(Fig. 1); after 18 days a cystogram showed only a small extravasation (Fig. 2); and 32 days after operation the bladder appeared normal and the catheter was removed. By this time the perineal wound was healed and the anal sphincter was felt to be intact. Forty-seven days after the initial injury the colostomy was closed and the patient was discharged home, continent of urine and faeces.

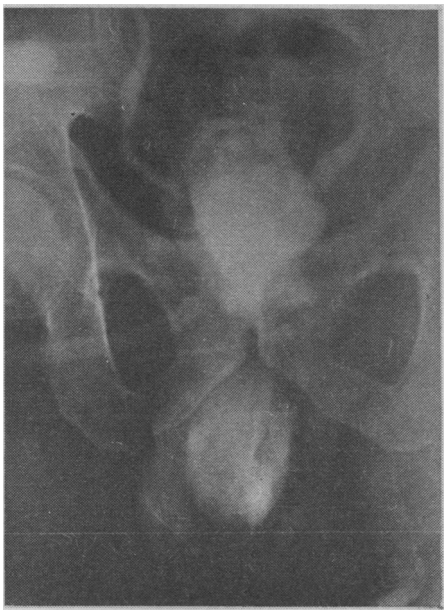

FIG. 1—Case 2. Intravenous pyelogram taken six days after operation.

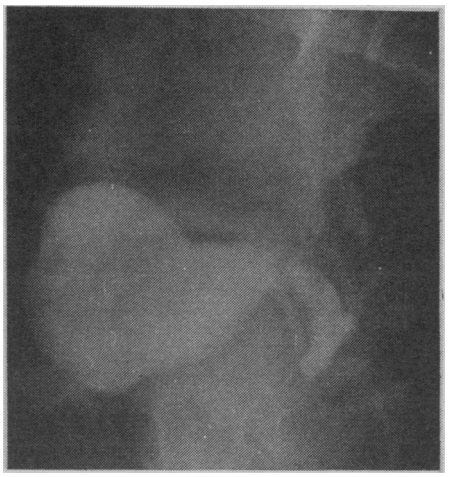

FIG. 2—Case 2. Cystogram taken 18 days after operation.

## Comment

When there is a history of a fall on to a sharp or protruding object, particularly if there is rectal bleeding, perforation of the rectal wall must be suspected. Abdominal examination may show signs of peritonitis. The urine should be examined; if it is blood-stained a cystogram may show the site and extent of damage to the bladder. Haematuria, however, does not necessarily indicate bladder perforation. Plain *x*-ray examination may show free gas under the diaphragm. A general anaesthetic is required for satisfactory examination, and proctoscopy will usually show the laceration in the anterior rectal wall. Laparotomy is indicated if there is any doubt about peritoneal involvement. At operation careful exploration must exclude other intraperitoneal injuries and a left iliac colostomy must be established. If the bladder is injured a perivesical drain and catheter are essential. After careful surgical toilet the perineal wound is left to heal by granulation.

I wish to thank Mr. L. T. Cotton for his advice in preparing this paper, Mr. R. M. Feroze for permission to publish details of the patient under his care and Mr. A. Yates-Bell for his help at the operation of the second case.

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# Peritoneal Dialysis for Lithium Poisoning

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Overdosage of lithium carbonate can cause serious intoxication, and several fatalities have been reported (Corcoran *et al.*, 1949; Hanlon *et al.*, 1949; Stern, 1949; Schou *et al.*, 1968; Hawkins and Dorken, 1969). There is no specific antidote, and treatment has been limited to supportive measures and attempts to increase the removal of lithium from the body. Saline infusion with or without forced diuresis and alkalinization of the urine is usually sufficient (Schou *et al.*, 1968; Thomsen and Schou, 1968). In two patients treated by haemodialysis (Amdisen and Skjoldberg, 1969; Hawkins and Dorken, 1969) serum lithium levels rose markedly again after the haemodialysis was stopped, and one died. Presumably this rebound effect was due to delayed redistribution of lithium from the tissues. In the following case of severe overdosage the patient was treated with peritoneal dialysis because it was thought that prolonged continuous dialysis would allow time for the plasma and tissue lithium levels to equilibrate.

