Pulmonary Embolism and Occult Right Ventricular Infarction

To the Editor:

We have read with great interest the article by Adams et al relating to the elevation of MB isoenzyme fraction of creatine kinase (CK-MB) after pulmonary embolism as an important contribution to the knowledge of manifestation of occult right ventricular infarction.

We have recently treated a 69-year-old woman who was admitted to the hospital because of a syncope without prodromal symptoms of ischemic heart disease. She developed progressive acute respiratory failure, so she was transferred to the critical care unit, where the laboratory findings were as follows: blood pressure 80/50 mm Hg, pulse rate 135 L/min, temperature 37.5°C, arterial blood gas fraction of inspired oxygen (PaO₂) 45 mm Hg, PaCO₂ 35 mm Hg, pH 7.46, HCO₃⁻ 25 mEq/L, SaO₂ 92 percent, Pa(A-a)O₂ 199 mm Hg, CK 730, and CK-MB 20. An ECG showed sinus tachycardia, right axis deviation associated with complete right bundle branch block (pattern S1Q3T3), ST depression in the precordial lead with T-wave inversion in 3 and aVF. Chest roentgenogram showed unilateral right pleural effusion and bilateral alveolar and interstitial infiltrates without cardiomegaly.

Given the information summarized above, pulmonary artery pressures were measured using a Swan-Ganz catheter; pulmonary pressures were pulmonary artery pressure (PAP) 60/25 mm Hg, pulmonary capillary pressure (PCP) 18 mm Hg, and pulmonary artery diastolic pressure (PAD) 16 mm Hg despite the x-ray film depiction of pulmonary edema. A two-dimensional echocardiogram (2D-ECO) showed tricuspid regurgitation, maximum velocity (Vmax) 3.13 m/s, tricuspid gradient 39 mm Hg, pulmonary artery pressure 49 mm Hg, right ventricular posterior wall akinesia, and normal left ventricular function. The patient who was suspected of pulmonary thromboembolism underwent a pulmonary arteriogram procedure with a Swan-Ganz catheter showing vessel cutoff and filling defects, finding severe hyperfusion of the upper and lower right lobes. Once the diagnosis was set, in situ thrombolytic therapy with 1 million IU urokinase was started.

Posterior evolution showed improvement of the hemodynamic alterations with PAP 24/12 mm Hg, PCP 10 mm Hg, PAD 5 mm Hg, pulse rate 100 L/min, blood pressure 120/80 mm Hg. Arterial blood gases were pH 7.53, PaO₂ 80 mm Hg, Pco₂ 53 mm Hg, HCO₃⁻ 29 mEq/L, SaO₂ 98 percent (FiO₂ 0.4). A 2D-ECO revealed tricuspid regurgitation, Vmax 2.6 m/s, tricuspid gradient 20.9 mm Hg, PAP 90 mm Hg, and right ventricular posterior wall hypokinesia.

Clinical and laboratory findings are similar to those exposed by Adams et al, which confirms the importance of monitoring CK-MB and 2D-ECO in patients with pulmonary thromboembolism in which ECG has excluded the initial diagnosis of ischemic heart disease. With this, we consider that occult right ventricular infarction secondary to pulmonary thromboembolism will be no more considered as an isolated case, to become a situation to worry about not only in the acute state but also as prognostic incidence.

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Exercise Testing in Evaluating High-risk Patients for Resection

To the Editor:

We read with interest the article by Holden et al on preoperative risk assessment for pulmonary resections. While the article states that arterial blood gases were obtained on all patients, these are not listed. We wonder if any of the patients who had complications or death had elevated PaCO₂ levels. This is generally held to be a relative contraindication to any pulmonary resection surgery.


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1 Holden DA, Rice TW, Stelmach K, Meeker DK. Exercise testing, 6-min walk, and stair climb in the evaluation of patients at high risk for pulmonary resection. Chest 1992; 102:1774-79


To the Editor:

Doctors Adams and Hazard raise an important issue regarding the significance of an elevated PaCO₂ in the preoperative evaluation of the patient who will undergo thoracic surgery. The 1990 position paper by the American College of Physicians states: "Severe chronic obstructive pulmonary disease increases the risk associated with lung resection and persistent elevation of arterial PaCO₂ to >45 mm Hg suggests a very high risk relative to the benefit of the procedure." Carbon dioxide levels were <45 mm Hg in all 11 patients experiencing no or only minor complications in our study (range 35-42.5 mm Hg), whereas 1 of the 5 patients who died had a PaCO₂ of 47 mm Hg. A second patient with a PaCO₂ of 44.6 mm Hg was extubated in 24 hours after a pneum-
monectomy. He was discharged home but died on the 85th postoperative day.

