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AMANTADINE AND DEPRESSION

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Since its introduction for the treatment and prophylaxis of influenza infection in 1963, amantadine has also been proved effective in a variety of neuropsychiatric disorders: it not only alleviates rigor, tremor and bradykinesia in drug-induced Parikinsonism and Parkinson's disease, but also improves vigilance in the latter disorder as well as in patients having suffered traumatic head injury and dementia processes. It improves fatigability in multiple sclerosis and has been used successfully in cocaine withdrawal.

Effects of amantadine on symptoms of affective disorders have been demonstrated in several trails administering it for varying purposes. Additionally, animal studies as well as clinical trials in men have shown a direct antidepressant activity of amantadine, which is presented here.

Amantadine appears to work through several pharmacological mechanisms, of which none could be identified as the one chief mode of action. It is a dopaminergic, noradrenergic and serotonergic substance, blocks monoaminoxidase A and NMDA-receptors and seems to raise beta-endorphin/beta-lipotropin levels. Amantadine has also recently been shown to have antiviral effects on Borna disease virus, which is suspected to possibly cause depressive disorders. All of these actions could constitute an antidepressant property, and it is suggested that amantadine might work as an antidepressant not through one, but through several mechanisms thought to be related to antidepressant activity.

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AMANTADINE TO AUGMENT ANTIDEPRESSIVE MEDICATION IN BORNA DISEASE VIRUS (BDV) INFECTED PATIENTS WITH AFFECTIVE DISORDERS

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Amantadine is known to have multiple pharmacological effects on the CNS, e.g., influence on the dopamine-, noradrenaline-, serotonin-system and NMDA-receptors. In addition, immune modulating as well as antiviral actions have been described. Amantadine was therefore mainly used for the therapy and prophylaxis of influenza infection, Parkinson-syndromes and to improve vigilance in disordered patients. The use of amantadine in the therapy of depressive disorders was also described, but high-lighted by the discovery of its antiviral effect on Borna Disease Virus (BDV), which may be causally related to subtypes of affective disorders.

BDV is well known as pathogenic in certain animal species. Symptoms in infected animals range from inapparent or subclinical manifestations to fatal neurologic disorders with neurobehavioral and/or emotional disturbances.

Psychiatric diseases were considered to be potentially associated with human BDV infections, since BDV-antibodies were detected in humans, and, recently, human strains of BDV were isolated from patients with recurrent mood disorders. However, the knowledge of the influence of BDV on the development and progress, e.g., of depressive episodes is still limited.

This report focuses on the use of amantadine to augment antidepressive therapy in BDV-infected patients (n = 20) with depressive episodes in bipolar and major depressive disorders with a special emphasis to clinical experiences.

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BIPOLAR II VERSUS UNIPOLAR ATYPICAL DEPRESSION IN PRIVATE PRACTICE OUTPATIENTS

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Objective: Differences between bipolar and unipolar atypical depression are not well studied. Aim of this study was to compare bipolar II with unipolar atypical depression in private practice outpatients

Method: Consecutive atypical depressed outpatients were interviewed with the Comprehensive Assessment of Symptoms and History, the Montgomery Asberg Depression Rating Scale, and the Global Assessment of Functioning scale.

Results: No significant differences were found between bipolar II (n = 38) and unipolar (n = 23) atypical depression on age at baseline/onset, gender, duration of illness, severity, psychosis, comorbidity, chronicity, and recurrences. Findings disagree with some reported differences between bipolar disorders and unipolar disorder (age at onset, female/male ratio, recurrences, comorbidity). Results support the view, based mainly on the better response to MAOI than to TCA, that atypical depression may be different from other forms of depression.

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BIPOLAR VERSUS UNIPOLAR PSYCHOTIC DEPRESSION IN PRIVATE PRACTICE OUTPATIENTS

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Objective: To compare bipolar with unipolar psychotic depression in private practice outpatients.

Method: 48 consecutive psychotic depressed outpatients were interviewed with the Comprehensive Assessment of Symptoms and History, the Montgomery Asberg Depression Rating Scale (MADRS), the Global Assessment of Functioning (GAF) scale, and the Brief Psychiatric Rating Scale (BPRS).

Results: 43.7% had bipolar I/II disorder, 56.2% had major depressive disorder. Of the variables investigated (age, duration of illness, severity, recurrences, atypical features, chronicity, gender, comorbidity, hallucinations, delusions) only depression severity, measured by MADRS and BPRS, was significantly greater in bipolar than in unipolar psychotic depression.

Conclusions: Bipolar psychotic depression was similar to unipolar psychotic depression on variables reported to distinguish bipolar from major depressive disorder (age, gender, recurrences, atypical features, comorbidity).

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CLINICAL EXPERIENCE WITH GABAPENTIN IN PATIENTS WITH BIPOLAR DISORDER: RESULTS OF AN OPEN LABEL STUDY

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Objectives: This study was carried out to evaluate the efficacy, tolerability and safety of gabapentin (GBP) used as adjunctive treatment in patients with bipolar disorder with acute symptoms and used as unique mood-stabilizer as prophylactic treatment.

Methods: Fifty patients fulfilling DSM IV diagnostic criteria for bipolar disorder underwent a 24-week, open trial treatment with

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GBP. Symptoms severity was measured using the Clinical Global Impression Scale (CGI) and the Brief Psychiatric Rating Scale (BPRS), the prophylactic effect was classified into one of three categories: complete, partial or no response.

