- ¹⁸ Lee KS, Weeks TA, Kao RL, Akaike N, Brown AM. Sodium current in single heart muscle cells. *Nature* 1979;278:269-71.
- ¹⁹ Brown AM, Lee KS, Powell T. Reactivation of sodium conductance in single heart muscle cells. J Physiol 1980;301:78-9P.
- ²⁰ Lee KS, Hume JR, Giles W, Brown AM. Sodium current depression by lidocaine and quinidine in isolated ventricular cells. *Nature* 1981;**291**: 325-7.
- Brown AM, Lee KS, Powell T. Voltage clamp and internal perfusion of single rat heart muscle cells. *J Physiol* 1981;318:455-77.
 Brown AM, Lee KS, Powell T. Sodium current in single rat heart muscle
- ²² Brown AM, Lee KS, Powell T. Sodium current in single rat heart muscle cells. J Physiol 1981;318:477-500.
- ²³ Isenberg G, Klockner U. Glycocalyx is not required for slow inward calcium current in isolated rat heart myocytes. *Nature* 1980;**284**:358-61.
- ²⁴ Lee KS, Lee EW, Tsien RW. Slow inward current carried by Ca²⁺ or Ba²⁺ in single isolated heart cells. *Biophys* J 1981;33:143a.
- ²⁵ Eckel J, Reinauer H. Characteristics of insulin receptors in the heart muscle. Binding of insulin to isolated muscle cells from adult rat heart. *Biochim Biophys Acta* 1980;**629**:510-21.

- ²⁶ Kao RL, Christman EW, Luh SL, Krauhs JM, Tyers GFO, Williams EH. The effects of insulin and anoxia on the metabolism of isolated mature rat cardiac myocytes. Arch Biochem Biophys 1980;203:587-99.
- ²⁷ Bahl J, Navin T, Manian AA, Bressler R. Carnitine transport in isolated adult rat heart myocytes and the effect of 7,8 diOH chlorpromazine. *Circ Res* 1981;48:378-85.
- ²⁸ Montini J, Bagby GJ, Burns AH, Spitzer JJ. Exogenous substrate utilization in Ca²⁺-tolerant myocytes from adult rat hearts. Am J Physiol 1981;H659-H663.
- ²⁹ Wittenberg BA, Robinson TF. Oxygen requirements, morphology, cell coat and membrane permeability of calcium-tolerant myocytes from hearts of adult rats. *Cell Tissue Res* 1981;216:231-51.
- ³⁰ Dow JW, Walker EJ. Features of cardiac myocytes in culture: characterisation of the failing cell. In: Longmore DB, ed. *Towards safer cardiac surgery*. MTP Press: Lancaster, 1981.

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Diaphragm pacing in ventilatory failure

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Abstract

Diaphragm pacing, which entails electrical stimulation to the phrenic nerve, is an effective means of managing patients with ventilatory insufficiency and intact lowermotor-neurone innervation of the diaphragm. The pacing apparatus is used to pace the right and left hemidiaphragms alternately to avoid fatigue, which may damage the muscle irreversibly.

Among the important benefits of pacing in quadriplegics with paralysis of respiratory muscles are the social and psychological advantages of not being dependent on a mechanical ventilator.

Introduction

Electrical stimulation of the phrenic nerve to effect ventilation for prolonged periods was first reported by Judson and Glenn in 1968. Since then the technique of diaphragm pacing has been used in over 180 patients in America, and we describe here what we believe to be the first case of its use in Britain.

Case report

A 42-year-old man was admitted to hospital in January 1980 having fallen headlong downstairs. On examination he was quadriplegic, with all respiratory movements being achieved by the sternomastoid and scalene accessory muscles. X-ray examination of the cervical spine showed an odontoid anomaly that was stable. He was observed initially and by the following day was becoming exhausted, so after endotracheal intubation positive-pressure ventilation was started and a tracheostomy performed. When the ventilator was stopped he could maintain a tidal volume of about 100 ml for up to six minutes. Operative treatment was not indicated, and as the neurological lesion was incomplete there was a slight chance of recovery of phrenic activity. X-ray screening of

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the diaphragm in March, however, showed no voluntary movement on either side, and his suitability for implanted diaphragm pacemakers was further investigated. Percutaneous stimulation of each phrenic nerve produced adequate tidal volumes and visible excursion of the diaphragm on screening.

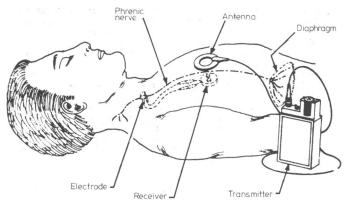
Receivers and electrodes of the pacemaker system were implanted by one of us (JJM) on the right side in September and on the left side six weeks later. The diaphragm was paced for short periods initially, but the duration of stimulation was gradually increased and the time spent on the ventilator became less. The pacing apparatus was adjusted to produce 12 respirations a minute with a tidal volume of about 440 ml during stimulation of the right and 460 ml during stimulation of the left hemidiaphragm with the patient supine. Arterial tensions of oxygen and carbon dioxide were in the normal range after four hours' continuous pacing on either side. The tracheostomy was maintained with a 24 French gauge plain Portex tube, which was occluded during the day, except when required for tracheal suction, to facilitate normal speech. At present each hemidiaphragm is paced alternately for eight-hour periods, so removing altogether the need for positive-pressure ventilation.

