

Successful weaning from mechanical ventilation using phrenic nerve stimulation

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Dear Editor,

Patients who survive cervical spinal cord injury at or above C4 may require long-term or permanent ventilatory support. We would like to report the first successful application of phrenic nerve stimulation (PNS) in Ireland to successfully wean prolonged mechanical ventilation in an adult patient after high cervical spinal cord injury.

A previously well 60-year-old male patient sustained a C2 odontoid peg fracture with spinal cord contusion from C1 to C3 following a fall from a horse. He was resuscitated at the scene of the accident, his lungs were mechanically ventilated and he was transferred to the Intensive Care Unit in the Mater Hospital. No surgical intervention was indicated and he was placed in a Miami J collar. One month after his injury, he remained fully alert and tetraplegic [C2

ASIA impairment grade A (complete) spinal cord injury]. The patient was dependent on full-time mechanical ventilation via a tracheostomy tube and despite minimal chance of meaningful improvement in his neurologic or respiratory status; he expressed a clear wish that long-term physiologic support should be continued.

An alternative to prolonged mechanical ventilation is electrical pacing or stimulation of the phrenic nerve leading to diaphragmatic contraction [1, 2]. Fourteen months after his injury, the patient underwent surgical implantation of the internal components of the phrenic nerve stimulator via bilateral second intercostal thoracotomy incisions. A small electrode was attached to each phrenic nerve and connected to a radio-frequency receiver which was placed in subcutaneous pocket on the anterior chest wall. Postoperatively, external rubberised circular antennae were taped onto the anterior chest wall above the subcutaneous receivers (Fig. 1). The antennae were connected to a stimulus controller and a rechargeable battery power source. The stimulus controller generates a radio-frequency signal at a fixed rate and amplitude in the external antennae which is conducted through the skin to the implanted receivers. The signal is demodulated and transmitted to the phrenic nerve electrode leading to contraction of the diaphragm.

Two weeks after surgery, PNS breathing was started for short periods during the day while the tracheostomy tube was disconnected from the ventilator. When we were confident that adequate tidal volume breaths (400–500 ml) were being generated by PNS, the tracheostomy cuff was deflated and the tube capped during PNS breathing. This resulted in normal humidification of inspired room air, a return of his voice during passive exhalation and increasingly long periods off mechanical ventilation. Six months after insertion the patient was on full-time PNS, requiring positive pressure ventilation for short periods only during

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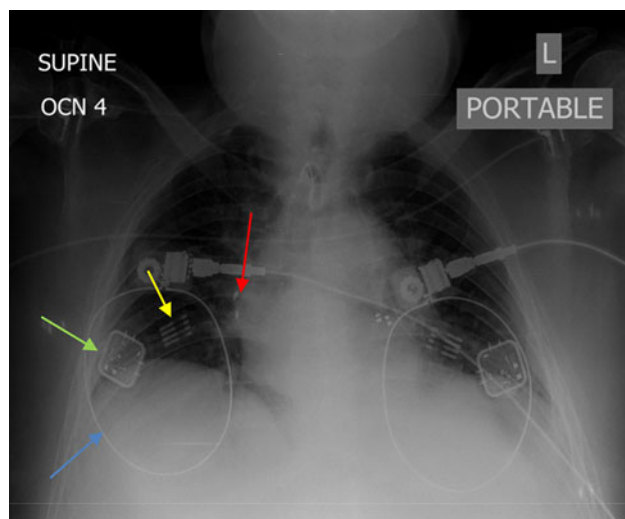


Fig. 1 Chest radiograph showing components of PNS; internal phrenic nerve electrode (*red arrow*), internal radio-frequency receiver (*green arrow*); internal junction box connecting the electrode and receiver (*yellow arrow*); external rubberised antenna (*blue arrow*)

daily cleaning and re-application of the antennae and the weekly battery change. At the night, he remained on PNS with the tracheostomy tube connected to a continuous positive airway pressure circuit to minimise the effects of upper airway obstruction during sleep. He had a reduction

in the number of respiratory tract infections and reported an improved sense of smell. Being weaned from long-term mechanical ventilation has made mobilising our patient both inside and outside the hospital less daunting and brought a welcome degree of independence to the patient himself. It is his hoped that eventually he will be cared for at home although his nursing requirements remains substantial. This case has encouraged us to consider deploying PNS in the small number of patients with high cervical spinal cord injury who require prolonged ventilatory support.

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