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IMPROVEMENT OF C-L SERVICES IN EUROPE

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Past: There has been a heated debate whether or not C-L psychiatrists should focus on a consult or a liaison model. Most literature has been produced by university hospitals and is not representative for the field.

Current state: The European C-L Workgroup Collaborative Study (ECLW CS) — a health service study in 56 European hospitals (both university and non-university hospitals) across 11 countries (MR4*-340-NL)- reported an average consult rate of 1% and almost non liaison activities. Consequently, C-L psychiatrists did not get their message across. From a quality perspective, this needs improvement.

Future directions: In the framework of the Biomed program there is a study focusing on the development and testing of a quality management system for C-L services (BMH1-CT94-1706), another one will produce a risk prediction instrument for complexity of medical, nurse and organisational care during hospital admission allowing for a more appropriate referral mechanism (BMH1-CT93-1180). In the Netherlands the national development of general hospital psychiatry has been supported by the government through a report called: "Beyond borders." It includes recommendations for hospital-wide guidelines for the approach towards for instance attempted suicide, confusion and alcohol abuse to be implemented through active participation of psychiatrists in general hospital staffs. This program has been inspired by guidelines of the UK Royal College of Physicians and Psychiatrists joint workgroup on the psychological care for the medically ill. Currently the feasibility of specific teaching programs for ward staffs provided by and supported with clinical C-L nurse services. All these efforts have been the result of national and international collaboration. These programs will improve the future quality and effectiveness of C-L service delivery. This will be reported in detail.

GUIDELINES FOR MANAGEMENT OF DELIRIUM: A CRITICAL REVIEW OF CLINICAL PRACTICE

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Delirium is defined as a transient and fluctuating organic mental syndrome of acute onset, characterized by a global impairment of cognitive functions, a reduced level of consciousness, attentional abnormalities, increased or decreased psychomotor activity, and a disordered sleep-wake cycle. Abnormalities in every aspect of the mental state have been found. The clinical presentation of delirium may vary considerably from patient to patient and in a given patient over a 24-hour period. Delirium is associated with higher mortality and complication rates, poor functional recovery and longer lengths of stay. The management of delirium is twofold: first, adequate treatment of the underlying causal factor(s) and second, symptomatic measures including psychological interventions, good nursing care and psychotropic medication. Of course, a correct diagnosis of delirium and its etiology is crucial. Symptomatic management of delirium is particularly based on clinical experience, since no systematic research has been done on the effectiveness of different interventions. Psychological measures and good nursing care include: providing a quiet, familiar, safe and supportive environment; avoiding extremes of sensory stimulation and information inputs; reorienting the patient on a regular basis; treating the patient in a calm, clear and reassuring way; close monitoring of the patient's mental state and behavior; and, in case continuous nursing care or attendance of a familiar person cannot be provided, employing physical restraints may be necessary to prevent (self) damaging behavior. The use of psychotropic medication in delirium is often necessary. Short-acting benzodiazepines are effective in the treatment of alcohol withdrawal delirium and hepatic encephalopathy, and may be used to ensure sleep in delirious patients. Haloperidol is the drug of choice for the treatment of agitation, psychotic symptoms and anxiety in delirium. It is advisable to provide adequate information and aftercare for the patient and his family and prevent posttraumatic (= delirium) stress symptoms.

S16. Continuum of spontaneous 'tardive' dyskinesia in schizophrenia

Chairmen: S Lewis, E O'Callaghan

THE RELATIONSHIP BETWEEN NEGATIVE SYMPTOMS AND TARDIVE DYSKINESIA IN SCHIZOPHRENIA

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Much of the efforts to determine the aetiology of tardive dyskinesia have been aimed at elucidating the relationship with symptomatology and drug treatment. Review of the literature demonstrates evidence of relationships with many clinical symptoms of schizophrenia but the most consistent findings have been with the 'negative' symptoms of the illness. Many studies, however, report associations with overall negative symptoms whereas the relationship may be more complex.

Results of a study of 185 patients with schizophrenia demonstrate an increase in overall negative symptoms in patients with dyskinesia compared to those without. This finding is confirmed by a stepwise regression procedure incorporating the effects of other parameters, such as drug treatment. However, the relationship does not appear to hold for certain aspects of what are assessed as negative symptoms, in particular affective blunting. The data from this study do not suggest a relationship with overall cognitive function. Thus the relationship seems to lie more with aspects of social dysfunction.

ABNORMAL MOVEMENTS IN NEVER MEDICATED NIGERIAN AND INDIAN SCHIZOPHRENIC PATIENTS

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242 Nigerian schizophrenic patients, mean age 42 years, were examined for dyskinesia, using the Abnormal Involuntary Movements

Scale (AIMS). 12 patients had never received antipsychotic medication and none had dyskinesia. Dyskinesia was found in 10–45% of patients who had received medication.

308 elderly individuals in Madras, India, were also examined for dyskinesia, using the AIMS. Dyskinesia was found in 15% of normal subjects (N = 101, mean age 63 years), 15% of first degree blood relatives of younger schizophrenic patients (N = 103, mean age 63 years), 38% of never medicated patients (N = 21, mean age 65 years) and 41% of medicated patients (N = 83, mean age 57 years).

