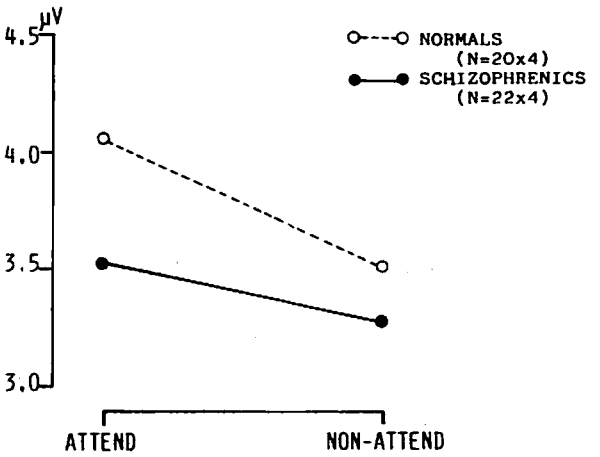


Fig. 1 *Mean amplitudes of Cz-N100.*

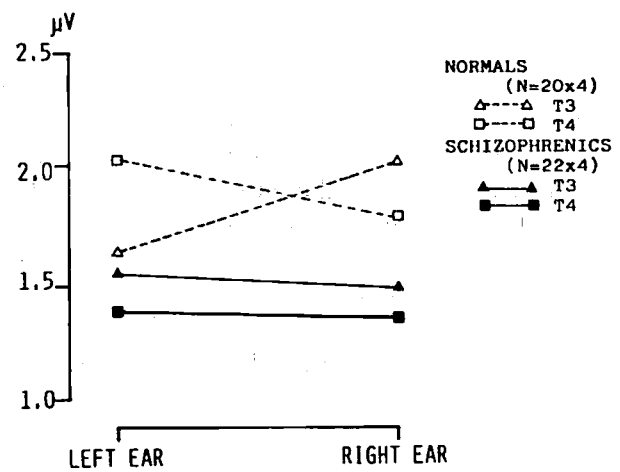


Fig. 2 *Mean amplitudes of T3-N100 and T4-N100.*

prominent in the schizophrenics than in the normals ["DIAG" × "CHANNEL": $F(1,319) = 3.53$, $p < 0.1$].

4. The amplitude of T3-P200 and T4-P200

As in the case of Cz-P200, the channel effect on the amplitudes of T3-P200 and T4-P200 was significant in the normals, as well as in the schizophrenics [$F(1,303) = 26.44$, $p < 0.01$; $F(1,335) = 5.16$, $p < 0.05$, respectively]. An interactive effect for "REGION" × "EAR" on the amplitudes of T3-P200 and T4-P200 was not observed in the normals or in the schizophrenics. In the normals, the amplitudes of T3-P200 and T4-P200 showed an effect for "REGION" [$F(1,303) = 15.87$, $p < 0.01$]. 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 [$F(1,335) = 0.11$, n.s.], and the amplitudes of T4-P200 were nearly equal to those of T3-P200 (Fig. 4).

5. The averaged amplitudes of the late positive components derived from the Cz region (Cz-LPCs)

In the normals, the channel effect on the Cz-LPCs was significant [$F(1,151) = 37.11$, $p < 0.01$], and the interaction of "CHANNEL" × "SYLLABLE (Target/Non-target)" was also significant [$F(1,151) = 7.85$, $p < 0.01$]. Thus, the target syllables in the attended ear produced the largest value of the Cz-LPCs in the normals. However, in the schizophrenics, neither a channel effect nor an interaction of "CHANNEL" × "SYLLABLE" was observed (Fig. 5).

6. T3-LPCs and T4-LPCs

In the normals, a channel effect and an interaction for "CHANNEL" × "SYLLABLE" were observed [$F(1,303) = 61.18$, $p < 0.01$; $F(1,303) = 8.37$, $p < 0.01$, respectively]. Furthermore, an interaction for "CHANNEL" × "SYLLABLE" × "REGION" was observed [$F(1,303) = 3.54$, $p < 0.1$]. That is, the effect of the interaction of "CHANNEL" × "SYLLABLE" was more prominent in T3 than in T4 (Fig. 6). In the schizophrenics, a channel effect was observed [$F(1,335) = 11.19$, $p < 0.01$], but an interaction for "CHANNEL" × "SYLLABLE" was not observed [$F(1,335) = 0.05$, n.s.].

The interaction of "REGION" × "EAR" was significant in the normals, as well as in the schizophrenics [$F(1,303) = 41.82$, $p < 0.01$; $F(1,335) = 23.48$, $p < 0.01$, respectively].

