ATRIAL FLUTTER–ATRIAL FIBRILLATION: IS A DISTINCTION CLINICALLY NECESSARY?

By ETIENNE O. ROBLES DE MEDINA, M.D., and FRITS L. MEIJLER, M.D.

In most cases, the distinction between atrial flutter and atrial fibrillation can readily be made from their ECG characteristics. Some cases, however, show characteristics of both. Because of the similarity in etiology, hemodynamic response, and current approach to pharmacological treatment, differentiation between the two arrhythmias may not be necessary. These authors weigh the evidence.

It is common practice in reading an ECG to try to distinguish between atrial flutter and atrial fibrillation. When characteristics of both arrhythmias are present, such terms as *atrial flutter-fibrillation* and *fibrillo-flutter* have been used. A WHO/ISFC Task Force on definition of cardiac arrhythmias[1] has recently defined atrial flutter as an arrhythmia that is characterized by rapid and regular electrical activity of the atria, usually in the range of 200 to 350/min. In typical cases, the atrial deflections, called flutter or F waves, are inverted in leads II, III, aV_F, and V_6, and no isoelectrical line exists between the F waves in these leads (Fig. 1). In uncommon cases, the atrial rate is usually less rapid, ranging from 100 to 250/min,[2] the polarity of the flutter waves is different, and an isoelectrical line may be seen between the atrial deflections. Occasionally, the polarity of the F waves may change during the same recording.

Conversely, atrial fibrillation has been defined as rapid and irregular disorganized electrical activity of the atria.[1] As a consequence, no P waves are discernible in the ECG, and the baseline shows irregularly spaced wavelets varying in size and direction (f waves) (Fig. 2). The f waves may be fine or coarse, depending on such factors as atrial volume, duration, and etiology of the arrhythmia. Coarse, high-amplitude f waves may mimic flutter waves. It should be noted that, with normal AV conduction, atrial fibrillation is typically characterized by random distribution of the R-R intervals; this characteristic, however, is not definitional of the arrhythmia, because this is an aspect of AV conduction[3] and possibly of the atrial activation pattern.[4]

Usually, the distinction between atrial flutter and fibrillation is not difficult; as previously mentioned, however, an arrhythmia may occasionally show characteristics of both (Fig.

From the Department of Cardiology, University Hospital, Utrecht, The Netherlands.

Dr. Robles de Medina is a professor of clinical cardiology.

Dr. Meijler is director of the Department of Cardiology.

3). Interesting as the differentiation may be from an electrophysiological point of view, the clinician who is treating the patient may rightly ask whether or not the distinction is of any clinical relevance. It seems that the most appropriate way to answer this question is to review both arrhythmias with regard to electrophysiological mechanisms, etiology, hemodynamic effect, response to treatment, and prognosis.

ELECTROPHYSIOLOGICAL MECHANISMS

Because this discussion mainly concerns the clinical aspects of atrial flutter and fibrillation, the consideration of their possible electrophysiological mechanisms pertains only to those aspects which may have therapeutic consequences. Three possible mechanisms have been considered in both arrhythmias, none of which has yet been established in humans: (1) rapid impulse formation in a single ectopic focus, (2) multifocal (polytopic) impulse formation, and (3) reentry within the atria.

A comprehensive survey of these theories, including arguments in favor of and against them, is given by Scherf and Schott.[5] Recent observations by Allessie,[6] using 192 simultaneously recorded electrographs to map the spread of activation during experimentally induced atrial flutter in the dog heart, showed

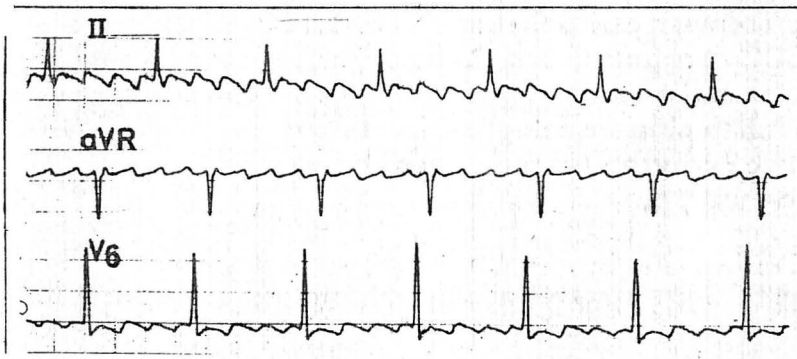

Figure 1. Common type of atrial flutter. Notice typical saw-toothed appearance of the baseline and inverted flutter waves in leads II and V_6. AV conduction ratio is 4:1. Leads are not simultaneous. This and subsequent ECGs have been recorded at a speed of 25 mm/sec.