We conclude that dyskinesia in elderly schizophrenic patients is an integral part of the illness and not associated with antipsychotic medication.

Results from a one-year follow-up of the 21 never treated patients will also be presented.

COMPARATIVE STUDIES OF ABNORMAL INVOLUNTARY MOVEMENTS IN NEVER-TREATED VS TREATED POPULATIONS WITH SCHIZOPHRENIA

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There is an important question to answer: is tardive dyskinesia due to neuroleptics only, or does schizophrenia itself represent a risk factor for these abnormal movements? A number of studies have been conducted in Casablanca to answer this question, some of them in collaboration with the Department of Psychiatry of Portland, Oregon, USA.

Some preliminary results of a ongoing study can be presented:

Methods and patients: 61 never medicated schizophrenics (G1), 45 treated schizophrenics (G2), and 25 normal controls (G3) were included, matched for sex, age and duration of illness. The mean age for the 3 groups was G1: 29.8 ± 6.5 years; G2: 28.9 ± 5.7 years; G3: 28.6 ± 3.7 years.

The mean duration of illness for G1 was 6 ± 5.0 years; G2: 5.3 ± 3.9 years.

The clinical assessment used the Abnormal Involuntary Movement Scale (AIMS), Each examination was videotaped and assessed in two ways: open and blind.

Results: The mean global score of AIMS for the open assessment was for G1: 3.5 ± 2.7, for G2: 3.2 ± 3.5 and for G3: 0.4 ± 0.7.

For G1, there was a positive correlation with age. The abnormal movements observed were firstly in the limbs, followed by the orofacial area, and by the trunk.

THE LONGITUDINAL ASSOCIATION OF COGNITIVE DYSFUNCTION WITH TARDIVE DYSKINESIA IN SCHIZOPHRENIA

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Though the subject of much controversy in a historical context, there is an increasing body of contemporary evidence that in schizophrenia the contribution of spontaneous, disease-related involuntary movements to tardive dyskinesia appears to have been underestimated. We have been studying correlates of such movement disorder among features of the illness in neuroleptic-treated populations. In younger outpatients, we found those with orofacial dyskinesia to evidence an increased ratio of minor physical anomalies of the head to those of the periphery, indicating an association with a relative predominance of early craniofacial dysgenesis, and greater neuropsychological impairment on frontal lobe testing; severity of movement disorder was associated both with this anomalies distribution index and with extent of neuropsychological impairment. In older inpatients followed longitudinally over 10 years, those with persistent orofacial dyskinesia showed poorer function in more basic cognitive domains than did those consistently without such movement disorder, though in neither group did that function change over the decade; the only patients to show significant deterioration in these cognitive domains were those evidencing the *de novo* emergence of orofacial dyskinesia, and this deterioration occurred only over the time-frame in which their dyskinesia developed. Orofacial dyskinesia emerging during long-term neuroleptic treatment in schizophrenia appears intimately related to features of the illness for which that treatment was prescribed; it would seem to reflect, at least in part, the neuroleptic-induced precipitation or enhancement of motor patterns intrinsic to the disease process.

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S17. Special aspects in the treatment of opioid addicts

Chairmen: M Gastpar, P Baumann

NEW ASPECTS IN THE PHARMACOKINETICS AND METABOLISM OF METHADONE

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Methadone (MTD) is a racemic drug, but (R)-MTD accounts for nearly all opioid effects of the racemate. However, most countries have introduced racemic MTD for maintenance treatment of opioid addicts. Plasma level monitoring of MTD has been introduced, but an optimal concentration range cannot be agreed upon, as almost all studies did not measure the plasma levels of the enantiomers. In situations of comorbidity, such as depression, comedications may be necessary, but little is known of their interactions with the stereoselective metabolism of methadone and their clinical consequences. We have conducted several studies on the metabolism of MTD in patients:

A. In Germany, until recently, only (R)-MTD has been used. For economic reasons, the racemic form is presently being introduced into this country. In a collaborative study, 22 patients under (R)-MTD treatment were switched to a double dose of (R,S)-MTD. Under racemic treatment, the (R)/(S)-ratios ranged from 0.63 to 2.40, and there was a significant decrease ($p < 0.005$) in the mean serum concentration/dose ratios of the active (R)-enantiomer before and after the change was measured (3.97 vs 3.33). This suggests self-induction of MTD metabolism, as already observed during maintenance therapy with racemic methadone. As a consequence, this may necessitate, in some patients, a dose adjustment.

B. 6 and 7 addicts treated with racemic MTD were comedicated with fluvoxamine and fluoxetine, respectively. Fluvoxamine (50–250 mg/day) addition resulted in a significant increase in the plasma concentrations of both enantiomers, while only those of (R)-MTD were increased by fluoxetine (20 mg/day). These results suggest that CYP2D6 (inhibited potently by fluoxetine) preferentially metabolizes (R)-MTD, and CYP1A2 (inhibited by fluvoxamine) contributes to the metabolism of both MTD enantiomers.