Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Blood Gas Derangements in Pulmonary Embolism

To the Editor:

Padmanabhan and Dhar (Chest, 1984; 86:927) recently reported a case of persistent hypercapnia secondary to extensive occlusion of the pulmonary artery. They stated that, "Hypercapnia has been documented only in states that prevent compensatory increases in ventilation." However, they cite a prior report in which a patient sustained a P[A-a]O₂ of 67 mm Hg despite mechanical ventilation with a minute ventilation of 20 to 25 L. In addition, they failed to note a patient reported by us who had a P[A-a]O₂ of 59 mm Hg while being ventilated with a volume cycled respirator at a rate of 26 L/min. Clearly, as the authors indicate, the presence of unexpected hypercapnia ought to raise the question of substantial occlusion of the pulmonary arterial bed. However, the authors do not explore the various associated disturbances of arterial oxygen tension. Their patient demonstrated substantial widening of the alveolar-arterial oxygen tension (P[A-a]O₂), a commonly reported finding, and they imply that hypoxemia is an inevitable accompaniment of such vascular disturbances. However, our patient in Denver, in whom a pulmonary angiogram revealed roughly 65 percent occlusion of the pulmonary arterial bed, demonstrated a much narrower difference; with an FIO₂ of 1.0, his PaO₂ was 397 mm Hg, a P[A-a]O₂ of approximately 113 mm Hg. This finding, a relatively modest disturbance of the oxygen transport, did more to obscure the diagnosis of pulmonary embolism in our patient than did the hypercapnia. We speculated that the early institution of assisted ventilation with high volumes may have prevented the development of diffuse airway narrowing and/or atelectasis, and the initial use of a FIO₂ of 1.0 may have minimized the impact of ventilation-perfusion imbalances. Given the nature of modern emergency medicine, wherein endotracheal intubation and volume-cycled mechanical ventilation may be initiated very quickly after the onset of impaired respiration, awareness of the possibility of mild disturbances of oxygen in the presence of massive thromboembolism is essential.

An additional set of questions raised by their case report relates to the origins of the vascular occlusion. The authors briefly speculate that pulmonary tuberculosis may have incited pulmonary arterial thrombosis, and they correctly note that such extensive thrombosis has not been previously reported. However, they do not mention whether the patient was black or had a sickle hemoglobinopathy. Massive vascular thrombosis has been reported in such patients with infection, metabolic derangements, or other physiologic disturbances. In the absence of evidence for deep-vein thrombosis of the lower extremities or abdominal viscera, in situ thrombosis must be considered equally probable to embolism.

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REFERENCES

1 Smith GT, Highland JW, PieMme T, Wells RE. Human systemic pulmonary arterial collateral circulation after pulmonary thromboembolism. JAMA 1964; 188:452

To the Editor:

Drs. Iseman and Haynes correctly state that we failed to note a report by them of a patient with severe hypercapnia in the setting of pulmonary embolism. This was not intentional, but an oversight. Their case report indicates, as did ours, that severe hypercapnia can occur secondary to massive pulmonary vascular occlusion.

The case report we cited of a patient with massive pulmonary embolism, whose PaCO₂ remained at 67 mm Hg in spite of a minute ventilation of 20 to 25 L on a ventilator, was not published by the authors as documentation of hypercapnia in spite of increased ventilation. (The reference provided by Dr. Iseman is in error.) We felt that there was enough evidence from the data provided to say that the hypercapnia seen was secondary to extensive pulmonary vascular occlusion. Our patient's large alveolar-arterial oxygen gradient in spite of mechanical ventilation with a large minute ventilation on an FIO₂ of 1.0, in the absence of a patent foramen ovale, led us to believe that it was secondary to the severe pulmonary vascular occlusion. We did not imply that such a severe disturbance in oxygenation was inevitable in this setting. We agree with Drs. Iseman and Haynes that atelectasis, with the resultant right to left alveolar shunt, and ventilation perfusion imbalance, with shift of perfusion to lung units with low ventilation perfusion ratios, are likely causes of hypoxemia in pulmonary embolism and, therefore, are amenable to correction by mechanical ventilation on a high concentration of inspired oxygen. We would like to add that, on spontaneous ventilation at the concentrations of inspired oxygen available without a ventilator, severe hypoxemia is most likely in the presence of massive pulmonary vascular occlusion.

We can only speculate on the etiology of the thromboembolic process. We do not feel, nor intended to give the impression, that tuberculosis incited the thromboembolic process. The possibility of this being thrombosis cannot be excluded. The clinical presentation and lack of obvious reason for thrombosis in situ favored thromboembolism. The patient was of Vietnamese origin with no evidence of sickle hemoglobinopathy. The patient's extremities were not explored at autopsy and deep venous thrombosis of the legs could not be excluded.

We would like to restate that, in the evaluation of severe irreversible hypercapnia, the possibility of extensive pulmonary vascular occlusion must not be ignored.

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