ADVERSE EFFECTS OF LOW CARBON DIOXIDE TENSIONS DURING MECHANICAL OVER-VENTILATION OF PATIENTS WITH COMBINED HEAD AND CHEST INJURIES

Report of Two Cases

BY

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SUMMARY

Two patients are described who had suffered injuries both to the chest and the head. Both patients had evidence of impairment of ventilation-perfusion relationships, and were ventilated mechanically. The large minute volumes which were used to inflate the lungs and to restore normal arterial oxygen tensions severely reduced the arterial carbon dioxide tension, and produced an arterial alkalaemia. The insertion of a deadspace into the ventilation circuit increased the arterial carbon dioxide tension; the arterial oxygen tension was not further impaired, and the levels of consciousness of both patients improved. A suitable deadspace allows normal carbon dioxide tension to be maintained when increased ventilation volumes are required for adequate mechanical ventilation.

Retention of carbon dioxide is uncommon in patients with acute impairment of pulmonary ventilation-perfusion relationships, and in these patients hypoxaemia and exhaustion from increased, yet inadequate, respiratory efforts are the usual indications for mechanical ventilation of the lungs (Björk and Holmdahl, 1965). Artificial ventilation at constant normal tidal volumes has been shown to aggravate atelectasis (Mead and Collier, 1959), while it is probable that large tidal volumes will contribute to the health of the lungs (Spalding and Smith, 1963), and substitute for the normal periodic deep breaths of spontaneous ventilation (Bendixen et al., 1964). Despite ventilation-perfusion irregularities, large tidal volumes reduce arterial and alveolar carbon dioxide tension, although hypoxaemia may persist (Haldane, 1922; Rahn and Farhi, 1964). In patients with chronic retention of carbon dioxide and in patients with respiratory muscular paresis, this reduction in carbon dioxide tension is either desirable or thought not severe enough to warrant consideration (Spalding and Smith, 1963). On occasion, however, acute reduction of carbon dioxide tension may have deleterious effects, and some observations on the consequences of inadvertent over-ventilation of patients with associated head and chest injuries are reported here.

MATERIAL AND METHODS

Femoral arterial blood was withdrawn daily from patients who were being maintained by mechanical ventilation. The blood was collected in syringes whose deadspace was filled with heparin, and the samples were analyzed immediately for pH, $P_{CO_2}$, and $P_{O_2}$. The methods of analysis, and of calculation of plasma bicarbonate have been described previously (Froman and Smith, 1966). The ventilation volumes were measured by the spirometer incorporated in the circuit of the mechanical ventilator.

ILLUSTRATIVE CASE REPORTS

CASE NO. 1. L.A., a 45-year-old motor-cyclist, was involved in a road traffic accident in which he sustained multiple rib fractures, fractures of the sternum and the right tibia, and a severe closed head injury. He was resuscitated with intravenous blood transfusion, and the samples were analyzed immediately for pH, $P_{CO_2}$, and $P_{O_2}$. The methods of analysis, and of calculation of plasma bicarbonate have been described previously (Froman and Smith, 1966). The ventilation volumes were measured by the spirometer incorporated in the circuit of the mechanical ventilator.

A rigid wide-bore plastic tube of 250 ml volume was then placed between the patient and his ventilator to serve as an additional deadspace. The arterial $P_{CO_2}$ rose by about 10 mm Hg, and the arterial pH, although still alkaline, was reduced accordingly. Arterial $P_{O_2}$ was
not adversely affected and, in fact, was slightly increased. Within half an hour there was improvement in the patient's level of consciousness, and he moved his left side purposefully in response to commands, although the right hemiparesis persisted. Systemic vital signs, pulse and blood pressure also improved and there was a well-marked peripheral vasodilatation. The improvement continued and, as the lungs recovered, he was gradually weaned back to spontaneous respiration after 4 weeks of mechanical ventilation, and made a satisfactory recovery. During this time the deadspace was removed experimentally for 24 hours, and then replaced, and the observations on blood gas tensions and pH were repeated (fig. 1).

CASE No. 2. C.H., a 23-year-old man, was a pillion-passenger on a motor-cycle which was involved in a road traffic accident. He suffered a severe fracture dislocation of the pelvis, a fractured fibula, chest wall contusions, and a closed head injury. At the time of admission, he was restless and aphasic, but he responded purposefully to painful stimuli. Thirty-six hours after admission, his respiration was becoming embarrassed, he was hypoxaemic, a tracheostomy was done, and he was mechanically ventilated. Two hours later, his level of consciousness had deteriorated; he was unresponsive to pain, and the right pupil had become dilated. An echo-encephalogram, however, did not show displacement of midline structures. At that time, arterial Pco₂ was very low, and he was hypoxaemic and alkalaemic (fig. 2).

