LITHIUM: AN ANAESTHETIC RISK

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SUMMARY
An abnormal recovery from an anaesthetic for electro-convulsive therapy is reported. A serum lithium concentration within the toxic range without any accompanying symptoms or signs was found subsequently. The dangers of anaesthesia for patients undergoing electro-convulsive therapy and who are also receiving lithium are discussed.

Lithium (usually as lithium carbonate) has been used for the treatment of manic depressive illness since 1949 (Cade, 1949). About that time it fell into disuse in general medicine because its toxicity had led to several deaths (Corcoran, Taylor and Page, 1949; Hanlon et al., 1949). It is now used widely both for the treatment and prophylaxis of manic depressive illness. It is considered to be safe when used in combination with other drugs and treatments, including electro-convulsive therapy (e.c.t.) (Bastrup and Schou, 1967; Schou, 1968).

Controlling the serum lithium concentration to within the therapeutic range of 0.8-1.4 m.equiv/l. can be difficult. It is generally agreed that the lithium concentration must not exceed 2 m.equiv/l. (Prien, Caffey and Klett, 1972).

This case report describes the problems which occurred when a depressed patient on lithium therapy received a general anaesthetic for electro-convulsive therapy.

CASE REPORT
A 64-year-old woman had suffered from recurrent depressive illnesses, although two attacks of manic behaviour had followed treatment for depression. She was first admitted to hospital in 1954 because of depression. Subsequently she had been admitted on fifteen occasions. As is common in the affective disorders, her attacks had become progressively more frequent and she had spent most of the previous 10 years, and all of the previous 3 years, in hospital. Each depressive illness lasted about 3 weeks, and was separated from the next illness by an interval of about 3 or 4 weeks when she was free from symptoms. At the beginning of each attack of depression she was suicidal but quickly passed into a state of depressive stupor. It was felt that an attempt should be made to relieve her recurrent and increasingly severe depressions with lithium carbonate. This was given in doses ranging from about 3.4 m.equiv/l. Lithium carbonate treatment was stopped and a week later the serum lithium concentration was 0.5 m.equiv/l. At this time, the patient received a further anaesthetic for e.c.t. using the same drugs and techniques and she recovered uneventfully.

DISCUSSION
At concentrations above 2 m.equiv/l., lithium usually produces somnolence, lethargy, muscle weakness, stupor and coma, but serum lithium levels of up to 3.6 m.equiv/l. without toxic symptoms have been reported (Allgén, 1969). These are also some of the toxic effects of the barbiturates and an additive effect between lithium and the barbiturates could be expected. The common causes of prolonged unconsciousness following barbiturate anaesthesia were excluded in this patient. Although depressed patients may have a low fluid intake she was given lithium carbonate 250 mg three times a day. Apart from lithium carbonate she was receiving no other drug therapy.

During the year the patient received two courses of e.c.t. Lithium carbonate therapy was being given during the second but not the first course.

The same drugs and techniques were used for all of her anaesthetics. The premedication was atropine sulphate 0.6 mg given intramuscularly 1 hour before anaesthesia which was induced with sodium methohexitone 60 mg followed by suxamethonium bromide 30 mg, both given i.v. IPPV with oxygen was given from a mask before and after each treatment. The problem occurred on one occasion during the second course of e.c.t. On examination before this anaesthetic she was depressed and withdrawn, which was characteristic of her psychotic state. She was physically normal and showed no signs of lithium toxicity, drowsiness, tremor or gastro-intestinal symptoms. She was given the usual anaesthetic and at no time was she cyanosed or hypotensive. Spontaneous respiration returned within the expected time but the patient could not be roused for over 2 hours, and she remained drowsy for the rest of the day. A venous blood sample was taken for estimation of urea and serum electrolytes (sodium, potassium, bicarbonate and chloride) and lithium concentration. Her blood urea and serum electrolyte concentrations were normal but the serum lithium concentration was 3.4 m.equiv/l. Lithium carbonate treatment was stopped and a week later the serum lithium concentration was 0.5 m.equiv/l. At this time, the patient received a further anaesthetic for e.c.t. using the same drugs and techniques and she recovered uneventfully.

not uraemic, nor was she on any other drug therapy. She had always recovered normally from anaesthetics before this episode, at times when her serum lithium concentrations were within the normal therapeutic range, and again she recovered normally a week after this event when her serum lithium concentration was 0.5 m.equiv/l. This suggests that the abnormally raised serum lithium concentration was responsible for this complication.

This risk can be minimized by monitoring the serum lithium concentration frequently, although it should be remembered that the concentration of lithium may change rapidly in association with an inadequate diet. It would perhaps be prudent to omit lithium therapy for at least 24 hours before an anaesthetic. On the other hand, since lithium is of doubtful value in an actual attack of depression (Noack and Trautner, 1951; Fieve, Platman and Plutchick, 1968), it might be better to avoid lithium altogether during a course of e.c.t.

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REFERENCES