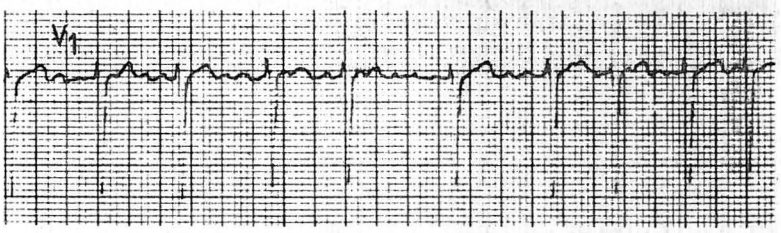

Figure 2. Atrial fibrillation. The baseline shows small, irregularly spaced wavelets that vary in size, shape, and direction. Typically, the R-R intervals are totally irregular.

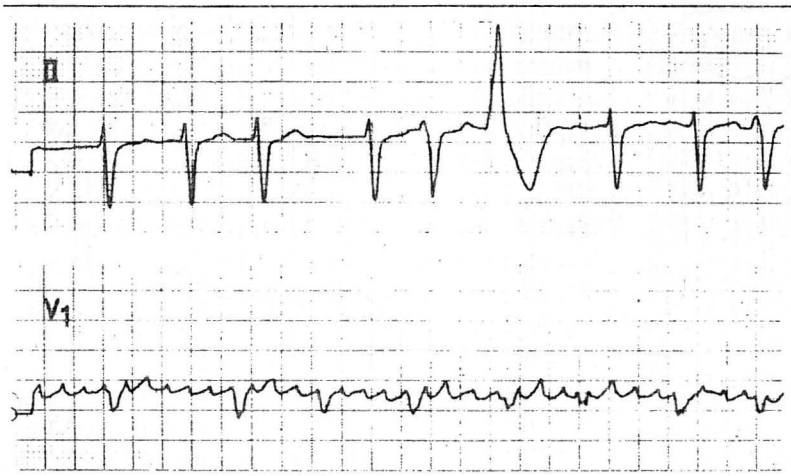

Figure 3. Atrial flutter-fibrillation. Nonsimultaneous recordings (digital to analog conversion) of leads II and V_1. The upper panel shows the characteristics of atrial fibrillation, whereas the lower strip suggests atrial flutter with variable AV conduction ratio. The atrial rate (428/min), however, is higher than is commonly seen with atrial flutter. (See Figure 1.)

that the arrhythmia was based on a continuous circus movement of the impulse within the atrial myocardium. The reentrant circuits were of a functional type, and their size and dimensions varied from case to case. If this finding also pertains to flutter in the human heart, it would result in the expectation that atrial flutter could be terminated by programmed electrical stimulation.

Coupled atrial extrastimuli, however, commonly fail to interrupt the arrhythmia,[7,8] whereas rapid stimulation of the atrium for a critical period of time and at a critical rate above the flutter frequency is highly successful.[8-10] According to Waldo et al,[10] these results argue against a macroreentrant circuit around the orifices of the great veins but do not allow the differentiation between enhanced automaticity and a small reentrant circuit. Conversely, in atrial fibrillation, disorganized, fragmented electrical activity makes it impossible to stimulate the atria. Hence, atrial fibrillation can only be terminated by drug treatment or by electric shock.

