as a function of their screening score to estimate the number of unascertained cases. Using this procedure, we project an additional 27 cases (3, 7 and 17, respectively), raising the overall rate to 19.7 per cent (60/305). Future references to Pitt's rate of late puerperal depression should use this adjusted figure. Hopefully, this correction will add further impetus to a research area pioneered by Pitt.

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CANCER AND DEPRESSION

DEAR Sir,

Brown and Paraskevas proposed (*Journal*, September 1982, **141**, 227–32) an intriguing theory that some cases of depression in cancer may be caused by immunological interference with the activity of serotonin. Tumor basic protein (TBP) appears ubiquitous among cancer cells (Caspary and Field, 1971). There appears to be no question that it contains a site which structurally is similar to the 9-residue peptide of myelin basic protein, which is responsible for experimental allergic encephalomyelitis (EAE) and which also binds serotonin. They also have shown that TBP binds serotonin.

During clinical EAE, very little circulating antibody is produced against the EAE active peptide. Therefore one must presume that the corresponding site in TBP also is a poor antibody producer. However, what antibody that is produced should *not* bind to serotonin since it and serotonin are complimentary to TBP and the EAE active peptide, which both bind serotonin. Therefore the *in vivo* interference of serotonin actions by TBP would be dependent upon large amounts of exposed TBP in areas that are sensitive to serotonin concentrations. The interference would not be due to antibody concentrations.

Another area which should be examined is the relationship of euphoria and demyelinating diseases. Contrary to what is seen in cancer, the active EAE peptide that binds serotonin is released (Cohen *et al*, 1975) in large amounts only for very short periods, e.g., during relapses in multiple sclerosis.

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ANTI-CHOLINERGIC DRUGS AND MEMORY

DEAR Sir,

The effects of anticholinergic drugs on memory have rarely been studied, yet knowing their nature is important. In 1982, Potamianos and Kellett reported their finding that anti-cholinergic drugs had an adverse effect on memory. In their study, geriatric patients performed significantly worse when on benzhexol than when on placebo. However, it should be noted that this applied only to three of their tasks: 'paired associated learning', 'short-story recall' and 'word list recall'. It did not apply to 'digit span'. This short term memory test was the only task they used, which did not require that the subject established mnemonic organization at encoding.

This is of particular interest in the light of recent results we obtained while studying memory in schizophrenia (Calev, 1981; Calev, Venables and Monk, 1983). We used two *long-term* memory tasks, which like many short-term memory tasks, minimized the need for the subject to use mnemonic elaboration at encoding. In the first task, the patients were instructed, before the recall test, to meaningfully sort and semantically organize the to-be-remembered words; so that the subject's spontaneous use of mnemonic organization at the encoding stage became redundant. In the second task, (recognition memory), patients were required to discriminate formerly presented target words from distractor words sampled from the same population; this task too was said to involve minimal need for mnemonic organization at encoding (e.g. Kintch, 1970; Koh, 1978). In both these tasks, we found no differences between two groups of chronic schizophrenics, of which only one was on anticholinergic medication (Disipal, procyclidine, and benzhexol). We have recently replicated these results.

Taken together, the results of these two studies seem to indicate that anti-cholinergic drugs affect memory tasks which require mnemonic organization at the encoding stage, but not all memory tasks. When mnemonic organization is either not essential (e.g. in 'digit span' and 'recognition') or artificially induced at encoding (e.g. our first task), no deficit is apparent. A literature search indicated that this conclusion also fits

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