

These findings suggest that those patients with extreme early onset (childhood) forms of schizophrenia may have more severe developmental brain anomalies compared to those with adult onset.

STRUCTURAL AND FUNCTIONAL IMAGING IN CATATONIA

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Introduction: Pathophysiology of catatonia in general and of posturing in particular is still unknown. We therefore performed structural measurements in CT and functional MRI activation studies in catatonic patients.

Methods: 37 catatonic Schizophrenic patients were investigated with CT and quantitative measurements were done. Furthermore we investigated two neuroleptic-naive catatonic patients exhibiting posturing with motor activation in F-MRI.

Results: Catatonic patients showed highly significant enlargements in CT of almost all inner and outer CSF spaces, mostly pronounced in frontal areas whereas parieto-occipital regions did not differ significantly from controls. In F-MRI both catatonic patients showed parietal hyperactivation which was not seen in 2 age and sex matched healthy controls, whereas frontal activation did not differ.

Conclusion: Catatonic patients seem to suffer from considerable brain atrophy and the parietal cortex may be central in the pathophysiology of catatonia.

BRAIN MORPHOLOGY IN DOWN SYNDROME AND SCHIZOPHRENIA

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Schizophrenia (SZ) is often presumed to be neurodevelopmental in etiology. The variability of brain morphologic changes reported in SZ is often cited as evidence of non-uniform etiology, and perhaps of genetic diversity. Since Down syndrome (DS) is a neurodevelopmental disorder of known (genetic) and generally uniform etiology, we compared qualitative measures on MRI scans in samples of 50 DS and 50 SZ group-matched on age and sex to matched normals to assess type and variability of brain pathology.

We found a wide diversity of morphologic abnormalities in both disorders, some which were clearly neurodevelopmental. In DS, given the uniform genetic lesion, we were surprised by the wide disparities in type, number and severity of brain abnormalities between individuals. This suggests the importance of modifier variables, (perhaps genetic) and suggests that similar mechanisms may be relevant in schizophrenia.

ACCELERATED INCREASE BRAIN VENTRICULAR VOLUME AT 2-YEAR RESCAN FOR CHILDHOOD ONSET SCHIZOPHRENICS

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Childhood onset schizophrenia (COS) (onset by age 12) is a severe, often treatment refractory form of the disorder. No studies have examined longitudinal change in brain anatomic images for very early onset cases. Our hypothesis was that adolescents with COS would exhibit greater ventricular enlargement than that for a matched group of 19 healthy controls. A group of patients with mean onset of illness of 10.2 yrs was scanned at mean age 14.7 and 16.8 yrs. An age and sex matched group of 19 healthy controls was also scanned, using the GE 1.5 T magnet. Our data showed that at two year MRI rescan 15 COS had a highly significant increase in ventricular volume ($p=0.003$) while rescan MRI for 19 healthy controls showed only a trend for increase ($p=0.11$) (Dx \times Time interaction = 0.005). There was no differential change in total cerebral volume. These data suggest an ongoing dynamic process of abnormal brain development for very early onset cases.

THE EFFECT OF CUMULATIVE NEUROLEPTIC EXPOSURE ON CAUDATE AND PUTAMEN VOLUMES

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We examined the effect of cumulative neuroleptic exposure on caudate nuclei, putamen and hippocampus volumes. We hypothesized positive correlations between cumulative neuroleptic exposure and caudate and putamen volumes but not with the hippocampus which is relatively lacking in D2-Dopamine receptor expression.

The volumes of the caudate nuclei, putamen, hippocampus and cranium were calculated from tracings of MRI images obtained on 70 schizophrenic males. Each patient's lifetime exposure to neuroleptics was estimated by a method developed at our center.

ANOVA's performed on the variables age, neuroleptic dose-years (NDY), caudate, putamen, hippocampus, and cranial volumes revealed a main effect of NDY on caudate ($F=18.57$, $p<0.0001$, $df=1,66$) and putamen volumes ($F=5.23$, $p<0.0254$, $df=1,66$) but no significant effect on hippocampus volumes ($F=0.01$, $p<0.9427$, $df=1,66$). A correlational analysis revealed that NDY was positively correlated with caudate