ETIOLOGY

Atrial fibrillation is a far more common arrhythmia than is atrial flutter; the underlying disease states and precipitating factors, however, are similar. Both arrhythmias are commonly indicative of organic heart disease, such as ischemic, hypertensive, and rheumatic heart disease, primary myocardial disease, congenital anomalies that are associated with dilatation of the atria (interatrial septal defect, mitral and tricuspid valvular disease), and inflammation or infiltration of the pericardium by pneumonia or by bronchogenic carcinoma. In addition, the association of atrial fibrillation and, less often, atrial flutter with hyperthyroidism has long been known. Almost any acute infectious disease or metabolic derangement, especially in the

postoperative phase of major surgery, may precipitate transient atrial fibrillation or flutter. In many cases, combined etiologic factors are operative, such as hypertensive and ischemic heart disease.[11]

As a complication of acute myocardial infarction, both atrial flutter and fibrillation may indicate incipient left heart failure or associated atrial infarction. Furthermore, both arrhythmias frequently occur as part of the bradycardia-tachycardia syndrome, a manifestation of the sick sinus syndrome.

Of particular interest is the low association with digitalis toxicity.[12,13] In most cases, the arrhythmias probably result from the underlying heart disease rather than from digitalis toxicity. Therefore, if atrial flutter or fibrillation develops in a digitalized patient and the ventricular rate is above 100/min, it is our policy to add a small dose of digoxin and monitor its effect on the arrhythmia, unless, of course, symptoms or other arrhythmias known to be associated with digitalis excess are present.[12] If no untoward effects occur following the additional dose of digitalis, the arrhythmia probably is not caused by digitalis toxicity. Indeed, the patient may benefit from a higher dose. If this approach is not feasible because of the lack of facilities to monitor the patient, it is best to stop the drug for one or two days and observe the arrhythmia. Determination of the plasma level of digoxin may also be helpful.

A similar diagnostic and therapeutic dilemma may arise when established atrial flutter or fibrillation is being treated with digitalis in order to slow the ventricular response. When anomalous QRS complexes occur during such treatment, one must decide whether one is dealing with ventricular ectopy, and hence the possibility of digitalis excess, or aberrant intraventricular conduction of supraventricular impulses, in which case more of the drug is required. Guidelines for this differentiation have been

given by Marriott and Sandler[14] and by Wellens et al.[15]

In addition, atrial flutter and fibrillation not infrequently complicate the Wolff-Parkinson-White (WPW) syndrome, especially in the presence of a left-sided bypass tract.[16] In such cases, extremely rapid ventricular rates may be life threatening (Fig. 4). In some cases of atrial fibrillation, no etiologic or known precipitating factor can be found, a condition referred to as lone atrial fibrillation. Such cases also occur with atrial flutter.

HEMODYNAMIC EFFECT

The hemodynamic effect of an arrhythmia is determined by the ventricular rate, the sequence of ventricular activation, the presence or absence of effective atrial contractions, and the underlying condition of the heart. The faster the ventricular rate, the shorter the time available for filling of the ventricles and coronary perfusion, resulting from abbreviation of diastole. In addition, myocardial oxygen demand is determined mainly by the ventricular rate.[17] When the mitral valve is normal, ventricular filling may not be compromised significantly unless the rate is excessively high, because ventricular filling is almost completed at the end of the early rapid filling phase. With mitral stenosis, however, ventricular filling continues during the whole of diastole, and shortening of filling time may seriously affect the circulation. It is therefore not surprising that patients with severe mitral stenosis often develop pulmonary congestion and pulmonary edema at the onset of atrial fibrillation or flutter.

In untreated atrial flutter, 2:1 AV block is usually present, with a ventricular rate of 125 to 175/min, usually 150/min. With 1:1 AV conduction, however, the ventricular rate may exceed 250/min (Fig. 5). With untreated atrial fibrillation, the ventricular rate is also high, usually in the range of 140 to 200/min. Extremely rapid rates may be seen in the WPW syndrome, when the effective refractory period of the bypass tract is short (Fig. 4).

The absence of atrial contractions with atrial fibrillation further contributes to an adverse hemodynamic effect, but this is also true for atrial flutter, where in each cardiac cycle one

or more atrial contractions occur during ventricular systole, when the atrioventricular valves are closed. With normal intraventricular conduction, the sequence of ventricular activation, and hence the sequence of ventricular contraction, is normal; this changes, however, in the presence of aberrant intraventricular conduction and may occur with both atrial flutter and fibrillation, contributing further to the altered hemodynamics.

