Acute Ventilatory Failure in Chronic Obstructive Lung Disease*

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Acute ventilatory failure, generally defined as an abrupt increase in carbon dioxide tension, is a common complication of chronic obstructive lung disease. When severe and uncompensated, coma and death may result unless heroic measures are taken to assist or stimulate respiration. However, milder episodes of carbon dioxide retention also occur in patients with the “emphysema-bronchitis syndrome,” especially during acute respiratory infections or with exacerbations of congestive heart failure. These milder episodes may respond promptly to treatment of the underlying cardiopulmonary disorder, requiring neither mechanical ventilatory assistance nor respiratory stimulant drugs. Such episodes have been reported previously by Cohn, Carroll and Riley and by Westlake, Simpson and Kaye and have been relatively common in our experience. The rapid “spontaneous” relief of acute ventilatory failure in these patients is worthy of emphasis since one might have erroneously ascribed their improvement to specific ventilatory stimulants or artificial respiration had such therapies been employed.

In the course of a long-term study of 175 patients with chronic obstructive lung disease, eight patients have been admitted to this hospital on ten occasions with episodes of acute ventilatory failure; in each instance, the carbon dioxide tension rose at least 15 mm Hg above its stable level. Lesser degrees of acute hypercapnia were noted on many other occasions, but these milder episodes were less clear cut and are not included in the present report. Of the ten definite instances of acute ventilatory failure, only one required tracheostomy and continuous respiratory assistance. This patient was admitted in a comatose state and became apneic shortly after hospitalization. After an erratic course, the patient gradually improved and was discharged from the hospital with a rebreathing mixed venous carbon dioxide tension (Pvco2) ranging between 52 and 58 mm Hg. He was readmitted with recurrent, acute hypercapnia within a few months; this second hospitalization is represented by episode 5 of Fig. 1.

In two other patients, respiratory stimulant drugs were used on a trial basis. Since the drugs had a dubious effect on the course of the hypercapnia, these two episodes have been excluded from the present report. In the remaining seven episodes (occurring in six patients) neither respiratory stimulants nor mechanical ventilatory assistance were employed. In all instances, the Pvco2 appeared to fall in response to therapy of the underlying cardiopulmonary disorder. Large doses of bronchodilators, especially theophyllines, were used, and patients were given low flow oxygen therapy, steroids, antibiotics, expectorants, digitalis, and diuretics when clinically indicated. Brief courses of intermittent positive pressure breathing were used in a few patients to assist in the administration of nebulized bronchodilator agents. However, such treatments were too brief and too infrequent to be considered as respiratory assistance. All reported mixed venous Pc02 values were determined by the rebreathing method of Campbell and Howell.

The course of the hypercapnia in the seven episodes receiving neither respiratory stimulants nor ventilatory assistance is depicted in Fig. 1. The mean admission Pvco2 was 78 mm Hg. This is in accord with the findings of other workers that the carbon dioxide tension cannot be much above 80

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mm.Hg prior to the institution of oxygen therapy, as otherwise the patient would have a degree of hypoxemia incompatible with life.

In three instances, peak P_{vco2} values were noted after a period of hospitalization rather than on admission. In episodes 3 and 4b, this may have been related to oxygen therapy administered during the first few days of hospitalization. In episode 6, the patient was admitted to the hospital because of an exacerbation of congestive heart failure, but with a P_{vco2} near her stable level. After three weeks of hospitalization, she developed acute bacterial pneumonia and an elevation of P_{vco2} was noted. The subsequent course is depicted in Fig. 1.

The carbon dioxide tensions returned to their stable levels within one to three weeks after the peak of hypercapnia; however, it should be noted that all patients were

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21437/)
chronically hypercapneic, both before and after the acute exacerbation. Four patients subsequently died (Nos. 2, 4, 5 and 6) but none of the deaths is known to be due to recurrent acute ventilatory failure. In patient 5, death occurred while the patient was still hospitalized, at a time when the Pco₂ had returned to stable levels. This death was sudden, unexpected and its mechanism is uncertain.

All episodes but 4b were precipitated by acute respiratory infection or by exacerbation of congestive heart failure. Symptoms apparently attributable to the acute hypercapnia were present in most. Somnolence was noted in episodes 1, 2, and 3, muscle twitching was prominent in episode 5, and confusion, amnesia, paraesthesiae, and ataxia were present in 4b. However, except for the last episode, symptoms were relatively mild, perhaps due to the fact that these patients were under continuous care on an out-patient basis and had been advised to report promptly any change in their symptomatology. Under circumstances such as these, mechanical respiratory assistance and respiratory stimulant drugs appear to be unnecessary, and could even be detrimental to the patient's course if employed indiscriminantly. The rapid improvement noted in many patients makes it difficult to assess the efficacy of any new form of therapy which is added to a program of supportive care.

It must be noted that the "routine" therapy used in these patients was vigorously applied. For example, bronchodilator agents were given to tolerance, usually with large doses of theophyllines by continuous intravenous drip. It must also be emphasized that patients such as these must be observed closely since their condition may suddenly deteriorate, necessitating the prompt institution of mechanical ventilatory assistance and/or analeptic agents.

**Summary**

Chronically hypercapneic patients with chronic obstructive lung disease are likely to develop acute increase in CO₂ retention under the stress of acute respiratory infection or with exacerbation of congestive heart failure. With milder episodes of acute ventilatory failure, patients usually respond satisfactorily to management of the underlying cardiopulmonary disorder, requiring neither analeptic drugs nor mechanical ventilatory assistance.

**Resumen**

Los pacientes en hipercapnia crónica con afecciones pulmonares obstructivas están propensos a experimentar un aumento súbito de retención del CO₂ bajo la influencia de una infección respiratoria aguda o de exacerbación de una insuficiencia cardíaca congestiva.

En episodios menos graves de déficit respiratorio estos sujetos responden bien a tratamiento del trastorno cardio-respiratorio básico, sin requerir medicación analeptica o métodos auxiliares de ventilación mecánica.

**Zusammenfassung**

Wahrscheinlich neigen Patienten mit chronischer Übersäuerung des Blutes an Kohlensäure, die an chronischem obstruktivem Emphysem leiden zu akuten Amstieg ihrer CO₂ Retention unter der Belastung einer akuten respiratorischen Infektion oder an einer Exazerbation ihrer dekompensierten Herzinsuffizienz. Bei leichten Episoden akuter ventilatorischer Insuffizienz reagieren die Patienten gewöhnlich befriedigend auf die und erfordern weder Analeptica noch eine Unterversorgung des cardiopulmonalen Grundfunktionsablauf der Atmung auf mechanischem Wege.

**References**


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