Atrial Flutter with 1:1 A-V Conduction*

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To date, there have been fewer than 50 cases of atrial flutter with 1:1 A-V conduction documented in the English literature. The purpose of this paper is to emphasize the diagnostic facets of this relatively uncommon rapid ectopic rhythm and to review the pharmacology of the various therapeutic agents used in its treatment. Two additional cases of 1:1 atrial flutter are reported, one occurring in a patient with chronic constrictive pericarditis.

**Case Reports**

Case 1: This 59 year-old white man was admitted to the Veterans Administration Hospital, Coral Gables, in 1957 because of chills, fever, sweats, cough and dyspnea. There was a past history of bronchiectasis, obstructive emphysema and pulmonary infarction.

The patient appeared markedly cyanotic and dyspneic. His blood pressure was 120/80, and the apical pulse was 140 and regular. There was marked clubbing and cyanosis. Severe wheezing associated with rales and diminished breath sounds was heard throughout both lung fields. The edge of the liver was 3 cm. below the right costal margin, and tender. The laboratory work-up revealed respiratory acidosis, with hypoxia and CO₂ retention. An electrocardiogram was interpreted as showing atrial flutter with 2:1 A-V block and cor pulmonale (Fig. 1). The atrial rate was 280/minute, the ventricular rate 140/minute. Prior to death the patient had frequent episodes of 1:1 A-V conduction, unresponsive to carotid sinus pressure (Fig. 2). Intravenous acetyl strophanthidin given under electrocardiographic control, however, increased the degree of A-V block. In spite of all measures, the patient's course was progressively downhill. Autopsy disclosed bronchiectasis, severe pulmonary emphysema and cor pulmonale.

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FIGURE 1—Case 1: Control electrocardiogram before onset of 1:1 A-V conduction. Atrial rate, 280; ventricular rate, 140. There is atrial flutter with 2:1 A-V conduction (occasionally 3:1 conduction). Incomplete right bundle branch block and a rare premature ventricular contraction are also present.
Case 2: A 24 year-old white man was admitted to the Veterans Administration Hospital, Coral Gables, in 1956 for episodes of palpitation, paroxysmal dyspnea and weakness precipitated by exertion or excitement. Past medical history was negative. Physical examination revealed a regular pulse of 140 per minute with a blood pressure of 110/90. There were signs of minimal right-sided heart failure. A chest x-ray film suggested calcification of the pericardium. An electrocardiogram revealed atrial flutter with varying degrees of A-V block and right ventricular hypertrophy. Cardiac catheterization was consistent with a diagnosis of constrictive pericarditis. Preoperative digitalis and quinidine failed to convert the atrial flutter to normal sinus rhythm. At thoracotomy, a thickened, partially calcified pericardium was excised and his postoperative course was uneventful.

FIGURE 2—Case 1: A. Six standard lead electrocardiogram showing atrial flutter with 1:1 A-V conduction. In lead I there is one brief period of 2:1 conduction. Atrial and ventricular rates are 300 per minute. B. Long leads I showing continuous atrial flutter with 1:1 A-V conduction. The rate is 300 per minute. C. Standard leads I and II after intravenous acetyl strophanthidin showing atrial flutter with 2:1 block: Atrial rate 300, ventricular rate 150 per minute.

FIGURE 3—Case 2: Control electrocardiogram demonstrating atrial flutter with varying degrees of block. Atrial rate 300. There is evidence of incomplete right bundle branch block with probable right ventricular hypertrophy.
Shortly after discharge, he was readmitted for severe palpitation brought on by exertion. A control electrocardiogram showed atrial flutter with 3:1 block; the atrial rate was 300/minute (Fig. 3). Minimal exertion precipitated an episode of 1:1 A-V conduction (Fig. 4). He experienced dyspnea, weakness, and severe palpitation coincident with his rapid heart action.

With re-digitalization, a high degree of A-V block was obtained and the patient has remained relatively asymptomatic. Several trials with large doses of quinidine by mouth failed to produce a normal sinus rhythm.