Despite these unfavorable effects, when the heart is normal the patient may tolerate the arrhythmias surprisingly well. With mitral stenosis or impaired contractile function of the left ventricle, however, even the most vigorous compensatory mechanisms may not be adequate to prevent congestive heart failure, a significant fall in blood pressure, or both. Similarly, because both arrhythmias are significantly associated with organic heart disease, no difference exists in this respect.

RESPONSE TO TREATMENT

The management of atrial flutter and fibrillation is discussed in detail in several textbooks.[18,19] This section focuses mainly on possible differences in therapeutic response. When treatment is indicated, because of subjective symptoms, signs of hemodynamic dete-

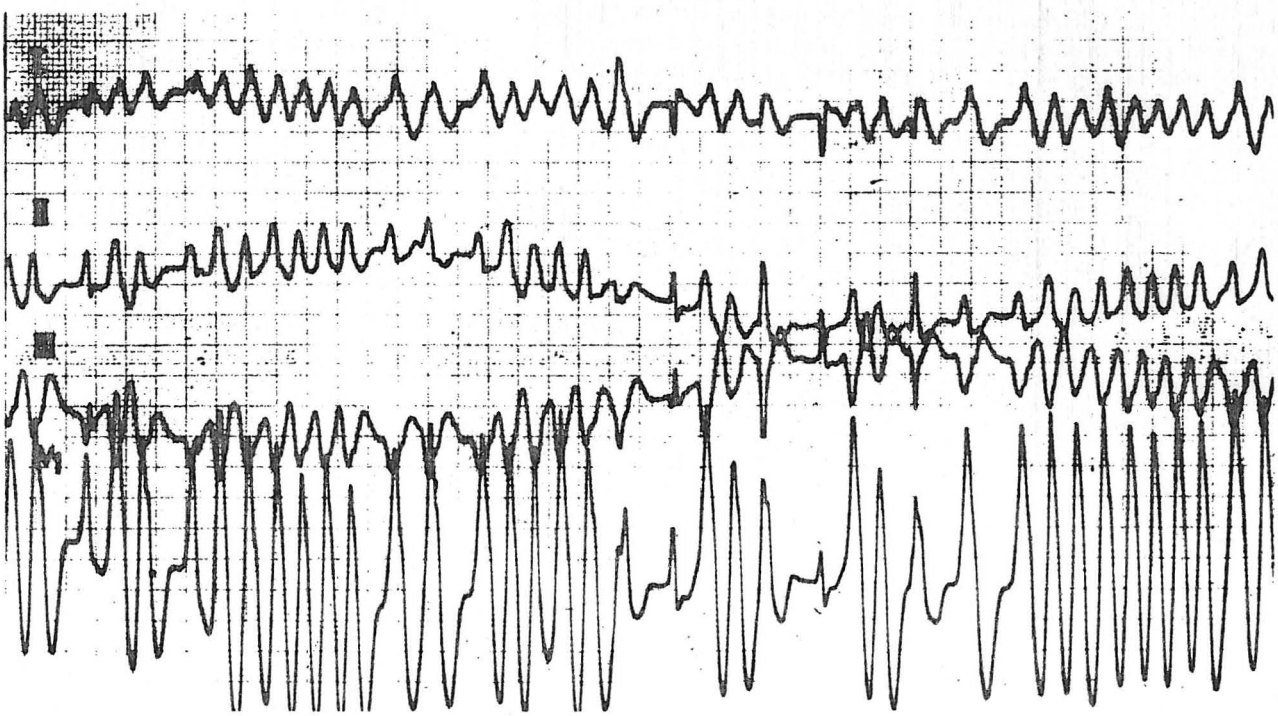

Figure 4. *Atrial fibrillation in a patient with the Wolff-Parkinson-White syndrome and a left-sided bypass tract. Notice extremely rapid ventricular response (shortest R-R interval is 160 ms). After long R-R intervals, QRS complexes show various degrees of normalization.*

