

BRIEF REPORTS

Brain Stem Auditory Evoked Potentials in Unmedicated Schizophrenic Patients

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Introduction

Numerous differences in auditory evoked potentials (AEPs) have been found between schizophrenic patients and normal controls. Roemer et al. (1979) have reported decreased stability in the AEP to click stimuli from 15 to 450 msec poststimulus. Shagass et al. (1977) have observed decreased AEP amplitudes to click stimuli over most of this epoch from electrodes placed in a variety of scalp locations. Freedman et al. (1983) have reported a shorter latency of P50 to click stimuli in unmedicated acute schizophrenics. Roth et al. (1980) found decreased P300 amplitude to rare auditory stimuli in medicated schizophrenic patients. Given these findings it is of interest to determine whether the first phase of auditory information processing, the first 6-8 msec post stimulus, is different in schizophrenics compared with controls, or whether differences in auditory information processing occur more than 8 msec poststimulus, that is, after the afferent volley has reached the inferior colliculus.

Pfefferbaum et al. (1980) found no differences in the latencies of auditory brain stem evoked potentials (BSEPs) between male schizophrenic patients, one-half of whom were medicated, and normal controls to binaurally

presented square-wave clicks of 50, 60, 70, and 80 db. Similarly no between-group differences were found to 80-db clicks presented with interstimulus intervals of 20 or 80 msec. Szelenberger (1983) found no differences in BSEP peak latencies between medicated male paranoid schizophrenics and normal controls. The purpose of the present report is to present BSEP data from a group of unmedicated schizophrenics that confirm and extend these findings.

Methods

Eight of nine schizophrenic patients were hospitalized at a university hospital. One of them was an outpatient. DSM-III diagnosis of schizophrenia was made independently by one of us (M.B.) and by another clinician. Patients were medication free for at least two weeks. Five of the patients were given 30 mg flurazepam hydrochloride (Dalmane) or 65 mg sodium amytal prn for insomnia up to 72 hr before testing. Nine controls were recruited by advertisement and screened for psychopathology and drug use. The mean age of the patient was 31.1 years \pm 8.47 and that of the controls 29.0 years \pm 2.15. There were four women in the patient group and two women in the control group.

All subjects were tested with three paradigms during the recording session. The paradigms were always run in the same order with the BSEP paradigm second. Subjects were tested in a darkened sound-attenuated chamber. Two thousand 70-db (SPL, alternating rarefaction and condensation) click stimuli of 0.5-msec duration were

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Table 1. Auditory Evoked Potentials in Unmedicated Schizophrenics and Controls^a

Group	Right ear stimuli ^b						Left ear stimuli ^c					
	Peaks			Interpeak latencies			Peaks			Interpeak latencies		
	1	3	5	3-1	5-1	5-3	1	3	5	3-1	5-1	5-3
Schizophrenics	1.791 (0.173)	4.000 (0.275)	5.825 (0.396)	2.211 (0.283)	4.035 (0.329)	1.822 (0.334)	1.839 (0.138)	3.872 (0.285)	5.879 (0.387)	2.033 (0.346)	4.081 (0.468)	2.047 (0.296)
Controls	1.691 (0.225)	3.766 (0.491)	5.816 (0.321)	2.835 (0.667)	4.125 (0.453)	2.050 (0.390)	1.944 (0.421)	4.061 (0.496)	6.086 (0.36)	2.116 (0.488)	4.417 (0.299)	2.025 (0.359)

^aStandard deviations are given in parentheses.

^bF values: groups, 0.064; groups × peaks, 1.1. *p* values: groups > 0.1; groups × peaks > 0.1.

^cF values: groups, 0.891; groups × peaks, 0.084. *p* values: groups > 0.1; groups × peaks > 0.1.

presented to one ear through headphones at the rate of 10/sec. Stimuli were presented to each ear with order of presentation randomized. Monopolar recordings were taken between a vertex electrode and the ipsilateral electrode, with an electrode on the forehead serving as ground. The potentials were amplified 100,000 times and subjected to digital filtering with a bandpass of 100 Hz to 2000 kHz. Brain electrical activity was sampled every 25 μ sec for 10 msec after each click. Peaks were identified visually, and the latencies of the first five peaks and the peak 3-1, peak 5-1, and peak 5-3 interpeak latencies were measured. For each ear the peak latencies and interpeak latencies were subjected to two-way analysis of variance with repeated measures and correction applied to the degrees of freedom.

Results

The results are presented in Table 1. For right ear stimuli, the F values for groups and groups by peak effects are 0.064 and 1.1, respectively. The F values for groups and groups by peak effects for left ear stimuli are 0.891 and 0.084, respectively. These F values have probabilities far greater than 0.1.

Discussion

Saletu et al. (1971a,b) have reported increased somatosensory evoked potential latencies in schizophrenic patients given a variety of neuroleptics. The shortened P50 latencies recorded by Freedman et al. (1983) increased to normal levels after these acutely psychotic schizophrenics were treated with neuroleptics. This raises the possibility that schizophrenic patients may have abnormally short BSEP latencies that are normalized by medication. However, the similar latencies in unmedicated schizophrenics and controls render this interpretation unlikely.

These data support the view that the earliest stage of auditory information processing is not abnormal in schizophrenic patients. Alternat-

tively stated, these data suggest that conduction velocity in the auditory nerve and the brain stem structures relaying auditory information is within normal limits in schizophrenics. This is consistent with the postmortem studies of Hankoff and Peress (1981), who found brain stem pathology in only one of eight schizophrenic patients.

This result is of importance given the panoply of neurophysiological findings in schizophrenia, particularly the wide variety of post-15-msec evoked potential abnormalities interpreted to reflect cortical and subcortical dysfunction. Normal BSEP latencies distinguish schizophrenics from alcoholics (Begleiter et al. 1981) and multiple sclerosis patients (Chiappa and Ropper 1982), who have prolonged peak and interpeak latencies. Well-documented neuropathology is also found in the latter two groups. This normal feature of schizophrenia in conjunction with abnormal anatomical and physiological findings in more rostral brain structures is useful in differentiating schizophrenia in a parametric fashion from other brain diseases.

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