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Late Asystole in High Cervical Spinal Cord Injury: Case Report

P. L. Silbert, MB BS,[1] M. J. E. Davis, MB BS, FRACP[2]
[1]*Neurology Registrar, Royal Perth (Rehabilitation) Hospital,* [2]*Cardiologist, Royal Perth Hospital, Perth, Western Australia.*

Summary

A patient with delayed episodes of extreme bradycardia, asystole and syncope occurring 5 to 9 weeks after traumatic high cervical spinal cord injury is described. Temporary transvenous ventricular pacing followed by oral propantheline was required to prevent further episodes. The investigation, physiology and treatment of this arrhythmia in tetraplegia are discussed.
Key words: *High cervical spinal cord injury; Late asystole; Cardiac pacing.*

Bradycardia occurs commonly following acute cervical spinal cord injury and tetraplegia. A recent study identified significant bradycardia (defined as heart rate less than 50 beats per minute) in 22 (26%) of 83 consecutive patients with traumatic quadriplegia. In all cases the bradycardia was limited to within 3 to 5 weeks following spinal cord injury (Winslow *et al.*, 1986).

We describe a patient with traumatic high tetraplegia who exhibited episodes of asymptomatic bradycardia from the time of his injury. These persisted beyond 5 weeks and became increasingly severe resulting in extreme bradycardia, asystole and syncope. These episodes were on occasions unrelated to any precipitating stimuli. The investigation, physiology and treatment of this arrhythmia in tetraplegia are discussed.

Case history

A 16-year-old school boy sustained a bifacetal fracture dislocation of C3 on C4 vertebrae after diving into a shallow water hole. On initial assessment he was completely tegraplegic below C4 spinal cord level with priapism, areflexia and flaccid paralysis of all limbs. The bulbocavernosus reflex was present with absent deep tendon, anal and cremasteric reflexes. His pulse was 72 beats per minute and regular, the blood pressure was 100/60. Radiography of the cervical spine showed a C3–4 anterior dislocation. A halo brace was applied under

Correspondence to: Dr P. L. Silbert, Medical Registrar, Royal Perth Hospital, PO Box X2213, Perth, Western Australia 6001.

general anaesthesia and the C3 on C4 dislocation reduced to an anatomical position. The patient remained intubated, and was nursed in a horizontal bed with a side-back-side regime with constant traction through the halo brace. His initial course was complicated by Staphylococcus aureus and Haemophilus influenza pneumonia, which were treated with intravenous Cloxacillin and Ceftriaxone. A tracheostomy was performed. Episodes of bradycardia to a heart rate of 26 beats per minute were noted during tracheal suctioning.

Fourteen days following injury, sensation was present in the C4 dermatome on the right with a flicker of muscle movement in the right deltoid. Sensation was present in the C5 dermatome on the left with a flicker of movement in the left deltoid and bicep muscles. Following complete resolution of the bronchopneumonia his ventilator time was gradually reduced so that at day 35 he was using the ventilator for 2 hours only each night, and was maintaining satisfactory arterial oxygenation at other times.

On day 40 whilst resting quietly he complained of nausea, became pale and then lost consciousness for a period of approximately 30 seconds. He had no recollection of the event other than feeling sick and dizzy prior to the episode. On days 42 and 44 he had similar episodes, and arterial blood gases taken immediately following the loss of consciousness on one occasion revealed pH 7·40, pCO_2 46 mm Hg, pO_2 98 mm Hg, Bicarbonate 29 mmol/l, Base Excess +4, and Oxygen Saturation 98%.

