The Clinical Features of Atrial Flutter and Their Therapeutic Implications*

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Analysis of 71 patients with atrial flutter delineated two categories of patients. One group (40 patients) had severe decompensated heart disease. Another group (31 patients) was free of structural heart disease or had disease not severe enough to result in congestive failure. Of the latter group 23 had an acute noncardiac illness. In 14 it was a disease of the bronchopulmonary system. Those patients with severe heart disease seldom reverted without electrical countershock during the first five days after hospital admission. The majority of the latter group reverted within five days without such therapy. Those observations are useful to the clinician who must select therapy for his patient with atrial flutter. Failure to appreciate this variability may lead to conflicting or misleading reports regarding the efficacy of newer forms of therapy.

The clinician faced with a patient with atrial flutter has been provided recently with a variety of therapeutic tools in addition to the traditional pharmacologic agents, digitalis and quinidine. He may choose external electrical countershock, rapid intra-atrial pacing, or a drug to produce β-adrenergic blockade. Each of these therapeutic modalities appears to offer certain advantages, but none has been viewed against the background of a large consecutive series of patients, which takes into account the clinical behavior of atrial flutter and its modification by the more traditional forms of therapy. In the absence of such comparisons and despite reports of successful restoration of sinus rhythm in up to 80 percent of patients by digitalis administration alone, some physicians have concluded that one or another form of intervention is to be preferred.

The clinical features of atrial flutter were examined on a number of occasions but infrequently in the past decade. Moreover, certain discrepancies exist in these reports. Certain of these discrepancies derive from differences in diagnostic criteria used to distinguish between atrial flutter and atrial tachycardia with A-V block and from the inclusion by some authors of coarse atrial fibrillation (“flutter-fibrillation,” “impure flutter”) in their analysis.

Accordingly, in order to provide a meaningful backdrop against which to examine the advantages and disadvantages of the various treatment modalities, we have reviewed the experience of one institution in a period during which a rather uniform approach to the treatment of this rhythm disturbance was used.

**METHOD**

The electrocardiograms of all patients coded “atrial flutter” from July 1, 1966 through June 30, 1970 were reviewed. Eighty-seven patients were found to meet the following criteria for atrial flutter: regular, continuous and uniform oscillations of the base line present in at least one lead in the presence of an atrial rate of 230 per minute or more. The clinical records of these patients were reviewed and abstracted.

During the study period, house officers at the Atlanta VA Hospital were taught to treat atrial flutter in the following manner: An initial evaluation was performed to determine the urgency of the situation and the status of antecedent drug therapy. A choice was then made between three alternative therapeutic interventions: (1) immediate application of electrical countershock in life-threatening situations; (2) withdrawal of digitalis therapy when digitalis toxicity was suspected; and (3) administration of glycoside. The drug was to be given in an amount and by a route designed to allow electrical reversion if a deteriorating clinical situation were detected after several hours of observation. If an improving clinical picture followed digitalis administration, it was to be continued along with other appropriate therapy. If reversion to normal rhythm did not ensue, electrical reversion was recommended after optimal hemodynamic improvement had been achieved.

**PATIENTS**

All patients, except one, were men. Their ages ranged from 34 to 83 years and averaged 61 years.
Sixty-nine (79 percent) were 50 years of age or more and 45 (52 percent) were 60 or more. The findings are in keeping with a population drawn from the Veterans Hospital and are consistent with the reports of previous investigators who found that men in middle or late life most commonly manifest this rhythm disturbance.

In nine patients atrial flutter appeared during the course of therapy of atrial fibrillation, their dominant rhythm disturbance. Three developed atrial flutter after receiving quinidine sulfate and in two it followed an attempt at electrical reversion of atrial fibrillation in patients on maintenance quinidine therapy. Atrial flutter appeared subsequent to digitalis administration alone in two men with chronic and two with paroxysmal atrial fibrillation.

Seven patients manifested recurrent paroxysmal atrial flutter intermixed with atrial tachycardia or atrial fibrillation or both. In three instances this occurred during acute myocardial infarction, and in two others the arrhythmias accompanied pulmonary emboli. The remaining two patients exhibited chronic, recurrent paroxysmal arrhythmias.

