

RESISTANT DEPRESSIONS — CONCEPT AND THERAPY APPROACH

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Concept: Resistant depressions are those cases that 12 weeks after peroral drug treatment remain refractory.

Phenomenological forms of the so called anxious-agitated and hostile patients were treated with sedative antidepressive agents for a period of 12 weeks, per os, and inhabited-aphatic and abulic patients with peroral administration of stimulative antidepressive agents during the same period. A total of 36 patients were treated in a five year period (1990–1995), which is 13.3% of the total number of depressive patients. Those were patients from the category of psychotic depression, with clear data on heredity, changed electropsychological profile of sleep, manic ideas, with the phenomenon of false background, suicidal attempts, associated disease, avoiding daylight and surroundings from eye sight.

Therapy approach: All those patients with the diagnosis of resistant depression following 12 weeks of peroral medication were administered infusions with antidepressive agents. Clomipramine and maprotiline were given in optimal therapy doses 5 times per week with a total of 15 applications per patient.

Summary: According to our opinion the basic infusion therapy with the aforementioned antidepressives is potentiated with additional levomepromazines, that is sulphides and further introduction of carbamazepines, bromocriptines and thyroxines. There were less patients for electro-convulsive therapy following this medication.

BIORHYTHMS, AFFECTIVE DISORDERS AND THE VULNERABILITY/STRESS THEORY. A THEORETICAL MODEL AND DIRECTION OF RESEARCH

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In recent years, it became clear that chronobiology will play a very important role in the medical practice and in psychiatry, too. Recent researches showed the importance of endogenous clocks in psychiatric practice, with a real "natural experiment," the seasonal affective disorders (Rosenthal 1988).

The author creates a heuristic model by connecting the vulnerability/stress theory with the imbalance of endogenous clocks; he identifies some possible markers of so called "biorhythms vulnerability": subclinical sleep/wake disorders, premorbid cognitive altered responses at exogenous zeitgerbers (including social ones) and biological — "energetic" ones from the perspective of the Traditional Chinese Medicine.

The author indicates three main directions of research:

1. To clearly identify the "biorhythmic vulnerability" markers with biological and psychometric methods.
2. To analyze the reliability of these markers.
3. Using the identification in prevention

NEUROENDOCRINE ABNORMALITIES IN BIPOLAR MANIA AND MAJOR DEPRESSIVE ILLNESS

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Bipolar mania and depression have long been considered mirror opposites of each other. However, from a neuroendocrine perspective they appear to share many similarities, which include hypercortisolaemia and non-suppression of plasma cortisol in response to dexamethasone. We have carried out a series of studies examining the

somatotropic axis and the serotonergic system in patients suffering from bipolar mania. Growth hormone (GH) plasma concentrations reflect a balance between the inhibitory effects of somatostatin and the stimulatory effects of GH-releasing hormone. Pyridostigmine, a cholinesterase inhibitor, increases cholinergic activity and in healthy volunteers increases GH secretion. Major depression is characterized by augmented pyridostigmine/GH responses indicating overactivity of central muscarinic function. We have found that the same is true of patients with bipolar mania. Acute administration of dexamethasone, in normal controls increases GH release by inhibiting somatostatin-ergic activity. However, major depressives and bipolar manics have blunted dexamethasone/GH responses indicating a defect at the site of the glucocorticoid receptor (dexamethasone's principal site of action). In order to determine whether these findings were true of non-somatotropic axes, we performed d-fenfluramine/prolactin (PRL) tests in patients with major depression and bipolar mania. Both sets of subjects had blunted d-fenfluramine-induced PRL responses indicating subsensitive serotonergic function. Even though bipolar mania and major depression are phenomenologically different, they appear to have certain neuroendocrine abnormalities in common.

ENDOGENOUS VS NEUROTIC DEPRESSION: IS THE ICD-10 CONCEPT OF RECURRENT AFFECTIVE DISORDER TENABLE?

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Objective: To determine the validity of the ICD-10 concept of affective disorders, we investigated 55 patients originally hospitalized with "neurotic depression" (n = 25) or "endogenous depression" (n = 30) as diagnosed according to ICD-9 criteria and the Newcastle Scale. All 55 patients fulfilled the criteria for the ICD-10 (F33) diagnosis of "recurrent depressive disorder".

Methods: All patients were tested for personality traits and social integration applying the following instruments: The Mannheim Interview for Social Support, Social Adjustment Scale, Inventory of Interpersonal Problems, the Five Factor Inventory (FFI). These were supplemented by the Symptom Check List 90 Revised (SCL-90-R), the Composite International Diagnostic Interview, and the questionnaire for assessing Motivation to Seek Psychotherapy (MSP). The ICD-9 diagnoses of neurotic and endogenous depression were corroborated using the Newcastle Scale. The t-test and Kolmogorov-Smirnov test were used to test sample means for significance.

Results: Three of the 7 instruments revealed slight to moderate dissimilarities between the two groups: Significant differences were found in 2 of the 9 categories ("aggressiveness" and "psychoticism") of the SCL-90-R, in 2 of the 4 MSP categories ("patient assessment of etiology" and "general expectations regarding therapy"), and in 1 of 5 subscales ("neuroticism") of the FFI.

Conclusions: Although our results tend to support the validity of subsuming "endogenous depression" and "neurotic depression" under the ICD-10 category of "recurrent affective disorder", the 2 groups did exhibit specific differences in personality traits, types of symptom, and motivation to seek psychotherapy.