Discussion

The diaphragm pacer uses integrated solid-state electronic circuitry to permit delivery of an adjustable electrical stimulus to the phrenic nerve. The apparatus has been described in detail by Glenn et al² ³ (Avery Laboratories, Farmingdale, New York 11735). An external battery-powered transmitter develops a radiofrequency signal, which is radiated from a loop antenna. This antenna is placed over a subcutaneous radioreceiver, which is usually implanted just above the costal margin in the mid or anterior axillary line. The receiver is encapsulated in an epoxy disc and covered in silicone rubber. It demodulates the signal, and the resulting direct-current pulses pass to an electrode placed on the phrenic nerve usually in the neck (figure). The delivery of a train of about 25 electrical pulses to the phrenic nerve over approximately 1.3 seconds produces contraction of the diaphragm. By using the controls on the external transmitter it is possible to alter the respiration rate, which represents the time interval between adjacent pulse trains, and the inspiration time, which is determined by the duration of each pulse train. The amplitude of the stimulating current determines the depth of inspiration and is also adjustable.

Glenn has had the most extensive experience with diaphragm pacing. He has reported its use in 36 patients with central alveolar hypoventilation, in one patient with chronic obstructive lung disease, and in 20 quadriplegics with paralysis of the

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Diaphragm pacer.

respiratory muscles.^{4 5} Patients with central alveolar hypoventilation were selected on the basis of several criteria, including the presence of clinical features of chronic hypoventilation and reduced ventilatory response to hypoxia and hypercapnia but near-normal results of tests of ventilatory capacity. There was clinical evidence of improvement in right heart failure after pacing was started and a fall in packed cell volume and pulmonary artery pressure. The patient with chronic obstructive lung disease was paced with the aim of being able to give regular safe treatment with oxygen despite dependence on hypoxic drive. Pacing over 42 months brought about a decrease in the frequency of acute exacerbations and other evidence of improvement including a fall in the packed cell volume.

Quadriplegic patients with total respiratory paralysis require ventilatory support at all times. Continuous pacing of one or both hemidiaphragms using electrical variables standard for intermittent pacing produces fatigue, however, which, if prolonged, irreversibly damages the muscle.⁶ ⁷ The hemidiaphragms are therefore paced alternately so that electrical stimulation of one side is never continued for longer than 16 hours. This gives each hemidiaphragm a rest period and avoids fatigue. Eight of the quadriplegic patients in Glenn's series were supported entirely by diaphragm pacing for up to 10 years. Among the important benefits of pacing in quadriplegics with paralysis of the respiratory muscles are the social and psychological advantages of freedom from dependence on a mechanical ventilator.

The aim of this report is to increase awareness of the availability of diaphragm pacing for use in the management of patients with ventilatory insufficiency and intact lower-motorneurone innervation of the diaphragm.

We are grateful to Dr C J Goodwill for allowing us to publish this report and to the staff of the intensive care unit for their help. We also thank Miss Maggy Gleeson for secretarial help.

ADDENDUM—Since this paper was submitted we have implanted a similar bilateral diaphragm pacing system in a 2-year-old boy with quadriplegia and consequent ventilatory failure. The system is functioning satisfactorily.

References

- ¹ Judson JP, Glenn WWL. Radiofrequency electrophrenic respiration. JAMA 1968;203:1033-7.
- ² Glenn WWL, Holcomb WG, Gee JBL, Rath R. Central hypoventilation; long-term ventilatory assistance by radiofrequency electrophrenic respiration. Ann Surg 1970;**172**:755-73.
- ³ Glenn WWL, Holcomb WG, Hogan J, et al. Diaphragm pacing by radiofrequency transmission in the treatment of chronic ventilatory insufficiency. J Thorac Cardiovasc Surg 1973;66:505-20.
- ⁴ Glenn WWL. Diaphragm pacing—present status. PACE 1978;1:357-70.
 ⁵ Glenn WWL, Hogan JF, Phelps ML. Ventilatory support of the quadriplegic patient with respiratory paralysis by diaphragm pacing. Surg Clin North Am 1980;60:1055-78.
- ⁶ Liu HM, Loew JM, Hunt CE. Congenital hypoventilation syndrome: a pathologic study of the neuromuscular system. *Neurology* 1978;28: 1013-9.
- ⁷ Oda T, Glenn WWL, Hogan JF, et al. Evaluation of electrical parameters for diaphragm pacing: an experimental study. J Surg Res 1981;30: 142-53.

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Severe metabolic alkalosis: a case report

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Abstract

A 45-year-old man who was admitted with nausea, vomiting, and abdominal pain was found to have severe metabolic alkalosis, with a Pa_{CO_2} of 11 4 kPa (85 5 mm Hg), Pa_{O_2} of 5.8 kPa (43.5 mm Hg), pH of 7.61, and plasma bicarbonate concentration of 82.0 mmol/1. He was treated with oxygen, intravenous physiological saline, and phenytoin and improved within 48 hours. Radiographs showed gastric outlet obstruction secondary to peptic ulcer, which was treated by surgery.

Though severe, the rise in carbon dioxide concentration in this patient was probably lifesaving. The Pa_{CO_2} was therefore allowed to fall gradually as the alkalosis was treated. The return of both Pa_{CO_2} and plasma bicarbonate values to normal in parallel suggests that hypoventilation compensated for the metabolic alkalosis and emphasises the importance of conservative treatment in cases of metabolic alkalosis.

Introduction

We report a patient with severe metabolic alkalosis, out of the range of most acid-base nomograms. The metabolic alkalosis was due to vomiting secondary to gastric outlet obstruction. The initial arterial plasma bicarbonate concentration was 82 mmol(mEq)/l, but it returned to normal within 48 hours of the start of treatment.

Case report

A 45-year-old man was admitted with a two-day history of nausea, vomiting, and abdominal pain. He had a history of alcohol withdrawal seizures. He had symptoms of peptic ulcer and an upper gastro-

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