PSYCHIATRY AND PSYCHODYNAMICS

DEAR SIR,

I read with interest B. A. Farrell's argument (*Journal*, July 1983, **143**, 1–7) that regardless of the Popperian insufficiencies of psychodynamic theory, common sense should give credence to certain of its ground rules as set out by Malan. Farrell must be aware of the problems inherent in judging such ideas on the basis of common sense, as have been pointed out by Bertrand Russell and others. These problems aside, the common sense attraction of Malan's notions, as cited, is surely a reflection of their being themes belonging to psychological theories in general rather than the particular property of a psychodynamic framework. The observations, predictions and many of the mechanisms implied in these notions can with a minimum of transcription be derived from any of a number of starting points conceptually dissimilar to the psychodynamic; for example, those of personal construct theory or social learning theory. It is this conceptual ubiquity that gives these notions the wide explanatory scope noted by Farrell.

Contemporary undergraduate medical training, contrary to the assertions in Farrell's article, now includes aspects of psychology, sociology and the philosophy of science. I think, therefore, that any agnosticism among mainstream psychiatrists concerning psychodynamic theory would take the form of informed scepticism, rather than the overawed puzzlement Farrell would have as the case.

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TARDIVE DYSKINESIA AND ANTI-PARKINSONIAN DRUG WITHDRAWAL

DEAR Sir,

The article "The Abrupt Withdrawal of Anti-parkinsonian Drugs in Mentally Handicapped Patients" (*Journal*, February 1983, **142**, 166–68) is inaccurate and confusing. Dr Carter states that numerous scales are available for the assessment of dyskinesia including the Abnormal Involuntary Movements Skill (NIMH, 1975) and that of Simpson and Angus (1976). The latter scale does not measure dyskinesia at all! Later he states that "a rating scale modified from that of AIMS with certain items such as micrographia excluded was used . . ." The AIMS contains no item for micrographia. This confusion extends to the result of the withdrawal, which in general, appears to be a mixture of parkinsonian plus acute dystonic reactions. The items mentioned in the 17 item scale which Dr Carter used are all parkinsonian items and do not relate to dyskinesia. It would be important to know the other items since the authors suggestion of tardive dyskinesia appearing after the withdrawal of antiparkinson agents is novel. More likely there was a rebound in parkinsonian side effects as has been reported previously—but this is unclear from the presentation.

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RATE OF DEPRESSION IN THE PUERPERIUM

DEAR Sir,

In 1968, Brice Pitt published an investigation of the frequency of depression in the late puerperium (Pitt, 1968). That study is still widely cited and justly so, since it was an early innovative work in this field. However, a reanalysis of the original data shows that the reported rate of 10.8 per cent is an almost 50 per cent underestimate. Rationale and computations for the corrected rate are described below.

Of 305 women completing a screening scale for depression in their third trimester and again at six to eight weeks postpartum, 38 had a difference score (postpartum score minus third trimester score) of 6 or greater; 74 had a positive difference score of less than 6; 193 a difference score of 0 or less (Pitt, 1980). In these categories, 34, 16, and 37 women were given a clinical interview using the Hamilton scale, and 27, 2 and 4 diagnosed as depressed, respectively (Pitt, 1968). Dividing the number of diagnosed cases, 33, by 305 produced the reported rate of 10.8 per cent.

Since only 87 women were interviewed, a question arises regarding the number of unascertained cases among the remaining uninterviewed subjects. Cross classification of the 87 interviewed subjects by screening and diagnostic status (Table I, Pitt, 1968) indicates that 79.4 per cent (27/34) of women with a difference score of 6 or greater were diagnosed as depressed; 12.5 per cent (2/16) of those with a positive difference score of less than 6; and 10.8 per cent per cent (4/67) of the rest. Unless interviewed subjects were diagnostically unrepresentative of other individuals in the same screening score category, we should apply these positive predictive values to the remaining 218 subjects

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