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EVENT RELATED POTENTIALS (CNV AND P300) IN CHRONIC OPIATE USERS: PRELIMINARY RESULTS

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a) Background: IV administration of flesinoxan (highly potent and selective 5HT1A full agonist) induces a significant and dose-dependant increase in ACTH, cortisol, prolactin (PRL), growth hormone (GH) and total neurophysins and a decrease in body temperature. There is no effect of age and gender on hormonal and temperature responses to intravenous flesinoxan. Effects of age and gender on neurophysins and temperature responses to flesinoxan was studied.

b) Design: Sample: 39 healthy volunteers (22 M, 17 F) - mean age 35.5 ± 10.7 years. Total neurophysins, vasopressin and temperature were assayed at 0, +15, 30, 60, 90 and 120 min after the injection of flesinoxan 1 mg/70 kg. We performed an ANOVA for the area tinder the curve relative (AUCr) values of temperature, total neurophysins, vasopressin and oxytocin responses, with age as a covariate.

c) Results: There was no significant difference between male and female neither for the AUCr values of temperature responses (−32.4 ± 22.1 vs −34.6 ± 29.3 °C min.) nor for total neurophysins (48.0 ± 133.9 vs 75.4 ± 148.2 pg min/l), vasopressin (18.3 ± 35.8 vs 19.0 ± 37.5 pg min/l) and oxytocin (29.8 ± 116.7 vs 56.5 ± 118.8 pg min/l) responses. Furthermore, there were no significant correlations between age and temperature (r = 0.06) or total neurophysins (r = 0.06), vasopressin (r = 0.008), and oxytocin (r = 0.07) responses neither among male nor female.

d) Conclusion: The sensitivity of 5HT1A receptors mediating neurophysins and temperature responses is neither age nor gender-related.

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REDUCED DOPAMINERGIC ACTIVITY IN DEPRESSED SUICIDES

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a) Background: Several data are available about the implication of the dopaminergic system in the control of inward-directed aggression. Previously, we suggested an involvement of D2-dopaminergic function in the expression of suicidal behavior by demonstrating a smaller growth hormone (GH) response to apomorphine, a dopaminergic agonist, in depressed patients with a history of suicide attempts in comparison to nonattempters. In the present study, the purpose was to analyze GH responses to apomorphine in depressive patients who later died by suicide.

b) Design: Sample: 8 male depressive inpatients who died by suicide within one year after hospitalisation, compared to 18 male major depressed inpatients who never attempted suicide. The two groups did not differ in mean age, weight or Hamilton Depression scores. Blood samples were collected at −20, 0, +20, 40, 60 and 120 min after injection of 0.5 mg apomorphine.

c) Results: Mean GH peak responses to apomorphine differed significantly between suicide completers and controls: for GH peak, 7.6 +/− 4.1 ng/ml vs 18.9 +/− 14.2 ng/ml, U = 30, Z = −2.33, p = 0.02.

d) Conclusion: Our results tend to confirm the role of dopamine in the biology of suicide in depression. However, this study should be controlled on a larger sample.

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THREE SYNDROMES OF SCHIZOPHRENIA – RESULTS OF AN EFA - CFA - CROSSVALIDATION APPROACH

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Background: Factor analyses of schizophrenic symptoms typically come to different solutions, although a model with three dimensions has been obtained with some consistency. We compared different factor structures of schizophrenic phenomenology in an unselected, hospital based patient sample.

Methods: Psychopathological symptoms were originally evaluated with the AMDP-system and afterwards grouped according to the SANS/SAPS-subscales. The sample included 1053 inpatients with schizophrenia. Exploratory and confirmatory factor analyses were conducted to test the hypothesis of one-, two-, three- and four-factor models.

Results: Results support a three-factor model, where dimensions can be termed as 'positive', 'negative' and 'disorganized' dimension, in line with previous reports. Factor loadings were not entirely pure in a sense, that two symptom subscales from the 'negative' factor also loaded on the 'disorganized' factor.

Conclusions: Despite mounting evidence, that schizophrenia symptomatology is not adequately represented by one or two dimensions, most studies continue to use such a simplified model. Forthcoming research should instead be based on three dimensions for the assessment of schizophrenia on a syndromal level beyond the nosological level, to reduce sample heterogeneity. However, further refinement of syndrome definition seems necessary, to obtain 'pure' dimensions.

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ALTERATIONS OF GAIT IN HEALTHY CONTROLS COMPARED TO DEPRESSED PATIENTS TREATED WITH TCA AND SSRI

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Objective: Parameters of human gait are important components of motor functioning controlled on the spinal level and by central motor programs. Gait patterns are altered in depression and may be associated with cerebral pathophysiology of the disease. In addition, tricyclic antidepressants (TCA), but not selective serotonin reuptake inhibitors (SSRI), may affect psychomotor dysfunction. Therefore, kinematic analysis of gait patterns was performed in patients with major depression treated with TCA and SSRI and in healthy controls.

Method: Spatial and temporal gait parameters including velocity, stride length, stance phase, and cadence, were measured in patients with major depressive disorder (MDD) (DSM-IV) (n = 18) treated with TCA and SSRI and in matched healthy controls. Depressive symptoms were documented by HAMD, Widlöcher Depressive Retardation Scale (WDRS), SHAPS-D and others.

Results: Spatial parameters including stride length (MDD: 146.42 ± 25.31 cm, CTRL: 159.65 ± 16.84 cm) and step length/leg length (MDD: 0.75 ± 0.06 cm, CTRL: 0.83 ± 0.10 cm) were decreased in patients. Temporal parameters including double limb support (MDD: 167.06 ± 30.54 ms, CTRL: 140.51 ± 20.60 ms) were increased and velocity (MDD 1.21 ± 0.13 m/s, CTRL: 1.48 ± 0.18 m/s) was decreased in patients, without significant differences in cadence (steps/min). Gait parameters in patients treated with SSRI were less altered than in the TCA group. Cadence correlated

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