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,