Discussion

Atrial flutter with varying degrees of A-V block is not uncommon. Katz and Pick1 observed an incidence of 0.54 per cent in patients having electrocardiograms at the Cook County Hospital.

Certain well-defined criteria have been considered necessary to establish a diagnosis of atrial flutter.2 The atrial rate should range between 225-360 per minute with constant atrial activity, manifested by the absence of an iso-electric period measuring 0.04 second or more. Often a sharp upstroke of the flutter wave and gradual downstroke is observed which is manifest electrocardiographically as the typical “saw tooth” appearance of the flutter waves. Flutter waves are usually perfectly regular and are best seen in standard leads I and II. There is occasionally some lengthening of the F-R interval and this accounts for the slight variation in the R-R interval which may be observed. Due to the usual presence of A-V nodal block of varying degree, the ventricular rate in atrial flutter usually ranges from 75 to 160 per minute.

To aid in determining whether a rapid ectopic rhythm is atrial flutter with 1:1 A-V conduction, the presence of atrial flutter with higher grades of block either before or after the onset of the 1:1 conduction is often necessary. The occurrence of auricular fibrillation or atrial flutter with a high grade of A-V heart block following digitalis therapy has also been observed to be helpful in defining a rapid ectopic rhythm as atrial flutter with 1:1 conduction.

When 1:1 A-V conduction has been observed, symptoms have included dyspnea, palpitation, syncope, angina pectoris and heart failure.3 It has been shown that with ventricular rate above 180 per minute, there is a precipitous fall in both cardiac output and coronary blood flow.4 This is not an absolute figure, and an individual patient may have relative coronary insufficiency and myocardial ischemia at a slower ventricular rate.

While atrial flutter with varying degrees of A-V block is usually diagnosed readily with the aid of the electrocardiogram, 1:1 A-V conduction has been confused with paroxysmal atrial, nodal and ventricular tachycardias and ventricular flutter. The ventricular complexes may be widened as a result of organic disease or as a result of the so-called “fatigue” of a bundle and add to the difficulty in establishing a correct diagnosis. Various attempts to increase vagal tone may be of invaluable diagnostic aid by increasing the degree of A-V block, and thereby providing an opportunity for clarification of the atrial mechanism.5

Digitalis, quinidine, and procaine amide have been recommended for the treatment of atrial flutter.6-10 Digitalis is generally considered the drug of choice in initiating the therapy of rapid atrial flutter.11-15 Both by a direct effect on the A-V node
and by a vagotonic action, digitalis slows A-V nodal conduction and protects the ventricle from the rapidly discharging atria.

Quinidine and procaine amide may terminate atrial flutter or atrial fibrillation by conversion to normal sinus rhythm. This appears to be accomplished by both direct and vagolytic effects on the atria. According to the classical concept of Sir Thomas Lewis, if the refractory period is increased to a greater degree than the conduction is slowed, then the head of a circus wave confronts refractory muscle and is stopped.

More recently the circus movement theory has been challenged and the concept of a rapidly discharging ectopic focus presented as an explanation for the atrial arrhythmias. Accordingly, the beneficial effects of quinidine and procaine amide in atrial flutter or fibrillation can be explained by their depressant effect on rapid ectopic auricular pacemakers.

Quinidine and procaine amide have often been considered etiological factors in the production of 1:1 A-V nodal conduction in patients with established atrial flutter. This dangerous complication of quinidine and procaine amide therapy results from a transient vagolytic effect on the A-V node. This increase in A-V nodal conduction, associated with drug induced slowing of the atrial flutter rate, may convert an atrial flutter rate of 340 per minute with 2:1 A-V nodal conduction to an atrial flutter with a rate of 280 to 300 per minute with 1:1 A-V conduction.

It would appear, therefore, that any attempt to convert atrial flutter to normal sinus rhythm with quinidine or procaine amide is hazardous if the conduction through the A-V node has not been previously delayed by adequate digitalis therapy.

REFERENCES