Results: 20 patients were displaying acute symptoms, 30 patients were in euthymic state and had to discontinuate the precedent treatment with lithium because of severe side effects. At the end of the study, 75% of the 43 patients treated with GBP for at least 24 weeks, had a positive response, as measured by changes in the CGI and BPRS scores. The prophylactic effect was complete for half of the euthymic patients. The average dose used was 900 mg. The only side effect observed was oversedation, decreasing with continuing treatment.

Conclusions: GBP was effective both in acute and maintenance phase treatment of patients with bipolar disorder. If confirmed in controlled studies, these findings suggest that GBP represents a well-tollerated, rapidly acting antimanic agent and mood stabilizer.

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Mon-P69

THE NIACIN SKIN PATCH TEST AS A DIAGNOSTIC AID IN PRIMARY CARE PSYCHIATRY

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The diagnosis of schizophrenia and manic depressive psychosis is at present made according to clinical criteria and by excluding organic brain disease. Oral nicotinic acid in doses of over 2 mg/kg produces in normal subjects marked skin flushing of the face and upper body, due to formation of prostaglandin D2 (PGD2) from arachidonic acid (AA) in dermal macrophages, but about one-third of all schizophrenic patients fail to flush one hour after 200 mg doses. For schizophrenic patients chosen for negative symptoms, absence of flushing rises to 50 per cent (Glen et al, 1996). Oral niacin can give rise to unpleasant skin flushing and recently Ward et al (1997) have described a skin patch technique which gives better separation between schizophrenia and other conditions. New data which we will present indicate separation between schizophrenia and bipolar manic depressive illness ($p < 0.01$ to $p < 0.001$, depending on time interval between application of patch and reading skin redness, and molar concentration of the test solution (methyl nicotinate)). Further work is in progress to evaluate the usefulness of the test as a diagnostic aid in primary care and in community mental health care.

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(2) Ward PE et al. (1997) *Schizophr. Res.* - in press.

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CHRONOBIOLOGICAL MODEL OF MOOD STABILIZERS EFFECT

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Polysomnographic effects of acute and long-term use of different compounds with normothymic activity were analysed in 23 patients with rapid cycling bipolar disorder. 7 of them were treated with lithium carbonate (LC), 9 - with carbamazepine (CRB) and 5 - with sodium valproate (SV). Sleep registration was done before treatment (after 2 weeks wash-out period), in 3–5 days, in 2 weeks and after 3 months of treatment. LC from first days inhibited REM-sleep and later activated slow waves sleep (SWS), on the contrary anticonvulsants rapidly stimulated SWS and secondary inhibited REM-phase. After long-term use all drugs had resembled effects on sleep characteristics. They inhibited activity of REM-sleep including prolongation of REM-latency, restored SWS and normalised ultradian distribution of sleep cycles during the night. Chronobiological model of rapid cycling bipolar disorder and mood stabilisers action have been proposed to explain the results.

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CHARACTERISTICS OF MIXED AND PURE MANIA IN BIPOLAR DISORDER WITH PSYCHOTIC FEATURES

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We investigated whether mixed mania, also called dysphoric or depressive mania, was phenomenologically distinguishable from pure mania in a cohort of bipolar patients with psychotic features. The method of this study has been described in detail elsewhere[1]. Eighty-nine consecutively hospitalized patients with current mixed and manic bipolar psychotic symptoms were included in this study. Index episode psychotic diagnosis and psychiatric comorbidity were assessed using the Structured Clinical Interview for DSM-III-R (SCID-P). Psychopathology was assessed by the Brief Psychiatric Rating Scale (BPRS) and the Hopkins Symptoms Checklist (HSCL-90). Awareness of illness was assessed with the Scale to Assess Unawareness of Mental Disorders (SUMD). Of the 89 DSM-III-R bipolar I patients with psychotic features, 61 (75.2%) had a pure manic episode and 28 (24.8%) had a mixed manic episode at the time of the admission. Among sociodemographic characteristics, unemployment status was found to be significantly more frequent in mixed mania group than in pure mania group (82.1% vs 57.4%, $p < .05$). Age of onset of bipolar disorder was earlier in mixed mania group than in pure mania group (22.6 ± 5.6 years vs 25.0 ± 7.7), but this difference was not significant. Obsessive-compulsive disorder comorbidity was found to be significantly more associated with mixed mania than with pure mania (21.4% vs 6.6%, $p < .05$). At the BPRS, grandiosity (3.7 vs 2.3, $p < .01$), unusual thought content (4.9 vs 3.7, $p < .02$), excitement (4.1 vs 2.5, $p < .01$), conceptual disorganization (3.4 vs 2.7, $p < .01$) and activation (2.7 vs 2.1, $p < .01$) were more frequent in pure mania than mixed mania group; conversely, motor retardation (1.9 vs 1.3, $p < .02$) and factor 'anergia' (1.5 vs 1.8, $p < .03$) were significantly more frequent in mixed mania than in pure mania group. At the HSCL-90, only the factor 'psychoticism' was found to be more frequently associated with mixed mania than pure mania (1.1 vs 0.7, $p < .05$).

Conclusions: Patients with pure mania are likely to present more severe psychomotor symptoms and thought disorders, while those

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