The remaining 71 patients had had at least one episode of what may be termed "pure atrial flutter." They form the focus for this report since each presented the clinician with an opportunity to select a treatment modality.

**OBSERVATIONS**

**Etiologic Considerations**

Fourteen patients had no clinical or radiographic evidence of cardiac enlargement, no cardiac failure, and no murmurs or gallop sounds. They were considered to be free of structural heart disease. Nine had severe pulmonary disease (Table 1). Minor electrocardiographic abnormalities in three of the five remaining patients were not considered diagnostic of structural heart disease. Left axis deviation was present in two, and a short PR interval (0.11 sec) was present in a third.

Fifty-seven patients were considered to have structural heart disease. Evidence of ischemic heart disease, cardiomegaly, or a diastolic murmur was documented in each. A clear clinical history of angina pectoris or of myocardial infarction was accepted as evidence of coronary atherosclerotic heart disease. When these findings were absent and no significant murmur or hypertension was present to account for cardiomegaly, the patient was given the diagnosis of cardiomyopathy. Table 2 depicts the etiologic breakdown of the group. Some patients exhibited more than one etiologic process but only the principal factor is tabulated.

Among the 57 patients with heart disease, evidence of decompenation was recorded in 40. Pulmonary congestion, the result of left ventricular failure, was the only manifestation recorded in 6, while 30 had signs of biventricular decompenation. Four men had right ventricular failure due to cor pulmonale. Thus, in 40 of 71 patients (56 percent) with pure atrial flutter, the rhythm disturbance accompanied advanced heart disease, but a sizeable minority of 31 exhibited no evidence of structural heart disease or heart disease insufficiently advanced to result in cardiac failure.

An acute noncardiac illness was present in 23 of these 31 patients (74 percent). Severe respiratory disease seemed to be the precipitating cause in 12 patients. Other forms of acute illness which appeared to be related to the atrial flutter were present.

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**Table 1—Atrial Flutter in Patients with Bronchopulmonary Disease, But No Structural Heart Disease**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advanced COLD</td>
<td>4</td>
</tr>
<tr>
<td>Advanced bronchogenic Ca</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>1</td>
</tr>
<tr>
<td>Severe acute pneumonia</td>
<td>2</td>
</tr>
</tbody>
</table>

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**Table 2—Atrial Flutter: Etiology of Heart Disease**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Patients, No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>No structural disease</td>
<td>5</td>
</tr>
<tr>
<td>Pulmonary disease only</td>
<td>9</td>
</tr>
<tr>
<td>Chronic coronary heart disease</td>
<td>18</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>3</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>14</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>12</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>6</td>
</tr>
<tr>
<td>Nonrheumatic valvular disease</td>
<td>1</td>
</tr>
<tr>
<td>Lymphosarcoma of heart</td>
<td>1</td>
</tr>
<tr>
<td>Myotonia dystrophica</td>
<td>1</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>71</strong></td>
</tr>
</tbody>
</table>

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**Table 3—Atrial Flutter: Associated Acute Noncardiac Illness**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>No Heart Disease</th>
<th>Compensated Heart Disease</th>
<th>Decompensated Heart Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchopulmonary disease</td>
<td>9</td>
<td>5*</td>
<td>7**</td>
</tr>
<tr>
<td>Acute alcoholism</td>
<td>1</td>
<td>3</td>
<td>2†</td>
</tr>
<tr>
<td>Advanced uremia</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Fractured femoral neck</td>
<td>1</td>
<td>1†</td>
<td>1‡</td>
</tr>
<tr>
<td>Dehydration/electrolyte abnormality</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Gram-negative sepsis</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>12</strong></td>
<td><strong>11</strong></td>
<td><strong>14</strong></td>
</tr>
</tbody>
</table>

*Two with cor pulmonale.  
**Four with cor pulmonale.  
†One with associated recent myocardial infarction.  
‡Associated acute myocardial infarction.
in 11 patients. Acute alcoholism accounted for four. Acute noncardiac illness was also present in 14 of the 40 patients (35 percent) who had findings of congestive heart failure. Table 3 details the acute illnesses which appeared to have been related to the onset of atrial flutter.

