which adequately controlled the blood pressure without adverse effect on erection.

From these observations it would appear that excessive alpha adrenoceptor stimulation, such as can result from the unmasking effect of a beta blocker, inhibits erection. It can be postulated that some patients may have an increased sensitivity of the alpha adrenoceptors involved in the erectile mechanism. Activation of the sympathetic nervous system during sexual arousal and anxiety (induced by previous failure of erection) would therefore inhibit erection in such patients by excessive alpha adrenoceptor stimulation. This hypothesis provides a rationale for a therapeutic trial of an alpha adrenoceptor blocking agent such as phenoxybenamine in the management of erection inadequacy.

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Reference

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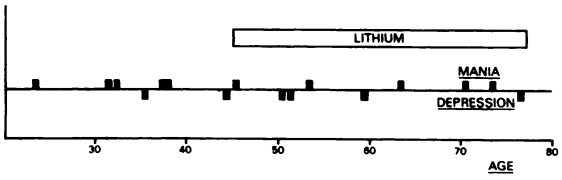
30 YEARS ON LITHIUM

DEAR SIR,

The account by Chiu *et al* (*Journal*, October 1983, **143**, 424–5) of renal function in the patient who must have held the world record for length of time on lithium is very interesting. It is also interesting to prepare from their data on his episodes of illness a chart which shows that the treatment may have helped him only modestly.



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RAPID EYELID TREMOR AFTER A MASSIVE PHENOTHIAZINE OVERDOSE

DEAR SIR,

A 20 year old man with 5 previous admissions, the first 17 months earlier, who was diagnosed variously as suffering from schizo-affective or manic-depressive (manic) psychosis was admitted after an overdose of 80 × 5 mg fluphenazine tablets and 6 × 0.5 mg benztropine tablets, 2 days previously. On examination he had a rapid twitching of his upper eyelids in both the closed and open position, synchronous with the tremor of his hands and tongue. There was a superimposed slow spontaneous blinking of 8 blinks per minute (normal ± 12 blinks per minute—Carney and Hill, 1982). Ocular movements were normal. The glabellar reflex showed non-habituation, and there were other Parkinsonian features. There were no signs of tardive dyskinesia; and there were no signs of psychosis.

He was given benztropine 2 mg intravenously. Within 20 minutes the eyelid tremor had markedly decreased. He reported feeling less stiff, and there was decreased jaw stiffness. The glabellar response remained non-habituated. He was subsequently given benztropine 2 mg bd orally. One week later, the tremor of the eyelids had ceased; but the fine tremor of the hands persisted, and blinking was still slow—10 blinks per minute. The glabellar reflex habituated after 3 taps.

Blink rate is decreased in Parkinson's disease (Hall, 1945), the latter sometimes being indistinguishable from drug-induced Parkinsonism (Baldessarini, 1980); but eye blinking is increased in schizophrenia and tardive dyskinesia (Stevens, 1978), and in Gilles de la Tourette syndrome (Cohen *et al*, 1980).

Penders and Delwaide (1971) showed a return towards normal eye movements in Parkinsonian patients treated with L-dopa or amantidine. Conversely, dopamine blockade by neuroleptics reduces the blink rate and thought disorder in schizophrenics (Karson *et al*, 1981a). Reduction of dopamine blockade as in Stevens' patients (Stevens, 1978) who were medication-free for 1–6 months, leads to an increase in blinkrate. Also, Karson *et al* (1981b) found an increased blink rate to apomorphine after haloperidol discontinuation. The central role of dopaminergic blockade in abnormal eye movements was further illustrated by the finding (Karson *et al*, 1983) of an inverse relationship between spontaneous blink rate and platelet monoamine oxidase activity.

Here, there was a decrease in spontaneous blinking, in keeping with the picture found in drug-induced Parkinsonism i.e. a dopamine blockade. The rapid tremor of the eyelids, synchronous with the tremor of the hands and ameloriated by benztropine, is thought