Atrial Flutter with 1:1 A-V Conduction*

Report of a Case in a Pregnant Woman Successfully Treated with DC Countershock

HOWARD F. SUSSMAN, M.D.** DARIO DUQUE, M.D.† and MILTON E. LESSER, M.D.‡

Miami Beach, Florida

INTRODUCTION

Atrial flutter is found in less than 0.5 per cent of patients clinically suspected of having heart disease.1,4,7 Atrial flutter with 1:1 A-V conduction is considered to be a rare entity. Jolly and Ritchie8 discussed atrial flutter in 1910; and in 1914, Ritchie9 published a monograph establishing the clinical features of this arrhythmia. The first documented case of atrial flutter with 1:1 A-V conduction was reported by Lewis3 in 1915. In 1956, Finkelstein and associates10 reported 40 additional cases which had been described in the literature and added six cases of their own. Since then, there have been scattered cases reported, bringing the total to approximately 60. The following electrocardiographic criteria are considered by Finkelstein et al., to be suggestive of atrial flutter with 1:1 A-V conduction: (1) the presence of undulating or oscillating (“sawtooth”) atrial waves occurring at a rate of 225 to 315 per minute and accompanied by ventricular response to each atrial impulse; (2) demonstration of atrial flutter with higher degrees of A-V block either before or after the onset of the 1:1 arrhythmia and (3) response to treatment of an ectopic rhythm with a rapid ventricular rate by either increasing degrees of A-V block or atrial fibrillation. The symptoms and signs of 1:1 flutter depend on the age of the patient, the ventricular rate, the duration of the tachycardia and the presence or absence of underlying heart disease.

CASE REPORT

The patient is a 32-year-old white woman, gravida IV, para 3-0-0-3, who had open heart surgery on January 27, 1960 for repair of a congenital inter-atrial septal defect with an anomalous right inferior pulmonary vein. Postoperative catheterization studies on May 16, 1960 showed complete physiologic closure of the inter-atrial septal defect. Following surgery, she has had frequent episodes of various cardiac arrhythmias, namely atrial flutter with varying 2:1 and 4:1 block; incomplete right bundle branch block pattern which was also present prior to surgery; intermittent sinus and nodal rhythm; sinus bradycardia with sinus arrhythmia and wandering pacemaker and nodal escape beats; nodal rhythm; sinus rhythm with wandering of the pacemaker into the A-V node, A-V nodal tachycardia with periods of A-V dissociation and nodal escapes; nodal rhythm with atrial premature systoles in bigeminy; normal sinus rhythm with A-V dissociation and interference and nodal rhythm with retrograde and orthograde conduction; nodal rhythm with bigeminal A-V nodal premature systoles, probably due to digitalis toxicity; and sinus arrhythmia.

Further review of the past medical history failed to reveal evidence of diabetes mellitus, hypertension, episodes of hypotension or electrolyte imbalance. Her three previous pregnancies ended in spontaneous delivery of full term normal infants. On January 29, 1964, the patient’s cardiac rhythm reverted from atrial flutter with predominant 2:1 and 4:1 A-V conduction to a nodal rhythm at a rate of 45 per minute. (Fig. 1). This nodal rhythm lasted until February 1, 1964, at which time atrial flutter with 2:1 A-V block was noted. On February 4, 1964, atrial flutter with 2:1 and 4:1 A-V conduction was again present. On February 5, 1964, at 8:45 a.m., an electrocardiogram showed an atrial flutter with 4:1 A-V block. Three hours later, however, at 11:45 a.m., the electrocardiogram showed an atrial flutter with 2:1 A-V block which later changed to atrial tachyarrhythmia at a rate of 280 per minute with 1:1 A-V conduction (Fig. 2). At that time, this seven weeks pregnant patient complained of weakness, dizziness, diaphoresis, cardiac palpitations and fear of impending death.

*From the Cardiovascular Division, Department of Internal Medicine, Mount Sinai Hospital.
**Chief Resident in Medicine, Mount Sinai Hospital.
†Fellow in Cardiology, Cardiovascular Division, Department of Internal Medicine, Mount Sinai Hospital.
‡Associate Attending, Cardiovascular Division, Department of Internal Medicine, Mount Sinai Hospital.
Figure 1: Nodal rhythm at a rate of 45 per minute, one of the standard and extremity leads, the other of precordial chest leads.
ATRIAL FLUTTER WITH 1:1 A-V CONDUCTION

Figure 2: Leads I and II show atrial flutter with 2:1 A-V block (atrial rate 280 per minute, ventricular rate 140 per minute), which changes in the third line from the top to atrial flutter with 1:1 A-V conduction (atrial and ventricular rates 280 per minute). The fourth line shows 100 watt/second synchronized DC countershock, followed by a long period of asystole. Leads I and AVF at the bottom show nodal rhythm at a rate of 71 per minute.