On day 45 whilst receiving passive physiotherapy to his upper limbs, he complained of nausea and dizziness and was noted to be pale. Tracheal suctioning was performed; the patient lost consciousness for approximately 30 seconds during which there was no palpable pulse. A similar episode occurred spontaneously whilst the patient was on a cardiac monitor, with a heart rate of 17 beats per minute noted. In view of the documented bradycardia and the history of loss of consciousness due to presumed extreme bradycardia/asystole, often without any predisposing stimuli, medical therapy with anticholinergic drugs was not attempted. A temporary transvenous ventricular pacing lead was inserted using a right subclavicular approach, with pacing set at a rate of 60 per minute on demand. On day 51 the pacemaker was turned off and Holter monitoring commenced. On day 52, 1 hour after being positioned to the right lateral position and associated with no other stimuli, the patient complained of nausea and faintness, but with no loss of consciousness. These symptoms lasted approximately 60 seconds, following which he vomited with subsequent resolution of symptoms. Analysis of the Holter monitor recording for this period revealed a 22-second period of asystole (Figure). Ventricular pacing was recommenced at 60 per minute and continued using the temporary transvenous pacing wire with pacing thresholds of 0·5 to 0·75 mA. On day 61, without preceding stimuli, the patient experienced nausea and presyncope. The heart rate was noted to be 7 beats per minute, and pacing spikes failed to capture the ventricle. The patient vomited and the heart rate increased to 50 beats per minute. Twenty minutes later a further episode of extreme bradycardia to 5 beats per minute occurred, the rate again increasing after vomiting. The threshold for pacing was found to be 12 mA,

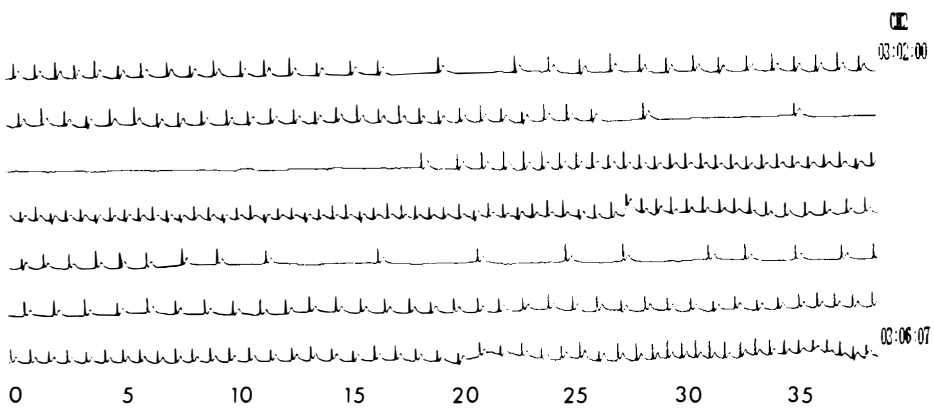

Figure Recording from Holter monitor demonstrating 22-second period of asystole. Enlargements at 25 mm per second of both Holter channels disclosed no atrial activity.

accounting for the failure to capture. The pacing lead was removed and no lead re-inserted, but propantheline 15 mg qid, with intravenous atropine before tracheal suctioning, was commenced with maintenance of a satisfactory heart rate and no further symptoms. On day 68 the intravenous atopine was ceased and the propantheline continued at the same dose. Seventeen weeks after injury the propantheline was gradually withdrawn over 3 days to facilitate bladder management. Holter monitoring was performed for 72 hours following cessation. Two asymptomatic periods of sinus arrest lasting 5 seconds were noted, but there was no symptomatic bradycardia. At review 6 months following injury, he remains neurologically unchanged, and asymptomatic from the cardiac point of view.

Discussion

Autonomic instability following cervical spinal cord injury is a common finding in the acute stages. Initial sympathetic hyperactivity has been well documented in animal studies and noted clinically associated with spinal cord pressure during cervical laminectomy (Greenhoot *et al.*, 1972). This phase is often missed in cases of spinal cord injury with the variable delays in transferring patients to intensive monitoring units. In cases of complete cervical spinal cord injury, an enhancement of the normal parasympathetic nervous system dominance on the heart occurs due to cervical sympathetic interruption. This results in a tendency to bradycardia, usually asymptomatic. In a minority of patients, superimposed vagal stimulation, through manoeuvres such as tracheal suctioning, may cause asystolic cardiac arrest (Frankel *et al.*, 1975).

