graphic changes which have resulted in a "graying of the population", the number of older individuals with chronic psychiatric illnesses (eg. schizophrenia; bipolar mood disorder) is also increasing. The same demographic trends are causing a significant increase in the incidence of neurodegenerative disorders (eg. Alzheimer's Disease) which are frequently characterized by behavioural changes in addition to their cognitive stigmata. This presentation will describe the prevalence and phenomenology of psychiatric syndromes and neurodegenerative illnesses in the elderly population, and will detail the differential diagnosis which the psychiatrist must invoke in order to arrive at a satisfactory diagnosis in the individual patient.

CLINICO-PATHOLOGICAL CORRELATIONS OF THE COGNITIVE IMPAIRMENT IN SCHIZOPHRENIA, TREATMENT IMPLICATIONS

M. Davidson [1], P. Powchick [2], K.L. Davis [2]. [1] *Chaim Sheba Med Cntr Tel-Aviv Israel;* [2] *Mount Sinai Med School NYC*

The debate between the Kraepelinian pronouncement that outcome of schizophrenia is invariably bleak and the view that outcome of schizophrenia in old age is variable focuses on cognitive capacities in old age and not on psychosis, which for many patients ameliorates. In addressing this debate, we studied cognitive functions in 400 institutionalized schizophrenic patients, between the ages of 25 and 85. To investigate the biological substrate for the cognitive impairment in geriatric schizophrenics, some of them had been followed until death and autopsy. Results indicated that 2/3 of the institutionalized geriatric schizophrenic patients experienced severe cognitive impairment but less than 10% of cognitively impaired schizophrenic patients met definite neurohistological criteria for Alzheimer's Disease (AD) and non had cholinergic deficits. On the other hand abnormal distribution of neuropeptides and synaptopysin was detected in these patients. Attempts to treat cognitive impairment in schizophrenia with behavioral interventions or currently available antipsychotic drugs have yielded only limited success. A reconceptualization of the treatment of schizophrenia is needed in which cognitive impairment becomes a target for pharmacological treatment. This can be achieved by conducting trials with compounds that are not antipsychotic but are believed to improve cognition, and/or by searching for antipsychotic compounds with cognitive enhancing properties. It is also essential to determine which of the novel anti-psychotic drugs can be given to agitated, psychotic, demented patients without further worsening cognitive performance.

A very preliminary study in which risperidone was given to geriatric schizophrenics suggests improvements in CGI, digit span, and MMSE scores without producing EPS. These suggestions are now investigated in large, double blind, placebo (and/or active) controlled studies.

ANTIPSYCHOTIC DRUG TREATMENT OF LATE-LIFE PSYCHOSES

D.V. Jeste, E. Rockwell, K. Warren, A. Schalz, I. Nabatian, D. Naimark. *VA Medical Center, San Diego, University of California, San Diego, 3350 La Jolla Village Dr. (116A1) San Diego, CA 92161*

Late-life psychoses include dementia with psychosis, psychotic depression, schizophrenia, delusional disorder, psychoses secondary to general medical conditions, and several other less common disorders with psychotic symptoms. Antipsychotics generally constitute the most effective treatment of late-onset psychoses. The risk of many adverse effects with antipsychotic drug treatment is, however, considerably higher in the aged. For example, we found the cumulative annual incidence of tardive dyskinesia with typical neuroleptics among patients over age 45 (mean age 66) to be 26%, which was five to six times greater than that reported in younger patients. Data concerning the use of the new serotonin-dopamine antagonists in elderly patients are relatively scanty. Initial studies suggest that clozapine in efficacious but its use in older patients is markedly limited by side effects such as anticholinergic toxicity. We recently examined the use of risperidone in 39 patients ranging in age from 45 to 100. Risperidone was clinically effective in a majority of these patients and was generally well-tolerated, but needed to be prescribed in lower dosages (3 mg/day or less) than those recommended for younger adults in order to reduce the incidence of adverse effects such as *postural* hypotension, sedation and extrapyramidal symptoms. Our preliminary data also suggested a mild but significant cognitive enhancing effect of low-dose risperidone in older psychotic patients. The mean Mini-Mental State Examination score increased from 24.2 to 28.2 after an average of 11 weeks of treatment ($p < 0.005$). We will discuss the risk: benefit ratio of the newer atypical antipsychotics in older patients.

PHARMACOLOGICAL MANAGEMENT OF PSYCHOSIS IN THE FACE OF DEMENTIA

B.A. Lawlor. *Dept. of Psychiatry, University of Dublin, Dublin 8, Ireland; Trinity College, Dublin 8, Ireland; St. James's Hospital, Dublin 8, Ireland*

Psychosis (hallucinations and delusions) occurs in up to one third of patients with Alzheimer's disease (AD) and can be the presenting and most disturbing feature of Lewy Body dementia (LBD). Typical neuroleptics are frequently prescribed to this patient population but are ineffective in many, produce side effects in most, and can be associated with fatal neuroleptic sensitivity in LBD. It remains unclear whether the lack of efficacy of typical neuroleptics in dementia patients is due to intolerance of higher doses or to resistance of psychotic symptoms to dopamine blockade. The side effect profile of atypicals appears more promising although efficacy remains to be demonstrated. Non-neuroleptic treatments can be useful, particularly where there is agitation or sleep disturbance associated with psychotic symptoms. In spite of the drawbacks, judicious use of neuroleptics in selected patients can be extremely beneficial. The following guidelines for neuroleptic use in dementia patients are based on the available empirical data: (i) Neuroleptics should be avoided if possible in patients with a history of neuroleptic sensitivity or suspected LBD. (ii) There is no clear evidence that one neuroleptic is any better than another and the choice of drug therefore depends on the side effect profile and clinician preference. (iii) The starting dose should be low and titrated upwards gradually, but in most cases, treatment doses should be in the range of 0.25–3.0 mg of haloperidol equivalents. (v) Although the ideal minimum (or maximum) period of treatment is unknown at present, because behavioral disturbances tend to be episodic and neuroleptic exposure is frequently associated with treatment emergent side-effects, exposure to neuroleptic should be time-limited and reviewed on a regular basis.

THE MANAGEMENT OF PSYCHOTIC SYMPTOMS IN DEMENTIA WITH LEWY BODIES (DLB)

I.G. McKeith, E.K. Perry, M.A. Piggott, R.H. Perry. *Department of Neurosciences and Psychiatry and the Institute for the Health of the Elderly, University of Newcastle upon Tyne, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne, England, NE4 6BE, UK*

DLB patients have higher rates of psychosis than in other dementia subtypes. Visual hallucinations (VH) occur in 80% of our autopsy confirmed DLB cases vs 19% of Alzheimer's disease (AD) and 0% of