other findings showing no short-term, but long-term memory problems resulting from these drugs (e.g. Crow and Grove-White, 1973; Safer and Allen, 1971).

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THE MANIA: MELANCHOLIA RATIO (1880–1910)

DEAR Sir,

I read Dr Edward Hare's paper (*Journal,* May 1983, **142,** 439–55) with interest and found his hypothesis concerning the slow epidemic aetiology of schizophrenia persuasive. However, he alludes to the declining ratio of mania to melancholia admissions between 1880 and 1910, and suggests that this may indicate a similar epidemic aetiology for the affective disorders. He adds that this change 'would certainly be hard to explain in sociological terms'.

From my own work on melancholia admissions in Edinburgh (*Journal,* in press), it seems that there was a progressive propensity, certainly from 1892 onwards, to admit non-delusional melancholics i.e. depressives were admitted more readily and with less severe illnesses. Hare's graph shows a decline in the diagnosis of both melancholia and mania from the early 1900's onward, presumably a result of the 'discovery' of schizoprenia. This decrease is sharper in mania than in melancholia, which shows that more schizophrenics were 'mis-diagnosed' as manic than as melancholics. This would tally with modern clinical experience, and would probably be even more prevalent in the days when the admission threshold for disturbed behaviour was higher—'manic' schizophrenics would be more likely to be admitted than 'melancholic' ones.

I suggest that the fall in the mania:melancholia ratio occurred on account of two factors—the increased admission of less disturbed melancholics, and the 're-diagnosis' of more manics than melancholics as schizophrenic. I do not think it is necessary to invoke an epidemic aetiology for the affective disorders to explain this change.

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ONE YEAR FOLLOW-UP OF TARDIVE DYSKINESIA

DEAR Sir,

A previous study of tardive dyskinesia (TD) in all known schizophrenics in Nithsdale found a point prevalence of 31 per cent (McCreadie *et al,* 1982). It was suggested that as a generation of schizophrenics has now been exposed to neuroleptics, thought to be the main aetiological factor in TD (Anonymous, Lancet, 1979), the community prevalence might have reached a plateau. A detailed review is being carried out, but the results of a one year follow-up are of interest.

The repeat census on 1.3.82 identified 136 schizophrenics, of whom 121 were members of the origial cohort. TD was assessed using the AIMS Scale (U.S. Department of Health, Education and Welfare, 1976) in all in-patients and day-patients, 98 per cent of out-patients, and 57 per cent of patients known only to their general practitioner (N = 122). If a rating of at least 'mild' on the global scale is taken as definite TD, then 27 per cent of patients had TD. Thus there has not been any increase in TD over twelve months; indeed, the prevalence has fallen slightly.

The 103 patients who were assessed in both 1981 and 1982 fell into four groups: 55 per cent did not have TD on either occasion, 18 per cent had TD on both occasions, nine per cent developed TD, and 18 per cent no longer had TD.

Methodological difficulties may explain some of the fluctuation in the latter group; for example, the assessment was brief and the sample of behaviour examined may not have have been typical. However there may have been a genuine decrease in TD in some patients, as the majority in this group had had an increase in neuroleptics over the year, a factor known to suppress TD (Carpenter *et al,* 1980).

If a move from 'absent' to 'mild' on the global scale