random. The number of decimal places worked out is immaterial; it is not the minutiae but the general pattern that is important. Correlations are given to three places, partly for conventional reasons and to allow others to work on the raw data if they so wish.

We do not agree that many of the features cannot be said to be either present or absent; one might as well say that nobody is entirely ill or entirely well. This may be literally true, but it is permissible and necessary to define "patients" according to convenient, if arbitrary, conventions. In the same way we used criteria for deciding whether a feature was to be regarded as present or absent. We would agree with Dr. Stanley that this aspect is important; and in due course we expect to be able to publish our criteria in greater detail. Knowledge, however, does not spring out fully armed, like Athene from the head of Zeus, and this is a report of work in progress. Incidentally, our method is founded on, and not a substitute for, clinical observation.

MARTIN ROTH.
M. W. P. CARNEY.
R. F. GARSIDE.

Department of Psychological Medicine,
Queen Victoria Road, Newcastle upon Tyne.

PATTERNS IN REACTIVE AND ENDOGENOUS DEPRESSIONS

DEAR SIR,

Dr. Foulds (*Journal*, November 1965) suggests that a psychiatrist who has the impression that a patient is suffering from a reactive depression would not ask about sleep disturbance with the same persistence as he would if he thought the illness was an endogenous depression. This is not so, because most British psychiatrists consider sleep disturbance to be an important differentiating symptom. Since the introduction of antidepressant drugs there has been a tendency to over-diagnose endogenous depression because of the supposed effectiveness of these drugs. In order to support the diagnosis of endogenous depression the average psychiatrist is likely to look carefully for sleep disturbance.

Dr. Foulds makes the erroneous assumption that reactive and endogenous depressions are equivalent to his neurotic and psychotic depressions. He regards delusion as an essential feature of psychotic depression. There are many patients with endogenous depression who are not deluded and will therefore be classified by Foulds as neurotic depressives.

FRANK FISH.

Department of Psychological Medicine,
The University of Liverpool.

EFFECT OF A DEPRESSIVE ILLNESS ON M.P.I. SCORES

DEAR SIR,

In their recent paper, Coppen and Metcalfe (*Journal*, March 1965) appear to make an important methodological point about test-retest reliability studies in general and those relating to the Maudsley Personality Inventory (M.P.I.) in particular. They say: "The stability of a test is often expressed in terms of the test-retest correlation coefficient, but our results show that this can be very misleading; groups of patients can evidence a considerable change in their scores even though the test-retest correlation remains high" (p. 238). Since their study differs in a number of important ways from a true test-retest reliability study, I question the validity of their discussion on this particular point.

In the first place, the test-retest correlation could be perfect, not merely "high", and yet the mean differences could still be as large as Coppen and Metcalfe report. There is no necessary relationship between the size of the mean difference and the correlation between the scores. Secondly, their data has only an indirect link with a true test-retest reliability study. Their experimental design specifically required that treatments be interpolated between the first and second testings. A control group, not undergoing any special treatment, would properly estimate repeat-test reliability over the same period of time. If a different value for this correlation coefficient were found in the experimental group, it would suggest that the treatment had had a differential effect on patients having different initial scores. Test-retest reliability is test-retest reliability and not just *any* correlation between repeated measurements.

Thirdly, the correlation coefficients quoted in their paper may not provide appropriate summaries of the data. It is apparent from Tables I and II that there are marked heterogeneities in their group of patients. Not only do the two treatment groups (E.C.T. and Drugs) give different mean scores on the M.P.I., but so do the three diagnostic groups. These differences and possible interactions between diagnostic group and the type of treatment could well invalidate all the correlation coefficients they compute. Far from being surprised how high or how low the correlations proved to be, they should regard it as remarkable that there is any correspondence at all with the data collected by others.

Finally, it should be clear that a test such as the M.P.I. should have two properties: there should be a relatively high stability—high repeat-test reliability and stable means—when no particular change is induced; and it should be sensitive to change when

effective treatments are applied. There is no paradox in this statement. Coppen and Metcalfe's own data give some demonstration that the M.P.I. has both properties.

