These findings support previous reports in which reduced nocturnal melatonin has been observed in major depression, particularly of the melancholic subtype (Brown *et al*, 1985; Claustrat *et al*, 1984). We therefore agree that urinary aMT6s is a practical index of melatonin output from the pineal gland and, like serum melatonin, does not appear to be increased in patients with anorexia nervosa at low weight.

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Lithium-Induced Paranoid Hallucinatory State

SIR: *Case Report*. A 38-year-old man with a history of manic depressive illness was admitted in a manic phase in July 1988. He was hyperactive, garrulous, aggressive, disinhibited, showed thought pressure, and was exposing himself. However, he had no perceptual disturbance or paranoid ideas. His sensorium was clear. Administration of haloperidol and chlorpromazine in high doses was ineffective, and caused intolerable sedation and extrapyramidal symptoms; neuroleptic medications were therefore discontinued. The patient was then given lithium carbonate (500 mg t.d.s.) and his blood lithium level was maintained at 0.45 mmol/l. This medication appeared to be well tolerated and improved his mood and behaviour, but unfortunately he experienced paranoid ideas towards the staff, became disorientated, and had visual and auditory hallucinations. He was convinced that he heard people telling him to do things and go to places such as shops. He was also convinced that he saw his brother speaking to the staff when, in fact, he never visited the ward. He came out of his room naked and alleged that someone had taken away his clothing. His paranoid ideas and hallucinations were most prominent at night and in the morning at the transition between sleep and wakefulness. His biochemical investigations and EEG revealed no abnormality. His lithium therapy was discontinued, and this resulted in complete abatement of the paranoid ideas and the hallucinations within 24 hours. His mood was then stabilised on carbamazepine, and he remained symptom-free on carbamazepine (100 mg b.d.) at follow-up some four months later.

It has been reported that certain individuals may be more vulnerable to the neurotoxic effects of lithium; in such cases psychotic manifestation can occur, even at therapeutic blood levels (Reynolds *et al*, 1982). My case highlights the uncommon lithium-induced psychotic phenomenon, which is probably due to the interaction of lithium and endogenous opioid systems. However, it appears to be a transitory reaction which has complete recovery at the discontinuation of the offending agent.

Sandyk & Gillman (1985) reported a case of lithium-induced visual hallucinations. Furthermore, it has been shown that lithium may interact with opioid receptors to produce increased activity of the endogenous opioid system (Stengaard-Pedersen & Schou, 1982). Increased activity of the endogenous opioid system has been linked to psychotic behaviour, including hallucinations (Berger *et al*, 1982).

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Prediction of Outcome After Treatment for Stuttering

SIR: Knowledge of variables that predict treatment success with adult stutterers is of utmost importance because of their strong tendency to relapse after therapy (Boberg, 1981). Consequently, Andrews & Craig's report (*Journal*, August 1988, **153**, 236–240) is extremely interesting because it claims to have identified three treatment goals (stutter-free speech,

normal communication attitudes, and an internalised locus of control) that predict long-term success following stuttering therapy. However, we have serious reservations concerning the validity of their predictors.

Firstly, treatment success was assumed when clients were speaking with a frequency of stuttering as high as 2% of syllables uttered during a telephone call made in the clinic to a stranger 10–18 months "after treatment concluded". It is generally accepted that stuttering treatment success can only be determined through multiple assessments over time in a variety of beyond-clinic conditions using evaluations of speech performance and speech quality (Bloodstein, 1987). Drs Andrews & Craig's criteria do not begin to approach this standard. The inclusion of up to 2% syllables stuttered, for instance, is not only incongruous with a successful treatment criterion (individuals may even require treatment for this level of stuttering), but it is also absolutely incompatible with their claim that those who met this criterion were "fluent" (see Table II of their paper).

Secondly, the authors claim that a single within-clinic telephone call has been "shown to be a valid and reliable measure of treatment outcome (Andrews & Craig, 1982)", yet their 1982 report makes no such claim at all. Such a claim would be quite difficult to justify, because the disorder is renowned for its variability across speaking situations (Bloodstein, 1987). The problem with the external validity of this task is magnified because their therapy actually trained this particular task in clinic conditions (see Andrews *et al*, 1987) but did not evaluate it outside of those conditions.

Thirdly, Andrews and his colleagues have indicated elsewhere (see Andrews *et al*, 1987) that many of their clients participate in self-help groups after formal treatment ceases in order to regularly practice their 'fluency skills'. If these subjects also participated in these groups then, arguably, treatment actually continued through to the point of "outcome" evaluation. Since the number of subjects engaged in these regular speech practice programmes was not indicated, it is possible that the "successful group" simply contained more members of these groups.

Fourthly, recent evidence demonstrates that clients' responses to the communication attitudes scale used in this study are largely based on the clients' current speech behaviour rather than their communication attitudes (Ulliana & Ingham, 1984). This factor may also operate in the clients' responses to the locus of control behaviour scale (Ingham, 1989). Consequently, it is entirely possible that the three treatment goals predicted "successful treatment" outcome because they yielded more speech performance information and not, as Drs Andrews & Craig claim, data on attitudes or perceived source of control.

Finally, a number of authorities have argued that a clinically valid outcome evaluation not only requires adequate beyond-clinic sampling but also appropriate measurement tools (Bloodstein, 1987; Boberg, 1981). Because Drs Andrews & Craig's therapy programme may ultimately produce speech that is unnatural sounding, it is now recognised that this type of therapy needs to incorporate evaluations for speech quality and is compromised if it does not (Bloodstein, 1987). For all of these reasons we believe that Drs Andrews & Craig's predictors of successful outcome have dubious experimental and clinical validity.

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Conversion Disorders and ECT

SIR: Dabholkar (*Journal*, August 1988, **153**, 246–247) reported that a 20-year-old male patient developed hysterical catatonia on two separate occasions following the accidental death of his father, and after villagers threatened to burn his house down, respectively. On the first occasion, his hysterical catatonia (and other conversion symptoms) were successfully treated with ECT, oral haloperidol (up to 5 mg per day), and "benzodiazepines and antidepressants in low dosage". Five months later (during which time the patient discontinued his medications), the patient re-developed hysterical catatonia, which was successfully treated with ECT and "oral diazepam in