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.

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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

positive lod with haptoglobin in our study and the negative lod found by Hodge *et al* was claimed by those authors to result from our failure to apply an age correction. Hodge *et al* make no such claim. They offer two explanations for the discrepancy (p. 252). The first is the possibility that HD may be caused by abnormal genes at more than one genetic locus. The second possibility is simply chance variation. As there are no firm data currently available to support the first possibility, I feel chance variation is the more likely. As with other statistical procedures, when a large number of independent linkage tests are simultaneously undertaken, some are expected to give a positive result by chance alone.

Conclusions concerning the exact linkage relations of the HD gene will have to await the results of further family studies or the development of a technique for detecting the abnormal gene product itself.

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PSYCHOLOGICAL PROBLEMS OF ACQUIRED DEAFNESS

DEAR SIR,

In his article entitled 'Some Psychological Problems of Acquired Deafness' (*Journal*, May 1982, **140**, 453–56), John Stevens described an important issue in geriatric medicine. Given the prevalence of sensory deficits in old age, and existing evidence of an association between mental health and sensory function, the psychological and social implications of acquired hearing loss do warrant further investigation. There are unfortunate flaws in the research Stevens described, however, and his conclusions reflect a certain naiveté.

While the methodology for assessing hearing handicap, and for evaluating benefits of aural rehabilitation, is in an early stage of development, both the characteristics and significance of hearing loss have been more precisely defined than Stevens' work suggests (Katz, 1978; Alpiner, 1978; Noble, 1978). Moreover, development of a measure of hearing handicap requires greater attention to the psychometric properties of the instrument than reported by Stevens.

With regard to Stevens' attempt at evaluation of the effect of hearing aid use on "handicap scores", his experimental design was clearly inadequate to this test. Comparibility of his groups was questionable from the outset, as there is reason to suspect that only more successful hearing aid candidates would "adjust" to use of aids and continue wearing them for six months. From the data presented in Table I, Stevens' Groups 1 and 2 differed not only with respect to treatment (with vs. without hearing aid) but also on the factors age and degree of hearing deficit, although a discrepancy between the text and labelling of Table I obscures the direction of these differences. As the variables age, hearing deficit and group were confounded both experimentally and in the data analysis, Stevens' conclusion that "each of these factors influenced the handicap score independently" was not warranted. In fact, previous research indicates there are significant interactions between these and related variables, with the result that consideration of multiple factors is necessary in the clinical assessment of hearing impairment (Noble, 1978; Giolas, 1982).

Criticism of the quality of Stevens' research does not diminish the importance of his topic. The work Stevens cited as suggesting the aetiological significance of hearing loss in paraphrenia (Cooper, 1976) was based on evidence that a significant proportion of patients with diagnoses of paraphrenia or paranoid psychosis have severe hearing impairment (Cooper *et al*, 1974). Supporting evidence for this aetiological role was recently provided by a demonstration that hypnotic suggestion of hearing loss can induce experimental paranoia (Zimbardo, 1981), and a case study in which improved hearing function (i.e. benefit from hearing aid) was followed by a dramatic reduction in paraphrenic symptoms (Eastwood *et al*, 1981).

Investigation for sensory deficit is preliminary to mental state examination of the elderly, as hearing loss has been shown to affect cognitive examination results (Ohta *et al*, 1981) and may well affect the reliability of responses throughout the interview. Conversely, clinical research in the area of compliance with recommendations for hearing aids indicates psychological state and social context may be critical factors in determining outcome of aural rehabilitation programmes (Alberti, 1977).

Results to date support the need for further study of these inter-relationships, and the application of findings in the assessment and treatment of geriatric populations. Investigations are most apt to advance knowledge in this area, and to result in practicable recommendations, if conducted conjointly by speci-