during five days. Estimation of patients clinical state and their brain functional state were performed before and after treatment. Before treatment all patients had compiaints of severe headache, emotional instability, disturbances of sleep cycles and other. Results of neuropsychological investigations by Luria's method showed the following mental disorders: pathological exhaustibility, derangement of memory accompanied by deficit of voluntary attention. Emotional disorders were presented as dysphoria with elements of apathy. EEG power mapping demonstrated as a rule two types of maps. The first was characterized by a significantly higher than normal level of power especially in alpha- and theta-bands. The second demonstrated a lower than in controls values of power in most spectral bands. Intrahemispheric coherences were decreased in the left hemisphere and increased in the right in comparison with normals of the same age. After treatment all patients mentioned improving of the their state with reduction of headache and normalization of the sleep cycles. Neuropsychological investigations showed decrease of mental impairments. EEG mapping also demonstrated tendency to normalization of power and coherence parameters in all patients. During and after treatment patients had no side and undesirable effects. So, results of our investigations allow to recommend drug Nimotop for treatment of patients after irradiation. As it was shown in experiments in vitro and in neurosurgical patients, Nimodipine possess antioxidant properties [3] From this point of view one of possible mechanisms of Nimodipine action in our patients could be its scavenging effect on free radicals which had developed after irradiation.

References

[1] Zubovsky G.A. and Kholodova N.B. Neurological status of subjects who participated in liquidation of the Chernobyl power plant accident consequences (1993) Medical Radiology, 38, 31-34

[2] Zhavoronkova L.A., Kholodova N.B., Charchenko V. and Zubovsky G.A. (1993) EEG and metabolic mapping data correlate with neurological disturbances in patients from Chernobyl. Brain Topography, 5, 296.

[3] Demchuk M. and Amcheslavskyi V. Nimodipine in the treatment of brain edema in neurosurgical patients. (1994) XIXth Collegium Internationale Neuro-Psychopharmacologicum Congress. 175S

P-9-5 Heterogeneity of biochemical dysfunction in Gilles de la Tourette syndrome (GTS)

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Background: Specific concern about tardive side-effects linked to the chronic use of neuroleptics to control tics in GTS have led to alternative therapeutic trials.

Study design: A cohort of 25 post-pubertal GTS patients (DSM IIIr Criteria, 19 M, 6 F, age: 18 ± 3.2) was submitted to two alternate open trial of 2 months duration each with either fluoxetine, a 5HT reuptake inhibitor (10 to 20 mg once a day) or clonidine, an α_2 noradrenergic agonist, used at low-dose (0.025 mg once to thrice a day). 16 patients were considered to have a positive response (more than 50% reduction of tics) to fluoxetine alone (n = 8) or clonidine alone (n = 8). A retrospective analysis of anamnestic data, completed with a parent interview by a blind research assistant allowed to compare fluoxetine responders (F+) and clonidine responders (C+) for indirect indices of monoaminergic dysfunction.

Results: The two groups differed significantly ($p < 0.05$) for a family history of Mood disorder: F(+) had more cases of major depression or suicidal attempt or alcoholic dependence or seasonal affective disorder in direct ascendants. Furthermore, enuresis during childhood development, compulsions to touch, imitate and "just right" obsessions in the tic history and actual sleep disorders with early awakenings were overrepresented in F(+). C(+) tended to differ from F(+) by the occurrence of stressful events during maternal pregnancy, short-sleeper habits sleep and locomotor hyperactivity without significant learning disabilities during childhood.

Conclusion: Chronic motor tics with a vocal component may be the "final common clinical expression" of distinct biochemical dysfunctions, as inferred from differential responses to distinct pharmacological probes in young adults with GTS.

