the patient is upset in a somewhat noisy and alarming way. As if this were not enough, a collection of ill-defined personality traits have been arbitrarily banded together (probably without any natural desire to form a cluster!); persons who possess one or more of these traits before or during their illness are then considered to have an "hysterical personality" which has predisposed them to develop hysteria.

Whitlock (1) in his paper on the aetiology of hysteria has decided to define his population as those showing clear-cut conversion or dissociation symptoms. The pitfalls of the diagnosis on the basis of such symptoms has been stressed by Slater (2); moreover, many such symptoms which have a time-honoured label as "hysterical" may well have a basis in some other pathology; for instance Stengel (3, 4) has shown that many "hysterical" fugues are depressive in origin; Walters (5) has made a plea for the abandonment of the term "hysterical pain" and the substitution of the term "psychogenic regional pain"; and I, for one, never see patients with so-called "globus hystericus" except in the setting of fairly severe anxiety states.

Woerner and Guze now attempt to "define" hysteria in a different way. Do they really believe that they have delineated a clinical entity by rating the patient for a whole list of symptoms, most of which are manifested by all patients with chronic neuroses? The authors state that a patient suffers from hysteria if he has at least 25 different symptoms drawn from ten "groups" of symptoms, in association with "a complicated medical history beginning before the age of 35, and the absence of any other diagnosis to explain the symptoms". The authors support their contention that they have defined a clinical entity on the basis of previous work (6) that patients, so defined are consistent in the subsequent course of their illness. But surely the authors must agree that the more severe and chronic any condition, organic or psychological, the less likely the patient is to get better.

If the authors had limited their conclusions to the statement that patients with more severe and prolonged neurotic states were more likely to have relatives who also suffered from some form of psychological instability, there would probably be few who would have disagreed with them. But their present conclusion that they have defined a clinical entity with a unique constitutional basis (in terms of the type of psychological disorders of their relatives) is of dubious value.

Medical science is now irrevocably "saddled" with this confusing term hysteria. We are really no nearer to defining what we mean by it than were the ancient Greeks. It is a pity that medical men did not eject the term from their vocabulary when it was proved that the uterus was not in the habit of wandering around the body. Whitlock (1) recalls the warning given by Charcot 80 years ago, and since ignored: "Bear well in mind—and this should not exact too great an effort—that the word hysteria means nothing."

R. P. Snaith.

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Dear Sir,

I welcome an opportunity to reply to Dr. Snaith's letter.

Dr. Snaith seems to be saying that because the label hysteria has been used in so many different ways in the past, often confusing and contradictory, it should be abandoned. In the final analysis perhaps he is right. Nevertheless, diagnostic names in medicine tend to persist when they have been in use for a long time. As I have indicated elsewhere, the syndrome that we have been studying was described by Briquet under the label of hysteria in 1859 so there is a precedent going back that far at least. Furthermore, I believe that most psychiatrists would agree that hysteria is the correct diagnosis for the patients we have been describing. Their usual argument is not that these patients are improperly labelled hysteria, but that they wish to use the term for other patients whom we are not prepared to label hysteria.

If Dr. Snaith believes, as his letter suggests, that follow-up and family studies do not serve to validate clinical diagnoses, I strongly disagree. I think that the results of our work indicate, subject to confirmation by others of course, that certain diagnostic criteria will predict the subsequent course and response to treatment of a group of patients, and that a similar disorder will be found in the families of these patients. Dr. Snaith may believe that this kind of observation is unimportant. While there is no arguing about taste in these matters, I cannot refrain,

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-