

EDITORIAL

Should the diagnosis of late paraphrenia be abandoned?¹

In a paper published in 1952 Roth & Morrisey (1952) used the term 'late paraphrenia' to describe a group of elderly patients with a well-organized system of paranoid delusions, with or without hallucinations, existing in the setting of a preserved personality and affective response. This and other studies (Roth, 1955; Kay & Roth, 1961; Post, 1966) reminded us that paranoid states could emerge for the first time after the age of 60. However, since its introduction the concept has come under fire as a valid diagnostic entity (Fish, 1960). The attack has come both from those who maintain that late paraphrenia is nothing more than the expression of schizophrenia with a late onset (Grahame, 1984), and from those who consider it as an early indication of organic dementia. Others have emphasized the heterogeneity of many accepted psychiatric categories and have viewed late paraphrenia as a mixture of disorders ranging from atypical affective psychosis, delusional disorder and symptomatic and functional schizophrenias, to extremes of paranoid personality type (Post, 1966; Holden, 1987), sometimes with and sometimes without detectable cerebral organic pathology. The course of late paraphrenia is chronic and only a subset of patients showed clear evidence of focal cerebral disease and cognitive impairment after many years of evolution (Kay & Roth, 1961; Hymas *et al.* 1989). The life expectancy of patients diagnosed as late paraphrenics appears to be only slightly shorter than that of a normal control population (Kay & Roth, 1961; Blessed & Wilson, 1982). Nevertheless, if the diagnosis of late paraphrenia includes a variety of underlying disorders, differences regarding the course and outcome of the disease are to be expected. In fact, a poorer outcome has been reported for late paraphrenics in whom cerebral organic factors were considered aetiologically important (Holden, 1987).

Late paraphrenia has never had a direct counterpart in the US literature. The conditions which it subsumes were undiagnosable under DSM-III (1980, until the revised version DSM-III-R (APA, 1987), by removing the upper age limit of 45 for the onset of schizophrenia, led to the preferred use of 'late onset schizophrenia'. Late paraphrenia has now been all but excluded from ICD-10 (WHO, 1990). Cases previously diagnosed as late paraphrenia will be accommodated under paranoid schizophrenia or persistent delusional disorder (Quintal *et al.* 1991). The latest draft of ICD-10 suggests that the category persistent delusional disorder, which excludes the presence of prominent hallucinations, should include late paraphrenia (WHO, 1990). This seems most unsatisfactory as many patients diagnosed as late paraphrenics will exhibit such symptoms.

As increased attention is being focused upon psychiatric problems in old age on both sides of the Atlantic, the time seems ripe for a closer examination of the delusional and hallucinatory states affecting the elderly. Late paraphrenia, still the preferred designation for these cases in Britain, is slowly losing its attraction as a diagnostic category in other parts of the world. Before it disappears from the current psychiatric nosology and becomes a concept of merely historical interest, it is necessary to evaluate the evidence for and against its validity as a diagnostic entity, and to determine whether there is a more useful nosological alternative.

In order to consider this issue it is important to examine the main findings of functional and structural studies of early onset schizophrenia, late paraphrenia and the chronologically intermediate group of late life psychoses emerging after the age of 45. In the unfortunate and somewhat paradoxical absence of neuropathological data on late paraphrenia to compare with the emerging findings in schizophrenia with early onset (Kirch & Weinberger, 1986), discussion on structural research will be limited to cerebral imaging.

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NEUROPSYCHOLOGY

Neuropsychological studies have shown a variety of cognitive deficits in schizophrenics (Cutting, 1985). As a group, they have lower IQ than their siblings or normal controls from the same social and educational background (Aylward *et al.* 1984). Other abnormalities include deficits on tests of mental flexibility, set shifting and category formation, which could be an expression of frontal lobe dysfunction (Goldberg & Weinberger, 1988). Impairment on tests of verbal and non-verbal memory may reflect temporal damage in these subjects (Kolb & Whishaw, 1983).

Miller *et al.* (1991) have recently used a comprehensive neuropsychological test battery to evaluate a group of 24 late-life psychotic patients (including late onset schizophrenia, schizophreniform disorder, delusional disorder and psychosis not otherwise specified starting after the age of 45 (APA, 1987) with mean age of 60.1. The results suggested cognitive impairment involving mainly frontal and temporal functions, in line with the findings reported in early onset schizophrenia (Kolb & Whishaw, 1983; Taylor & Abrams, 1984).

