

**Conclusion:** ECT can be useful in the therapy of schizoaffective disorder.

- (1) Miroslava Jasovic-Gasic. *Is ECT Efficient in Therapy Schizoaffective Disorders*. ECNP Congress, 1996.
- (2) Shapira B. et al. *Enhanced Serotonergic Responsivity Following Electroconvulsive Therapy in Patients with Major Depression*. British Journal of Psychiatry, 1992; 160: 223.

### Wed-P36

#### SOME PERSONAL EXPERIENCE OF PSYCHOTHERAPEUTIC AID ON THE EXAMPLE OF A PSYCHO-NEUROLOGICAL OUT-PATIENT CLINIC OF A MOSCOW DISTRICT

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From 1995 to 1996 more over 200 patients addressed to the out-patient clinic of the one of the regions of Moscow to get psychotherapeutic aid. All of them were examined beforehand by psychiatrist. After preliminary diagnostic test 108 patients (35 men and 73 women) were selected. The variety of age of the patients was from 19 to 63 years old, 22% of which were officially invalidated in connection with their mental disease. The data distribution table N 2 shows and compares the results of the work with the patients according to the psychiatric diagnoses (ICD-10) and psychotherapeutic methods applied. The methods of psychotherapeutic treatment accounted to:

1. Weekly individual conversations with psychoanalytic orientation per 50 minutes each. It included from 7 to 12 talks (22 persons).
2. Weekly hypnosis group sessions including some elements of assertiveness training (41 persons).
3. Psychocorrectional groups for communication in which patients searched some affinity (45 persons). In the group for affinity more than half of the persons had severe disorders, mainly schizophrenia.

**The Findings:** In the process of individual psychodynamic psychotherapy the patients' attitude was becoming smoother to the environment for they realized their inadjustability in behaviour as well as their character peculiarities. After attending the group of hypnosis neurotic symptoms of the patients disappeared to some extent and were not so vivid. For example, either they again managed to use Underground without any fear, or they manager (twice as less) to reduce their doses of tranquilizers and antidepressants taken. In the process of attending a serie of the group of affinity a patients' low self-appraisal and inferiority complex disappeared, but their search for affinity and emotional syntonia increased. We obtained the increase of the level of adjustability with 11 out 24 schizophrenic patients. I suppose that it should be urgent to arrange psychocorrectional groups as well as apply reinforcing psychopharmacologic therapy during the periods of remission of the patients that suffer schizophrenia provided there is a regional out-patient clinic.

### Wed-P37

#### VITAMIN E: AN ALTERNATIVE TO ANTICHOLINERGIC DRUGS?

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The degeneration of nigral neurons due to the oxidative formation of free radicals (Fr. R.) and the depletion of Fr. R scavenger enzymes, is the underlying process of neuroleptic induced pseudo-parkinsonism (Ps.P), as of parkinson (P.D). The phenotiazines form

Fr.R. intermediates, during their metabolism. Vitamin E. (V.E), protects cell membrane from damage, by Fr. R. since it attenuates the oxidation of unsaturated fatty acids (U.F. Ac). Our survey compared of V.E - effective in treating P.D - versus placebo, on neuroleptic treated schizophrenic patients' extrapyramidal side effects (E.S.Ef), in order to use it as a valid alternative to the anticholinergic drugs, of limited efficiency and of unpleasant and even harmful side effects.

**Method:** Thirty chronically hospitalized schizophrenic patients (16 male, 14 female), on the average 53 years old, after a two-week washout of neuroleptic and anticholinergic, were given haloperidol and V.E (2000 lu/Day) or matching placebo capsules, double-blind. After psychiatric and medical testing, patients were repeatedly (3 days after washout and then every 2 weeks, thrice) compared on two dyskinesia and E.S.Ef scales (SAS and AIMS). The study was monitored from outside and took about 7 month. 21 patients (11 on V.E, 10 on placebo) finished the trial. Dropout was due to persistent side effects.

**Results:** Using anova procedures no difference could be observed between both groups. The only exception was a strong trend on the AIMS between the two groups ( $F = 3.86$ ,  $DF = 13.15$ ,  $P = 0.16$ ).

**Conclusion:** V.E seems not to be effective in treating neuroleptic induced Ps.P in chronically hospitalized schizophrenic patients. The study although based on firm theoretical grounds did not support the hypothesis. The reasons may be, the small sample used, the possibility of the dopaminergic system of such long lasting patients, being damaged and an inappropriate dosage as no dosage changes were made in V.E during the trial.

### Wed-P38

#### THE DRUG PRESCRIPTION IN SCHIZOPHRENIA PATHOLOGY IN FRANCE

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From an épidémiological study realized in France during two resumptions in April 1995 and in April 1998 it is possible to have an analysis of modes practice concerning neuroleptic prescription in the schizophrenia on more of 1.000 files to each stop.

The analysis will focus on two aspects:

1. - characteristics of the neuroleptic processing and the other psychotropics processings associates.
2. - the evolution of these characteristics in 3 years considering the evolution of the references.

The population of schizophrenic patients seems to be distribute by an heterogeneous manner: there are 3 types of patients which can be differentiate by the drug treatment.

