POSTOPERATIVE VENTILATORY FAILURE IN A PATIENT WITH PRIMARY ALVEOLAR HYPOVENTILATION

A Case Report

S. A. HARGRAVE, J. S. LEGGE AND K. N. V. PALMER

SUMMARY
A slightly obese woman developed severe ventilatory failure which required assisted ventilation on the first day after an emergency appendicectomy. Subsequent investigation revealed a mild restrictive ventilatory defect, hypoxaemia and hypercapnia and an abnormal ventilatory response to carbon dioxide. The patient was considered to have primary alveolar hypoventilation and, because of this, was abnormally sensitive to narcotic analgesics.

A 50-year-old woman (weight 67 kg, height 1.5 m) was admitted as an emergency with a 30-hour history of central abdominal pain radiating to the right iliac fossa. On examination she was found to be slightly obese and pyrexial (38.4°C). There was tenderness and guarding in the right iliac fossa. A diagnosis of acute appendicitis was made and the appendix was removed. The anaesthetic was sodium thiopentone 250 mg, suxamethonium 75 mg with endotracheal intubation and controlled ventilation with oxygen, nitrous oxide and halothane. Muscle relaxation was maintained with alcuronium 10 mg and reversed by neostigmine 4 mg with atropine 1.2 mg injected intravenously. There was some difficulty in establishing adequate spontaneous respiration immediately after the operation, but half an hour later her condition was considered satisfactory and she was returned to the ward.

The initial postoperative course was uneventful. She received methadone 7.5 mg intramuscularly at 3 and again at 13 hours after the operation. There was no abnormal response to the first dose, as observed clinically. She was first noticed to be drowsy and confused on the following morning, 18 hours after the operation. The arterial oxygen tension (Pao₂) was 59 mm Hg, the arterial carbon dioxide tension (Paco₂) 68 mm Hg and the arterial pH 7.22. Two hours later, and in the absence of specific treatment, her condition had deteriorated further and she became unconscious with Cheyne-Stokes respiration. The Pao₂ was at this stage 32 mm Hg, Paco₂ 80 mm Hg and pH 7.20. After endotracheal intubation, manual pulmonary ventilation was instituted and aminophylline 250 mg, 10% mannitol 200 ml and levallorphan 1.5 mg were injected intravenously. Following this, her condition improved rapidly and she regained consciousness. The rest of her stay in hospital was uneventful.

Lung function measurements were made 9 days after operation and again 3 months later. The results are shown in Table I together with the predicted normal values for a woman of her age, height and weight. On both occasions there was evidence of a mild restrictive ventilatory defect with hypoxaemia and mild hypercapnia. Figure 1 shows the ventilatory response to carbon dioxide 3 months after operation. It can be seen that the patient showed no increase in ventilation as the end tidal Pco₂ rose from 55 to 70 mm Hg whereas in 3 normal subjects the ventilation increased three-fold when the end tidal Pco₂ rose to these levels.

Primary alveolar hypoventilation is seen most commonly in severely obese patients (Burwell et al., 1956). Such patients may develop the so-called Pickwickian syndrome which is characterized by episodes of stupor or even unconsciousness. However, primary alveolar hypoventilation has also been described in the non-obese, where it has usually been associated with a neurological disorder (Rodman et al., 1962; Garlind and Linderholm, 1958). In both types of patient the main abnormality is thought to

S. A. HARGRAVE, M.B., CH.B.; J. S. LEGGE, M.D.; K. N. V. PALMER, M.D., F.R.C.P.; Department of Medicine, Foresterhill, University of Aberdeen AB9 2ZD.
be a diminished central response to carbon dioxide. Lawrence (1959), however, described a patient with idiopathic alveolar hypoventilation who weighed only 167 lb. He had polycythaemia and cor pulmonale, but at post-mortem examination, no lesions of the brain or lungs was demonstrated.

The patient described here was slightly overweight but could not be considered obese. There was no clinical evidence of neurological disease so that she appears to have primary alveolar hypoventilation of unknown cause. The episode of severe ventilatory failure after operation was probably brought about by the administration of narcotic analgesics to a patient who was not, at that time, known to have a markedly diminished ventilatory response to carbon dioxide. This concept is supported by the rapid return of consciousness and adequate ventilation following the administration of a morphine antagonist and respiratory stimulants.

REFERENCES