Arrhythmia History

Previous episodes of atrial arrhythmia were known in only six patients, all of whom had heart disease. Three had undergone electrical reversion of atrial fibrillation seven months to two years previously. Two had had episodes of flutter and one of them had both flutter and fibrillation during the year prior to admission.

In 68 of the 71 patients atrial flutter was almost certainly of recent onset. The rhythm disturbance was first documented on admission in 53 patients, while in 15 it began during hospitalization. Symptoms suggestive of the onset of the rhythm disturbance one week to one month prior to admission were obtained in five. The arrhythmia was documented prior to admission in the remaining three. Two exhibited a truly chronic course. The rhythm had been present in one, a man with myotonic dystrophy, almost continuously for 25 years. In the other, a 76-year-old man with angina, congestive failure and left bundle branch block, it had been persistent for five years and paroxysmal for ten prior. In the third patient the rhythm disturbance was recorded one month prior to admission, but treatment had not been instituted.

Electrocardiographic Features

The atrial rate prior to initiation of therapy ranged from 230 to 350 per minute. Atrial rates less than 250 per minute were unusual and tended to occur predominantly in association with advanced heart disease. No difference was found in the mean atrial rate of the 32 patients receiving digitalis prior to admission and those of the 39 who were not.

Of the 39 patients who were not receiving glycoside therapy at the time of their initial evaluation for atrial flutter 31 (80 percent) exhibited 2:1 A-V conduction. Of the remainder, two had 1:1 conduction and six had degrees of block greater than 2:1. One patient without evidence of heart disease and another with mild hypertenion and acne alcohol withdrawal exhibited 1:1 conduction. Their ventricular rates were 280 and 230 per minute, respectively. In only four of the six with block greater than 2:1 was it sufficiently advanced to result in ventricular rate less than 100 per minute. Two of these four had acute myocardial infarction, one had myotonic dystrophy, a condition known to produce abnormal A-V conduction, and the other may have been an example of the sick sinus node syndrome.

A-V block greater in degree than 2:1 was initially present in 11 of 32 (34 percent) of those receiving digitalis prior to their evaluation for flutter. One of these cases was judged to be the second example of sick sinus node syndrome. The ventricular response of the patient did not increase despite withdrawal of the glycoside.

Therapeutic Observations

Among the 71 patients, the clinical situation was judged to require immediate electrical reversion in nine instances. Digitalis toxicity was suspected in five others, and the drug was withdrawn. No therapeutic intervention was undertaken in six. The remaining 51 patients were managed initially with digitalization, with continued maintenance dosages of digitalis, or with increased amount of digitalis in the manner described.

No Specific Therapy

Six patients received no specific therapy directed toward atrial flutter because their arrhythmia was transient or because of the degree of A-V block which was present (Fig 1).

Digitalis Withdrawn

Digitalis was withdrawn in five patients (Fig 2). The presence of ectopic ventricular beats or of a high degree of A-V block necessitated this step in two. In three others no superimposed rhythm disturbances were present but excessive digitalis administration was suspected. In one the diagnosis was cardiomyopathy and in another it was severe pul-

![Figure 1. Course of atrial flutter in six patients receiving no specific therapy directed toward atrial flutter.](image1.png)

![Figure 2. Course of cardiac rhythm in five patients who entered on digitalis therapy and in whom drug was discontinued.](image2.png)
monary insufficiency, conditions thought to increase susceptibility to glycoside toxicity.

Subsequent events suggested that toxicity had existed in one of these five, a patient with cardiomyopathy. Ectopic ventricular beats appeared after countershock (5-watt seconds) (Fig 3) even though he had received no glycoside therapy for 72 hours.

**Digitalis Administration**

Fifty-one patients were initially treated with digitalis (Fig 4). Thirty subjects who had not been on glycoside therapy received “digitalizing” doses in the manner described (see "Methods"), while 12 with a history of maintenance digitalis therapy were treated with increments of digoxin in accordance with their estimated individual need. Nine others were continued on maintenance dosages.

