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PROTEIN TAU - A MARKER FOR "EARLY FORMS" OF ALZHEIMER'S DISEASE

M. Schmolke, M. Riemerschneider, K. Buch, A. Kurz, W. G. Guder. *Inst. F. Klin. Chemie., Städt. Krankenhaus Bogenhausen, Englschalkingerstr. 77, München, Germany.*

Protein tau (tau), a main constituent of neurofibrillary tangles, is elevated in cerebrospinal fluid (CSF) of mild to moderate and severe forms of Alzheimer's disease (AD) [1-4]. To prove tau as a marker for early AD forms we investigated four groups of patients: a) very mild AD in patients having a cognitive performance above the conventional "cut off" level of dementia (Mini Mental State Examination score (MMSE) = 25-28; n=15), b) milde to moderate AD (MMSE<24; n=18), c) other forms dementia (OFD; n=8) and d) cognitively health controls (n=30). AD diagnosis was established according to NINCDS-ADRDA criteria. Tau was determined by an enzime linked immunoassay [5]. CSF-tau concentrations (mean ± SEM) of very mild (677 ± 113 ng/l) and mild to moderate AD (757 ± 117 ng/l) were significantly higher than controls (175 ± 12 ng/l; $p<0.01$ Kruskal-Wallis). Tau of both AD groups did not differ significantly. No correlation was seen with the severity of cognitive impairment (MMSE). Tau was slightly elevated in OFD (<360 ng/l) except in Steele-Richardson-Olszewski-Syndrom which is accompanied by neurofibrillary tangles (400; 610 ng/l; n=2). In conclusion, measurement of CSF-tau seems to hold promise as a biological marker in the early diagnosis of AD and the differential diagnosis of dementia.

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Neuropsychological Assessment in Treatment - Resistant Schizophrenics

Ilia Theotoka (*), G.M.Papageorgiou, E. Polyzopoulos, Ch. Istikoglou, C. Vlachos
Department of Psychiatry, Evangelismos General Hospital and (*) Dromokaition Mental Hospital, Athens, Greece

The aim of this study was to investigate the Neuropsychological profile of non-responders to classical antipsychotics under treatment with risperidone. Twelve DSM-III-R schizophrenic inpatients, treatment resistant to classical antipsychotics and under current treatment with risperidone were assessed neuropsychologically using Benton, Rey and WAIS-R tests, with the question whether a more favorable response with risperidone was connected with a better neuropsychological profile. BPRS scores were used for psychopathological assessment.
After 45 days of treatment, there wasn't a statistically significant difference between the various patients in our sample, but those who had more favorable course under risperidone had an overall better neuropsychological profile
This kind of assessment might be useful for the recognition of potentially good responders to the atypical neuroleptics.

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MENTAL ILLNESS, HEMISOHERIC DYSFUNCTION, PSYCHOTHERAPY

M. Trimarchi, L. L. Papeschi, P. Guasco, M. Nicolini, P. Russo, C. Persegani, E. Lugaresi. *Brain Health Centre, Via Antonio Bertoloni, no. 29, Rome, Italy.*

The main psychiatric illnesses show a specific underlying hemispheric dysfunction. In the case of mood disorders, for instance, it has been demonstrated that manic symptomatology is likely caused by a left hemisphere dysfunction that may lead to an exaggerated non-dominant arousal (Stratta P et al, 1995). The clinical heterogeneity of depressive disorders may be associated with variations in both left frontal and right posterior dysfunctions (Bruder GE, 1995). By interpreting recent findings that have emphasized specific inter- and intra-hemispheric anomalies in schizophrenia (Trimarchi M. et al 1995) found a probable functional etiology of schizophrenia in these alterations. Psychotherapy cannot disregard hemispheric functional asymmetries which take place in psychiatric disorders. Neuropsychophysiology applied to psychological treatment enables therapists to intervene on cerebral dysfunctions, taking advantage of: 1) information characteristics management and 2) the specific functional mechanisms of the two cerebral hemispheres (Trimarchi M, Papeschi LL, 1986). Neuropsychophysiology-oriented psychotherapy bridges hemispheric dysfunction, symptoms and psychological treatment (Trimarchi M. et al, 1996), leading to symptomatology reversal which is positively related to timeliness of treatment. Our clinical data show how it is possible to modify cerebral dysfunctions through specific hemispheric stimulations, enabling us to test its psychotherapeutical effect both before and after treatment.

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COMPUTER MODEL FOR CHOICE TACTICS OF TREATMENT MAJOR DEPRESSION

R. Vovin, C. Mazo, M. Ivanov. *Department of Biological Treatment of Psychiatric Patients, Bekhterev Research, St. Petersburg, Russia.*

The objective of this trial is the choice optimum therapeutical tactics with computer models of treatment for endogenic depressions. The research computer model was based on 104 patients with DSM-III-R major unipolar depressive disorder. 70 patients were treated by SSRIs: Fluoxetin (Lilly) - 20 patients, Fluvoxamin (Duphar) - 20 patients and Sertralin (Pfizer) - 30 patients. The other 34 patients were treated with TCA - Amitriptylin. The created database contains 42 formalized attributes (anamnes data, condition of patients and parameters of an expert estimation of the answer on therapy Hamilton Scale ($HAM\text{-}D_{17}$) reduction based on two databases were allocated - "responders" to TCA and "responders" to SSRIs. Medical statusmetry method (by Razorenov G.) was used. An expert model was developed for forecasting results on therapy SSRIs and TCA. It includes 9 information attributes: duration of disease, early postnatal harmfulments, premanifest subclinical affective oscillation, age of manifest, quantity transferred affective phases, provoking factors, depression with anxious features, additional symptoms in structure of phase, duration of urgent depression. Models were constructed by medical statusmetry method (error of model is 12.5%). 3 patients did not submit decisive rule in groups of patients receiving therapy SSRIs, in case of application TCA - 2 patients. Application of computer experiment allows us to proceed from the group to individual forecast.

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