In contrast, the cognitive profile of late paraphrenics remains largely unknown. Naguib & Levy (1987) used the Mental Test Score and Digit Copying test to evaluate 43 patients. They performed significantly worse than age matched controls on these tasks and showed some deterioration in a 3.7-year follow-up study, though their scores remained above the cut-off point for a diagnosis of dementia (Hymas *et al.* 1989). Further examination of this issue is required using more detailed neuropsychological tests and perhaps an even longer period of follow-up.

The finding by Miller *et al.* (1991) that patients with late-life psychoses present a cognitive profile similar to that described in schizophrenia should be interpreted with caution. Different neuropathological processes could elicit identical neuropsychological deficits. Moreover, the results of studies which have attempted to relate psychopathological symptoms, cognitive deficits and localization of the brain lesions in psychoses have been negative (Feinstein & Ron, 1990).

NEUROIMAGING

Neuroradiological abnormalities in schizophrenia have been extensively investigated in the last few years. There are indications that some schizophrenics show mild ventricular enlargement or cortical atrophy on CT-studies (Lewis, 1990). Similar results have been reported in MRI studies of schizophrenics with early onset (Waddington *et al.* 1990).

For schizophrenics with a late age of onset (APA, 1987) there is only a single published quantitative study, reporting that the mean ventricle-to-brain-ratio (VBR) was higher in late onset schizophrenics than in healthy controls, but lower than that found in a group of age-matched Alzheimer's disease patients with hallucination and delusions (Rabins *et al.* 1987). The authors further suggested that the late onset cases were comparable to schizophrenia with early onset, since the ratio of the VBR controlled for age was similar in both groups (Rabins *et al.* 1987). MRI studies of late life psychotics have shown that the proportion of such patients with subtle brain abnormalities appears to increase with the sophistication and sensitivity of the neuroimaging technique involved. The white-matter lesions demonstrated by MRI in a high proportion of these patients (Breitner *et al.* 1990; Miller *et al.* 1991) are, perhaps, an indication of the organic substrate in late-onset psychotic illness.

Naguib & Levy (1987) described a normally distributed mild ventricular enlargement in a sample of 43 late paraphrenics, when these patients were compared to healthy age-matched controls (see also Burns *et al.* 1989). Flint *et al.* (1991) recently reported that cerebral infarcts were demonstrated more frequently in the brain scans of late paraphrenics without hallucinations than in those with such symptoms. The former were also less responsive to antipsychotic medication. A similar approach to the question of heterogeneity in late paraphrenia has been used by R. J. Howard *et al.* (1992). Late paraphrenics without first rank symptoms (FRS) exhibited ventricular and sulcal enlargement comparable to that in demented patients suffering from Alzheimer disease, while those with FRS showed brain scans similar to healthy age-matched controls (Howard *et al.* unpublished results). There have as yet been no MRI studies of late paraphrenia.

CONCLUSION

First, these results indicate that in contrast to the subtle brain lesions described in only a proportion of schizophrenics with early onset, organic factors could play a more important role in the causation of late onset psychotic symptoms (Miller *et al.* 1991). Secondly, the neuroimaging findings suggest that late paraphrenics might eventually be differentiated by the characteristics of their brain lesions (Flint *et al.* 1991; Howard *et al.* 1992).

Neuropsychological and neuroradiological findings suggest that schizophrenia might be associated with different and often mild brain disease, which in turn may lead to cognitive impairment. It is not yet clear, however, whether schizophrenia with late and early onset is the expression of the same neuropathological process, or whether a neurodegenerative mechanism in late onset cases may contrast with the neurodevelopmental mechanism proposed for early cases (Weinberger, 1987).

Late paraphrenia and late life psychosis (as defined by Miller *et al.* 1991) are very similar in clinical terms; both seem to constitute a heterogeneous group of paranoid disorders starting in later life. Patients with typical schizophrenic symptoms beginning after the age of 45 (late onset schizophrenia) represent only a subgroup of what is called 'late paraphrenia' or 'late life psychosis'. The high prevalence of subtle brain lesions in association with psychotic symptoms of late onset would seem to be qualitatively and quantitatively different from the findings reported in early onset schizophrenia. MRI studies in association with a comprehensive cognitive assessment of late paraphrenics may help to clarify the real status of this condition. Until such results are available, we will not be able to decide whether we can afford to abandon the potentially useful diagnostic category that late paraphrenia has provided over the past 35 years.

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