This 48-year-old man, whose brother recently had an acute myocardial infarction, had a recent history of chest discomfort and palpitations. His physician had started him on Inderal outside the hospital but he developed dyspnea and Inderal was quickly cut to tiny doses. The day he entered the hospital he again had chest pain and palpitations.

The admission ECG, taken at 2:35 p.m., is the first one shown and is normal. The second record, taken
at 4:10 p.m., was recorded when his blood pressure became almost unobtainable (90/?), his heart rate rose, and he developed congestive failure with dyspnea at rest and rales half-way up both lungs. This second ECG, shows Atrial Flutter with 1:1 AV response and a heart rate of 238/min. There are ST depressions present, but no T abnormalities. The third ECG, taken the following morning (and after conversion to normal sinus rhythm followed I.V. digoxin) showed antero-lateral T abnormalities (compare to tracing one). These latter changes eventually evolved into negative T waves in subsequent ECGs.

The arrhythmia (second ECG) is called flutter rather than atrial tachycardia because the rate (238/min.) is too rapid for atrial tachycardia which occurs at rates under 200/min. Flutter rates occur at 250-350 and occasionally 400/min.

Atrial flutter rarely occurs in normal persons and hence, by itself, alerts one to an abnormal cardiac situation. This is not the case for atrial fibrillation, which not only is 20 times more common than flutter, but can occur in normals.

The conditions producing atrial flutter include those producing atrial enlargement, e.g., interatrial septal defects, mitral or tricuspid valve disease; metabolic states such as thyrotoxicosis, beri beri, alcoholism; pericarditis alone or associated with pneumonia, car-


dias or pleural tumors; atrial damage in coronary disease. Chronic flutter is very rare and no emboli result from atrial flutter in contrast to atrial fibrillation.

The form of atrial flutter shown here with 1:1 AV response, i.e., without any AV blocking, is rare and is usually a cardiac emergency. Hemodynamic deterioration is rapid and may require electroversion of the flutter. In this man, it is likely that dropping the Inderal to a low, and probably ineffective, level allowed a sympathetic system rebound after beta blockage. Thus the AV node was under less vagal control and allowed for free access to the ventricles of every one of the 238 atrial beats per minute. AV node blockage with digitalis, and especially for the congestive failure, is crucial for the patient. He probably had either an infarction or severe ischemia and his coronary system should eventually be evaluated.

A history of atrial flutter in an insurance applicant, therefore, requires considerable review.

You are invited to comment on these records. In addition, we will consider for publication electrocardiograms sent to this section of the Journal.