

groups (1) Responsive to monotherapy. (2) Responsive to second line treatment. (3) Poor response to second line treatment.

Results: Lateral ventricular enlargement, and dilatation of the sylvian fissure was associated with poor response to monotherapy, but only infratentorial atrophy was associated with poor response to second line treatment. The neuropsychological impairment of the resistant group was most pronounced in tests of speed, and perseveration.

Conclusion: In late life depression the response to treatment, is adversely affected by organic brain abnormalities, characterised by diffuse atrophy, mental slowing, and impaired frontal lobe functioning.

WHAT ARE THE FACTORS INFLUENCING PRESCRIBING OF COGNITION ENHANCERS

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To discover 1., to what extent patients' wishes and the extent of any abnormality of brain performance influence the frequency with which cognition enhancers (CE; in Germany mainly Ginkgo biloba, nimodipine, piracetam, secale alkaloids, xanthine derivatives) are prescribed and 2., the medical practitioners' expectations of the effectiveness of such medications, we performed a representative survey (145 family physicians (FP), 14 primary care neuropsychiatrists (NP); response rate 83.2%) in southern Lower Saxony.

Two different written sample case histories were presented to these physicians in a face-to-face interview. Case one described a slight unspecific memory and concentration problem in an otherwise healthy 70 y old woman with or without expressed wish for medication, case two a moderate dementia of Alzheimer's type (Version B) or vascular type (Version A).

Regardless of the wish of the patient and type of the abnormal brain function about 70% of all participating doctors would prescribe those drugs, even though about 56% had doubts about their effectiveness. About 28% expected a positive effect on brain performance. A nearly equal proportion of doctors would continue an existing CE-drug regimen as would prescribe one.

In conclusion, the prescription of CEs is influenced less by medical criteria than by factors which concern doctor-patient relationship.

NR10. Schizophrenia: psychopharmacology and neuropsychology

Chairmen: K Aitchison, A Mortimer

VISUAL FORM PERCEPTION IN SCHIZOPHRENIA: FURTHER EVIDENCE FOR A DISORDER OF SEMANTIC MEMORY

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A considerable literature has documented that patients with schizophrenia perform poorly on tests of visual perception, particularly those involving recognition of complex forms and judgements of facial expression. Left unanswered by this work is the question of

whether such deficits are neuropsychologically specific or whether they merely form part of a general pattern of poor performance in schizophrenia.

We examined aspects of visual object perception in a group of 40 patients meeting DSM IV criteria for schizophrenia. All patients were assessed using the Visual Object and Space Perception Battery (VOSP) of Warrington and James (1991), which includes tests sensitive to different kinds of visual impairment seen in neurological patients. Patients who showed poor performance in one or more aspects of the tests were subjected to more detailed 'single case' analysis using a battery of additional tests distinguishing different levels of visual analysis. All patients were also administered tests of IQ and general intellectual function.

In the group screening phase of the study the patients showed a low frequency of poor performance on basic level 'presemantic' tasks such as identifying shapes from fragmented outlines. Those who failed tended to show overall intellectual impairment. Substantially worse performance was observed with tests requiring higher level form representation, especially when these required semantic information, eg naming objects.

Single case analysis supported the view that the bulk of the deficit in schizophrenia is found on tasks which require semantic level analysis and presemantic aspects of visual analysis are spared or relatively spared in the disorder.

LONG-TERM RELAPSE PREVENTION WITH CLOZAPINE IN SCHIZOPHRENIA

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The atypical neuroleptic clozapine has superior antipsychotic efficacy in patients refractory to treatment with classical neuroleptics. Moreover, clozapine is used for prevention of relapse in patients who respond to clozapine as acute treatment. Unfortunately, systematic studies of relapse rates of clozapine are lacking but clinical reports on successful long-term treatment with clozapine (Povlsen et al., 1985; Kuha et al., 1986) — e.g. in terms of social and occupational re-integration — suggest that clozapine might effectively prevent schizophrenic relapse.

In this study we calculated relapse rates and mean duration of hospitalizations in 53 outpatients treated with clozapine up to 8 years. 11 patients were changed to clozapine during their first hospitalization, the remaining 42 patients were pretreated for relapse prevention over mean \pm SD 41.8 \pm 24.1 months with typical neuroleptics. The mean \pm SD duration of continuous clozapine treatment (including intermittent hospitalizations) was 60.3 \pm 22.8 months, the actual mean \pm SD daily clozapine dosage 214 \pm 135 mg (range 50–550). During a cumulative observation period of 2995 months in 53 patients, 40 relapses were observed under clozapine. In the 42 patients who had previous relapse prophylaxis with typical neuroleptics, the yearly relapse rate calculated from the raw data was reduced from 0.56 to 0.16 under clozapine. For statistical analysis the mirror image design was used. Using similar intraindividual observation periods (mean \pm SD 41.8 + 24.1 months) and excluding one previous hospitalization (where medication was changed to clozapine) there was still a significant superiority ($p < 0.01$) of clozapine as compared to typical neuroleptics indicating a high efficacy in prevention of schizophrenic relapse.