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-motor-neurone 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

- Judson JP, Glenn WWL. Radiofrequency electrophrenic respiration. *JAMA* 1968;**203**:1033-7.
- Glenn WWL, Holcomb WG, Gee JBL, Rath R. Central hypoventilation; long-term ventilatory assistance by radiofrequency electrophrenic respiration. *Ann Surg* 1970;**172**:755-73.
- 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.
- Glenn WWL. Diaphragm pacing—present status. *PACE* 1978;**1**:357-70.
- 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.
- Liu HM, Loew JM, Hunt CE. Congenital hypoventilation syndrome: a pathologic study of the neuromuscular system. *Neurology* 1978;**28**:1013-9.
- 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.

(Accepted 18 August 1981)

Severe metabolic alkalosis: a case report

SHAHROKH JAVAHERI, EDWARD A NARDELL

Abstract

A 45-year-old man who was admitted with nausea, vomiting, and abdominal pain was found to have severe metabolic alkalosis, with a P_{aCO_2} of 11.4 kPa (85.5 mm Hg), P_{aO_2} of 5.8 kPa (43.5 mm Hg), pH of 7.61, and plasma bicarbonate concentration of 82.0 mmol/l. 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 P_{aCO_2} was

therefore allowed to fall gradually as the alkalosis was treated. The return of both P_{aCO_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-

Pulmonary Division, Boston University Medical Center and Boston City Hospital, Boston, Mass 02118, USA

SHAHROKH JAVAHERI, MD, assistant professor of medicine (present address: Laboratory of Normal and Pathological Physiology, University of Gent, B-9000 Gent, Belgium)

EDWARD A NARDELL, MD, assistant professor of medicine (present address: Harvard Medical School, Department of Medicine, Cambridge Hospital, Cambridge, MA 02139, USA)

intestinal x-ray examination six years earlier had shown a scarred duodenal bulb and acute duodenal ulcer. He was taking hydrochlorothiazide for hypertension. There was no history of chronic obstructive pulmonary disease.

On admission the patient was stuporous and had dry skin. His reflexes were normal. The blood pressure was 130/70 mm Hg, pulse rate 92/min, respiratory rate 12/min, and rectal temperature 38.3°C. Packed cell volume was 49.8% and white blood cell count $15.3 \times 10^9/l$. Blood urea nitrogen was 10 mmol/l (28 mg/100 ml). Serum amylase activity was normal. Serum sodium was 142 mmol(mEq)/l, potassium 2.0 mmol(mEq)/l, and chloride 50 mmol(mEq)/l. Arterial carbon dioxide tension (P_{aCO_2}) was 11.4 kPa (85.5 mm Hg) (at 37°C) and oxygen tension (P_{aO_2}) 5.8 kPa (43.5 mm Hg), pH 7.61, and the calculated plasma bicarbonate 82.0 mmol(mEq)/l. Initially he was treated with naloxone without response. Oxygen, intravenous physiological saline, potassium chloride, and phenytoin were administered. He improved within the next 48 hours (see table) but developed aspiration pneumonia while in the intensive care unit.

Acid-base variables during patient's stay in hospital (values corrected to patient's body temperature)

Time:	Admission	6 hours later	12 hours later	24 hours later	48 hours later
F_iO_2	0.21	0.40	0.35	0.35	0.21
P_{aO_2} (kPa) ..	5.8	14	10.9	12.7	10.5
P_{aCO_2} (kPa) ..	11.4	9.7	10.3	7.5	4.7
pH	7.59	7.56	7.47	7.43	7.43
HCO_3^- (mmol/l) ..	82	65	55	35.5	23.8

F_iO_2 = Fractional concentration of inspired oxygen.

Conversion: SI to traditional units— P_{aO_2} and P_{aCO_2} : 1 kPa $\approx$ 7.5 mm Hg.

Two days after admission his serum sodium was 146 mmol/l, potassium 3.8 mmol/l, and chloride 102 mmol/l. Blood urea nitrogen was 5.7 mmol/l (16 mg/100 ml) and serum creatinine 80 μ mol/l (0.9 mg/100 ml). Radiographs of the upper gastrointestinal tract showed gastric outlet obstruction secondary to peptic ulcer, for which he underwent surgery.

His pulmonary function tests showed a forced vital capacity (FVC) of 4.0 l (80% of predicted), forced expiratory volume in one second (FEV₁) of 3.39 l (90% of predicted), FEV₁ as a percentage of VC of 84%, and maximum mid-expiratory flow rate of 3.98 l/second (108% of predicted).

Arterial blood gas analysis—Arterial blood gases were analysed by appropriate electrodes at 37°C (model 813, Instrumentation Laboratory, Lexington, Mass.) PCO_2 and PO_2 electrodes were calibrated by gases of known concentration as well as human blood tonometered with the same gases. The pH electrode was calibrated with precision buffers. The patient's arterial blood gas values were corrected to his body temperature and plasma bicarbonate was calculated from the Henderson-Hasselbalch equation by using a Severinghaus blood gas calculator.

