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Correspondence

PROGNOSIS OF SCHIZOPHRENIA BEFORE AND AFTER PHARMACOTHERAPY

DEAR SIR,

I find it difficult to find a valid basis for the article by Michael Pritchard (*Journal*, December, 1967, p. 1345). The amazing degree of disagreement on claims and counter-claims for therapeutic results is attributable to the conventional application of diagnostic entities. Much time has been spent on relating the assumed causes of illness to the assumed effects of specific treatments. The initial hopes for curative effects of insulin coma and other somatic treatments for schizophrenic patients turned into disappointment when it became evident that with the passage of time differences between treated and untreated patients diminished so far as the further course of the illness was concerned. But the anticipation of permanent therapeutic results can only be based on the evidence of permanently-produced changes in biological systems. Only in the case of psychosurgery does this requirement seem to be met. So far as insulin coma and convulsive treatments were concerned no assertions of permanently induced changes were made, but on the contrary they were rigorously refuted to defend their safety. This leaves us in the highly peculiar situation in which the historical association of a given treatment, administered at a particular point in time, is regarded as the determining influence on the short and long term outcome.

One of the most important advances in somatic treatment concerns compensatory treatment of chronic disorders with drugs. The fact that a therapeutic effect, once achieved, can be maintained for long if not infinite periods has contributed greatly to the growing number of ambulatory patients with actual or latent psychoses. Neither logically nor pharmacologically can a course of treatment directed at the symptomatology at a given point in time be regarded as a decisive event in changing the patient's prognosis. Nor for that matter does it suffice to use the mere fact of drug treatment for statistical purposes without evaluating the treatment in terms of adequacy, type of drug, duration of treatment, effects and complications. There surely is no entity 'drug treatment' comparable to appendicectomy or radiation treatment.

It is not the clinical diagnosis of schizophrenia, but the specification of particular symptom constellations which determines what constitutes drug-treatable pathology. In the absence of any final knowledge of cause and effect relationship between illness and treatment, a later relapse need not be related to the efficacy of treatment with excellent immediate results. Nor does it follow that maintenance of good health is evidence of good permanent therapeutic results. Psychiatric illnesses, like other illnesses, manifest themselves in very different symptomatologies at different times. I doubt that this poses a conceptual challenge for the cardiologist who depends on presenting pathology as much as on his knowledge of the underlying disease process.

While the author acknowledges that continued use of the drugs after discharge "might have lessened the re-admission rate", he leaves us with the tacit assumption that a history of drug treatment, good or bad, is sufficient to evaluate the impact on the outcome of schizophrenic disorders. He fails to explain on what basis drug treatment, given for a short time, can be expected to exert a long-term, if not life-lasting effect.

FRITZ A. FREYHAN.
New York University Medical Center,
Department of Psychiatry,
New York, N.Y. 10011.

DEAR SIR,

I am grateful to Professor Freyhan for raising some theoretical issues which I should perhaps have discussed in my papers. The studies which I reported were, however, of an empirical nature, and set out to discover, firstly, whether two groups of patients diagnosed as suffering from schizophrenia and admitted to the same psychiatric hospital at different periods differed in immediate and long-term outcome, and, secondly, whether any such differences could be explained in terms of the treatment they received. This involved no particular assumptions concerning either the causes of the illness or the effects of "specific" treatment. Nor did it assume, because all the patients were diagnosed as schizophrenic, that they were necessarily suffering from a single diagnostic entity, though it seemed reasonable to believe that the two groups as a whole were diagnostically comparable.

Clearly Professor Freyhan is right when he says that 'drug treatment' is of no value as an entity, but at

the time of the study only two drugs were used—chlorpromazine and reserpine—and, though some comparison between them was made in the results, the aim was not to compare the effectiveness of different drugs. For this purpose something other than the clinical diagnosis may well be required, such as the "target symptom" approach originally proposed by Professor Freyhan, or the more recent "target function" concept of Irwin (1968). What, I think, was shown by the comparison between the two treatment eras was that, as a group, patients treated with drugs had a better short-term outcome, and that whereas with earlier somatic treatments, such as deep insulin, the patients who responded favourably were probably those "destined for a more benign course of illness" (Freyhan, 1955), pharmacotherapy seemed to be effective in a wider range of patients, including those who showed less hopeful prognostic features.

It is, however, in relation to the long-term prognosis that most of the theoretical difficulties arise. As it happens, the comparison of the two groups showed no difference in this respect, but Professor Freyhan asks on what theoretical basis any long-term effects could be expected from short-term drug treatment. He states, "This leaves us in the highly peculiar situation where the historical association of a given treatment, administered at a particular point in time is regarded as the determining influence on the short- and long-term outcome." This, together with his statement that "the anticipation of permanent therapeutic results can only be based on the evidence of permanently produced changes in biological systems", seems to imply a belief in a solely somatic basis for the illness or group of illnesses that we call schizophrenia.

As far as neurotic illness is concerned, rather than a highly peculiar situation there is a widely held view that the historical association of given psychological events or circumstances at a particular point in time does have a determining influence on subsequent liability to illness. With schizophrenia, an increasingly widely accepted view is that we are dealing with a group of disorders in which somatic and psychological factors contribute in varying proportions. Thus, a combination of a genetically determined predisposition with certain adverse psychological experiences in childhood may, with exposure to some current stress, result in an illness which may be further aggravated by the distortion of reality which is produced. If the latter is speedily reduced or removed, then may there not be less psychological damage and perhaps less likelihood of subsequent illness? Professor Freyhan himself has earlier expressed this view—"It seems, furthermore, justified to assume that course and prognosis are favourably influenced by the speed of resocialization, which somatic and psychological therapies facilitate through rapid reduction, or removal, of disturbing symptoms that disorganize the inner continuity of the schizophrenic individual" (Freyhan, 1955).

In short, to deny that short-term treatment might have long-term effects seems to imply a static somatic approach and ignores the dynamic interplay of aetiological factors which probably determines the manifestation of this illness. In such a complex nexus, alteration of one element could theoretically have far reaching and long-term effects.

Finally, may I be permitted to draw attention to a regrettable statistical error on p. 1347 of my first paper? The first χ^2 should read 7·96, which gives $p<0·05$, but fortunately this does not affect the conclusions of the study.

MICHAEL PRITCHARD.

The London Hospital Medical College,
Turner Street,
London, E.1.

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5-HYDROXYTRYPTAMINE IN THE HINDBRAIN OF DEPRESSIVE SUICIDES

DEAR SIR,

I have read with great interest the paper by Drs. Shaw and Camps and Mr. Eccleston (*Journal*, December, 1967, pp. 1407-1411).

While I feel that their work is important I wish to comment on their interpretation of the data and on the assumptions underlying the work.

It is true that the figures quoted do show a just significant difference between the 5-hydroxytryptamine (5-HT) contents of depressed v. control hindbrains if Student's t test is used ($p<0·05$).

However, if the control and depressed groups are pooled and the two groups are distributed above and below the median value of the pooled figures the following results:

	Above Median	Below Median	
Controls	10	7	$\chi^2= 2·67$
Depressives	3	8	$p>0·1$

The really disappointing feature of their results (on which they do not comment) is the lack of any difference between the 5-HT levels of the depressed Suicides v. the other Suicides ($t = 1·42$, $p>0·1$). This is the critical comparison to make if one is to