PHILIP LEVY.

Department of Psychology,
University of Birmingham.

TRIAL OF OXYPERTINE FOR ANXIETY NEUROSIS

DEAR SIR,

In the October issue of this *Journal*, McAllister takes us to task for concluding on the basis of intercorrelations of approximately 0·20 or less between scores on Cattell's I.P.A.T. Anxiety Scale and independent clinical ratings of anxiety that "The I.P.A.T. Anxiety Scale does not appear to be a valid technique for the assessment of anxiety states". He does so on the grounds that the I.P.A.T. Anxiety Scale is mainly a measure of anxiety as a personality trait and that it may be valid for this purpose without necessarily having any significant correlation with ratings of anxiety as a state. We wish to make four points in reply.

First, we doubt whether it was improper of us to assess the validity of the scale by comparing test scores and clinical ratings of anxiety. Indeed, this procedure is explicitly recognized by Cattell, who on p. 9 of the *Manual* describes the intercorrelation between test scores and psychiatric assessments of anxiety as being one of the three "most conclusive ways possible" of determining the scale's external validity. It would thus appear that McAllister's views are at variance with those of the author of the scale.

Secondly, while agreeing that in general it is quite legitimate to draw a conceptual distinction between measures of personality traits and of clinical states, we doubt whether such a distinction can be applied unambiguously in the present case. In particular, it is difficult to reconcile McAllister's views with Cattell's description of the "overt symptomatic" score which is distinguished precisely to provide "*a record of actual symptoms*" (p. 6, our italics).

Thirdly, even if McAllister were right to draw this distinction with respect to the I.P.A.T. Anxiety Scale, this has no relevance to our conclusion, since at no time did we question the scale's validity as a *personality* measure.

Fourthly, we question McAllister's interpretation of the scale's purpose, which he maintains is to measure predisposition to anxiety. We, on the basis of the evidence cited in our article, suggest that the scale measures neuroticism. Since our study was not specifically designed to adjudicate between these rival interpretations, we do not wish to be dogmatic on this point. We may note, however, that our interpretation is consistent with the findings of Bendig (1960), who on the basis of an extensive factor-analytic study of anxiety and neuroticism inventories (which included the Cattell Scale) suggested that "Anxiety and Neuroticism are both manifestations of a more general emotionality factor and are not separate dimensions within commonly used inventories . . ." (p. 167).

J. B. KNOWLES.

Department of Psychology,
Queen's University,
Kingston, Ontario, Canada.

N. KREITMAN.

M.R.C. Clinical Psychiatry Research Unit,
Graylingwell Hospital,
Chichester, Sussex.

REFERENCES

BENDIG, A. W. (1960). "Factor analyses of 'anxiety' and 'neuroticism' inventories." *J. consult. Psychol.*, **24**, 161–168.

CATTELL, R. B. (1957). *Handbook for the I.P.A.T. Anxiety Scale*. Champaign, Ill.

PHENOTHIAZINE TREATMENT IN SCHIZOPHRENIA

DEAR SIR,

To test the hypothesis that phenothiazine treatment in schizophrenia loses much of its effectiveness if its initiation is delayed, we recently studied the records of 109 schizophrenic patients.

All these patients had a well-confirmed diagnosis of schizophrenia (made independently by at least two psychiatrists), were less than 45 years old, had graduated high school, and had been in-patients in this hospital at some time more than three years prior to the study.

Our basic assumptions were that all these patients must have begun their schizophrenic illness at around the same age, and that those who had first received phenothiazines at early ages would, therefore, tend to have received them at an earlier stage in their illness than those who first received them at later ages.

As an index of how well or badly the patients did, we used the percentage of lifetime after the first psychiatric consultation spent in mental hospitals.

A Pearson correlation coefficient was calculated between the ages at which phenothiazines were first given, and the following index: