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LIPID PEROXIDATION AFTER ELECTROCONVULSIVE THERAPY

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Objective: Until recently, there is no credible evidence that electroconvulsive therapy (ECT) causes structural brain damage. Nevertheless, the cognitive deficits associated with ECT raise the specter of brain damage, particulary in specific brain regions (i.e. hippocampus). Most recently, it has been hypothesized that ECT-induced memory dysfunction results from neuronal insults due to excessive release of excitatory amino acids and oxidative stress.

Methods: We investigated serum malondialdehyde (MDA) levels as an index of lipid peroxidation using a newly developed highly specific HPLC-method. We examined 6 patients with severe major depressive disorder (DSM-IV criteria) who did not response of treatment with various anti-depressants. These patients did not receive any drugs for at least 21 days before and during the series of ECT (exception: anaesthesia: thiopental, muscle relaxation: succinylcholine). Fasting serum samples for MDA-analyses were drawn 5 min before and 5 min, 60 min after ECT. MDA serum levels above 1.90 nmol/ml were considered pathological with respect to cerebral damage.

Results: MDA (nmol/ml) 5 min before ECT (mean values ± SE): 1.62 ± 0.14; after 5 min: 2.27 ± 0.19; after 60 min: 1.76 ± 0.11. Thus, samples from ECT treated patients 5 min after ECT were found to have increased amounts of MDA levels, which were normalized after 60 min ($p < 0.05$).

Conclusion: Although the number of the investigated patients is low, these findings might provide an additional role of oxidative stress in the pathogenetic mechanisms underlying the cognitive deficits associated with ECT. This model offers multiple testable hypotheses (i.e. pharmaceuticals strategies for oxidative stress prevention and treatment) which will be discussed.

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SALIVA METHANOL VALUES IN ALCOHOL WITHDRAWAL

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Objective: It has been suggested that serum methanol levels exceeding 1 mg/dl are highly suggestive of long-term alcohol intoxication and can be used as a marker for chronic alcohol abuse. Recently, it has been shown that saliva methanol values in subjects not suffering from chronic alcoholism were moderately correlated with breath and saliva ethanol values. Furthermore, a threshold saliva methanol value of >0.3 mg/dl has been defined possibly to indicative heavy drinking. However, no data are available about methanol levels in patients with alcohol dependence undergoing withdrawal from alcohol.

Methods: Serum methanol concentrations (SMC) and saliva methanol values (SMV) were assessed in 8 male chronic intoxicated. Fasting samples were taken at admission (D0), at the first (D1) and third day (D3) of withdrawal treatment. Saliva (0.2 ml) was sampled with the "salivette-tubes" (SARSTED) and the cotton swab was centrifuged 30 minutes afterwards. Methanol was analysed by routine Headspace-gaschromatography (Perking-Elmar 2000/Germany).

Results: Samples from patients with alcohol dependence were found to have significant increased amounts of methanol levels. Blood alcohol concentration at admission (BAC in promille): 1.98 ± 0.35. Mean values ± SE at admission: SMC (("normal range": <0.1 mg/dl): 1.52 ± .75 ($t = 4.89$, $p = .002$); SMV: 2.39 ± 1.41 ($t = 4.32$, $p = .003$). SMC and SMV steadily decreased during the observation period and were in a normal range within 24 hours.

Conclusion: Saliva procedures are valuable in determining high methanol presence in intoxicated patients suffering from alcoholism. The present findings are consistent with other observations. We would like to suggest further investigations whether saliva methanol values correlate with the severity of alcohol withdrawal symptomatology.

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THE SCHIZOPHRENIC COMMUNICATION SCALE (SCD): A MEASURE OF SCHIZOPHRENIC DISSOCIATION

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Background: Numerous authors try to define the cognitive dysfunction underlying the disorganisation syndrome specific to schizophrenia. Our hypothesis is that schizophrenic patients exhibit a deficit in integrative processes of contextual information and a lack of "theory of mind". Directly from this hypothesis, we propose a simple seven items scale for the assessment of Communication Disorders which are specifics to schizophrenic disorganisation. These items are directly the expression of the hypothesis of cognitive dysfunction: three concern the contextual information processes and four concern the "theory of mind".

Method: The conversational situations supposed to facilitate the emergence of these cognitive dysfunctions are define for each item. A guideline for assessment is added. Thirty schizophrenic patients were assessed to the SCD and to the Thought Language and Communication Scale proposed by Andreasen and scores were compared to others psychiatric patients.

Results: The SCD appears to be more sensitive and more specific than the TLC with a good inter judge consistency.

Conclusion: The Schizophrenic Communication Disorder Scale (SCD), directly conceived from our hypothesis of schizophrenic cognitive dysfunction, seems to be a useful implement to evaluate specific cognitive dysfunction which emergence is facilitated by conversational situations defined by the scale.

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