

**Structural Changes After Anterior Capsulotomy for Obsessive Compulsive Disorder**James A. Taren<sup>1</sup>, George C. Curtis<sup>2</sup>, Stephen S. Gebarski<sup>3</sup><sup>1</sup> Neurosurgery, University of Michigan Medical Center, Ann Arbor, Michigan; <sup>2</sup> Psychiatry, University of Michigan Medical Center, Ann Arbor, Michigan; <sup>3</sup> Radiology, University of Michigan Medical Center, Ann Arbor, Michigan

Stereotactic anterior capsulotomy in five patients with intractable obsessive compulsive disorder (OCD) resulted in progressive clinical improvement which correlated with serial magnetic resonance imaging evidence of progressive local and distant alterations in brain structures. Volumetric changes were noted in the caudate, mamillary body, thalamus, hippocampal formation, and the lesion site in the anterior limb of the internal capsule. These changes support the hypothesis of a neuroanatomic substrate for OCD.

**ENHANCEMENT OF CREATIVE MATURITY AFTER AN ACCIDENT.**

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Clinical account of two cases of increased creative maturity following accidental injuries. The first involved an eighteen year old man who fell flat on his back and was unable to breathe for half a minute. He later stood up without assistance. He refrained from speaking for one day and from grooming himself for three days. He soon resumed a congenial manner, and months later, friends observed an increasingly mature level of behavior. This increase was complimented by an increase of creativity in his fine art endeavors. His enhanced creativity continued for the duration of his life. The second case involved a nineteen year old woman who was thrown from a car during an accident without losing consciousness. The woman subsequently earned her Ph.D. and produced three biographies considered definitive in her field. She feels her enhanced creative faculty stems from the accident. The suggested mechanism, as in W. B. Cannon *Wisdom of the Body*, stems from the body's ability to marshal nervous and chemical resources for restoration. The dynamics of this mechanism differ from my previous report on Delayed Psychosomatic Responses, Life Sciences, v.4, pp.2047-56 (1965).

**The Cognitive Functioning in Depression: A Neuropsychological Approach.**Sanja Totić, Dragan Marinković, Brankica Aćimović, Tibor Babinski and Vladimir R. Paunović. Institute of Psychiatry, University Clinical Center, Belgrade, Yugoslavia.

The cognitive impairment in depression includes qualitative and quantitative changes and spans a range of cognitive functions. A number of studies suggest a "subcortical" pattern of cognitive dysfunction in depression. Luria-Nebraska neuropsychological battery was administered to 15 male unmedicated inpatients who met DSM-III-R criteria for major depression and 15 male, healthy volunteers as control group. The aim of study is to compare the cognitive performance between two groups and to determine, if any, specific neuropsychological profile of depressed patients. The results indicate the right frontal and right parieto-occipital dysfunction in depressed patients and the "subcortical" pattern of cognitive dysfunction. This finding suggest that dysfunction of subcortical brain regions may be responsible for the cognitive impairment observed in depressed patients.

**VENTRICULAR VOLUMES IN PATIENTS WITH TEMPORAL LOBE EPILEPSY AND PSYCHOSIS**Daniel Umbricht, Houwei Wu, William Barr, Neil Schaul, Jeffrey Lieberman, Hillside Hospital, Psychiatry Research Dept., P.O. Box 38, Glen Oaks, NY 11004

A similar neuropathology could be one reason for the similarities of the psychosis in patients with temporal lobe epilepsy (TLE) to schizophrenia. We investigated whether these two patient groups show comparable increases of ventricular volumes indicating a common neuropathology differentiating them from non-psychotic patients with TLE and from normal controls. The volumes of the lateral ventricles of 9 patients with TLE and psychosis were measured on high resolution MRI brain scans and compared to the corresponding values of 9 patients with TLE and no psychosis, 9 schizophrenics and 9 normal controls, matched on age, sex and duration of epilepsy, respectively. The schizophrenic patients showed significantly larger volumes than the normal controls, while the values of both TLE groups lay in between, neither significantly different from each other nor from the two other groups. We found no evidence that patients with TLE and psychosis show an increase of ventricular volumes similar to the one observed in schizophrenic patients. In so far as the neuropathology underlying ventricular enlargement in schizophrenia is related to psychopathology, this study does not support the notion that schizophrenia-like symptoms in patients with TLE are based on a similar neuropathology.