Novel Insights from Clinical Experience

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A Rare Instance of 'Cardio-Respiratory Pacing': Permanent Pacemaker Insertion for Symptomatic Bradycardia in a Quadriplegic Man Dependent on Diaphragmatic Pacing by Phrenic Nerve Stimulators

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Established Facts

- Spinal cord injury above T6 results in dysautonomia including hypotension and bradycardia.
- Electromagnetic interference is a serious concern in patients with cardiac pacemakers.

Novel Insights

- We describe a patient who required cardiac pacemaker insertion in the presence of phrenic nerve stimulators for ventilator support.
- This is the first reported instance of the successful combination of cardiac and respiratory pacemakers without electromagnetic interference.

Key Words
Permanent pacemaker · Diaphragmatic pacing · Phrenic nerve stimulator · Electromagnetic interference

Abstract
Patients with high spinal cord injury may present with significant cardiac dysautonomia. There is a dearth of data regarding electromechanical interference to cardiac pacemakers from phrenic nerve stimulators which are used in such patients for respiratory support. We report an instance of bipolar lead permanent pacemaker insertion for ventricular standstill in a man with quadriplegia following C2 fracture and the measures we adopted to minimise electromagnetic interference with phrenic nerve stimulators. To the best of our knowledge, this is the first reported instance of successful pacemaker insertion in a quadriplegic patient on long-term diaphragmatic pacing.

Introduction

High spinal injury results in altered autonomic state and consequent haemodynamic instability. Life support systems in such patients can cause electromagnetic interference (EMI) to permanent pacemakers. We present a case which highlights these issues.

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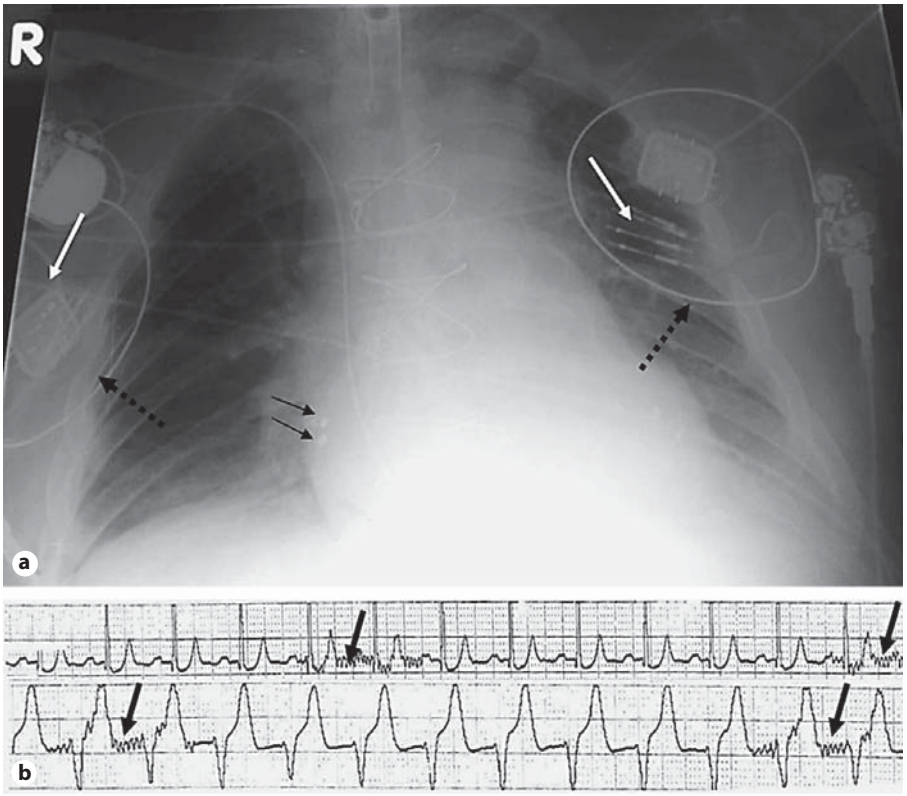

Fig. 1. a Chest X-ray: PNS components include bilateral internal electrodes (black arrows), implanted radio receivers (white arrows) and external antennae (black square dot arrows). The cardiac pacemaker can be seen at the right shoulder with the VVI lead in situ. The tip of the bipolar pacing lead was positioned at the apex of the right ventricle. A tracheostomy tube and sternal wires from the thoracotomy are also evident. **b** ECG trace in sinus rhythm (top) and in VVI mode (bottom). High-frequency oscillations (arrows) denote periods of phrenic nerve stimulation at a rate of 12 per min. It is evident from the lower trace (bottom) that ventricular pacing is not inhibited by the PNS impulse. Also, there is no evidence of any reciprocal interference to PNS function.