Discussion

This patient suffered from the most severe metabolic alkalosis ever reported as a result of vomiting secondary to gastric outlet obstruction. Hydrochlorothiazide worsened the process. Hypoventilation, which prevented a lethal alkaline pH, was at least partly due to the depression of central and peripheral chemoreceptors by the alkalaemia. The patient was not taking any drugs which would depress respiration, and he showed no response to naloxone.

Although grand mal seizure may lead to carbon dioxide retention and might have further augmented the rise in P_{aCO_2} , its contribution to hypoventilation in this patient was uncertain. A severe grand mal seizure is often associated with lactic acidosis resulting in an increased anion gap ($(Na^+ + K^+) - (Cl^- + HCO_3^-)$). This patient had a low anion gap initially. Nevertheless, if our patient had seizure-induced carbon dioxide retention, the contribution of such a sharp increase in carbon dioxide concentration to the rise in plasma bicarbonate concentration was minimal. Furthermore, chronic obstructive lung disease and carbon dioxide retention on that basis were ruled

out by his history and by normal pulmonary function values and P_{aCO_2} 48 hours after treatment. The initial alveolar-arterial difference for oxygen tension, (A-a) DO_2 , was also normal.

It has been suggested that compensatory carbon dioxide retention in metabolic alkalosis is limited since the impending hypoxaemia and concomitant hypokalaemia¹ prevent hypoventilation. Many patients with severe metabolic alkalosis and pronounced compensatory carbon dioxide retention have been hypoxaemic,² however, and it is now well-known that the carotid body is less sensitive to hypoxaemia in the presence of alkalaemia.³ This patient initially had a P_{aO_2} of 5.8 kPa (43.5 mm Hg).

We have shown⁴ that percentage regulation of hydrogen in the arterial blood of normal subjects with steady state metabolic alkalosis is independent of changes in plasma potassium concentrations. Other reported cases, moreover, have shown severe carbon dioxide retention compensatory to metabolic alkalosis with potassium depletion and hypokalaemia.² This patient's initial plasma potassium concentration was 2.0 mmol/l. Therefore, it does not follow that hypokalaemia would necessarily limit hypoventilation. On the contrary, respiratory muscle weakness secondary to hypokalaemia might itself have aggravated hypoventilation, although this probably did not contribute to carbon dioxide retention in our patient since his reflexes were intact.

Several recent studies have attempted to define more fully the normal ventilatory response to metabolic alkalosis. For a bicarbonate of 82 mmol/l our regression equation predicts a P_{aCO_2} of 10.4 kPa (75 mm Hg), while that of van Ypersele de Strihou⁵ predicts a P_{aCO_2} of 12 kPa (90 mm Hg). This patient's P_{aCO_2} of 11.4 kPa (85.5 mm Hg) was therefore, predicted reasonably accurately given the need to extrapolate beyond the data used to generate either regression equation.

Although very severe, the rise in carbon dioxide concentration in our patient was potentially lifesaving. It was therefore important to allow the PCO_2 to fall gradually in response to treating the metabolic alkalosis. This gradual return of P_{aCO_2} to normal concomitant with the return of plasma bicarbonate values to normal also suggests that hypoventilation was compensating for metabolic alkalosis in this patient. Moreover, the rapid recovery of our patient underscores the importance of conservative treatment (volume expansion, potassium, and oxygen) even in the most severe cases of metabolic alkalosis; the normal kidney has a large capacity for excreting bicarbonate.

References

- Goldring RM, Cannon PJ, Heinemann HO, Fishman AP. Respiratory adjustment to chronic metabolic alkalosis in man. *J Clin Invest* 1968; **47**:188-202.
- Lifschitz MD, Brasch R, Cuomo AJ, Menn SJ. Marked hypercapnia secondary to severe metabolic alkalosis. *Ann Int Med* 1972; **77**:405-9.
- Hornbein TF, Roos A. Specificity of H ion concentration as a carotid chemoreceptor stimulus. *J Appl Physiol* 1963; **18**:580-4.
- Javaheri S, Shore NS, Rose BD, Callahan BJ, Kazemi H. Respiratory compensation in metabolic acidosis and alkalosis in normal man. *Am Rev Resp Dis* 1980; **121**:356.
- van Ypersele de Strihou C, Frans A. The respiratory response to chronic metabolic alkalosis and acidosis in disease. *Clin Sci Mol Med* 1973; **45**: 439-48.

(Accepted 14 August 1981)

MANY attempt to cure a cold, by getting drunk. But this, to fay no worfe of it, is a very hazardous experiment. No doubt it may sometimes succeed, by suddenly restoring the perspiration; but when there is any degree of inflammation, which is frequently the case, strong liquors, instead of removing the malady, will increase it. By this means a common cold may be converted into an inflammatory fever.

(Buchan's *Domestic Medicine*, 1786.)