Sounds Like a PE to Me

To the Editor:

I wish to comment upon the April ACCP-SEEK Board Review Question of the Month (April 1997), in which a 23-year-old woman presents with sharp left scapular pain, dyspnea, abdominal pain, and nausea. Because the patient had taken a 1,200-mile automobile ride and because the arterial blood gas revealed a pH of 7.50, PaO2 of 60 mm Hg, and Pco2 of 30 mm Hg, we are initially encouraged to believe that this case represents pulmonary embolism (PE). However, the chest roentgenogram revealed free air under the central tendon of the diaphragm and air outlining the wall of a section of bowel. The free air suggested acute perforation of an abdominal viscus.

I am troubled by the explanation of the hypoxemia and hypocarbia, which has been attributed to “splinting and hyperventilation.” Assuming the patient was breathing room air, the alveolar-arterial oxygen difference was 52 mm Hg. In this patient with presumably normal lungs, hyperventilation would lead to hypocarbia, but should not lead to severe hypoxemia. Splinting and atelectasis may result in some degree of hypoxemia, but such an abnormal alveolar-arterial difference should be accompanied by radiographically evident atelectasis. The case illustrated some very important points, but unexplained severe hypoxemia, a clear chest roentgenogram, and a 1,200-mile automobile ride should always make PE a suspect, even if (especially if) there is a concomitant intra-abdominal event.

The free air is a great finding, but I maintain that you would also have to look for PE. A better lesson would be the frequency with which PE presents as a complication of another illness.

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REFERENCE
1. ACCP-SEEK board review question of the month. A woman with shortness of breath and left pleuritic pain. Chest 1997; 111:1115-16

To the Editor:

The author of the question was asked to see this young woman because of the possibility of pulmonary embolism (PE). She indicated that she had been binge drinking over the entire spring break. On examination, there was rebound tenderness. Her actual blood gases were as recorded; indeed, the alveolar-arterial oxygen pressure gradient [P(A-a)O2] was 52 mm Hg. A perforated duodenal ulcer was repaired and the patient recovered uneventfully.

This degree of hypoxemia, widened P(A-a)O2, and hypocarbia is readily explained by her pain, referred from a perforated viscus to her scapula with splinting; radiographically evident atelectasis need not be present. A normal chest radiograph is uncommon in patients with PE.

Chest strapping, which simulates splinting, causes nonuniformity of ventilation as measured by the single breath nitrogen test and modest intrapulmonary shunting. With a fraction of inspired oxygen of 1.0, a decrease in mean PaO2 from 609±5.1 to 569±11.8 mm Hg has been reported. It has also resulted in a decreased closing capacity from 29.7±1.1% to 24.0±3.0%. Finally, in patients with strapped chests breathing air, the PaO2 was reduced to a mean of 81 mm Hg (range, 74 to 88 mm Hg), a calculated mean P(A-a)O2 of 24 mm Hg (range, 17 to 31 mm Hg).

Philosophically we apply the razor of William of Occam, who wrote, “Pluritas non est ponenda sine necessitate” (“Entities should not be multiplied unnecessarily”)—ie, the principle of parsimony or, in the modern vernacular, “keep it simple” (KIS). There can be only one answer to Board examination questions. One must choose the single best answer. The perforated viscus is a sufficient explanation of the facts in this question. It is the single best answer.

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REFERENCES

Truth in Billing

To the Editor:

Dr. Block (March 1997)1 is to be congratulated for the standard he upholds in documenting the care of teaching patients, a standard to which all clinician-teachers ought to subscribe. Attending physicians (and I see many) who continue only to countersign their residents’ notes are not only untruthful when they bill, they are also practicing inferior ICU care.

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REFERENCE
1. Block AJ. The truth is the truth, and HCFA knows it. Chest 1997; 111:531

Does the Type of Surgical Operation Influence the Changes Observed in Fibrinolytic Activity After Cardiopulmonary Bypass?

To the Editor:

Most cardiac surgical operations are performed with cardiopulmonary bypass (CPB). It has long been recognized that CPB results in abnormal hemostasis that may lead to postoperative complications.1,2 Several studies have shown that these patients have an intra- and postoperative activation of the fibrinolytic system, which may be one of the possible culprits of the