Clinical Presentation

A 67-year-old quadriplegic presented to the accidents and emergency department with a 3-day history of increasing dyspnoea. He had sustained a type 2 odontoid peg fracture 10 years previously, which had resulted in quadriplegia and diaphragmatic paralysis. Bilateral phrenic nerve stimulators (PNS) inserted by a low open thoracotomy approach provided daytime ventilatory support and mechanical ventilation via a tracheostomy was used at night. Prior to admission, he managed his own business successfully and coped well with his ventilatory support. He did not have a history of cardiovascular disease or other illnesses. He was admitted to the intensive treatment unit with sepsis secondary to a lower respiratory tract infection. Multiple episodes of presyncope and one documented episode of ventricular standstill (>5 s) occurred which prompted consideration of a permanent pacemaker. We noted that his PNS (Atrostim Jukka™; Atrotech Ltd., Tampere, Finland) generated a 9-volt impulse in bursts of 5 impulses (10–12 per min). Concerns were raised about interference between a permanent pacemaker and the PNS. A literature search revealed that there were no published reports of cardiac pacemaker insertion in patients with spinal cord injury and phrenic nerve palsy, although there are rare reported instances of cardiac pacemaker implantation in patients with congenital central hypoventilation syndrome on diaphragmatic pacing [1, 2]. After consideration, a bipolar lead VVI pacemaker was inserted by the right subclavian route (St Jude Verity™ ADx XL SC 5056; basal rate 60 bpm, hysteresis 50 bpm). The patient made a good recovery and was discharged a week later. Chest X-ray prior to discharge confirmed satisfactory lead position and ruled out serious complications (fig. 1a). Pacemaker interrogation on days 1, 30 and 180 showed no pacemaker interference during phrenic nerve stimulation (fig. 1b). He has not had any syncopal episodes in the following 2 years.

Discussion

Spinal cord lesions rostral to C3 result in complete diaphragmatic paralysis and require respiratory assistance by diaphragmatic pacing using PNS [3] or chronic ventilatory support. The PNS device consists of internal electrodes placed adjacent to the phrenic nerves on either side by open thoracotomy or thoracoscopy, connected to subcutaneous radio receivers under the anterior chest wall and an external transmitter with circular antennae powered by a 9-volt battery (fig. 2). The electrical impulse is transmitted by radiofrequency from the external antennae to the implanted receivers resulting in coordinated phrenic nerve stimulation. Patients with a level of spinal cord injury above T6 are at risk of dysautonomia from direct damage to the thoracic sympathetic outflow. This

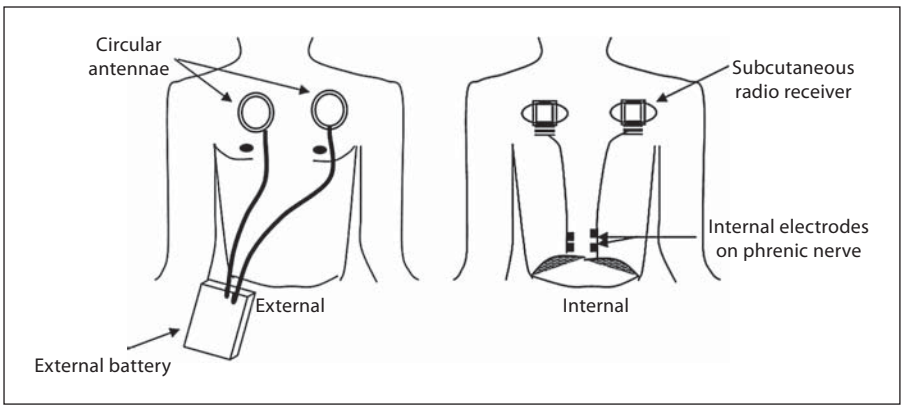

Fig. 2. PNS for diaphragmatic pacing.

may present in an acute, subacute or chronic pattern [4] and may be triggered by infections or procedures such as urinary catheterization and tracheal suction. Significant bradycardia may require permanent pacemaker implantation.

Our patient presented some unique challenges. He was dependent on diaphragmatic pacing following his spinal cord injury and the episodes of ventricular standstill were precipitated by intercurrent infection. There were concerns about EMI between the PNS and the permanent cardiac pacemaker. We used a bipolar ventricular electrode to minimize the interference from the phrenic nerve electrodes and opted for a right subclavian route to achieve minimum interference and maximum separation of the pacemaker pulse generator from the PNS system (fig. 1). We were successful in preventing significant electrical interference as evidenced by the Holter monitor trace post-procedure (fig. 2). One explanation may be that given the relatively low amplitude of the PNS signal seen on the surface ECG compared to the QRS voltage (although this may not accurately reflect the intra-thoracic PNS signal intensity), interference is easily negated by adjusting pacemaker sensitivity. Conversely, the bursts of PNS activity may not be affected by the permanent pacemaker, as the frequency of the ventricular pacing impulse is less than the PNS impulse and, furthermore, the PNS is not set in 'demand' mode. This allowed our patient to maintain his quality of life by continuing to use the PNS in the daytime which he preferred over 24-hour mechanical ventilation.

Conclusion

EMI is an important concern with implanted cardiac pacemakers and defibrillators [5, 6]. The presence or absence of clinically significant EMI depends on the strength of the source of interference, the distance from the source and the time of exposure to the source. Our case highlights a rare cause of EMI which was circumvented by appropriate choice of electrodes and optimum positioning of the pacemaker system.

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