P-9-6 Pyritinol improves tardive dyskinesia in double-blind cross-over placebo-controlled trial

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Pyritinol (former chemical name pyrithioxine) is a scavenger of free radicals and central cholinomimetic agent (Benešová, Krejčí, and Pavlík, 1991). Its therapeutic efficacy in tardive dyskinesia (TD) was tested in a 12-week double-blind cross-over placebo-controlled trial. To our knowledge, pyritinol was used in the treatment of TD for the first time. Pyritinol (600 mg/day perorally) or placebo were administered for the 6 weeks after a one-week single-blind placebo period. The next 6 weeks the medication was reversed. The neuroleptic dosage was kept constant for the whole trial.

Twelve of seventeen psychiatric inpatients (5 men and 7 women, 11 diagnosed as schizophrenia and 1 as schizoaffective psychosis according to DSM-III-R) completed the trial. The withdrawal of 5 patients was unrelated to the experimental therapy. Mean age (±SD) of the patients group was 55.1 ± 13.8 years. TD was assessed from videotape by two independent raters, using the Abnormal Involuntary Movement Scale (AIMS).

Pyritinol was significantly better than placebo in improving TD as measured by the total score of the AIMS (ANOVA, df = 2; F = 5.20; P = 0.01). Simpson and Angus Rating Scale for Extrapyramidal Side Effects, Brief Psychiatric Rating Scale (BPRS) and Structured Adverse Effects Rating Scale (SARS) did not detect any treatment emergent symptoms on 5% level of significance.

Our results support both hypotheses on the importance of free radicals (Cadet et al., 1986) and central cholinergic hypofunction (Klawans, 1973) in TD development.

References

Benešová, O., Krejčí, I., Pavlík, A. (1991) Nootropic drugs. Avicenum, Prague, 47-49.

Cadet, J.-L. Lohr, J. B., Jeste, D. V. (1986) Free radicals and tardive dyskinesia. Trends Neurosci., 9, 1986, 107-108.

Klawans, H. L. (1973) The pharmacology of tardive dyskinesias. Amer. J. Psychiatry 130, 82-86

P-9-7 Therapeutic effect of diltiazem in tardive dyskinesia

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Introduction: Calcium channel-blockers are used primarily to treat hypertension, cardiac arrhythmias and angina pectoris. Since 1987, some reports have appeared in literature about using this group of drugs in the treatment postneuroleptic tardive dyskinesia. The various studies have yielded conflicting results as to efficacy in the treatment of tardive dyskinesia [1-3].

Methods: Place of study: out-patients and in-patients of the Institute Psgchiatry and Neurology Study design: double-blind, duration 4 weeks Investigated groups: treatment with diltiazem 60 mg/24 h, and for comprising group treatment with diazepam 10 mg/24 h. Evaluation: The Brief Psychiatric Rating Scale (BPRS Overall and Graham 1988), Clinical Global Impression (CGI), The Tardive Dyskinesia Rating Scale (TDRS Simpson and all 1979), side effects (ARI), physical examination, a routine blood tests, ECG. Assessment time: 0, 7, 14 and 28 days. Including criteria: schizophrenic patients (ICD — 10 diagnostic criteria) with postneuroleptic tardive dyskinesia. Excluding criteria: concurent somatic illness, alcohol or drugs abuse any psychiatric disorder, other than the target discorder.

Results: Characteristic of patients and results are summarized in Tables 1 and 2.

Table 1. Characteristic of study subject

Drug	Diltiazem	Diazepam
Number of patients	16	16
Out patient	9	14
In patient	7	2
Mean age (years)	43	49
Gender male	7	6
female	7	10
Maintenance neuroleptic therapy (years)	15.3 ± 2.9	12.6 ± 1.51
Duration of TD (years)	5.0 ± 0.8 range 4.92	10 ± 0.5 range 9.5
Symptoms of TD (before treatment) and number of patients	entire body - 16 facial and oral - 16 lower extr. - 15 upper extr. - 10 neck and trunk - 5	facial and oral - 16 lower extr. - 16 entire body - 14 upper extr. - 6 neck and trunk - 2
Concomitant medication	In consequence of chronic psychosis all the patients received antipsychotic	