1% (Ungerstedt, 1971). Many researchers counter this argument by pointing out that these systems are highly arborized and have a wide sphere of influence. That these neurones arborize widely is true, but their influence is probably no more widespread than that of cholinergic or other putative neurotransmitter systems (Aston-Jones et al., 1983).

Is it not reasonable to suggest that disorders of higher cognitive functioning such as schizophrenia, must primarily involve neurones at the highest cortical level? Yet at present we concentrate our energies on monoamine neurones in the medulla, pons and mesencephalon. Whatever the sphere of influence of these neurones there can be little doubt that they are relatively small in number and anatomically vary little from the rat to man.

Surely the time has come to look at these systems realistically and focus our attention on neurones at a higher level. In this regard psychiatrists are obviously dependent on advances in neurophysiology and neurochemistry. Whilst waiting for such advances, let us not delude ourselves into believing, we have found the root of madness.

TIMOTHY G. DINAN

Wellcome Research Fellow, St. George's Hospital Medical School, London SW17

References

ASTON-JONES, G., SHAVER, R. & DINAN, T. (1983) The physiology of cortically projecting neurons in rat basal forebrain. Society for Neuroscience. Abstr. 106, 15, 355. UNGERSTEDT U. (1971) Stereotaxic mapping of the monoamine pathways in the rat brain. Acta Physilogica Scandinavica Suppl, 367, 1-48.

BRIEF PSYCHOTHERAPY IN FAMILY PRACTICE

In response to Dr Williams' letter (Journal, January 1984, 143, 101-2), the points made had indeed been covered in the original article (Journal 1983, 143, 11-19), and we acknowledged the high probability of a type two error. The figures provided by Dr Williams are quite correct, but one would expect there to be a differential effect between treatment groups for a larger number of subjects to demonstrate an effect. In fact, a reverse trend was found in that the control group improved more than either of the treatment groups.

Secondly the problems of finding patients suitable for controlled therapeutic trials of psychotherapy were discussed, and Dr Williams has merely emphasised those issues. He goes on to point out that "The results of such a study are applicable to only seven per cent of

those patients with significant psychiatric morbidity who present to general practitioners, and thus of limited relevance to the practical management of psychiatric disorder in general practice". What he appears to have failed to appreciate is that it is in fact only these patients with persistent psychiatric morbidity in whom we were interested. The vast majority of psychiatric disorders presented by patients in general practice remit (Johnstone & Goldberg, 1976). There were 128 patients who were persistently symptomatic for at least six months of whom 27 were allocated to the control group. Of the remaining 101 persistently psychologically symptomatic patients, 35 refused interview, 25 declined treatment and 12 dropped out of therapy leaving 36 patients who completed psychotherapy. A more realistic appraisal then is that 36 out of 101 patients with the type of disorder specified, persistent psychological morbidity for at least six months, might be suitable for dynamic psychotherapy.

We look forward with interest to the results of the studies by workers at the General Practice Research Unit with regards to social casework in the primary care setting as until now only anecdotal evidence of its efficacy exists.

HENRY BRODATY
GAVIN ANDREWS

The Prince Henry Hospital, P.O. Box 233, Matraville, N.S.W. 2036, Australia

Reference

JOHNSTONE, A. & GOLDBERG, D. P. (1976) Psychiatric screening in general practice: A controlled trial. *The Lancet*, i, 605–8.

MIANSERIN AND WARFARIN

DEAR SIR,

I refer to the letters of Warwick and Mindham (Journal, September, 1983, 143, 308) and Ancill and Pinkerton (Journal, February, 1984, 144, 213-4) concerning a case of concomitant administration of warfarin and mianserin which resulted in an abnormally high prothrombin time. I would like to report a case where such an interaction did not occur.

An otherwise healthy 53 year old female developed cardiac arrhythmias and pulmonary oedema while on tricyclic antidepressant therapy for a severe depressive phase of a manic-depressive psychosis (ICD 9, 296.1). On recovery she was stabilised on digoxin and anticoagulant therapy with warfarin 8 mgs. daily, a dose she has remained on since. A month after commencing warfarin she was prescribed mianserin on the grounds that it is non-cardiotoxic. She responded to a dose of mianserin built up to 120 mgs./day, but