Pacemaker-induced Friction Rub and Apical Thrill

To the Editor:

I read with interest the article by Flickinger et al,1 which appeared in the July 1992 issue of Chest. One of the most frequent findings in patients with inferior wall myocardial infarctions is an ordinary friction rub, with or without pericardial effusion. Thus, the conclusion that the “vibration induced by pacemaker leads rubbing against intracardiac structures” was the cause of the rub heard in this patient seems problematical. Since the insulation material of modern pacing leads has the properties of a very low coefficient of friction and a very small cross-sectional diameter, the concept that enough friction could be developed to generate a sound of sufficient amplitude to be heard, although perhaps possible, is a difficult one to embrace. An additional mechanism that might well be considered is mechanical interference with valvular function. That the sound was described as loudest during sinus rhythm and least heard during pacing could be attributed to decreased right ventricular pressures and volumes commonly found during ventricular pacing. This could account for the decreased sound amplitude, since pressure of the heart against the pericardium would be decreased as well. Finally, one should also consider that the pacing catheter could have had enough compressive torque to force the infarcted inferior wall against the pericardium, thus causing the described sounds. Although the indicated cause of the sounds heard is possible, other more plausible root causes should also be considered.

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REFERENCE


Diagnosis of Acute Pulmonary Embolism in Patients With COPD

To the Editor:

The article by Lesser et al,1 which appeared in the July 1992 issue of Chest, was of interest to us. There are several issues that need to be addressed before a final conclusion can be drawn regarding the utility of the PaCO2 in the diagnosis of pulmonary embolism in patients with obstructive lung disease. As they mentioned, none of their patients had hypercapnia in the range of that in the patients that we described;2 the highest PaCO2 in the patients with embolic disease appeared to be in the neighborhood of 40 mm Hg prior to embolus. One patient with a pulmonary embolus that they mention in the discussion demonstrated a PaCO2 rise from 51 mm Hg to 68 mm Hg. This does not seem to be reflected in any of the tables or the graphic displays. Certainly, patients can retain CO2 as a result of an increase in dead space, but all the patients we have seen with hypercapnia as a result of a pulmonary embolus have been hemodynamically unstable with massive emboli—a finding confirmed by others.2

Until more supporting evidence is available, we continue to caution our housestaff that in patients with COPD with baseline hypercapnia who have normalized or reduced their PaCO2, the diagnosis of pulmonary embolus must be considered, particularly if the patient is still unstable and dyspneic. Most of our patients with exacerbations of COPD demonstrate hypercapnia, not hypo-

REFERENCE

1 Lesser DA, Jackson WE, Steinberg HD. Chest 1993; 103:1026-7