Brit. J. Psychiat. (1982), **141**, 425–431

Correspondence

WHAT SCHNEIDER REALLY SAID

DEAR SIR,

The investigation reported by Lewine *et al* (May, 1982, **140**, 498–502) and some of the studies to which they and also Berner and Küfferle (June, 1982, **140**, 558–65) refer, are based on a misunderstanding of Schneider's First Rank Symptoms in Schizophrenia.

Reading once again the relevant section in the 1959 translation of the 1956 edition of Schneider's book, and comparing it with the seventh German edition of 1965, has confirmed that this misunderstanding does not arise from any faults of the translation. It just will not do to take in isolation as a starting point of any research the statement where Schneider proposed a group of symptoms, which when "undeniably present and no basic somatic illness can be found . . . make the decisive clinical diagnosis of schizophrenia", as was done by Lewine *et al*.

It would lead too far to summarize Schneider's views on the meaning of "symptom" in conditions whose psychopathology, alone, was known at his time, or to expound his views on the provisional nature of our classifications of the endogenous psychoses. It shall suffice to quote from p. 133 of the translation: "Among the many abnormal modes of experience that occur in schizophrenia, there are some which we put in the first rank of importance, not because we think them to be "basic disturbances" but because they have this special value in helping us to determine the diagnosis of schizophrenia as distinct from non-psychotic abnormality or from cyclothymia. The value of these symptoms is, therefore, only related to diagnosis; they have no particular contribution to make to the theory of schizophrenia, as Bleuler's basic and accessory symptoms have or the primary and secondary symptoms which he and other writers favor". Later, he disclaims the existence of a common structure for all these symptoms of first rank importance. Schneider (p. 134) does wonder, however, whether loss of identity, diffusion of thought, and all passivity experience may not be regarded as a group which presented the "lowering of the barrier between the self and the surrounding world . . .". This proposition might perhaps be tested more specifically by Lewine *et al* employing not (as reported in their paper) all their 100 subjects, but only the 80 who had a Catego diagnosis of schizophrenia.

Schneider made it abundantly clear that he regarded differential diagnosis between schizophrenia, cyclothymia, and intermediate conditions as a matter concerning the use of definitions rather than of basic understanding. He chose as symptoms of first rank only those which could be clearly and sharply identified, while recognizing that there were schizophrenics without them. Thus, he did not include among his first rank symptoms affective flattening, incongruity, or formal thought disorder. It was for this reason that so much weight was given to Schneider's first rank symptoms by workers in epidemiology when they constructed their present mental state measures. The nature of first rank symptoms, and why they sometimes occur in patients who cannot be given a diagnosis of schizophrenia, are matters which should be investigated, for instance, by testing the interesting German hypotheses summarized by Berner and Küfferle, among them the concept of dynamic derailments.

FELIX POST

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HYSTERECTOMY FOR MENORRHAGIA

DEAR SIR,

It is unclear how Dr Gath and his colleagues (*Journal*, April, 1982, **140**, 335–50) validated the "menorrhagia of benign origin" for which the women in their study underwent hysterectomy. The assumption that women who complain of heavy periods actually suffer from a significant increase in menstrual blood loss is the crucial factor that has bedevilled both research into, as well as management of, "Menorrhagia", and its clarification is particularly relevant when such a high proportion of women with this complaint are shown to be psychologically disturbed.

It is easy to understand why a woman will feel miserable when she becomes anaemic as a result of haemorrhage from a pedunculated fibroid and why she should feel better following its removal. On the other hand, a woman who is miserable for other reasons may be sensitive to a relatively minor change in her menstrual pattern and also complain about this; she might even have a small, unrelated, fibroid: not only would this also be called "Menorrhagia of benign

origin", but psychological improvement following hysterectomy would be either unexpected or, if it occurred, coincidental.

The possibility exists that for a significant number of these women their "menorrhagia" might have been an expression of psychological vulnerability rather than uterine pathology and this is supported by the absence of uterine pathology from so many of them, the infrequency of anaemia and the fact that a considerable number of them remained psychologically disturbed following hysterectomy. This explanation also accords with the findings of a study in preparation, in which 62 per cent of women who complained of menorrhagia had little evidence of significant menstrual bleeding, but considerable evidence of psycho-social disturbance.

The danger with a hysterectomy being performed for the complaint of menorrhagia is not that women with significant bleeding will become depressed as a result, but that this operation may be inflicted upon depressed women with little evidence of abnormal bleeding.

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ANXIETY MANAGEMENT TRAINING

DEAR SIR,

I should like to make three brief comments on the paper by Ramm *et al* on "anxiety management training . . ." (*Journal*, April 1982, **140**, 367–73). Firstly, no matter what was recorded in the patients' diaries, do the authors really believe that the negative self-instruction group religiously repeated such self-depreciatory and pessimistic comments? Secondly, I was under the impression that in clinical trials a putative treatment is compared with the best available or, at worst, a placebo rather than a noxious procedure.

Finally, I consider it unethical to ask patients to repeatedly make such statements as "really going crazy . . . going to make a fool of myself . . . getting steadily worse . . ." when in stressful situations, and am most surprised that the MRC supported the project.

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Dr Watt's letter seems to assume that patients are very unwilling to repeat self-depreciatory or pessimistic comments, but we have not found this to be so in our own experience. During exposure treatment not a few patients even spontaneously try out such methods without being asked to do so. Some report the approach to be helpful, and there are many anecdotes of the therapeutic effect of such paradoxical intention, especially in the writings of Victor Frankl. His classic advice to patients who had anxiety about their heart was 'go out and have a heart attack right now', and I myself have found this approach calming during realistic danger. There was thus good reason to believe that negative self-instruction, far from being a 'noxious procedure', might well reduce anxiety occasionally. Our results bore this out to some extent and found no untoward effects from the approach. However, there is still no 'best available' treatment for anxiety states which is demonstrably better than placebo.

Current treatments of phobias and compulsive rituals by exposure in vivo are effective but were also initially thought by many to be too unethical to try. Only when clinicians carefully explored what actually happened rather than prejudge the issue was this significant advance made in treatment. Such experiments are an essence of clinical research, provided that the effects are always carefully monitored, with the patient's wellbeing constantly in mind.

ISAAC MARKS

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GENETIC LINKAGE ANALYSIS AND AGE-OF-ONSET CORRECTION

DEAR SIR,

In his otherwise excellent article on 'The Search for Genetic Linkage in Schizophrenia' (*Journal*, May 1982, **140**, 432–37), Dr Watt makes some rather misleading statements concerning a linkage analysis of Huntington's disease (HD) published by Brackenridge *et al* (1978), a paper of which I was a co-author. We did not report linkage between HD and haptoglobin as suggested by Dr Watt, but rather a maximum lod of 1.88 (at $\theta = 0.05$). This is suggestive of linkage but in the lod score method, linkage is conventionally not "proved" until a maximum lod in excess of 3.0 is obtained.

Quoting Hodge *et al* (1980), Dr Watt claims our investigation did not employ a sufficient correction to account for young individuals in our pedigrees who were not showing HD but could be presymptomatic carriers of the abnormal gene concerned. Our investigation was in fact the first published HD linkage analysis to employ such a correction and it was done on the basis of each subject's age and the population distribution of onset age; the same method was employed by Hodge *et al*. Dr Watt suggests the