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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References

CALEV, A. (1981) *Post-organisational Memory Deficit in Severely Disturbed Schizophrenics.* Doctoral Thesis, University of York.

—— VENABLES, P. H. & MONK, A. F. (1983) Evidence for distinct verbal memory pathologies in severely and mildly disturbed schizophrenics. *Schizophrenia Bulletin,* **9,** 247–64.

CROW, T. J. & GROVE-WHITE, I. G. (1973) An analysis of the learning deficit following hyoscine administration to man. *British Journal of Pharmacology,* **49,** 322–7.

KINTCH, W. (1970) Models for free recall and recognition. In: *Models of Human Memory* (ed. D. A. Norman). New York: Academic Press.

KOH, S. D. (1978) Remembering of verbal materials by schizophrenic young adults. In: *Language and Cognition in Schizophrenia* (ed. S. Schwartz). Hillsdale, New Jersey: Lawrence Erlbaum.

POTAMIANOS, G. & KELLETT, J. M. (1982) Anti-cholinergic drugs and memory: The effects of Benzhexol on memory in a group of geriatric patients. *British Journal of Psychiatry,* **140,** 470–2.

SAFER, D. J. & ALLEN, R. P. (1970) The central effects of scopolamine in man. *Biological Psychiatry,* **3,** 347–55.

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

can be taken as the firmest evidence of the development of TD, then the annual incidence rate of the condition in Nithsdale schizophrenics is three per cent.

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References

ANONYMOUS (1979) Tardive dyskinesia. *Lancet, ii,* 447–8.

CARPENTER, W. T., REY, A. C. & STEPHENS, J. H. (1980) Covert dyskinesia in ambulatory schizophrenia. *Lancet, ii,* 212–3.

MCCREADIE, R. G., BARRON, E. T. & WINSLOW, G. S. (1982) The Nithsdale schizoprenic survey: II. Abnormal movements. *British Journal of Psychiatry,* **140,** 587–90.

U.S. DEPARTMENT OF HEALTH, EDUCATION AND WELFARE (1976) Abnormal Involuntary Movements Scale (AIMS). In *ECDEU Assessment Manual* (ed. W. Guy), pp 534–7. Rockville, Maryland: U.S. Department of Health, Education and Welfare.

RENAL FINDINGS AFTER 30 YEARS ON LITHIUM

DEAR Sir,

John Cade reported his original findings on the use of lithium salts in the treatment of 10 patients with mania (Cade, 1949). The fifth patient, B.D., continued to take lithium for 32 years until his death in 1980 at the age of 77 from a cardiac infarction. Two years before his death he was studied with renal function tests and renal biopsy.

History of psychiatric illnesses: He was born in 1903. He was a second year medical student when he had his first manic illness in 1926. Further episodes of illness occurred in 1934 and 1935. In 1938 there was an episode of "deep depression—which was like a deep dark trough of despair from which even God could not rescue me." In 1940 there were 2 episodes of mania each requiring hospital admission. Another depressive illness occurred in 1947.

In 1948 there was another episode of mania. Cade's notes read "recurrent mania, present attack has lasted 2½ months and shows no signs of abating. At present restless, noisy, elated, flight of ideas, constantly striding about, gesticulating and up to all sorts of antics. Eating very well, but thin".

30/7/48 "Commenced lithium citrate gr. 20 t.d.s."

2/8/48 "Marked improvement. Far less flight of ideas and much less restless".

12/8/48 "Slowly settling down. Less motor restlessness. Flight of ideas still marked".

16/8/48 "Greatly improved. Flight of ideas has disappeared".

21/8/48 "Quiet, pleasant and rational".

27/8/48 "Normal. To continue with lithium indefinitely".

In 1948, the patient was aged 45 and he said "Since I took the lithium I had no more severe mania or deep depression like the 1938 episode." Despite lithium, however, he was in hospital on 9 subsequent occasions. There were five depressive episodes in 1953, 1954, 1962 and 1979, and 4 manic episodes in 1956, 1966, 1973 and 1976. B.D. never married. He left medical school in the second year and entered the public service and worked until he retired at 65.

Mild tremor of the hands, and severe polydipsia, polyuria and nocturia were the main side effects of lithium treatment, though it is difficult to define the progression of the last symptoms. Thyroid function tests were normal. His plasma lithium levels were monitored from 1972, and levels between 0.6–1.2 mmol/l were kept till 1975 when levels of 0.6–0.8 were obtained. Daily dosage varied but it is estimated that in 32 years he would have taken 14.5 kg of lithium salts. Lithium toxicity was never recorded. In the last two years of his life he developed mild angina. In June 1980 he developed severe chest pain and died in hospital on the same day, of myocardial infarction.

Renal investigations: These were carried out in August 1978 after 30 years on lithium treatment.

Tests of distal tubular renal function revealed that the polyuria was associated with a marked defect in urinary concentrating ability. This urinary concentration defect was resistant to the action of exogenous vasopressin, confirming its nephrogenic origin. The defect was also more marked than in any other patients we have studied on lithium (Walker *et al,* 1982). A significant impairment of urinary acidification was also present. However, measurements of determinants of glomerular filtration rate were not different from age-related normal values (Table). The urinary sediment and urinary protein excretion were also entirely normal.

A percutaneous renal biopsy demonstrated the specific distal tubular lesion associated with lithium therapy that has previously been described (Burrows *et al,* 1978). The lesion consists of vacuolation of the epithelial lining cells of the distal nephron (distal convoluted tubular and collecting duct), with the appearance of variable degrees of periodic acid Schiff (PAS) positive material in granules and strands particularly at the periphery of the cells. There was a mild to moderate degree of interstitial fibrosis. Tubular atrophy and glomerular sclerosis were also present similar to the changes of chronic focal interstitial nephropathy described in other patients on long term lithium therapy (Hestbech *et al,* 1977). However, the