A 250-ml deadspace was then introduced between the tracheostomy tube and the ventilator; the arterial Pco₂ was increased, the arterial Po₂ was maintained, and the patient's level of consciousness improved within the hour. He mouthed his name, responded to questions, and he moved his right side purposefully, although there was a left hemiparesis. He continued to improve, and mechanical ventilation with an added deadspace was maintained for a further 2 weeks. He was then weaned back to spontaneous respiration.
Ventilation with constant low tidal volumes is suggested that normal blood gas tensions contribute materially to survival in severe head injury. Reversed by deep breaths (Bendixen et al., 1964). Known to produce atelectasis and this will increase the inspired carbon dioxide tension. Increase arterial desaturation; this tendency can be recovered when the same rate of ventilation was sustained. Henderson was not due to mechanical impedance of venous return, nor to change in hydrogen ion concentration, but that it was the result of abstraction of carbon dioxide, and that the blood pressure recovered when the same rate of ventilation was maintained with expired air which served to increase the inspired carbon dioxide tension.

Maclver, Frew and Matheson (1958) have suggested that normal blood gas tensions contribute materially to survival in severe head injury. Ventilation with constant low tidal volumes is known to produce atelectasis and this will increase arterial desaturation; this tendency can be reversed by deep breaths (Bendixen et al., 1964). There is also evidence that high tidal volumes may maintain alveolar patency (Hedley-Whyte, Laver and Bendixen, 1964). Optimum alveolar gas tension requirements and pulmonary inflation in patients with acute chest injuries may be satisfactorily obtained by a combination of high tidal volumes, a moderately high inspired partial pressure of oxygen, and a suitable additional deadspace.

REFERENCES


**EFFETS OPPOSES DES BASSES TENSIONS DU CO₂ PENDANT L'HYPERVENTILATION MECANIQUE DES PATIENTS AVEC LES ATTEINTES COMBINEES A LA TETE ET A LA POITRINE: RAPPORT DE 2 CAS**

**SOMMAIRE**

On décrit l'état de deux malades ayant souffert tous deux d'atteintes à la poitrine et à la tête. Tous deux présentaient une détérioration du rapport ventilation-perfusion et furent ventilés mécaniquement. L'amplitude des volumes-minute qui furent utilisés pour gonfler les poumons et pour restaurer les tensions normales de l'oxygène artériel ont réduit sérieusement la tension du CO₂, et provoqué une alcalémie artérielle. L'insertion d'un temps mort dans le circuit de la ventilation a augmenté la tension du CO₂ artériel; la tension de l'oxygène artériel n'a pas été détériorée davantage, et les seuils de la conscience se sont amoindris chez les deux patients. L'insertion d'un temps mort convenable permet de maintenir d'une tension normale du CO₂, lorsque des volumes accrus de ventilation sont exigés pour compenser la ventilation mécanique.

**NACHTEILIGE WIRKUNGEN NIEDRIGER KOHLENDIOXYDSPANNUNGEN BEI PATIENTEN MIT KOMBINIERTEN KOPF- UND BRUSTVERLETZUNGEN WAHRENDE EINER MECHANISCHEN HYPERVENTILATION: EIN BERICHT ÜBER 2 FALLE**

**ZUSAMMENFASSUNG**


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**BOOK REVIEW**


The *Clinical Anesthesia* series has justifiably earned itself a high reputation for gathering together in each volume material written by experts which would be difficult to find without much search in the library. The intention is to keep the clinical anesthetist up to date with modern anaesthetic practice and outlook, and it is through his eyes that this volume must be viewed. Respiratory physiology has been such a focal point of interest to anaesthetists, and so many papers and indeed books have appeared on this field, that perhaps it is no wonder that the present volume finds it more difficult to strike the reader as being quite so valuable as most of the others in the series. The team of experts were chosen with care and every one of them deserves high praise for having written his chapter in an excellent manner. Nevertheless, there is little new material and the reviewer is left with the impression that this is the mixture as before. To the British anaesthetist the literary style does not make easy reading, and unless he has already developed a considerable interest in respiratory physiology and disease, he may well complain that no obvious attempt is made to simplify and clarify what is acknowledged to be a difficult field of physiology and medicine both in understanding and terminology. Perhaps the most interesting of the chapters, because it deals with a field of activity not highly developed in this country, is the one on "The preparation of the patient with chronic lung disease for operation and anaesthesia". This contains an interesting and well illustrated account of what is known in the United States as Inhalational Therapy. So important is this method of treatment regarded there that a corps of trained individuals known as Inhalational Therapists has developed, complete with its own discipline, qualifications and place in the hospital community. Also in this chapter is a well described and excellently illustrated account of physiotherapy for these patients. For knowledgeable readers this book is a useful compilation of physiological and clinical material, written by men who know what they are talking about, and which will serve as a guide to American thought and practice. They will find it stimulating to compare their own work and the consensus of opinion in the British Isles, with that set out here.

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