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"