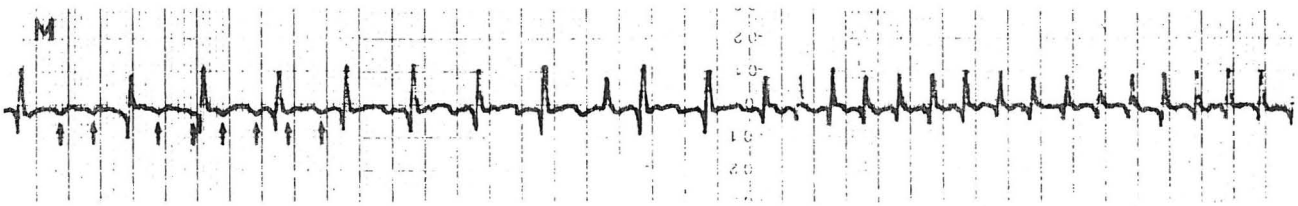

Figure 5. *Atrial flutter with variable AV conduction ratio. On the right, transition into 1:1 AV conduction is seen, with a ventricular response of 300/min. Arrows point to flutter waves.*

rioration, or both, the primary concern is the fast ventricular response. If the situation is urgent, the treatment of choice is cardioversion by DC shock. In this respect, a difference in response may be seen between the two arrhythmias, because atrial flutter is extremely sensitive to DC shock, often responding to 50 W or less. Atrial fibrillation is less sensitive to DC shock, especially when the fibrillatory waves are fine. Thus, an overall difference exists in the amount of energy required to convert the arrhythmias to sinus rhythm.

With regard to pharmacological management, both arrhythmias show an identical response to beta blockers and calcium antagonist drugs (e.g., verapamil), which increase AV block and temporarily slow the ventricular response but usually will not convert the arrhythmia to sinus rhythm. The latter can be achieved in a high percentage of cases by the oral administration of high doses of quinidine. Slowing of the atrial rate and, as a consequence, decreased concealed conduction in the AV node may cause a paradoxical increase in ventricular response with consequent deterioration of the hemodynamic status. Therefore, attempts at conversion with quinidine are best accomplished by combining the drug with digitalis or a beta blocker to protect against untoward acceleration of the ventricular rate. We prefer to combine quinidine with a beta blocker instead of digitalis to enable DC shock when drug conversion fails.

When the primary goal of treatment is to slow the ventricular response, rather than conversion to sinus rhythm, digitalis is the drug of choice. Clinical experience has shown that, in this respect, established atrial flutter is far more resistant to digitalis than is atrial fibrillation. High doses are often required in atrial flutter, with consequent narrowing of the range between therapeutic and toxic doses. Therefore, many clinicians now prefer to combine the usual doses of digitalis with a beta blocker. When the arrhythmias are of recent onset, a fair chance of conversion to sinus rhythm exists a few hours after digitalization. It has been stated that this is not the effect of digitalis but rather the result of spontaneous subsidence of the arrhythmia. Although this is difficult to disprove, the clinical impression is that digitalis works. Atrial flutter often converts to atrial fibrillation, with sinus rhythm supervening after cessation of digitalis. In patients with the WPW syndrome and atrial flutter or fibrillation, digitalis is basically contraindicated because it shortens the effective refractory period of the accessory pathway[16] and may thus enhance the ventricular response.

When the primary goal of treatment is to slow the ventricular response, rather than to convert to sinus rhythm, digitalis is the drug of choice

Another important aspect of therapy is the problem of anticoagulation. It is generally accepted that atrial fibrillation is associated with an increased risk of atrial thrombosis and embolic complications, especially in the presence of mitral stenosis.[11,20] No convincing evidence exists, however, that atrial fibrillation *per se* increases the risk of atrial thrombosis and embolism. Thus, the increased incidence of embolism in patients with left heart failure and atrial fibrillation[21] may result from thrombosis in the ventricles or in the atria, secondary to atrial dilatation. Therefore, only those patients in whom atrial fibrillation is caused by mitral valve disease or left heart failure require anticoagulation. Anticoagulants have also been advocated one to two weeks prior to cardioversion, to prevent embolism after restoration of sinus rhythm; no definite proof is available, however, to support this practice.

Data on thromboembolic complications during atrial flutter are lacking, probably because of the rarity of the chronic form of this

arrhythmia. Nonetheless, although atrial contractions are generally maintained during flutter, it does not seem unreasonable to follow the same indications for anticoagulation as are followed in atrial fibrillation.