7. The effect of medication on the latencies of the N100 and P200 components

Next, the effect of medication was examined. Judging from the results of the ANOVA, the effect of the factor "MED (Medicated/Non-medicated)" and the interaction of "MED" × "REGION" on the latencies of the N100 and P200 components were significant [N100: $F(1,319) = 22.29$, $p < 0.01$; $F(1,319) = 7.13$, $p < 0.01$; P200: $F(1,319) = 6.45$, $p < 0.05$; $F(1,319) = 9.67$, $p < 0.01$, respectively]. As is shown in Fig. 7, the latency of T3-N100 for the non-medicated group was shorter than that of the medicated group, while the latency of T4-N100 was nearly equal

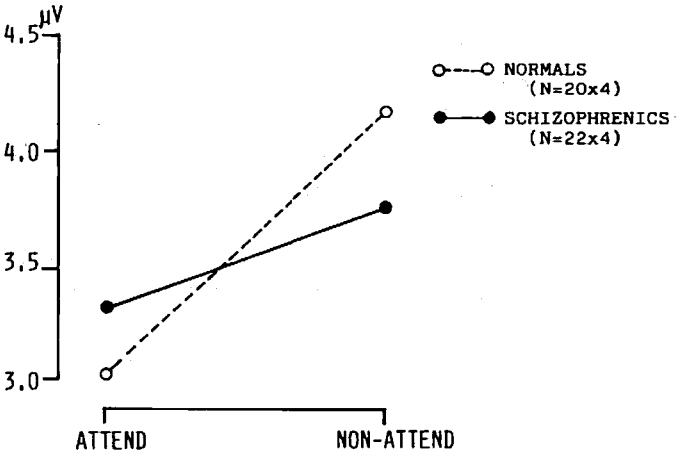


Fig. 3 *Mean amplitudes of Cz-P200.*

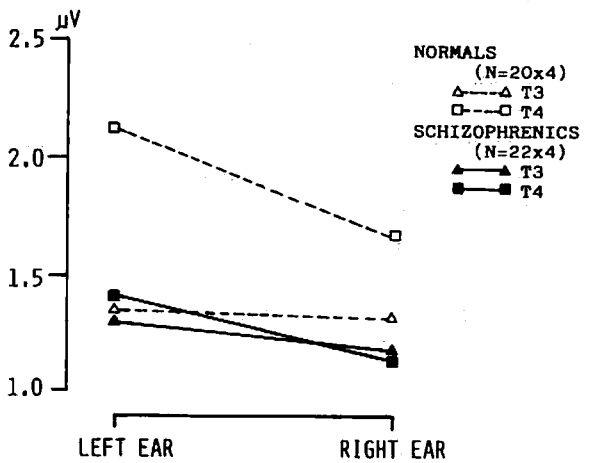


Fig. 4 *Mean amplitudes of T3-P200 and T4-P200.*

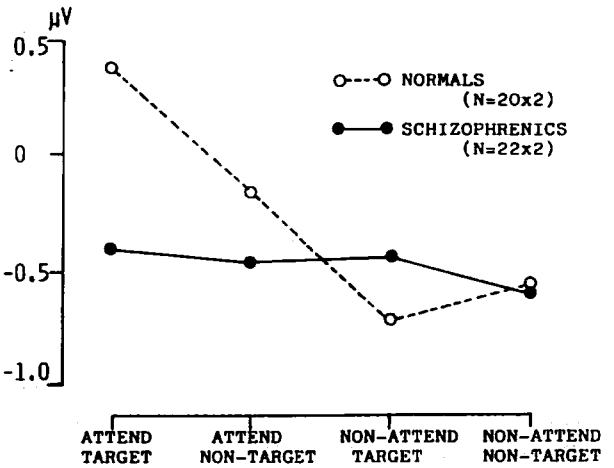


Fig. 5 *Mean LPCs (Cz).*

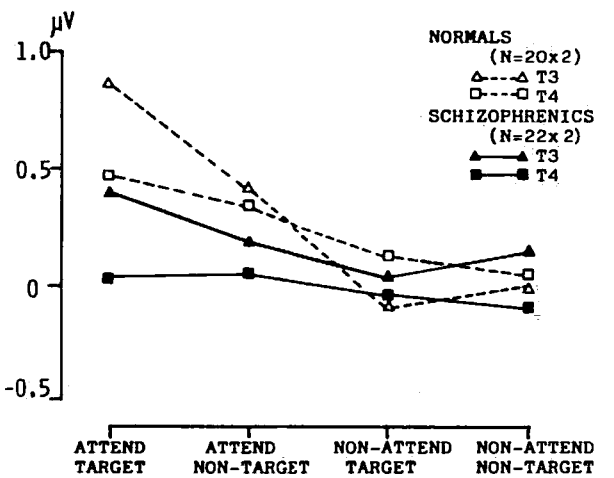


Fig. 6 *Mean LPCs (T3, T4).*

in both groups. The latency of T3-N100 for the non-medicated group was also shorter that of T4-N100 for the non-medicated group.

