Atrial flutter (AFl) may exist with or without underlying structural heart disease. Typical AFl presents as a “sawtooth” pattern on the ECG— with inverted flutter (F) waves in the inferior leads and upright F waves in V1. This morphology offers no direct clues as to the underlying cardiac disorder, if any. Occasionally we have encountered giant F waves, most prominently in lead V1, reaching 5 mv or more in height—sometimes exceeding the QRS voltage. The significance of this pattern has not been investigated and reported on. To determine if giant F waves in V1 provide any insight into the presence/type/absence of specific underlying cardiac pathology, the history of 6 consecutive patients with giant F waves was reviewed. Upon review, the only factor common to each patient was the presence of or history of pulmonary hypertension. Right ventricular dilation and/or dysfunction and right atrial enlargement with or without tricuspid insufficiency were present in each by echocardiography. Giant F waves appear to occur in the setting of right heart dysfunction in patients with a history of or the continued presence of pulmonary hypertension. Their detection should indicate the need for right heart evaluation.

Atrial flutter (AFl) is a common arrhythmia¹ that may occur in the presence or absence of underlying structural heart disease and may occur in conjunction with or in the absence of periods of atrial fibrillation.² Most often, atrial flutter takes the form of a right atrial reentrant arrhythmia revolving around the RA isthmus in a clockwise or counterclockwise direction, in which it is referred to a “typical” AFl and produces a “sawtooth” pattern to the flutter waves—especially in the inferior ECG leads; other forms morphologically and/or in location have been considered as “atypical” flutter.⁵,⁶

Among the many structural heart disease alterations to which AFl has been linked is pulmonary hypertension,⁷ whether from congenital⁸ or acquired disorders. However, no specifics of the morphological characteristics of the flutter in the setting of pulmonary hypertension have been described. Accordingly, we found it of note that in six consecutive patients who presented with “giant” flutter waves—5 mv or greater in height in lead V1, often taller than the QRS complex in the same lead, each had a common underlying finding: that of a history of or active presence of pulmonary artery (PA) hypertension and structural right heart alterations.

Methods

The records of six consecutive patients who pre-
sented in atrial flutter with “giant” flutter waves as defined above were reviewed retrospectively. Patient ages were 36 to 73 years; 5 were women. Each underwent echocardiography in addition to other tests deemed clinically necessary in their particular circumstances.

Figure 1-3: 12 lead electrocardiograms from three of the six patients that demonstrate “giant” flutter waves. In the remaining 3 patients, the flutter waves strongly resembled those in panel 2.
Patient #1 was a 36 year old female with primary pulmonary hypertension who was treated with an atrial balloon septostomy. Her pre-procedure PA pressures were 84/48 mm Hg. Patient #2 was a 38 year old female with idiopathic pulmonary fibrosis who ultimately underwent lung transplantation. Her pre-operative PA pressures were 99/52 mm Hg. Patient #3 was a 59 year old female with a history of rheumatic aortic and mitral valve disease who was s/p mechanical mitral and aortic valve replacement. She also had chronic obstructive pulmonary disease. Her estimated systolic PA pressure by echocardiography was 48 mm Hg. Patient #4 was a 73 year old female with a history of rheumatic mitral valve disease, having undergone valvuloplasty in 1954 and eventually prosthetic valve replacement 50 years later. Her estimated PA systolic pressure by echocardiography was 45 mm Hg. Patient #5 was a 37 year old female with primary pulmonary hypertension who ultimately underwent bilateral lung transplantation. Her pre-operative PA pressures were 92/49 mm Hg. Patient #6 was a 62 year old male with a familial hypertrophic cardiomyopathy and mitral insufficiency who was treated medically. Her estimated PA systolic pressure by echocardiography was 50 mm Hg.

Results
In each of the 6 patients described, the ECG presentation of their atrial flutter revealed “giant” flutter waves. None of the patients had atrial fibrillation in the time frame of their atrial flutter. There was no underlying left heart disease in the three patients with primary pulmonary hypertension or idiopathic pulmonary fibrosis. Representative ECGs from three of the patients are shown [figures 1-3]. In addition to the presence of “giant” flutter waves on their electrocardiogram, and an underlying pathology that included the presence of or history of pulmonary hypertension, each of the patients on echocardiography had findings of right ventricular dilation and/or mechanical dysfunction and visually assessed right atrial enlargement. RA planimetry was not routinely performed in all patients. Four also had more than trace tricuspid insufficiency. Flutter was treated medically in each patient and none has undergone electrophysiologic study. In only 1 patient (patient #1) was the flutter “typical” RA isthmus-dependent by 12 lead ECG (not shown).

Discussion
Atrial flutter is a commonly encountered atrial tachyarrhythmia. It may occur as an isolated disorder (“lone”) or alternate with “lone” atrial fibrillation or it may occur in the setting of demonstrable underlying cardiac disease. To date, however, the magnitude of the flutter waves on the electrocardiogram has not been a focus of diagnostic interest. In the six patients we encountered whose data are described above, the magnitude of the flutter waves in ECG lead V1 was “giant,” that is, 5 mv or more. In each there was an underlying common finding of primary or secondary pulmonary hypertension with right ventricular and right atrial enlargement. We have not encountered “giant” flutter waves in any other setting. Thus, we suggest that the detection of “giant” flutter waves should indicate a high degree of suspicion for pulmonary hypertension and right heart pathology and lead to an appropriate evaluation if not already performed.

References