In cases of spinal cord injury, before attributing bradycardia to autonomic instability, hypoxia must be excluded. Hypoxia may result from high cervical quadriplegia with impaired diaphragmatic function, superimposed atelectasis, consolidation and respiratory depression due to sedation or narcotic analgesia. Tracheal suctioning may cause both transient hypoxia and vagal stimulation, resulting in bradycardia. Whilst our patient's initial episodes of bradycardia, on days 1 to 10, were related to tracheal suctioning, the episodes of extreme bradycardia occurring from days 40 to 61 were, with one exception, not associated with tracheal suctioning or any other external stimulus. Hypoxia was considered unlikely to be contributory since arterial blood gas analysis performed immediately following an episode of extreme bradycardia with presyncope showed adequate oxygenation. Jennett (1970) has demonstrated that the response of heart rate to hypoxia is identical in normal subjects and those with chronic (greater than 2·5 months) complete cervical spinal transection. All patients showed an increase in heart rate with hypoxia and a subsequent decrease with reoxygenation, indicating that mechanisms other than cervical sympathetic outflow must be of importance in regulating heart rate, in particular in response to hypoxia and presumably other stresses. Other possible mechanisms of cardiac arrhythmias in these patients include brain stem injury or ischaemia, myocardial injury and vasovagal responses to physical or extreme emotional stresses.

Heart rate is under direct nervous system control from the medulla. Acute interruption of the cervical sympathetic outflow results in parasympathetic dominance initially. However, previous studies have suggested that the autonomic instability and tendency to bradycardia is limited to the 3 to 5 weeks, following the spinal cord injury (Winslow *et al.*, 1986). In this case, although asymptomatic bradycardia in response to vagal stimuli (tracheal suctioning) was demonstrated in

the initial weeks after injury, a previously unreported tendency to delayed episodes of extreme symptomatic bradycardia and asystole with no precipitating stimulus was also seen. These occurred between days 40 and 61 until they were controlled with medical therapy.

The method of treatment of bradycardia following spinal cord injury depends upon several factors. These include the age and general health of the patient, possible contributory medications, the time after spinal cord injury, and the level of spinal cord injury and whether it is complete or incomplete. It is also important to consider whether the patient is symptomatic or asymptomatic during the episodes, and whether they are felt to represent a threat to life. In our case we documented a 22-second period of asystole and presumably the episodes associated with loss of consciousness represented an even longer period of asystole. Whether the patient would have spontaneously regained cardiac activity without the external stimulation that occurred with each episode is unknown. It was noted that following vomiting the patient's heart rate returned to normal, although this was associated with external stimulation through repositioning the patient. The nausea and vomiting that occurred with each episode we believe to be presyncopal rather than vagal in origin as it was abolished by ventricular pacing.

As the bradycardia is due to dominant and/or excessive parasympathetic nervous activity, anticholinergic drugs such as atropine and propantheline may be effective as in this case. Sympathomimetic drugs such as isoprenaline may also be effective but are significantly more arrhythmogenic. In most cases bradycardia occurs in response to vagal stimuli and cholinergic blockade is therefore preferable. Atropine can be used intravenously or intramuscularly prior to any vagatonic manoeuvres such as tracheal suctioning. Although propantheline, a long acting anticholinergic agent, has been unsuccessful in previous cases (Frankel *et al.*, 1975), its initiation in combination with intermittent atropine was associated with symptomatic improvement in this case. Possible side-effects of propantheline therapy include dry mouth, blurred vision and difficulties with bladder control. Thus, in this case propantheline was gradually withdrawn 17 weeks after injury, to allow establishment of bladder function and control, without detrimental cardiovascular effects.

Winslow *et al.* (1986) stated that pacemaker insertion did not prolong life. Temporary pacing is indicated where medical therapy is ineffective and to protect the patient if vomiting, with its potential for aspiration in tetraplegics, occurs due to these episodes. Permanent pacing is unlikely ever to be indicated as bradyarrhythmia will ultimately resolve.

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