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, 1993 issue of Chest, 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 dependant. Specifically, the composition of the topical coolant, and whether or not an insulating pad was used, 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 avoided, or an insulating pad is used.

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 colleagues 1 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

<table>
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<tr>
<th>Hospital Day</th>
<th>CK</th>
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<tr>
<td>1</td>
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transferred to the intermediate ICU with a diagnosis of presumed sepsis. He required dopamine for only 5 h. An ECG performed for a complaint of chest pain that morning showed ST-segment elevation in leads 1, aVL, V₅, and V₆, with a smaller degree of elevation in lead 2. Cardiology consultation was obtained. Subsequent ECGs performed over the following days showed a return of the ST segments to baseline, with nonspecific ST- and T-wave changes. Serial cardiac enzymes were as follows:

Based on the elevated creatine kinase (CK) and ECG changes, the patient was transferred to the ICU with the diagnosis of acute myocardial infarction. He was started on heparin and nitrates. Clinical examination was consistent with congestive heart failure, and captopril was added for afterload reduction. An echocardiogram showed a mildly dilated left ventricle, diffuse LV hypokinesia, and a moderately depressed LV ejection fraction. The right ventricle was poorly seen, but mild dilation and depressed function was suspected. A left pleural effusion was identified as well. On the eighth hospital day, a cardiac catheterization was performed and was normal. The patient was discharged after 10 days in the hospital in stable condition, to be followed up by cardiology as an outpatient.

This patient would appear to conform quite closely to the clinical and laboratory picture which characterized the patients in the report by Blum et al. The only departure in this particular case is the initial presence of global, rather than regional wall motion abnormalities and depressed LV (and perhaps RV) function on echocardiogram. This may be explained by the fact that our patient appeared to be truly septic before the onset of the cardiac event. Therefore, sepsis-associated myocardial depression may have been superimposed on dysfunction due to ischemia/infarction.

Our patient did quite well with conservative management and never received thrombolytic therapy. We agree that it would be most useful to obtain angiographic data prospectively in future patients who appear to have infective myocardial infarction, before the institution of thrombolysis, and we would suggest that in young patients without obvious coronary risk factors who present with a similar picture, that approach be taken unless clinically unfeasible at the time. It would also seem reasonable to perform endomyocardial biopsy at the same time to rule out the possibility of myocarditis, especially if no evidence of thrombosis is apparent on the angiogram.

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To the Editor:

The patient presented here had a global ischemia, perhaps reflecting true classical myocarditis with left ventricular (LV) depression. At a later date, when he developed the acute myocardial infarction pattern, an echocardiogram was not performed and may be in this stage, the myocardium would have a regional contraction defect as our patients.

The enigma is to differentiate between true classical myocarditis and acute myocardial infarction when there is a global contraction defect. Therefore, the case presented here is not quite typical of that "syndrome."

It is well-known that infection and especially sepsis cause stimulation of the immunologic system by monocyte-liberating cytokines such as tumor necrosis factor (TNF), interleukin (IL)-1B, IL-6, and IL-8. The IL-1, for example, directly and indirectly activates platelets and causes them to be in "a hypercoagulable state," to secrete platelet-aggregating factor and other growth factors that activate the clotting proteins and the "clotting cascade" through Von Willebrand's factor, fibrinogen, fibrinogen, and other adhesive molecules."

The global, rather than regional wall motion abnormality and depressed LV function, can be caused by cytokines that are secreted by monocytes and lymphocytes during sepsis and cause myocardial depression, for example, TNF and IL-6 are secreted from monocytes and macrophages, and IL-2 is secreted from T-lymphocytes and natural killer cells.

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Pulmonary Vascular Lesions in Chronic Thromboembolic Pulmonary Hypertension

To the Editor:

In the March issue of Chest, Moser and Bloor claim that plexogenic (sic) lesions occur in the small pulmonary arteries of patients with chronic thromboembolic pulmonary hypertension. Figures 4 and 5 in their article illustrate, however, organized and recanalized thrombi rather than plexiform lesion as stated in the respective legends. Plexiform lesions occur in lateral branches of muscular pulmonary arteries measuring between 100- to 150-μm diameter, shortly after their origin from larger muscular pulmonary arteries. They are slightly more common in supernumerary arteries than in symmetrical regular dichotomous branches. The typical plexiform lesion has a striking appearance and has two basic elements. The first consists of a circumferential dilatation of a pulmonary artery branch close to its origin from a parent artery. In this dilated segment, there is destruction of the arterial wall with pronounced thinning of the media, loss of smooth muscle cells, disappearance of the internal elastic lamina, and sometimes also the external elastic lamina. The second element is a plexus of narrow slit-like channels within the dilated segment. These channels which are lined by cells with hyperchromatic nuclei resemble a vascular plexus. The distal part of the plexiform lesion