Tues-P8

IMPROVEMENT OF NEUROCARDIAL VAGAL DYSFUNCTION AFTER SUCCESSFUL ANTIDEPRESSIVE TREATMENT WITH ELECTROCONVULSIVE THERAPY (ECT)

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Objective: To assess autonomic neurocardial function (ANF) in relation to response to antidepressive treatment with electroconvulsive therapy (ECT).

Methods: Using a standardised measurement of heart rate variability we prospectively evaluated ANF in 10 patients (m/w 3/7; mean age 44.1 years) with treatment refractory major depression (DSM-III-R) and in 26 age matched healthy controls. Exclusion criteria were cardiac, pulmonary or neurological diseases, thyroid disease, diabetes mellitus, alcoholism or drug dependence. ECT was applied using a Thymatron DG™ (methods see 1). Four patients took lorazepam during the course of ECT, the others were free from medication for at least four days. Patients were re-tested 24–48 hours following their last ECT. Artefact-free single ECG sweeps from a 5-min recording while resting were digitally stored using the software package "Neurodiag" (H. Lambeck, Munich; modified version of the ProSciCard).

Results: Compared to controls we found a significantly higher mean resting heart rate and a reduction of the mean coefficient of variance (CVr; $p < 0.005$) as well as of the root mean square of successive differences (RMSSDr; $p < 0.01$) in depressive patients. They also showed a marked reduction of the HF-power (spectral analysis). The response to ECT was excellent in five, moderate in three in poor in the other two patients. ECT resulted in a marked increase of the mean CVr and RMSSDr as well as in an increase of the mean HF-power ($p = 0.06$). Patients, in whom depressive symptoms did not clearly improve, showed either no change or a slight deterioration in each HRV-parameter.

Conclusion: In contrast to a recently published report by Schultz et al. 1997 (to the best of our knowledge the only one on this issue), our data suggest a marked improvement in ANF after successful treatment of depression with ECT.

(1) Agelink MW et al.: Benefit and risk of electroconvulsive therapy in medically ill patients of old age. Nervenarzt 1998; (in press).

Tues-P9

ADRENALINE AND IMIDAZOLINE DRUG-INDUCED PLATELET AGGREGATION IN PATIENTS WITH MAJOR DEPRESSION

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Major depression is associated with supersensitive α_{2A}-adrenoceptors (increased receptor density and aggregation) (1) and increased density of imidazoline receptors (radioligand receptor binding and immunodetection of receptor proteins) (2, 3) in blood platelets. The functions of imidazoline receptors (I_1-and I_2-type) in blood platelets are not known. The aim of this study was to compare the aggregation response induced by adrenaline (α_{2A}-adrenoceptor-mediated) with that of imidazoline drugs in control subjects (5 men, 6 women; age, 38 ± 4 years) and in patients (5 men, 4 women; age, 37 ± 3 years) with major depression (DSM III-R). Platelet aggregation was measured photometrically as described previously (1). Platelet counts were similar in control subjects (298 ± 35 × 1000/µl) and depressed patients (268 ± 24 × 1000/µl). In drug-free (more than 2 months) depressed patients, the primary aggregation response induced by adrenaline (0.01–100 µM) was potentiated, which indicated increased α_{2A}-adrenoceptor sensitivity (controls, slope, EC50: 1.07 ± 0.13 µM, aggregation, EC50: 0.67 ± 0.09 µM, n = 11; depressed, slope, EC50: 0.59 ± 0.07 µM, aggregation, EC50: 0.44 ± 0.05 µM, n = 9, $P < 0.05$). In control subjects, as well as in depressed patients, agmatine (up to 1 mM, n = 4), one endogenous ligand for imidazoline receptors, cirazoline (mixed α-adrenoceptor and $I_{\frac{1}{2}}$ imidazoline drug, up to 370 µM, n = 3), antazoline (mixed α-adrenoceptor and I_2 imidazoline drug, up to 370 µM, n = 6) and valldemossine (LSL 61122, selective I_2 imidazoline drug, up to 370 µM, n = 6) did not induce platelet aggregation. In contrast, moxonidine (0.1–370 µM), a putative selective I_1 imidazoline drug, induced a weak aggregation response (slope, EC50: 3.81 ± 0.81 µM; aggregation, EC50: 3.13 ± 0.95 µM, n = 3) that was antagonized by the selective α_2-adrenoceptor antagonist RX 821002 (1 µM, methoxyidazoxan), indicating that this drug behaves as an α_{2A}-adrenoceptor agonist on platelet aggregation. These preliminary results further support the existence of supersensitive platelet α_{2A}-adrenoceptors in depression, and suggest that the activation of platelet imidazoline receptors is not associated with the induction of aggregation, even when these receptors are abnormally up-regulated in major depression.

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(1) Garcia-Sevilla et al., Arch. Gen. Psychiatry 43: 51–57 (1986)
(2) Piletz et al., Arch Gen. Psychiatry 48: 813–820 (1991)
(3) Garcia-Sevilla et al., Arch. Gen. Psychiatry 53: 803–810 (1996)

Tues-P10

CHOLESTEROL AND SEROTONERGIC CONTROL OF PROLACTIN SECRETION IN DEPRESSED AND SUICIDAL PATIENTS

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In depressed and suicidal patients a reduced response of PRL and cortisol to *d*-fenfluramamine has been described. On the other hand a correlation between serum cholesterol levels and suicidal behaviour has been reported so that some authors considered the cholesterol to be responsible of the lower serotonergic activity. The aim of our study was to seek the correlation between cholesterol and central serotenergic activity in depressed patients and/or in suicidal behavior. Study participants comprised 18 subjects, 12 patients (mean age: 46.1 ± 15.5; M/F: 1/2) and 6 controls (mean age: 39.1 ± 9.5; M/F: 1/2). The *d*-fenfluramine challenge test was performed and the serum lipid and protein profile was evaluated in a blood sample, within 24 hours from the act fulfillment. Results showed a reduced total serum cholesterol in suicidal patients and a decreased response of PRL to *d*-fenfluramine challenge test in most depressed patients. The correlation between total serum cholesterol concentration and the PRL peak was not statistically significant. While the serotonergic activity was related to the severity of depression on the HRSD, serum cholesterol levels were related to the total serum protein, BMI, item 3 of HRSD and item 7 of Z-SDS.