however, from suggesting to him that he consider an analogous situation with another illness, namely schizophrenia.

It has been shown repeatedly that there are certain clinical criteria associated with a poor prognosis, and that the relatives of patients with these clinical criteria tend to have an increased risk of a similar disorder. Such patients are frequently labelled "process" schizophrenia, "true" schizophrenia, or simply "schizophrenia" to contrast them with patients having a better prognosis whose relatives show a different pattern of illnesses, principally a higher rate of affective disorders. The diagnosis of "reactive" schizophrenia, schizophreniform, or schizoaffective is usually used for this second group of patients. This differentiation is considered important, valid, and useful by many thoughtful investigators. Would Dr. Snaith say that this is all nonsense because everyone knows that the label schizophrenia has been used in so many different ways by so many different workers that we would be better off dropping the term entirely? Perhaps this analogy is not entirely fair since other workers have not confirmed our findings yet, but I think there is enough merit in it to suggest that Dr. Snaith rethink his argument.

Finally, I want to challenge Dr. Snaith's assertion concerning our diagnostic symptoms that "most . . . are manifested by all patients with chronic neuroses". Our experience and the published data of others indicate, on the contrary, that it is an unusual patient with chronic anxiety neurosis, chronic obsessional neurosis, chronic alcoholism, chronic depression, chronic schizophrenia, etc. who manifests most of these diagnostic symptoms. Further, in a paper soon to be published in the *British Journal of Psychiatry*, Woodruff demonstrates that it is very rare for patients suffering from chronic medical illnesses to fulfil our diagnostic criteria for hysteria. I don't know how, until we know more about aetiology or pathogenesis, Dr. Snaith expects to define any clinical entity except by the presence or absence of specific symptoms and the demonstration that a particular pattern of symptoms predicts something important like prognosis or familial illness.

We think that our use of the term hysteria means quite a good deal clinically; our published data so indicate. It seems a bit absurd to refute our data with an 80-year-old quotation attributed to Charcot.

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INDICATING TENSION DURING RECIPROCAL INHIBITION

DEAR SIR,

We were interested to read the paper by Seager and Brown (1) setting out the details of their apparatus for indicating tension during treatment by reciprocal inhibition. We certainly agree that a reliable and valid method of monitoring anxiety is of great value and, in spite of the small number of papers dealing with this, it is our experience that many therapists are already using various physiological indicators according to the instrumentation available in their department. The authors' statement that "any of the physiological responses to anxiety could be used" would seem, however, to be an oversimplification. Since the earlier works of Cameron (2) and Lacey *et al.* (3, 4), there has been experimental evidence for the concept of response specificity, i.e. the tendency for an individual to react predominantly by certain physiological responses and to show relatively limited activation of others. This means that the particular measure for an individual patient will be idiosyncratic and a standardized indicator becomes a doubtful proposition.

Furthermore, Malmo and Shagass (5) have contended that the actual symptomatology will itself be related to this idiosyncrasy of autonomic function, which leads to the hypothesis that for patients with certain neurotic symptoms success in behaviour therapy may require the monitoring of a particular index of anxiety, while different symptoms require a different index, even though clinically both conditions would be described as phobic anxiety and might be treated by relaxation. This suggests that highly detailed noting of the patient's complaints and anxiety signs might offer a lead to the best channel for recording autonomic change. If, however, as has been suggested by later work—Oken *et al.* (6), Johnson *et al.* (7), the response bias is not stable over time and indeed changes from stimulus to stimulus, then a single technique for monitoring becomes of even less value.

In spite of this, Wenger's (10) original studies show that of all the variables that load on the autonomic factor palmar conductance has one of the highest weightings in children, though less so for adults, where heart period and sub-lingual temperature precede. On this basis there is something to be said for skin resistance measures. Our own experience has been disappointing, however, for the skin response seems to be particularly prone to habituation, i.e. to gradual diminution without a corresponding lessening of the patient's anxiety response, both subjectively and according to other indices. A rela-

tionship is recognized between such phenomena of habituation and habit extinction, but this cannot be considered to be absolute, and the difference may prove to be allied to the components of the orientation reaction (Sokolov, 8). There is also the shifting baseline to contend with, as Seager and Brown note, and while this can be adjusted during the session or removed instrumentally, Wilder's (9) work on the Law of Initial Values indicates that different degrees of anxiety might be involved. Since, for a constant stimulus, the response will be larger if the subject is close to his resting level than if he is in a state of anxious arousal, the sensitivity of the indicator is lowered when the patient is not relaxed. This obviously reinforces Seager and Brown's statement on the importance of the patient being fully relaxed for efficient monitoring and therapy. We do not seek to denigrate the use of skin resistance in monitoring, but to counteract the impression that it is a straightforward and reliable method of measuring anxiety, including the fluctuations produced in reciprocal inhibition. We have separately tried many indices and have found that in cases where the GSR was unfruitful the finger pulse volume (in two cases—DAB) and muscle tension (in seven cases—NK—measured by EEG electrodes on scalp and forearm) have proved helpful. Muscle tension is also, of course, a direct monitor of the therapeutic method, i.e. muscle relaxation. There appears also to be some promise in the waveform of respiration, though we are not aware of a satisfactory method of quantifying this effect.

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DEPRESSION: A PHYLOGENETIC VIEW

Dear Sir,

May I comment on the letter by Dr. Price (*Journal*, January, 1968, p. 119), in which he promotes a phylogenetic hypothesis of depression?

In principle I support him heartily in such an approach. However, I feel unhappy when observations based on animal behaviour are directly, or even indirectly, applied to man, unless, as in the case of Lange's hibernation hypothesis (1928), measurable variables can be compared.

Again, Dr. Price bases his argument on a fundamental dichotomy within the depression complex, while remaining quite ready to admit to a lack of unanimity on such a clinical division.

My own experience makes me support those who see reactive and endogenous depression as part of the same disease spectrum. Therefore, I incline to favour the more unitarian proposition (1965) in which clinical depression is equated phylogenetically with a "basic emotional state", postulated to be the normal mood-level of early man. In contemporary man, by contrast, normal mood could then be regarded as being one of relative elation, this having been superimposed on the primitive mood-control system by "evolution".

Clinical depression in such a framework can then be considered to be a reversion to the basic or primitive state.

Such a reaction might be triggered, as suggested elsewhere (1967), by stress on man's central physiological clock. This structure may respond by reverting to its inherent (? 25 hour per day) circadian rhythm, while at the same time releasing basic mood.

Like Dr. Price's, this hypothesis is open to corroboration by testing. In some respects the latter lends itself more easily to experimentation. Professor Jenner (*Journal*, December, 1967, p. 1447–1448) has already intimated that he might undertake the appropriate isolation experiment if a suitable subject became available.

I should add that this latter hypothesis does imply that depression has a detrimental connotation phylo-