showed again significant differences in Cz and Fz. In post hoc tests, these results were due to a significant difference between medicated patients and controls. The present findings indicate a significant effect of medication on attention/task related auditory information processing in schizophrenic patients.

88. PERCEPTUAL DEFICITS IN SCHIZOPHRENIA MAY BE EXPLAINED BY ABERRANT GAMMA RANGE OSCILLATIONS

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Problems in cognition in schizophrenia may be explained by a problem in the timing of neural processes across different cortical regions. We explored a possible connection between backward masking deficits in schizophrenia and aberrant gamma range cortical oscillations. Backward masking is a procedure that assesses the earliest components of visual processing. On this procedure, the visibility of a briefly-presented target is reduced by a visual mask. Schizophrenic patients consistently show deficits on this procedure. As the interval between target and mask increases, backward masking performance sometimes shows an oscillating pattern, which is thought to reflect gamma oscillations of visual pathways. Previously, we found that normal controls showed an oscillating pattern, whereas schizophrenic patients did not show such a pattern.

We recently developed a computerized version of backward masking that is well suited for modeling the performance pattern in masking. Intervals between target and mask were set regularly at 13 ms increments, and a threshold procedure was used to equate subjects for initial performance. On this procedure we found the predicted qualitative differences between patients and normal comparison subjects. There was a significant oscillating component for the comparison subjects, (i.e., a sine wave fit the data), but there was no such component for patients. The pattern of results suggest that a fundamental abnormality in establishing cortical oscillations may partially account for backward masking deficits in schizophrenia. It is likely that sensory and perceptual neurocognitive deficits in schizophrenia may have common roots in a failure to establish cortical oscillations.

89. ALTERED PLATELET MITOCHONDRIAL COMPLEX I ACTIVITY IN SCHIZOPHRENIA

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Mitochondrial abnormality has been observed in several neurological disorders. Changes in energy metabolism and thereby disturbed neuronal function, is an attractive explanation for some of the pathological aspects observed in schizophrenia. Indeed, PET studies have suggested that a change in energy metabolism in cortical and subcortical structures is involved in the pathophysiology of schizophrenia. An electron microscopy study in autopsied anterior limbic cortex of schizophrenic patients revealed deformation and reduction in the number of mitochondria. Our results show that in platelets of medicated and unmedicated schizophrenic patients at acute relapse complex I activity, but not complex IV, is increased significantly (240%, p < 0.001). No such change was observed in patients with affective disorder. To find out whether the increase in complex I activity is state dependent we have measured its activity in patients with acute relapse, chronic patients with positive symptoms and patients with residual schizophrenia and compared them to normal subjects. Complex I activity was significantly increased in both the acute and chronic patients with positive symptoms while reduced in chronic patients with residual schizophrenia as compared to normal subjects. A positive correlation was observed between complex I enzymatic activity and mRNA of 24 kDa iron-sulfur flavoprotein subunit of complex I, which has catalytic propiexie. mRNA levels of subunit 75 kDa, the largest transmembranous iron-sulfur flavoprotein which demonstrate no catalytic activity, was similar in all groups. If complex I abnormality in platelet reflects brain alterations, it may further support the relevance of abnormal energy metabolism to the pathophysiology of schizophrenia and might become a useful peripheral marker for this disorder.

90. A LONGITUDINAL STUDY OF HIPPOCAMPAL VOLUMES IN SCHIZOPHRENIA


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Cross-sectional MRI brain studies have shown a similar degree of hippocampal volume reduction in patients with chronic schizophrenia and first-episode psychosis. It remains unclear whether these abnormalities predate the onset of symptoms, occur during the initial development of psychosis or whether they progress over the course of the illness.

We examined hippocampal volumes longitudinally (time 1/time 2) in patients with chronic schizophrenia (n = 11), first episode psychosis (n = 22), an ‘at-risk’ group (n = 27) and control subjects (n = 23). At time 2, 11 of the ‘at-risk’ group had developed a psychotic illness. Hippocampal, whole-brain and intracranial volumes were estimated from high-resolution 1.5-mm cortical images.

There was no group by time interaction for either left ($F(4,77) = 0.2, p = 0.93$) or right ($F(4,77) = 0.4, p = 0.77$) hippocampus (covaried for intracranial volume. In addition, there was no group by time interaction for whole brain volume, covaried for intracranial volume ($F(4,77) = 1.5, p = 0.20$).

These data did not show longitudinal changes in hippocampal volume in patients with chronic schizophrenia, first-episode psychosis or the at-risk group who developed psychosis, consistent with the neurodevelopmental model. It is possible that the manual tracing technique used is not sensitive enough to identify small hippocampal changes in small samples.