### Wed-P39

#### MEMBRANE PHOSPHOLIPID ABNORMALITIES AS A BIO-CHEMICAL BASIS FOR THE NEURODEVELOPMENTAL CONCEPT OF SCHIZOPHRENIA

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The neurodevelopmental hypothesis is supported by changes in brain morphology, by behavioural abnormalities prior to the development of overt schizophrenia, by the increased risk of schizophrenia associated with viral infections during pregnancy and with perinatal complications, and by an association with minor physical abnormalities. The hypothesis fails to account for the genetic basis

of schizophrenia or to provide a biochemical basis. Most of the observations which support the neurodevelopmental hypothesis can be explained on the basis of genetically determined abnormalities in membrane phospholipid metabolism which can be attenuated or exacerbated by various environmental events. The phospholipids provide the main structural basis for all neuronal membranes and influence the behaviour of all membrane-bound and membrane-associated proteins such as receptors, ion channels and ATPases. The phospholipids also modulate the actions of all neuronal cell signalling systems, and the fatty acids they contain provide many of the second messengers which are activated as a result of receptor occupancy. The phospholipids provide a unique site where genes and environment interact: basic phospholipid structure depends on the enzymes involved in their synthesis and breakdown, whereas those enzymes depend on the environment for the supply of the fatty acids which are major components of the phospholipids. The phospholipid hypothesis proposes that in schizophrenia there are reduced rates of incorporation into phospholipids and increased rates of loss from phospholipids of the long chain polyunsaturated fatty acids (LCPS) which make up about 20% of the dry weight of the brain. Because phospholipids are so important in the brain, such defects could lead to abnormal brain morphology and synaptic remodelling, abnormal susceptibility to viral infections and perinatal hypoxia, insults which are known to interfere with phospholipid metabolism, and the onset of overt symptoms around or soon after puberty when phospholipid metabolism is known to change. This concept provides novel proposals for improved treatment of schizophrenia.

### Wed-P40

#### P50 IN DEFICIT AND NON DEFICIT SCHIZOPHRENIA

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Sensory gating impairments have been found in schizophrenia, with prevalence estimates as high as 90%. Schizophrenic patients show a diminished suppression of the auditory - evoked P50 potential to the second (test) of two paired click stimuli as compared with control subjects. The P50 suppression measure was calculated as the ratio of the test amplitude to the conditioning amplitude (C/T ratio). In order to investigate the relationship between P50 suppression and negative symptoms, we have compared P50 measures between 19 deficit and 32 non deficit schizophrenic patients. The 51 patients were all neuroleptic-treated at the time of the testing and the Schedule for the Deficit syndrome (Kirkpatrick et al., 1989; Ribeyre et al. 1994) was used to classify patients as either deficit or non deficit subtypes.

P50 measures were obtained at vertex during a conditioning-testing paradigm.

P50 amplitudes, latencies and C/T ratio were similar for both deficit and non deficit subgroups. These results suggest that sensory gating impairment is not related to the clinical subtype of schizophrenia.

### Wed-P41

#### ACTIVE <sup>3</sup>H-DOPAMINE UPTAKE OCCURS IN PLATELETS AND IN ARTIFICIAL NEURONAL CELL LINES OF HUMAN ORIGIN BUT NOT IN LYMPHOCYTES

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As lymphocytes have been proposed as a peripheral model of dopamine reuptake in brain neurones, which might play a role in several psychiatric diseases, kinetic and pharmacological properties of <sup>3</sup>H-dopamine uptake by native human lymphocytes were investigated.

Our results suggest that the transport of <sup>3</sup>H-dopamine measured with lymphocytes after separation over Ficol-Paque™ or Percoll™ is mainly generated by platelets which are always part of freshly prepared lymphocyte suspensions.

The investigations were extended to well defined cell lines in order to compare the pharmacological properties of native and artificial cells without any influence of contaminating cells such as platelets in lymphocyte suspensions. The artificial cell lines MOLT-3 and EBV-transformed lymphoblasts were not able to transport <sup>3</sup>H-dopamine which is consistent with our hypothesis that native lymphocytes do not exhibit a dopamine uptake. The investigation of the human neuroblastoma cell line IMR-32 demonstrated a GBR-12909 and cocaine-sensitive specific transport of dopamine, whereas dopamine transport in platelets is performed by a imipramine-sensitive serotonin transporter.

Our results do not support the existence of a dopamine transporter in human lymphocytes and demonstrate the possibility of verifying experiments conducted with crude native cells by the use of artificial, but homogeneous cell lines.

### Wed-P42

#### INFLUENCE OF SEVERITY AND DURATION OF PSYCHOTIC STATE ON THE LEVEL OF MIDDLE MOLECULES

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It is known, that various toxic conditions are accompanied by increased level of middle molecules (molecular weight 500–5000 Dalton) in blood plasma. It is considered, that the increase of middle molecules (MM) concentration in plasma in schizophrenia proves endotoxiosis hypothesis.

It was studied the level of MM in plasma in schizophrenia. There was examined 14 patients: 10 patients with paranoid schizophrenia (F 20.0, ICD-10) and 4 patients with postschizophrenic depression (F 20.4). Patients with organic or somatic pathology were excluded.

In schizophrenic patients with acute episode, characterized by severe paranoid and hallucinosis symptomatology (6–7 CGI score, less than month duration) the MM plasma concentration has exceeded 3–4 time the normal level. In psychotic patients with less severe states (4–5 CGI score) the duration of which was more than month, the MM concentration was less increased and exceeded the normal level by 1.5–2 times.

In all patients with postschizophrenic depression (3–4 CGI score, state duration more than month) the MM level did not exceed norm.

Control MM level was  $0.45 \pm 0.06$  g/l.

It was found the tendency to negative relationship between MM plasma concentration and duration of psychotic symptomatology and positive relationship between MM plasma concentration and severity of psychotic symptomatology.