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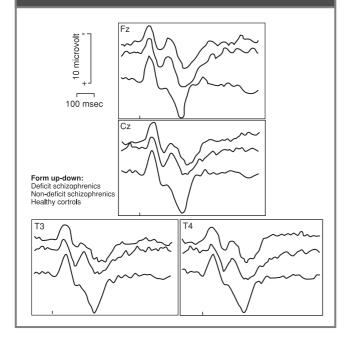
HE BROKEN P300 IN SCHIZOPHRENIA:

CAN IT BE FIXED? (1)

New Frontiers in Psychiatry / Psikiyatride Yeni Ufuklar

Schizophrenia is probably the most devastating illness that psychiatrists treat. An estimated 1% of the population has schizophrenia, which typically begin in the teens or early 20s, leaving most of those affected unable to return to normal lives. Since Thomas Szasz (1961) who believe in that people with schizophrenia have a fake disease: "to be a true disease it must somehow be capable of being approached, measured, or tested in a scientific fashion"; neuroscientist focus on this psychiatric disease have come a long way. With the help of advanced neuroimaging techniques, we are now able to measure directly the deviation of the neuronal activity in the brains of

Figure 1: The grand averages of a group of schizophrenic patients and healthy controls. Both defict and non-deficit schizophrenic patients have smaller P300 amplitude than healthy controls (detalis of patients have been given and can be found in the reference Gonul et al 2003).



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schizophrenic patients. However, a relatively old finding is still one of the major evidence for alteration in the normal physiology of neurons in schizophrenia: reduced P300 amplitude.

Until Sam Sutton and his colloquies discovered the P300 component of the event-related potential (ERP) in 1965; ERPs called evoked potentials, which were primarily investigated as a possible means of testing the integrity of sensory pathways (Sutton et al 1965). In contrast P300 component was demonstrated to reflect a cognitive process re-

lated to failure of an expected event to occur could be elicited in the absence of a stimulus.

At late 1960s and early 1970s two groups of investigators attached electrodes to the scalps of schizophrenic patients and controls to measure the newly reported P300 (Roth and Cannon 1972, Levit et al 1973). Both groups found that P300 amplitude was lower in persons with schizophrenia than in controls. Since than, this finding was confirmed many times from different researchers from different countries. This finding replicated in different types of schizophrenic patients like deficit and non deficit types (Figure 1).

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