Communications for this section will be published as space and
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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 doc-
dumented only in states that prevent compensatory increases in venti-
lation." However, they cite a prior report in which a patient sustained
a PaCO₂ 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 PaCO₂ 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 distur-
bances. 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
during mechanical ventilation with high volumes may have
prevented the development of diffuse airway narrowing and/or
atelectasis, and the initial use of an 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 thromboem-
bolism 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 pulmonary embolism has been reported in such patients with
infection, metabolic derangements, or other physiologic distur-
bances. 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.

Michael D. Iseman, M.D., F.C.C.P.
National Jewish Hospital, Denver, and
J. Brecard Haynes, M.D., F.C.C.P.
Vanderbilt University School of Medicine,
Nashville

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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 a 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
inherent 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 pulmo-
nary 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 thromboem-
bolism. 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 irrevers-
ible hypercapnia, the possibility of extensive pulmonary vascular
occlusion must not be ignored.

Krishnan Padmanabhan, M.D., F.C.C.P.
Coney Island Hospital
Brooklyn
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Breakage of Alligator Forceps In Transbronchial Biopsy

To the Editor:

In their letter in the June issue (Chest 1984; 85:837), Malik and Behera describe a rare complication during fiberoptic bronchoscopy: the breakage of the biopsy forceps. We have recently come across the same complication while performing a transbronchial biopsy.

We were trying to obtain a specimen following Zavalai's technique for transbronchial biopsy1 with an Olympus BT-B3R bronchoscope and an alligator forceps (Olympus FB 15 C) in a 55-year-old woman whose chest film showed a pulmonary solitary nodule in the right lower lobe.

Upon the retrieval of the first sample we noticed that a forceps' jaw was missing. A chest film showed the broken fragment within the nodule (Fig 1). Using an identical forceps we obtained three more samples that established the diagnosis of adenocarcinoma. When thoracotomy was performed, this diagnosis was confirmed and the broken piece recovered.

Usual complications of transbronchial biopsy are well known: bronchial bleeding and pneumothorax,2 exceptionally fatal bleeding.3 However, in our review of the literature we have not found any reference to this rare complication. In our patient the fracture of the forceps jaw can only be attributed to an intrinsic metal defect and not to overuse metal fatigue as suggested by Malik and Behera, since our forceps had only been used twice before.

J. P. Masa-Jimenez, M.D.; H. R. Verea-Hernando, M.D.; M. T. Martin-Egana, M.D.; J. Fontan-Bueso, M.D., Respiratory Unit, Juan Canalejo Hospital, La Coruña, Spain

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