Fig. 8 illustrates similar findings for the P200 component. The latencies of N100 and 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 normals.

## Discussion

Hink et al. (1978) recorded ERPs from the Cz, C3' and C4' regions (lateral to C3 and C4 by 10% of the inter-aural distance, respectively) in normal subjects during syllable discrimination tasks similar to those employed in this study. They found that the N1 component derived from the Cz region was enhanced for all stimuli in the attended ear, while the P3 component was enhanced only for 'target' stimuli in that ear. They also found that the N1 amplitude was larger for the side contralateral to the ear of stimulation, and that there were no significant asymmetries in P3 activity.

In this study, we compared ERP-waveforms derived from the Cz, T3 and T4 regions during syllable discrimination tasks in normals and schizophrenics. The results we obtained for the normals can be summarized as follows. 1) In all three regions, the N100 amplitude was enhanced for stimuli in the attended channel (ear), while the P200 amplitude was reduced for the same stimuli. 2) The LPCs, by which the P300 amplitudes were represented, were enhanced for the target stimuli in the attended channel. 3) The N100 amplitude was larger for the side contralateral to the ear of the stimulus presentation than for the ipsilateral side. 4) The P200 amplitude of T4 was larger than that of T3, irrespective of the side of the stimulus presentation. 5) The LPCs for the target stimuli were larger at T3 than at T4.

Hence, it can be properly concluded that the results of Hink et al. (1978) concerning ERPs derived from the Cz region were replicated in this study. Although Hink et al. did not mention the P200 component in their report, it was observed in our study that the P200 amplitude displays channel effect inverse to that for the N100 component.

It has been reported by other investigators that a prolonged negative component with a peak latency of 200 msec can be observed through subtraction of the ERPs elicited by stimuli in the unattended channel from those elicited by stimuli in the attended channel (Näätänen et al., 1978; Hansen and Hillyard, 1980). We did not examine the existence of such a prolonged negative activity in this study. It can be speculated, however, that if such a prolonged negative shift overlaps with the exogenous N100 and P200 components, an increase in the N100 amplitude, as well as a decrease in the P200 amplitude, could result from this overlapping. However, the possibility that the channel effect works on the P200 amplitude in an independent manner from that of the N100 amplitude also remains.

Based on our results, we can add the following new finding to the results of Hink et al. That is, attentional effects contribute to ERPs derived from the T3 and T4 regions to the same degree as to those from the Cz region. Furthermore, in contrast to Hink et al., we observed a lateral asymmetry for P300. That is, the LPCs elicited

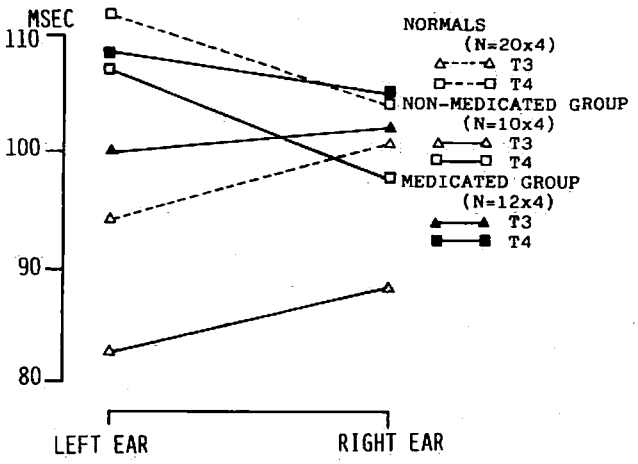


Fig. 7 *Mean latencies of T3-N100 and T4-N100.*

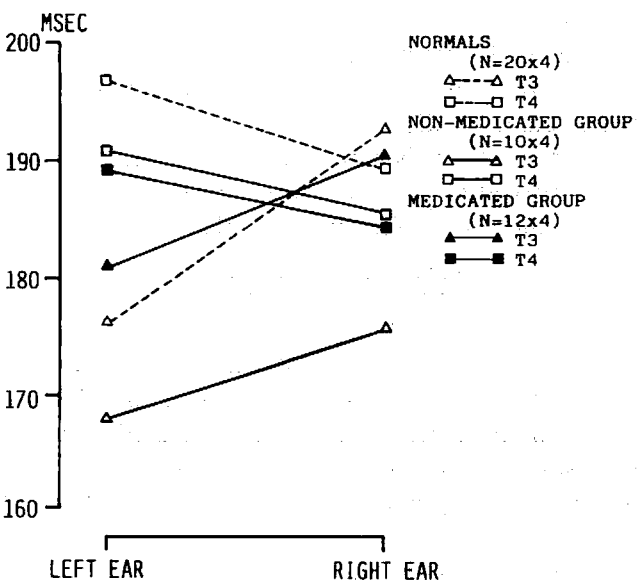


Fig. 8 *Mean latencies of T3-P200 and T4-P200.*

by the target stimuli displayed a larger value at T3 than at T4. This difference may have been brought about through the difference in the EEG-source sites in our two studies. From our result, it can be suggested that the process of target detection in verbal tasks, such as those employed in this study, exclusively produces larger values for LPCs in the dominant hemisphere than in the non-dominant hemisphere.