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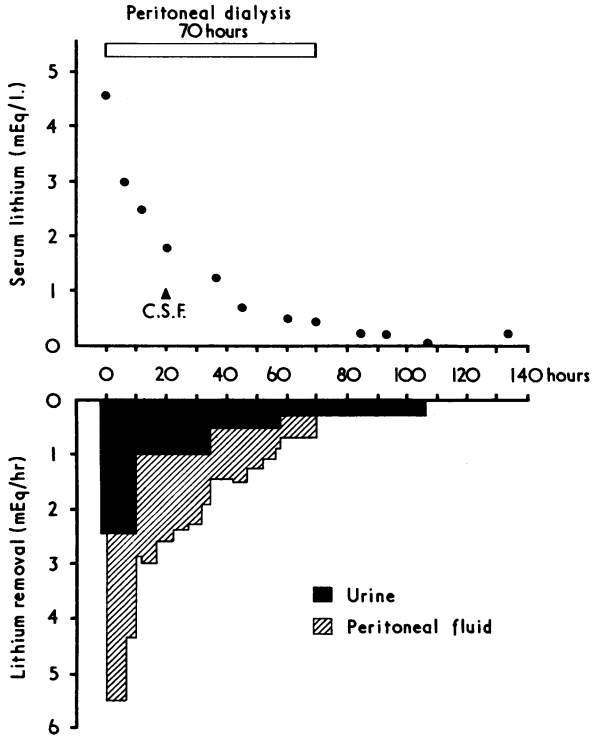
## Case Report

A 45-year-old man who had been on lithium carbonate 2,700 mg daily for recurrent mania developed on the day before transfer to our department the classical picture of lithium intoxication, with retching, tremor, myoclonic contractions, and a decreased level of consciousness. On admission he was comatose, responding intermittently to painful stimuli. Respiration was regular. The head remained preferentially turned to the left. The pupils were central,

equal, and normal in size. Jerky eye movements were present. The face was expressionless. Arms and legs were in extension and a fine tremor (8½ to 9 contractions per second) with myoclonic contractions was noted. Cog-wheel rigidity was present. After stimulation, and occasionally spontaneously, paroxysms of increased tremor and myoclonus occurred, most pronounced in the left arm, while he simultaneously reiterated in clonic fashion, "ja, ja, ja, ja." The reflexes were depressed and symmetrical. The blood pressure was 130/70 mm Hg, the pulse was regular at 120 per minute, and there were no signs of cardiac failure.

Peritoneal dialysis was started three hours after admission, when the serum lithium level was 4·6 mEq/l. (normal therapeutic range 0·8 to 1·6 mEq/l.) The catheter was introduced in the midline below the umbilicus and 2 litres of dialysate was exchanged every hour. The ensuing serum lithium levels and the removal of lithium from the body, expressed in mEq per hour in dialysate and urine, are shown in the Chart.




Serum lithium levels and lithium removal during peritoneal dialysis.

Because of the patient's extreme hyperactivity physiotherapy was impracticable, and he would not retain a pharyngeal airway. Only with much difficulty was a stomach tube passed. Benztropine mesylate 1 mg intravenously did not suppress the muscular activity, which was suggestive of a disturbance in the extrapyramidal system, but 15 mg of diazepam intravenously had some effect. Thirty-six hours after the start of dialysis the patient responded to simple commands, and after a period of aphasia complete consciousness returned on the fourth day. The neurological findings also reverted to normal. Dialysis was stopped after 70 hours, when the serum lithium level had fallen to 0·5 mEq/l. There was no subsequent rebound.

## Comment

The mortality from severe lithium intoxication is high—in the reported cases nearly half of the patients who developed coma or subcoma died (Corcoran *et al.*, 1949; Hanlon *et al.*, 1949; Stern, 1949; Schou *et al.*, 1968; Hawkins and Dorken, 1969). Usually death was not due directly to lithium intoxication but to pulmonary complications of the coma. Our case showed the difficulties in nursing such patients. The viscous secretions which constantly accumulated in the upper respiratory tract in the acute phase were almost impossible to remove owing to the patient's hyperkinetic state and his tendency to chew through anything placed in his mouth.

Peritoneal dialysis was preferred to haemodialysis because of the known delay in intracellular and extracellular lithium levels reaching equilibrium (Schou *et al.*, 1968). Though it has been suggested that lithium is distributed evenly throughout the body water Schou *et al.* (1968) have found in postmortem studies that much higher concentrations of lithium are present in bone and muscle than in liver. Furthermore, the passage of lithium across the cell membrane is slow. In-vitro and in-vivo experiments of Ljungberg and Paalzow (1969) on dogs showed that the distribution rate for lithium between plasma and erythrocytes was from 8 to 10 hours. This slow exit of lithium from the cells and the relatively large stores in muscle and bone would account for the rebound phenomenon after haemodialysis.

The half-life of the serum lithium levels in this patient was 14 hours during dialysis, which compares favourably with a half-life of 30 to 100 hours found in patients treated conservatively (Schou *et al.*, 1968). The determination of lithium clearances showed that the renal clearance varied from 10 to 12·5 ml per minute and that the peritoneal dialysis clearance was slightly higher at 13 to 15 ml per minute. Thus peritoneal dialysis can remove large quantities of lithium from the body. Though repeated haemodialysis would do the same, peritoneal dialysis has the added advantage of being more generally practicable.

We are grateful to Dr. H. M. van Praag and Dr. T. van Manen for their advice during the treatment of this patient.

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