Physical examination revealed an anxious, dyspneic, slightly cyanotic, acutely ill woman, with rapid pulsations of the chest wall, clamminess of the skin, pallor and faint peripheral pulses. There was no clubbing of digits or ankle edema. The blood pressure was 90/60 mmHg, respirations 32 per minute and regular, and temperature was normal. Neck veins were flat. Lungs were free from rales. Examination of the heart revealed a rapid rate of over 200 per minute. No correlation could be made between the apical, jugular and carotid pulses. The abdomen was soft and non-tender. There was no hepatomegaly or splenomegaly. Carotid sinus massage, first right and then left, each of eight seconds duration failed to effect any change on the electrocardiogram. Her condition rapidly worsened, the blood pressure dropped to 70/40 mmHg. She was still fully conscious and oriented and was given 100 mg. of meperidine (Demerol) intramuscularly, and 100 mg. of pentobarbital (Nembutal) intramuscularly. Fourteen minutes

Figure 3: Wandering pacemaker from S-A node to A-V node at a rate of 56 to 53 per minute.
later, an additional 100 mg. of pentobarbital was given intravenously because of the persistence of extreme apprehension. One minute later, the patient was given 100 watt/second synchronized direct current countershock across the intact precordium, which resulted in nodal rhythm (Fig. 2). Immediately thereafter, the entire clinical status of the patient markedly improved. The blood pressure returned to a level of 110/70 mm.Hg (normal for the patient being 110/60 to 128/80), the cyanosis and pallor disappeared and once again the peripheral pulses became full and easily palpable. Prior to the 1:1 A-V conduction, she had been fully digitalized and was receiving maintenance doses of 0.5 mgm. digoxin orally and Pronestyl 500 mg. four times a day. Immediately following the countershock, the procainamide hydrochloride (Pronestyl hydrochloride) was discontinued and quinidine was started at a dose of 200 mg. four times a day. The quinidine, however, was discontinued three days later on February 8, 1964, because of gastrointestinal intolerance and oral procainamide hydrochloride 500 mg. four times a day was once again instituted. Serum electrolytes and blood urea nitrogen before and after the episode of atrial flutter with 1:1 A-V conduction were stable and within normal limits. The patient was never placed on anticoagulants.

On April 6, 1964, the cardiac rhythm was wandering pacemaker from S-A node to A-V node at a rate of 56 to 53 per minute (Fig. 3). Her obstetrician reported that as of June, 1964, the pregnancy was progressing well. Her only complaint was mild exertional dyspnea, although the lungs were free from rales. An electrocardiogram on June 16, 1964, revealed atrial flutter at a rate of 272 per minute with variable A-V conduction (Fig. 4A). Following this a fetal electrocardiogram revealed fetal sinus arrhythmia at an average rate of 136 per minute (Fig. 4B). On September 6, 1964, the patient had a spontaneous vaginal delivery of a normal 6 pound 11 ounce girl.

COMMENTS

Accepted etiologic factors in the production of 1:1 A-V conduction in atrial flutter include (a) serious myocardial damage, (b) increased sympathetic tone (such as occurs during exertion, emotional strain and induction of anesthesia), (c) quinidine or procainamide therapy when given during atrial flutter with partial degrees of A-V block and (d) atropine given intravenously to patients with rheumatic heart disease and atrial fibrillation or atrial flutter with 2:1 A-V block.

Atrial flutter with 1:1 A-V conduction represents a cardiac emergency because of the rapid ventricular rate and requires prompt treatment. It may represent a catastrophic event depending on the duration of the tachycardia and the underlying disease. (myocardial infarction, uremia, congenital heart disease, congestive heart failure, pulmonary emboli, or degenerative heart disease such as progressive muscular dystrophy).

The electrocardiographic diagnosis of atrial flutter with 1:1 A-V conduction is frequently complicated by the development of aberrant ventricular conduction secondary to functional fatigue of the bundle branches due to the rapid rate.

Procainamide and quinidine are known to decrease the flutter rate and to have transient vagolytic effect on the A-V
Quinidine or procainamide slows the atrial rate and/or increases the conductivity of the A-V node to a point where the conduction tissues become responsive to each atrial impulse instead of to a fraction of the atrial impulses, whether 2:1, 3:1, 4:1, etc. 4-10, 11-13 It has thus become common practice to administer digitalis prior to quinidine. 14 Digitalis tends to decrease the refractory period of atrial tissues and to increase the refractory period of the conduction tissues, thereby tending to change atrial flutter into atrial fibrillation and to permit fewer atrial impulses to reach the bundle of His. 14 However, digitalis does not always prevent the development of 1:1 A-V conduction. 4-14 Although digitalis increases A-V block, exercise in digitalized patients with persistent atrial flutter may temporarily decrease the degree of block and thus produce periods of rapid ventricular rate. 11

Carotid sinus pressure is usually ineffective in slowing the ventricular rate. 15 Digitalis, quinidine and procainamide may not be effective in converting the 1:1 flutter.

Since the work of Lown and associates, 16 first published in 1962, DC countershock has been firmly established as a treatment of choice in the conversion of many arrhythmias, especially in life-threatening situations.

Several thousand patients have now been treated by this modality with few untoward effects.

A review of the literature, however, does not show any instance of DC countershock being used to terminate the threatening situation of 1:1 conduction supervening in atrial flutter. Nor is there any evidence in the literature that DC countershock has been employed in patients who are pregnant.

In our patient, a single shock sufficed to change the rapid conduction defect to a slower nodal rhythm, a rhythm which the patient had manifested several times prior to the emergency situation. That this treatment was clinically effective was further shown by the immediate elevation of her blood pressure to normotensive levels without the concomitant use of vasopressor agents.

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References