While the majority of studies examining preoperative risk have acknowledged that hypercarbia represents a relative contraindication to lung resection, a threshold value of 45 mm Hg has not been formally evaluated. Most studies have not listed PaCO₂ values, although it is likely the majority of patients had a PaCO₂ value <45 mm Hg. Smith et al included one patient with a PaCO₂ of 48 mm Hg who suffered a subendocardial infarction postoperatively but survived. Interestingly, despite a PaCO₂ of 48 mm Hg, his maximal oxygen consumption on a preoperative exercise test was 17.0 ml/kg/min—a value that suggested he would tolerate surgical resection.

We would agree the presence of hypercarbia suggests the patient is at high risk for lung resection. Until we have viable treatment alternatives to surgical resection for bronchogenic cancer, however, it is reasonable to continue attempts to redefine acceptable operable criteria. Exercise testing may identify a subgroup of patients who, despite not meeting gas exchange criteria, would tolerate lung resection. Finally, if the PaCO₂ is used to deny the patient a potentially curative therapy, the clinician must be sure the hypercarbia reflects a limited ventilatory reserve and not an underlying metabolic alkalosis or respiratory drive problem.

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Phrenic Nerve Injury

To the Editor:

We have carefully read the article on phrenic nerve injury by Dr. De Vita, which appeared in the March, 1983, issue of Chest,¹ and enjoyed it. The article provides useful information on the various techniques to evaluate phrenic nerve function. We felt, however, a few suggestions were in order.

Although Dr. De Vita has taken the time to thoroughly evaluate his patients in order to determine the incidence of phrenic nerve injury after cardiac surgery, the authors fail to discuss the fact that the incidence of postoperative phrenic neuropathy is technique dependent. Specifically, the composition of the topical coolant²³ and whether or not an insulating pad was used⁴ have both been shown to affect the incidence to phrenic neuropathy. We would suggest that the high incidence of phrenic neuropathy observed in this study may be a result of the technique of topical myocardial cooling used (ice slush) rather than from the sensitivity of the method used to identify phrenic neuropathy. Other investigators, as well as ourselves have noted a much lower incidence of phrenic neuropathy when ice slush is avoided²³⁵ or an insulating pad is used.⁴

We would also like to point out that Curtis et al observed a similar frequency of spontaneous resolution of phrenic neuropathy. In that study, 78.1 percent of postoperatively elevated hemidiaphragms resolved after 1 year vs a (calculated) 72 percent incidence of radiographic resolution in De Vita et al's study.

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REFERENCES

“ Infective” Myocardial Infarction

To the Editor:

It was with particular interest that we read the article by Blum and colleagues¹ in the April 1993 issue of Chest. At the time of publication, we had on our service a patient who seems to meet all of the characteristics of infective myocardial infarction. We would like to add this case to the literature concerning this syndrome.

A 22-year-old black man was admitted to our service with complaints of chills and fever, which began the evening before admission. Other associated symptoms were a nonproductive cough, weakness, dizziness, sweats, and nausea for 2 days. He also stated an episode of transient loss of consciousness the day before admission. He related a 3-month history of intermittent sharp pain in the left lateral chest and left upper quadrant. His past history was noncontributory and he denied drugs, alcohol, or HIV risk factors. Admission examination revealed these vital signs: temperature 99.6°F, pulse 103, respirations 20, BP 100/44. Physical examination was unremarkable except for decreased breath sounds bilaterally (left more than right) and left upper quadrant guarding and tenderness. Arterial blood gas showed moderate hypoxemia, with a PaO₂ of 67 mm Hg and saturation of 94 percent on room air. Chest x-ray film demonstrated a left lower lobe infiltrate on lateral view. The WBC count was elevated at 20,300 and the ECG showed a sinus tachycardia. He was admitted with a diagnosis of pneumonia and started on intravenous erythromycin. In the early morning, his BP dropped to 80 mm Hg systolic and his temperature was 101.6°F. He received 4 L of crystalloid and his BP rose only to 70 mm Hg. Ceftriaxone was added to his regimen, dopamine was started, and he was

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Communications to the Editor