1979 in our acute spinal cord injury unit there have been almost equal numbers of divers who sustained their injuries in the two sites. In most cases the cervical spine was fractured and the spinal cord crushed when the top of the head struck the bottom of the lake or pool and not when the head struck the water.

We estimate, from the experience in our unit and from Botterell and colleagues' data,[5] that at present there are approximately 150 major spinal cord injuries per year in Ontario. Approximately 10% of these injuries, or 15 per year, are due to diving accidents. There are probably an equal number of cervical fractures without cord injury (but sometimes with nerve root injury), as well as instances of drowning resulting from paralysis due to spinal cord injury.

Table III—Neurologic recovery among the 38 divers

	Neurologic grade	
No. of patients	At time of admission	At time of follow-up
16	1	1
3	1	2
3	1	4
3	1	*
3	2	2
2	2	4
3	2	7
2	4	7
1	4	8
1	4	†
1	7	10

*These patients died within a year of their accident.
†This patient was lost to follow-up.

Because diving is a major cause of spinal cord and nerve root injury among the young people of Ontario, a concerted effort should be made to bring this fact to their attention. For example, educational programs about the hazards of diving into shallow water should be conducted in the spring and summer each year. The medical profession, the Canadian Red Cross Society, the Royal Life Saving Society, St. John Ambulance, the ministries of health and education, and the media could each play a role. Signs warning against the hazards of diving into shallow water should be mandatory at public pools and beaches, and their use encouraged in all private swimming areas.

Reckless diving must be regarded as one of summer's main hazards.

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Tachy-bradycardia syndrome related to lithium therapy

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Lithium salts, widely used in the treatment of manic–depressive illness, have been described as causing repolarization abnormalities detectable on surface electrocardiograms.[1] Recently sinus node dysfunction and sinoatrial block have also been reported.[2–4]

A case of tachy-bradycardia apparently related to lithium therapy during an acute inferior wall myocardial infarction is reported.

Case report

A 57-year-old man was admitted to hospital with a 2-day history of recurrent syncope. First he had experienced severe retrosternal pain radiating to the left arm and shoulder, associated with nausea, sweating and weakness. The pain lasted 3 hours and subsided spontaneously. One day later he began to have the attacks of syncope, which began suddenly and without warning, and after 2 days of numerous attacks he was admitted to the intensive care unit.

Risk factors for coronary artery disease included a 7-year history of diabetes mellitus controlled by diet, and 20 years of smoking a pack of cigarettes a day. During the past 14 years the patient had had numerous admissions to hospital for severe manic–depressive psychosis. Various medical regimens had met with poor results until lithium carbonate therapy was instituted. His symptoms had been well controlled for 4 years before this admission, when he was taking 900 mg of lithium carbonate per day.

The physical examination yielded essentially normal findings except for an atrial gallop at the apex and sinus bradycardia (heart rate 50 beats/min). A 12-lead electrocardiogram showed evidence of acute inferior wall myocardial infarction.

Shortly after the patient's admission, paroxysms of atrial fibrillation with rapid ventricular response developed. At the end of the dysrhythmia several periods of asystole lasting 6 to 9 seconds occurred in association with loss of consciousness (Fig. 1). A total of 2.4 mg of atropine was given intravenously over 5 minutes. Within the next 30 minutes several episodes of symptomatic tachy-bradycardia were ob-

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served. Thus, a temporary demand pacemaker was inserted. Serial enzyme measurements and electrocardiograms confirmed the diagnosis of recent acute inferior wall myocardial infarction. The serum lithium level at the time of admission was 1.46 mmol/l, and because of the cardiac rhythm disturbance the drug was discontinued.

Over the next 7 days sinus rhythm predominated but was interrupted by brief paroxysms of atrial fibrillation that were no longer followed by periods of asystole. After the 10th hospital day sinus node function was assessed by rapid atrial pacing, intravenous infusion of atropine and isoproterenol, and carotid massage. The results were normal (the corrected sinus node recovery time was 350 ms) and the prophylactic pacing was discontinued.

From the 11th to the 20th hospital day the patient remained free of cardiac dysrhythmias but began to show evidence of grandiose thinking and signs of hypomania. Because the symptoms could not be controlled by conventional measures, therapy with lithium carbonate was reinstituted and monitored. No dysrhythmia was observed until the 29th hospital day, when syncope again developed along with paroxysmal atrial fibrillation followed by long periods of asystole.

Permanent programmable demand ventricular pacing was undertaken, with the pacemaker set at a rate of 50 beats/min. After the patient's discharge from hospital a 24-hour Holter electrocardiogram showed persistent sinus bradycardia and paroxysmal atrial fibrillation. with normal pacemaker parasystole. No further episodes of syncope were reported.

Discussion

Sinus bradycardia is common after an acute inferior wall myocardial infarction, having a frequency as high as 41%.[5,6] This abnormality is usually transient, limited to the acute stage of the infarction and responsive to either atropine or isoproterenol infused intravenously.[7-9] In the patient I have described there were several atypical features: (a) the sinus bradycardia persisted for a long time after the myocardial infarction presumably occurred; (b) the bradycardia was resistant to a therapeutic dose of atropine given intravenously; and (c) there were periods of asystole, which demonstrated an unusual depression of the automaticity of subsidiary pacemakers. These abnormalities are compatible with the so-called tachy-bradycardia syndrome.[10]

The sinus node dysfunction in this patient was clearly related to the lithium carbonate therapy since the dysrhythmia gradually abated after the drug was discontinued and did not recur until lithium therapy was resumed (Table I). It is possible that only after the sinus node and junctional tissue had been rendered vulnerable by ischemic injury were the toxic effects of lithium fully realized.

From this case it is recommended that lithium salts be used with caution in patients with myocardial ischemia or pre-existing sinus node dysfunction.

Table I—Temporal relationship between cardiac dysrhythmia and serum lithium level

Day in hospital	Serum lithium level (mmol/l)	Rhythm
1–4	1.46	Atrial fibrillation with rapid ventricular response followed by long periods of asystole
5–20	0	Sinus rhythm
20–30	1.2	Atrial fibrillation with rapid ventricular response followed by long periods of asystole

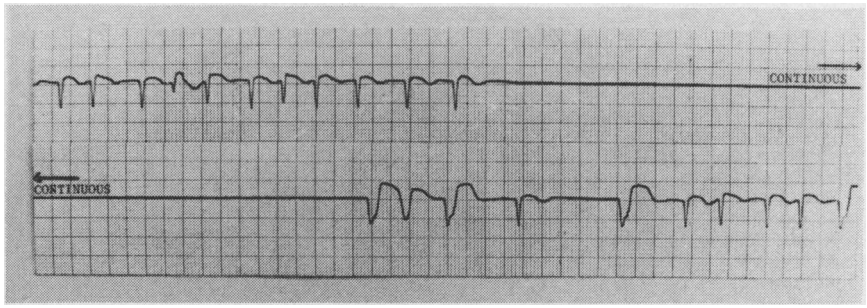

FIG. 1—Atrial fibrillation with rapid ventricular response, and 7-second periods of asystole terminated by ventricular escape beats.

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