

Editorial

Diaphragm pacing

Diaphragm pacing is a technique of artificial ventilation using electrical stimulation of the phrenic nerves to produce diaphragm contraction. Electrotherapists of the nineteenth century were well acquainted with phrenic nerve stimulation but Sarnoff and colleagues¹ in 1948 were the first to show that the technique could maintain adequate ventilation. Sarnoff used percutaneous and transcutaneous electrodes, clearly not suitable for longterm use, and further clinical progress had to await the development of miniature stimulators that could be implanted. In 1966 Glen and coworkers² first treated a patient with chronic ventilatory failure, using a radiofrequency system with an implantable receiver and electrode but an external power source. This group has subsequently treated more than 80 patients and has made scientific contributions to all aspects of diaphragm pacing.³ Only in 1980 was the first patient treated in Britain.⁴ Worldwide more than 600 patients have been treated in this way.

Pacing can be considered only for patients with chronic ventilatory failure whose lungs are relatively normal and who have no appreciable impairment of function of the distal phrenic nerves or diaphragm. Patients with disorders causing a lower motor neurone lesion of the phrenic nerve or a myopathy of the diaphragm are not suitable. The most frequent indications for pacing are high cervical cord lesions (either traumatic or postmeningitic) and central alveolar hypoventilation.

Before the implantation of electrodes the function of the phrenic nerves and diaphragm requires careful assessment. Patients with cord transection not uncommonly have an injured phrenic root. Assessment is best done by percutaneous supramaximal phrenic nerve stimulation in the neck with surface electrodes. When nerve and muscle function is normal a vigorous contraction of the diaphragm is observed on x ray screening. Where possible it is advisable to measure

transdiaphragmatic pressure (Pdi)—with oesophageal and gastric balloon catheters—and phrenic nerve conduction times.^{5,6} Some prolongation of conduction time may not exclude successful diaphragm pacing provided that the contractile response of the diaphragm is good. Twitch transdiaphragmatic pressures at the lower limit of normal may be difficult to interpret in patients with longstanding diaphragm paralysis and disuse atrophy of the muscle. In most patients with central alveolar hypoventilation breathing during voluntary manoeuvres is normal, so that diaphragm strength can be assessed by measuring Pdi at total lung capacity or, most usefully, during a maximum sniff.⁷

Modern equipment for pacing consists of a small receiver, about 4 cm in diameter and 1.5 cm thick, supplying a unipolar electrode sited against the posterior aspect of the phrenic nerve, preferably in its upper thoracic course. Each phrenic nerve has its own receiver and electrode. The receivers are positioned subcutaneously on the anterior rib cage or abdomen. The external part of the system consists of the transmitter with its battery power supply and antennae. The loop antennae are taped on to the skin over the implanted receivers and deliver intermittent, very short range signals. The current cost of equipment* is £10 000 for a unilateral system and £15 000 for bilateral pacing.

The electrodes are best placed at thoracotomy, through the second interspace anteriorly.⁸ Cervical placement is occasionally preferable—for example, when there is thoracic deformity—but may exclude the accessory nerve, a branch of C5, or other low contributions to the phrenic nerve. For bilateral pacing the electrodes are sited at two operations one to two weeks apart. Diaphragm pacing is started 12–14 days after surgery, initially for a few minutes at a time; stimulation periods are then extended gradually, thereby conditioning the diaphragm. The problems of fatigue are minimised by using the lowest possible stimulation frequencies and respiratory rates that produce adequate ventilation.

To achieve adequate ventilation continuously by unilateral pacing the stimulation settings required will sometimes produce diaphragm fatigue. Daytime unilateral pacing, combined with nocturnal mechan-

Address for reprint requests: Dr J Moxham, Department of Thoracic Medicine, King's College Hospital, London SE5 8RX.

*Available from Avery Laboratories Inc, 145 Rome Street, Farmingdale, New York 11735, or Avery Laboratories AG, Fraumunsterstrasse 13, 8001 Zürich (tel 010-41-1-211 2047/52).

ical ventilation, may be appropriate for occasional patients. To achieve full time ventilatory support both hemidiaphragms should be paced, alternately or simultaneously. Although alternate pacing for 12 hours on each side is effective, bilateral pacing is more efficient. Gradual conditioning of the diaphragm eventually enables the use of low stimulation frequencies and low respiratory rates that minimise the risk of fatigue or long term damage to phrenic nerves or diaphragm muscle. In young children unilateral pacing is inadequate and bilateral pacing may initially produce fatigue. With gradual conditioning of the diaphragm, however, continuous support can be achieved.⁹ Patients with long term diaphragm paralysis have substantial disuse atrophy of the muscle that is gradually reversed by pacing, and the diaphragm may double its capacity to generate pressure over six months.¹⁰ Repetitive low frequency stimulation leads to histological and biochemical changes in the diaphragm, with an increase in oxidative capacity and decreased fatigability.¹¹

The results of diaphragm pacing in tetraplegic patients are good provided that the lesion does not affect the origin of the phrenic nerve. Full time support is normally possible after three to four months' conditioning by bilateral pacing. Glenn and colleagues³ have reported data on 30 patients, aged 7–71 years, who have had satisfactory pacing for a mean period of 50 months. At King's College Hospital, where six tetraplegic patients have had pacing in the last few years, results have also been satisfactory. Five patients have been followed up for one and a half to six years, and three are now being cared for at home. One patient died with respiratory infection after five years of pacing. An important point is that pacing may not provide adequate ventilation if metabolic requirements rise or if infection impairs the function of the lungs. There is, however, no evidence that infection is more frequent in patients having pacing than in those being ventilated conventionally. Overall, severe problems associated with diaphragm pacing are infrequent. From their early experience with 20 tetraplegic patients Glenn and colleagues reported three deaths related to pacing, but such deaths have become unusual in recent years. The results of diaphragm pacing for disorders of central respiratory control are less good than those for tetraplegia. In many patients upper airways obstruction is a severe problem during pacing and tracheostomy is required. In patients with hypoventilation secondary to brainstem lesions both aspiration pneumonia and problems with swallowing are common. For many patients with central alveolar hypoventilation, particularly those without identifiable lesions of the central nervous system, ventilation is a major problem only at night, and alternative methods of ventilatory support may be preferable.

Patients who are treated with diaphragm pacing

require careful long term follow up. Mechanical failure (most commonly battery failure) is possible, so that alarm systems and the immediate availability of alternative mechanical ventilation are essential. The need for accurate assessment, careful surgery, optimal pacing schedules after operation, and long term follow up means that diaphragm pacing should be undertaken at specialist centres, where for carefully selected patients pacing can bring freedom from the mechanical ventilator and increased mobility, and facilitate rehabilitation towards a more normal life socially and domiciliary care.

JOHN MOXHAM
DENNIS POTTER

*Departments of Thoracic Medicine and of
Anaesthetics and Intensive Care
King's College Hospital
London SE5 8RX*

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J Moxham and D Potter

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