Dr Berrios ignores the literature on brainstem auditory hallucinosis. Cambier *et al* (1987) reported four such cases with typical religious/patriotic/music-hall themes, attributed to auditory deafferentation. In fact, the site of lesion was ambiguous, although all had brainstem lesions and none cortical ones. All were bilaterally deaf and auditory nerve lesions could not be excluded audiologically; the brainstem neurological symptoms resolved but not the deafness. A further case with an apparently pure brainstem lesion had primitive auditory hallucinations during one night of insomnia.

I conclude that Dr Berrios's review (and mine for that matter) were both incomplete, and it is unfortunate, but predictable, that his review is now taken as the last word up to 1989 on musical and related hallucinations.

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Failure to convulse with ECT

SIR: Failure to convulse with electroconvulsive therapy (ECT) has been estimated to occur on up to 21% of occasions that the electrical stimulus is administered (Pettinati & Nilsen, 1985). The topic has received much attention in recent years in the *British Journal of Psychiatry* (e.g. *Journal*, January 1988, **152**, 134–136; *Journal*, April 1988, **152**, 571; *Journal*, May 1988, **152**, 712–713). Following my own correspondence (*Journal*, May 1990, **156**, 747–748) I have been made aware of the Royal College's recommendations (Freeman *et al*, 1989) concerning what should be done if there is no observable seizure during ECT. While welcoming these guidelines, it is interesting to contrast them with those of the American Psychiatric Association (1990). The two regimens differ, for example, in:

(a) the number of restimulations that can be applied
(b) the requirement that a designated time elapse between restimulations
(c) the magnitude of the parameters of the repeat stimuli.

Although unsafe practice cannot be inferred from diverse practice, there clearly remains a need for a more uniform protocol, validated by empirical data. It is sobering to recall that when ECT was first attempted, in mid-April 1938, the initial stimulus of 70 volts for 0.2 seconds was unintentionally subconvulsive. Cerletti remembered (Cerletti, 1950) that a voluble discussion then broke out among the spectators, who included Bini, Longhi, Accornero, Kalinowski and Fleischer. Most objected to a further shock. The patient himself protested: "Non una seconda! Mortifera!" (Not another! It will kill me!). With some trepidation Cerletti decided to administer another stimulus of 110 volts for 0.5 seconds and the patient convulsed. It is a sad reflection on our discipline and, in this era of quality assurance, perhaps unsatisfactory that the protocols that have been devised to allay Cerletti's trepidation continue to lack empirical support and are often conflicting.

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Anorexia nervosa in the elderly

SIR: I read the report of "anorexia nervosa" in a 73-year-old woman by Cosford & Arnold (*Journal*, February 1991, **158**, 286–287) with interest. This lady suffered her (first?) episode of marked weight loss, food avoidance (why?), fear of weight gain and amenorrhoea after a loss at the age of 23 years. She received an unstated treatment over nine months as an in-patient. Fifty years later she "suffered a relapse, with severe weight loss, a distorted body image and a fear of becoming fat". This time she was put on a strict diet and was discharged after five months. This makes 14 months in-patient treatment. Is there an alternative explanation? Was it an atypical affective disorder. Did the passage of months (*and* the affective episode) provide the 'cure'?

Bernstein (1972) described the successful treatment of a 94-year-old woman with "anorexia nervosa"

with electroconvulsive therapy. The reader was left wondering why this lady with a delusion that her food was being poisoned was not diagnosed as chronic paranoid schizophrenia.

Surely it would be wiser to apply the terms atypical anorexia nervosa or anorexia nervosa-like to such cases (Arya, *Journal*, February 1991, **158**, 285–286) until more is known about their (psycho-) pathogenesis.

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Chronic fatigue syndrome

SIR: I read with interest the article by Hickie *et al* (*Journal*, April 1990, **156**, 534–540) about the chronic fatigue syndrome (CFS). It inspires a few reflections concerning the criteria and the study, and concerning the existence of the CFS itself.

Their criteria are of two orders: psychological and immuno-infectious. It risks associating two different types of patients: some with post-infectious syndrome and some with psychological problems. Fatigue is one of the most common symptoms in medicine (Adams, 1980; Bugard, 1989). Patients with 'fatigue', 'concentration/memory impairment' and occasional 'lymphadenopathy' may have diagnosis of CFS. What is the frequency of lymphadenopathy in the general population? What do we know about relations between fatigue, depression and immunological deficit? Is not chronic fatigue usual in infectious diseases and after?

What is the interest of a control group "selected from the in-patient and out-patient psychiatric services"? Why not from medical units? Do the authors suppose that the difference between the group of patients with CFS and the control group is necessarily non-psychiatric?

The authors find 29 cases of depression among 48 patients with CFS but they say CFS is different from depression and near medical pathology. Is CFS a medical equivalent of depression (Rodin & Voshart, 1986)? We might test this hypothesis by using antidepressant drugs in CFS or looking for the presence of the same biological disorders both in CFS and depression. The GHQ and Zung scores are elevated as with medical patients. Severe depressive disorders are rare in CFS. However, this quantitative result does not exclude the association of depression and CFS.

The authors make the interesting hypothesis of clinical similarities between CFS and depression. They show it to be false, but CFS could be culturally differentiated depression with overmedicalisation. The patients with CFS had the "conviction that they are physically ill" and "they held this belief and rejected psychological interpretations". According to Balint (1972), a medical rather than a psychological diagnosis is favoured, which could increase diagnosis of CFS and reduce those of depression or hysteria and other equivalent diagnoses in DSM–III–R (American Psychiatric Association, 1987). As stated by the authors, the patients with CFS have a "reluctance to accept psychological interpretations of their somatic symptoms" and have a high score on the denial subscale. Criteria for CFS must be more stringent (e.g. previous history of documented infectious illness, no depression, presence or not of psychiatric symptoms, etc).

Further studies are required to remove the confusion between CFS and depression. They should analyse the psychological context at the beginning of CFS and its psychological evolution. They will define whether fatigue is a result of, or only increased by, infectious disease, whether depression is cause or consequence, and whether CFS is a morbid entity or the somatic expression of a psychological disorder.

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Family intervention

SIR: There is a point raised by McCreadie *et al* (*Journal*, January 1991, **158**, 110-113) which merits special emphasis. The authors noted the disappointing results of family intervention when compared with previous accounts (see, for example, Smith & Birchwood, *Journal*, May 1990, **156**, 654–660). The authors, in their discussions of this discrepancy,