treatment. Not one "endogenous" factor showed a significant association.

Using Column A (which is a more realistic and conservative estimate than Column B), diurnal variation is no longer significant, and psychomotor retardation is thus the only one of the six "endogenous" factors to be significant. However, the three "reactive features" which were significant in Column B remain so.

Perhaps the most significant aspect of Table II is the large number of "mixed" (really undiagnosable, according to our criteria) cases. When the more conservative method of distributing the patients was used, 32 per cent. of the patients did not fall into either the "reactive" or "endogenous" group.

To turn to Foulds's claim that the use of "adequate personality and steady course under endogenous, and their opposites under reactive", is inadmissible: These factors were originally studied as continuous variables, in which case the extremes might have validity. Furthermore, using "course of illness" as an example, if fluctuating course as reactive feature were removed, this would create a bias in the direction of making the diagnosis of "endogenous". To remove both is to ignore what may be significant components of the syndrome.

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DEAR SIR,

Professor Fish (*Journal*, January, 1966) says that I make the erroneous assumption that reactive and endogenous depressives are equivalent to my neurotic and psychotic depressives. But I criticized Carney, Roth and Garside for using terms from two different universes of discourse (endogenous and neurotic)! As the two dimensions (endogenous-exogenous and psychotic-neurotic) are used by psychiatrists, they are very far from being orthogonal. When I have been wanting to dichotomize depressives into psychotic and neurotic and some wayward psychiatrists have written endogenous or reactive, I have asked them to use psychotic: neurotic. Almost invariably endogenous and psychotic have been associated, and so have reactive and neurotic. I dislike endogenous: exogenous because it is an aetiological classification (without adequate basis and with less likelihood of inter-judge agreement than presence or absence of delusions) amidst surrounding phenomenological classes.

With regard to sleep, my more general point was that clinicians often confirm their hunches because they so arrange the situation that there is no possibility of disconfirmation. I could have made this point better had I said paranoid rather than reactive depressive.

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DEAR SIR,

Recent correspondence in the *Journal* on the nature of depressive illness is rather disturbing: it is especially a ground for despondency that controversy remains after so many years' discussion, although this is one of the occasions when clinical experience and more academic studies appear to be in agreement. One is bound to ask just what fundamental advances have been made in psychiatry for which administrators and the pharmaceutical industry are not responsible.

It is a part of human experience that some suffer changes in mood for which they can find no explanation, while others suffer from a change in mood for which an environmental cause is only too clear. Those who experience both types of mood change at one time or another can distinguish them not only by the presence or absence of an environmental cause, but also in the quality of the mood change. When they suffer reactive depression they have suffered a stress which they are, at least temporarily, unable to withstand; they lie awake thinking of the problem at night, and then sleep through the alarm clock; they forget the problem temporarily at a party and feel happier until they are again reminded of it.

These are also the symptoms of a neurotic depression, and when one moves from normal experience to experience of disease one finds neurotic depression affecting one sort of person, who experiences one set of symptoms and shows one type of response to treatment; and endogenous depression affecting another type of person, with different symptoms and a different response to treatment: and none of these differences looks like a merely different point along the same line. If neurotic depression and endogenous depression were merely quantitatively different one would have to place the endogenous depression at the more severe end of the scale; and yet we can find mild depressions which share the basic symptomatology of severe endogenous depression, which are milder than other depressions which share the symptomatology of a non-pathological reactive

depression. I have already mentioned the different quality of the depression when environmentally and non-environmentally determined in the normal: in illness, too, are we really describing the same thing in the endogenous and neurotic types of depression when we use the word "depression" itself? This one word has in English to cover the world-embracing pessimism of the one and the self-pity of the other. The response of a more severe illness to treatment should be less satisfactory, and yet most of us are more successful at treating the endogenous variety of depression: sufficiently so for us to justify our over-diagnosing the condition. I have met those who in discussion deny any essential difference between the two types of depression and yet advocate classification according to the response to different treatments. When one asks what factors they seek to elicit in deciding which is likely to be the most successful line of treatment, they talk in similar terms to those the rest of us use in describing endogenous and neurotic depression.

Some writers have objected to the use of such words as "good personality" and "inadequacy". Surely these can be used as objective descriptive terms as well as moral value judgments. I may or may not prefer subjectively a pint of beer to a half pint of beer, but I can either way measure the difference in volume. We are, of course, all inadequate at some things or in some roles at some time and therefore we make no attempt to aspire to such things; but when we call patients "inadequate" we are labelling a persistent pattern of behaviour mal-adaptive even to the lowest of roles compatible with survival in our society. Really such a standard is very low: I have sometimes been puzzled by an apparent inconsistency between a patient's symptomatology, behaviour and response to treatment on the one hand, and my initially favourable assessment of personality on the other; when I discuss aspects of the case with someone who has known the patient longer and more intimately and suggest that the patient may be slightly inadequate, they marvel at my delicacy.

For the present one should not quibble over one's preference for the terms "reactive" and "neurotic" and whether they are essentially similar, once one has accepted that there is such a phenomenon as a reactively precipitated endogenous depression, which is symptomatically like any other endogenous depression. I use the term "reactive depression" to describe a normal phenomenon and also occasionally use it to describe a depression presented to me as a result of severe stress in a relatively stable individual. (Even then I speculate why this particular individual shows the abnormal behaviour of seeking psychiatric help!)

