Diving: a frequent and potentially preventable cause of spinal cord injury

CHARLES H. TATOR, MD, PH D, FRCS[C]; VIRGINIA E. EDMONDS, RN; MARY L. NEW, MPH

In our recent retrospective study of 358 patients in Toronto with acute spinal cord injuries, we identified four main causes of these injuries: traffic accidents (34%), work-related accidents (29%), sports and recreational accidents (15%) and falls at home (9%).[1] Of the 55 sports and recreational injuries 38 (11% of all 358 injuries) were due to diving accidents. Kurtzke,[2] however, in a detailed epidemiologic analysis of spinal cord injuries, documented a lower incidence of such injuries due to diving in several other countries. This difference prompted us to analyse further the 38 spinal cord injuries due to diving in our series in a search for ways of preventing such injuries.

Methods

We reviewed the records of patients with acute spinal cord injuries admitted to either Sunnybrook Medical Centre or the Toronto General Hospital during the 25-year period 1948 to 1973. For inclusion in the study a patient must have been admitted to either hospital within 30 days of injury, and the record must have contained complete documentation of the injury, treatment and result. Patients who had received major treatment in other institutions before referral or who had spinal injuries without cord involvement were excluded. Similarly, those with nerve root involvement only and those with injuries at the L2 vertebral level or below were excluded. Neither hospital admitted children with spinal cord injuries. A total of 358 patients were included in the study. The information about them was computer processed by the University of Toronto Computer Centre with the use of the Statistical Package for the Social Sciences.[3]

The severity of the spinal cord injury was assessed in terms of the residual motor and sensory function below the level of the injury. The injuries were graded from 1 to 10: grade 1 denoted a complete cord deficit, with complete absence of voluntary motor function and sensation below the level of the injury; grades 2 to 9 denoted partial cord injuries. Frankel and colleagues[4] used a similar grading system.

In each case the grade was established retrospectively from the patients' records at the times of admission, discharge and final follow-up, which for patients with grade 1 lesions was at least 1 year from the time of injury, and for those with grades 2 to 9 lesions at least 1½ years. These follow-up times were thought to be sufficiently long to encompass most of the neurologic recoveries (or deteriorations) that could have occurred.

Results

Of the 38 patients with spinal cord injuries due to diving accidents 32 were male; this sex distribution is very close to that for cord injuries of other causes. However, the divers were significantly younger than the other patients, the median ages being 21 and 34 respectively.

Most of the diving injuries occurred in vacation districts, 35 in lakes; only 1 injury occurred as a result of diving into a swimming pool. Most occurred during the summer months, particularly July, and late afternoon was the most frequently recorded time of injury.

All the diving injuries but two were cervical, and the commonest vertebral level of injury was C5–6 (Table I). The commonest type of injury to the vertebral column was a posterior fracture-dislocation (Table II). Two patients in the series had no radiologically demonstrable vertebral injury.

Only 6 of the 25 patients with complete spinal cord injuries at the time of admission showed any recovery at the time of follow-up (though 3 had died by this time), whereas 9 of the 12 patients with partial cord injuries who were available for follow-up examination showed improvement (Table III).

Discussion

We found young men to constitute most of the victims of diving accidents resulting in acute spinal cord injuries. Most of the injuries were very severe (three were lethal), and almost all were cervical. Significant recovery of neurologic function was rare among those with complete cord injuries but common among those with incomplete injuries.

As expected, the peak incidence of diving injuries in Ontario was in the summer, mostly in the cottage and lake districts. However, it was surprising to find the marked preponderance of lakes over pools as the site of injury in the 1948–73 study period, because from 1974 to

Table I—Vertebral level of 38 acute spinal cord injuries in divers

Level	No. (and %) of patients
C4–5	9 (24)
C5	6 (16)
C5–6	15 (39)
C6	3 (8)
C6–7	3 (8)
C7–T1	1 (3)
T12	1 (3)

Table II—Type of injury to the vertebral column in the 38 divers

Injury	No. (and %) of patients
None	2 (5)
Anterior dislocation	2 (5)
Anterior fracture-dislocation	9 (24)
Posterior fracture-dislocation	14 (37)
Compression fracture	7 (18)
Burst fracture	3 (8)
Body fracture	1 (3)

From the acute spinal cord injury unit and the division of neurosurgery, Sunnybrook Medical Centre, University of Toronto

Reprint requests to: Dr. Charles H. Tator, Head, Acute spinal cord injury unit, Sunnybrook Medical Centre, 2075 Bayview Ave., Toronto, Ont. M4N 3M5

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

K.C. WONG, MD, FRCP[C]

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-

Reprint requests to: Dr. K.C. Wong, Bluffton Community Hospital, 139 Garau St., Bluffton, OH 45817, USA