PROGNOSIS

The excellent prognosis in cases of lone atrial fibrillation is an indication that atrial fibrillation *per se* is compatible with normal life activities and normal life expectancy. Godtfredsen,[11] however, stressed the complex interplay of several factors in the evaluation of the prognostic implications of atrial fibrillation. Patients less than 70 years of age and patients with mitral valve disease, thyroid heart disease, and lone fibrillation had a relatively good prognosis when ventricular rate was controlled, whereas patients more than 70 years of age and patients with atherosclerotic heart disease, aortic valve disease, or moderately severe congestive heart failure had a poor prognosis (i.e., a five-year survival of less than 50 percent). Again, data on prognosis with chronic atrial flutter are lacking, most cases eventually converting to atrial fibrillation.

CONCLUSION

In the majority of cases, the distinction between atrial flutter and atrial fibrillation can readily be made from the classical ECG characteristics of these rhythms. In such cases, the arrhythmias must be classified under their respective headings, if only to facilitate future clarification of their electrophysiological mechanisms or to study the results of new therapeutic interventions. The latter aspect is exemplified when contemplating electrical treatment (atrial overdrive or DC shock). In those relatively rare cases, however, in which an irregular supraventricular tachycardia shows characteristics of both atrial flutter and fibrillation, a forced diagnosis is not clinically relevant because of the similarity in etiology, hemodynamic response, and the present approach to pharmacological treatment. Such cases may justifiably be classified as *atrial flutter-fibrillation* (Fig. 3), which is a more appropriate term than *fibrillo-flutter.*•

REFERENCES

1. Robles de Medina EO *Amer Heart J 95:*796, 1978
2. Coumel P in *Cardiac Arrhythmias* (Krikler DM, Goodwin JF, eds.), WB Saunders, London, 1975, pp 111-143
3. Abildskov JA, Moe GK in *Mechanisms and Therapy of Cardiac Arrhythmias* (Dreifus LS, Likoff W, eds.), Grune & Stratton, New York, 1966, pp 135-143
4. Bootsma BK, Hoelen AJ, Strackee J, Meijler FL *Circulat 41:*783, 1970
5. Scherf D, Schott A *Extrasystoles and Allied Arrhythmias,* William Heinemann Medical Books Ltd, London, 1973, pp 400-418
6. Allessie MA: Mechanism of atrial flutter in the isolated canine heart. *Abstract Book VIIIth European Congress of Cardiology Paris,* 1980
7. Wellens HJJ *Electrical Stimulation of the Heart in the Study and Treatment of Tachycardias.* Stenfert Kroese, Leiden, 1971, pp 27-39
8. MacLean WAH, Karp RB, Kouchoukos NT et al *Circulat 54 (Suppl II):*II-167, 1976
9. Puech P, Grolleau R, Latour H et al *Arch Mal Coeur 66:*159, 1973
10. Waldo AL, MacLean WAH, Karp RB et al *Circulat 56:*737, 1977
11. Godtfredsen J: Atrial fibrillation. Etiology, course and prognosis. Thesis, Copenhagen, 1975, pp 45-47, 142-144, 180
12. Pick A *Circulat 15:*603, 1957
13. Delman AJ, Stein E *Circulat 29:*593, 1964
14. Marriott HJL, Sandler IA *Progr Cardiovasc Dis 9:*18, 1966
15. Wellens HJJ, Bär FWHM, Lie KI *Amer J Med 64:*27, 1978
16. Wellens HJJ in *The Conduction System of the Heart* (Wellens HJJ, Lie KI, Janse MJ, eds.), Stenfert Kroese, Leiden, 1976, pp 569-587
17. Sonnenblick EH, Ross J, Braunwald E *Amer J Card 22:*328, 1968
18. Friedberg CK *Diseases of the Heart.* WB Saunders, Philadelphia, 1966, pp 543-562
19. Bigger JT in *Heart Disease* (Braunwald E, ed.), WB Saunders, Philadelphia, 1980, pp 691-743
20. Askey JM *Amer J Card 9:*491, 1962
21. Freeman I, Wexler J *JAMA 184:*1007, 1963