Nevertheless, placing a patient in one or other category, endogenous or reactive, and keeping him there is often not easy, and this is most particularly true of those who remain as out-patients (in a case seen for the first time as an in-patient, it is much easier to assess the extent of the depression, of the crisis or of the patient's vulnerability to crisis). We cannot always reach a definite diagnosis either by making an algebraic sum of a list of symptoms or by persuading ourselves that we can see an overall picture. If our overall impression and our algebraic sum disagree we have to discard one or the other, perhaps later to decide that the wrong one was discarded (or to quote an apocryphal saying of Sir Thomas Beecham, "I once made a mistake. I thought I was wrong when I was not.") And this brings us to the interpretation of the patient's symptoms: if he expresses guilt feelings, are these delusional, are they a painful insight into feelings of personal inadequacy, or are they mere histrionic breast-beating? It would be clearly bad psychiatry in the *clinical* situation not to try to understand what lies beyond the patient's words, but how far can we go in explaining a symptom without deceiving ourselves into believing that we can fathom the unfathomable? In the *research* situation one risks either treating a lot of heterogenous information as one factor, or finishing up by demonstrating the bi-polarity of one's own presuppositions.

Should we accept the patient's own explanations: if the classical endogenous depressive is most depressed in the morning and the neurotic depressive most depressed because of being most tired in the evening, what do we make of the woman who is depressed in the mornings "because my husband leaves me for work" and cheerful in the evenings "because he is back"; or of the patient who is regularly depressed in the afternoon without obvious environmental explanation? In other words, is it the time of day that is the guide, or is it the explicability or otherwise of the fluctuation regardless of the time of day? The neurotic depressive does suffer fluctuations from day to day or week to week, both with and without psychogenic explanation: the latter might justifiably be called an "endogenous" variation, but in using the word endogenous have we made a semantic trap for ourselves?

If a patient has difficulties in getting off to sleep, rewakes fitfully throughout the night, is our cry of triumph on discovering that he now wakes at 6.30 a.m. instead of 7.0 a.m., justified: and if not at 6.30 a.m. why at 5.30 a.m. or 4.30 a.m.? One need not make too much of a single symptom if there are others to choose from, but some of the milder endogenous depressives seem to have only isolated pointers

to the nature of their disease or perhaps nothing typical of either type of disease, leaving one to make a presumptive diagnosis either on reactivity or by accepting or rejecting minor flaws in personality.

Mention of personality brings us to the question, "Is an endogenous depression in a neurotic personality a likely diagnosis?" I have always felt that those types of personality classically described as predisposed to affective psychosis have been fundamentally unneurotic, possibly even the very antithesis of neurotic; and therefore everyone else must be less predisposed and "everyone else" may or may not include the neurotic types of personality. Whenever I have diagnosed an endogenous depression in a neurotic personality I have had cause to revise my diagnosis, and almost invariably it has been because, while I had fancied I could see the point in time when a neurotic personality became endogenously depressed, I could never find the point of return. Was this because the endogenous depression had itself acted as a psychogenic stress and produced a superadded neurotic depression which took over from it *pari passu* as it improved, or was it because the original point of departure had existed only in my imagination?

Should response to treatment be a guide to diagnosis? If what has appeared to be a lifelong neurotic condition makes a sustained fundamental improvement, was one's (and other people's, both lay and medical) original diagnosis wrong, or have the symptoms of a neurotic process and the signs of a drug-induced hypomania cancelled one another out. Does the successful response to the imipramine group by the milder featureless depressions I have already described justify one's labelling them "endogenous"?

Should so-called "depressive equivalents" show the same pattern of behaviour as the overt depressive illness itself and the same sort of response to treatment? Some of those who present with an apparently causeless anergia are like the featureless mild endogenous depressives. What is one to make of it when they are most anergic in the mornings, when this is such a common phenomenon in the normal and may have changed in the patient only quantitatively? In my own experience they do less well on antidepressants than the mild endogenous depressives, and yet may do well with E.C.T.

W. J. Stanley's letter causes me most dismay. I am not quite clear what part or parts of the phrase "pseudo-scientific, mathematical or statistical approach" the "pseudo" actually refers to; my impression is that it refers only to "scientific", but that he is complaining of mathematical and statistical approaches without qualification! One must state categorically that unless psychiatrists learn to measure, fundamental advances will continue to come from elsewhere; clinical description and categorizing without measurement do nothing more than mark out the ground to be surveyed. It is doubtful whether they have much more to offer us except a source of futile bickering. If some psychiatrists contrast endogenous and exogenous depressions, while other contrast psychotic (what *is* the definition of that word?) and neurotic depressions, how but by measurement will we determine whether the two groups are talking about the same things? How far has knowledge been advanced when yet another author describes a contrast between S types and J types of depression?

My knowledge of the history of the basic sciences is partial, but most seem to have remained a mixture of anecdote and speculation until measurement and instruments for measurement have come along. Measurement is necessary to unite hypotheses with anecdotal observation! W. J. Stanley persists in a fallacy that statistics are inapplicable to rough scales of measurements, whereas the truth is that fine scales of measurement just do not need statistics. Our knowledge of heat really begins with the thermometer, but had the Ancients given a score of two to "heat" and one to "cold" (and bought themselves a computer) they would have got further than they did. How without being able to measure neuroticism and endogenous depression can we determine how frequently the two occur together (which does have some bearing on how frequently we should diagnose and treat it)? If we assume that the neurotic personality only differs quantitatively from the normal, how can we confirm or correct that assumption without measurement? How will we determine whether there is a separate condition called neurotic illness? How indeed, will we ever know whether "quantitative variation from the normal by more than two standard definitions" is just a 20th century synonym for "an Act of God".

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[*Note:* This correspondence has been re-opened by request.]