It has been reported by some authors that schizophrenics demonstrate smaller amplitudes for the N100 and P300 components (e.g., Roth et al., 1981). However, there seem to have been few investigations in which schizophrenic deficits in information processing, specifically disturbances in attentional functioning, have been studied in relation to ERPs recorded during relatively complex psychological tasks that have required subjects to activate selective attentional functioning. Saitoh et al. (1983) recorded ERPs in schizophrenics during dichotic detection tasks. They reported that schizophrenics demonstrated disturbances in both the 'stimulus set' and the 'response set' as defined by Broadbent (1971).

In this study, the schizophrenics 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 than the normals. Assuming that the channel effect for the N100 and P200 components is reflected in the prolonged negative shift as described previously, it can be speculated that the negative shift overlaps with the P200 component, but not with the N100 component, in the case of schizophrenics. In other words, the negative shift in schizophrenics can be supposed to appear after some delay with a low amplitude. Therefore, it can be speculated that the attentional functioning for channel selection in schizophrenics is ineffective. Moreover, the following two possibilities can be assumed. Information processing may begin with some delay in schizophrenics; or the processing speed in schizophrenics may be slow.

In regard to the P300 component, it has been strongly argued that it reflects target detection or stimulus evaluation (Hillyard et al., 1978; Donchin, 1979). The schizophrenics failed to display enhanced LPCs (representing the P300) to the target stimuli in this study. This result does not necessarily suggest that the schizophrenics were not motivated. In order to check this point, we calculated the Error Indexes (EIs) according to the following formula.

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

The EIs were then utilized as indexes of performance level. The mean EI of the schizophrenics was 25.4%, with that of normals being 5.7%. 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 expected EI value if they had counted the number of target stimuli in a random manner.

Hence, it can be concluded that schizophrenics have some disturbances in the stage of information processing which is reflected in the LPCs. These disturbances are thought to be responsible for the low performance levels in schizophrenics. Differing from our speculations, Desmedt (1980) has interpreted the P300 component as reflecting a post-decision closure mechanism. From his standpoint, our result concerning LPCs could alternatively indicate that schizophrenics have

some disturbance in the post-decisional mechanism, but not in the stimulus evaluation process. Although not shown in this report, a positive correlation between the LPCs and performance level was found for the normals in this study. Therefore, Desnedt's interpretation of P300 cannot be maintained.

Concerning ERP-asymmetries in schizophrenics, Shagass et al. (1977) and Roemer et al. (1978) have reported that schizophrenics display a more marked waveshape instability in the left hemisphere than in the right hemisphere. Hiramatsu et al. (1983) found that a schizophrenic patient with persistent auditory hallucinations demonstrated an abnormality of the N1-P2 component of the AEP exclusively in the vicinity of the T3 region. Saitoh et al. (1983) speculated from their results concerning ERPs during dichotic detection tasks that schizophrenics have a left-hemisphere dysfunction.

In this study, the non-medicated group of schizophrenics demonstrated shorter latencies for the N100 and P200 components exclusively in the T3 region than the normals. The medicated group of schizophrenics displayed latencies for the N100 and P200 components nearly equal to those of the normals even at the T3 region. Schizophrenics have been reported to display shorter latencies for the N100 and P200 components (Saletu et al., 1971; Roth et al., 1980). Furthermore, neuroleptics have been considered as delaying these latencies (Saletu et al., 1971; Sakalis et al., 1972; Roth et al., 1972). However, few reports have mentioned asymmetries in latencies. Seven out of the ten non-medicated patients in our study were in an active phase at the time of the experiment. Therefore, it can be speculated that schizophrenics demonstrate shorter latencies for ERPs at the T3 region during their active phase, and that neuroleptics normalize the shortened latencies. A lateralized effect for neuroleptics has also been noticed by Mintz et al. (1982).

In this study, the schizophrenics displayed smaller amplitudes for the N100 component at the T4 than at the T3 region. In addition, 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. Moreover, the schizophrenics failed to show an asymmetry in P200 amplitudes between the T3 and T4 regions. All these results seem to indicate that schizophrenics have difficulty in the integration mechanism of both hemispheres as well.

It can be concluded that schizophrenics seem to have two kinds of hemispheric dysfunctions, one being a dysfunction of the left hemisphere itself, with the other being a dysfunction in the integration mechanism of both hemispheres. It can also be concluded that neuroleptics may improve the left hemisphere dysfunction.

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