

Correspondence

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Tracings of Left Heart Failure—Not Mitral Regurgitation

To the Editor:—We read with interest the report by Schaerf *et al.* of a case of amniotic-fluid embolism complicated by congestive heart failure.¹ However, we believe that the authors' diagnosis of acute mitral regurgitation was not justified on the basis of the pulmonary-artery wedge pressure tracing alone, without supporting evidence such as the onset of a new systolic murmur. Assuming the occluded pulmonary artery and electrocardiographic tracings were recorded simultaneously, then the abnormal waves appear to be "a" waves, not the "v" waves of mitral regurgitation. The waves, as shown, follow atrial depolarization and precede ventricular depolarization (fig. 1A). True "v" waves are shown in figure 1B, which represents the occluded pulmonary-artery-pressure tracing in patients with mitral regurgitation.

Abnormally large "a" waves recorded from right atrial, left atrial, or pulmonary-artery catheters in the occluded position can result from atrial contraction against a closed tricuspid or mitral valve such as in junctional rhythm or A-V dissociation, or from atrial contraction against a stenotic atrioventricular valve. More commonly, in early acute heart failure large "a" waves arise from the increased atrial pressure necessary to pump a given volume of blood into a relatively *noncompliant* ventricular chamber. We believe that the authors documented well the onset of heart failure in this patient, with cardiac output decreasing in spite of an increased pulmonary-artery wedge pressure, and that the "a" waves as shown on the wedge pressure tracing are further evidence of left ventricular failure, but not of mitral regurgitation.

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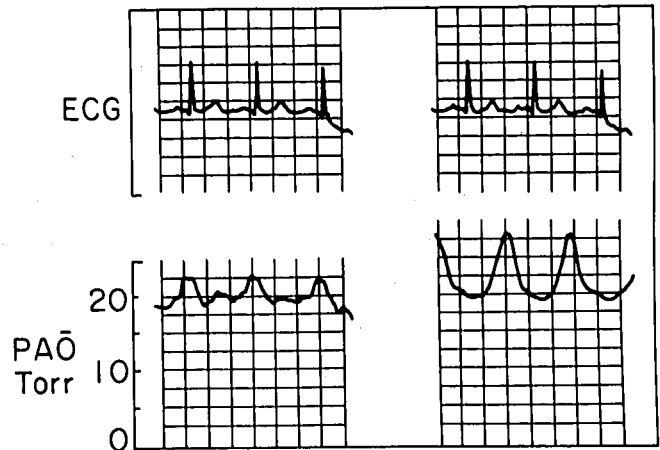


FIG. 1. A (left), electrocardiographic (above) and occluded pulmonary-artery pressure (below) recordings as reported by Schaerf *et al.*¹

B (right), Representative "v" waves as recorded from occluded pulmonary-artery-pressure catheters in patients with mitral regurgitation at Emory University and as reported by Forrester *et al.*²

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2. Forrester JS, Diamond G, Chatterjee K, et al: Medical therapy of acute myocardial infarction by application of hemodynamic subsets. *N Engl J Med* 295:1356–1362, 1976

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Dose Limits to Acute Nitroprusside Therapy Challenged

To the Editor:—At the end of a recent excellent article by Dr. Michenfelder on the toxicity of nitroprusside in the dog, he suggests that nitroprusside, when administered acutely, be limited to a maximum total dose of 1.0 to 1.5 mg/kg.¹ I believe this to be

an unwarranted recommendation. Upon reviewing my clinical practice with nitroprusside over more than 10 years, I find that I have exceeded this dose on more than 100 occasions without difficulty. I have limited the dose to a maximum of 10 μ g/kg/min, but