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CEREBROLYSIN IN NEUROLEPTIC COMPLICATIONS

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Cerebrolysin (EBEWE Austria) has a neurotrophic and antioxidant effect an is used for treating protracted extrapyramidal complications resulting from neuroleptic therapy (tardive dyskinesia, parkinsonism). 30 patients with endogenous psychoses (schizophrenia, shizoaffective and affective psychosis) were studied who developed tardive dyskinesia (10 patients), parkinsonism (11 patients) and a combination of tardive dyskinesia and parkinsonism (9 subjects). During registration of neuroleptic complications, standard scales were used - ESRS, AIMS, SARS. Cerebrolysin was presecribed i.v. drop infusions every other day for 28 days. The dosage was made up 5.0-10.0. It was found to be equally effective in treating both tardive dyskinesia (responders 60%, partial responders 20%) and parkinsonism (responders 54% and partial responders 18%). Therapeutic efficacy decreased in combination tardive dyskinesia and parkinsonism (responders 22.2%, partial responders 44.4%). Treatment with cerebrolysin reduced extrapyramidal symptoms and reduced the severity of other adverse effects of the use of psychotropic drugs.

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AMYLOID β BINDING TO LIPIDS AND APOLIPOPOTEINS OF THE HDL

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It is known that in normal human plasma and cerebrospinal fluid the soluble form of Alzheimer's amyloid beta protein (sAβ) is complexed to high density lipoprotein (HDL) and that Aβ protein participates in lipid metabolism. Nevertheless it is not known to which HDL structural constituent sAβ is primarily bound. We report on further studies of the Aβ to HDL interaction in an *in vitro* system using biotinylated synthetic A 1-40 as an sAβ tracer and normal human plasma HDL. Purified HDLs were incubated with the peptide followed by the lipoprotein repurification by Size Exclusion (SE) HPLC. DSD/PAGE following by Immunoblot and N-terminal sequence analysis of the biotin-Aβ positive protein bands revealed that Aβ is bound to many apolipoproteins of the HDL, mainly apoA-I, apoA-II, apoE and apoJ. On the other hand, reconstituted protein free HDL also binds Aβ peptide and inhibits its aggregation, as intact HDL does. This was assessed by SE-HPLC, SDS/PAGE, Immunoblot analysis and ultrastructural electron microscopic studies of Aβ morphology and Congo Red staining for β amyloid fibrils. Aβ binding to both lipids and apoliproteins may modulate the peptide to HDL association and be particularly relevant for AB biochemistry and physiology.

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ANTICONVULSANTS NORMOTHYMICS: PROGNOSIS OF EFFICACY

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Secondary prevention of relapse in affective and schizoaffective psychoses still remains important because it involves the problem of reducing the considerable number of relapses and thus a high level of social functioning. Comparative efficacy of normothymic drugs and the different indications of this drug administration with consideration to the prognosis of prophylactic efficacy is uncertain and evidence suggests there is no direct correlation between serum concentrations of anticonvulsants and the clinical prophylactic efficacy. The object of the study was to find out early pharmacokinetic predictors of prophylactic efficacy of carmbamazepine (CBZ) oxcarbazepine (OCB) and sodium valproate (SV). 76 patients with affective or schizoaffective disorders between 18 and 62 years were examined. 30 patients received CBZ, 20- OCB and 16 - SV. Estimation of plasma concentrations of all three and their metabolites by high performance liquid chromatography took place in a one week interval in the first month of treatment and monthly during a year of follow-up. Plasma concentration of CBZ was an average of 6.6 ± 1.27 mkg, OCB - 0.189 ± 0.28 mkg/ml. No relationship was found between plasma concentrations and prophylactic efficacy but some positive and negative correlations were found with concentrations of their metabolites. These finds can be used for the prediction of individual prophylatic efficacy at the initial stages of therapy.

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ANTERIOR CINGULATE CORTEX IN SCHIZOPHRENIA: A STEREOLOGICAL STUDY

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In order to explore the role of the anterior cingulate cortex in schizophrenia, we performed a quantitative analysis of cortical thickness and neuron densities in 8 patients with schizophrenia (6 men, between the ages of 36 and 77 and 2 women aged 51 and 71 years), and 10 age- and sex-matched controls. Paraffin-embedded blocks from the anterior cingulate cortex were cut into 50 um thick sections. The total and laminar thickness and neuron densities were estimated bilaterally on cresyl-violet-stained sections (Terry et al, 1987). In particular, neurondensities were measured using the optical dissector, an unbiased stereological counting method implying that all regions within the structure of interest have an equal chance of being analysed (i.e., that there is no bias in sampling), and that counts do not depend on variables such as the size and shape of neurons. In patients with schizophrenia, the total cortical thickness of both left and right anterior cingulate cortex was decreased compared to controls (left: 1.8+0.07 vs 2.2+0.08, right: 1.9+0.1 vs 2.4+0.07, $p<0.01$). Laminar thickness in layer V of both left and right anterior cingulate cortex showed as significantly lower in schizophrenics compared to controls (left: 0.60+0.04 vs 0.73+0.02; right: 0.63+0.033 vs 0.85+0.04). There was a significant increase in neuron density in layer V of the left and right anterior cingulate cortex in patients with schizophrenia (left, 39722+2225 vs 32764+1869; right, 35757+1496 vs 29885+2046). After correction for cortical narrowing, no statistically significant differences were found in total and laminar neuron densities between the diagnosis groups. Consistent with previous observations, our data reveal a significant decrease of cortical thickness in the anterior cingulate cortex bilaterally in patients with schizophrenia compared to controls. This difference is not due to neuronal loss but might be the consequence of synaptic or neuropil alterations in the course of this disease.

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