Reversion to sinus rhythm occurred in 13 of 30 patients (43 percent) not previously receiving digitalis and in 9 of 21 (43 percent) of those who were. Fourteen of these 22 reversions took place in the first 24 hours and only 4 occurred subsequent to the fifth hospital day.

Digitalis administration was followed by con-

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**Figure 4.** Course of cardiac rhythm in 51 patients who initially were given digitalis therapy.

**Figure 3.** Countershock in patient with suspected digitalis toxicity. Low energy shock was followed by atrial fibrillation and by increased ventricular ectopic activity. Intravenously administered diphenylhydantoin therapy controlled latter and sinus rhythm resumed within 24 hours.
tined atrial flutter in 11 (36 percent) and by atrial fibrillation in 6 (20 percent) patients who had not previously received the drug. Slowing of the ventricular rate, a consequence of an increased degree of A-V block, was the rule. Of the 21 who had been receiving digitalis, 10 (48 percent) exhibited continued atrial flutter and two (10 percent) atrial fibrillation.

Four of the eight patients in whom digitalis administration produced atrial fibrillation subsequently were reverted to sinus rhythm. Countershock was used in two and normal rhythm appeared concomitant with the administration of 800 mg of quinidine sulfate per day in the other two. Two others died with atrial fibrillation and the remaining two were discharged with it.

Among the 21 patients in whom atrial flutter persisted, 4 were discharged in the rhythm, and 2 died with it. Sinus rhythm was restored in the remaining 15. This was achieved by means of countershock in 14 and occurred concomitant with the administration of 800 mg quinidine sulfate daily in one. One electrical reversion was carried out after conversion of atrial flutter to atrial fibrillation by intra-atrial pacing.

Reversion to normal sinus rhythm following digitalis administration was far more common in patients with little or no heart disease. Among 26 patients without evidence of failure treated in this manner, sinus rhythm was restored in 12 (46 percent) as it was in 3 of 4 patients (75 percent) with only pulmonary congestion. Only 7 of 32 (22 percent) with biventricular congestive failure reverted.

Viewed from a slightly different perspective an even more impressive separation is apparent. Fifty-four patients received neither quinidine nor electrical countershock during the first five days of their hospitalization. Twenty-four reversions took place. Thirteen followed increments of digitalis, five followed continued maintenance, two followed withdrawal of the glycoside, and four occurred spontaneously. Sixteen of 25 patients (64 percent) without heart failure, 4 of 5 (80 percent) with mild heart failure, and 4 of 24 (17 percent) with moderate or severe failure returned to sinus rhythm.

Electrical Reversion

Electrical reversion was attempted 28 times in 27 patients (Table 4). On nine occasions this was the initial therapy (Fig 5). In two it followed withdrawal of digitalis (Fig 2). In 16 patients (17 attempts) it followed therapy with digitalis (Fig 4). Four of these had converted to atrial fibrillation, one, as a result of intra-atrial pacing.

Standard techniques were used. A synchronized capacitor discharge was administered by means of anterior and posterior paddles following anesthesia or sedation.

Normal sinus rhythm resulted from 21 of the 23 reversion attempts for atrial flutter, in 3 of the 4 for atrial fibrillation. Once ventricular fibrillation occurring during cardiac catheterization of a patient with atrial flutter reverted. An energy level of 200-watt seconds was exceeded only once. In none of the three instances of failure of electrical reversion were energy levels greater than 150-watt seconds attempted.

Only one significant complication of the countershock was experienced. Undetected faulty synchronization resulted in a capacitor discharge of 100 watt seconds into a large T wave (Fig 6). The ensuing ventricular fibrillation was promptly terminated with an additional shock. No untoward effects were detected, and the patient is grouped with those who reverted successfully.

Quinidine Therapy

Eleven patients received quinidine. The sulfate salt, 800 mg daily in divided doses, was given prior to planned electrical reversion in eight patients, five with atrial flutter and three with atrial fibrillation following digitalis therapy. Sinus rhythm returned prior to countershock in one of the patients with atrial flutter and two of the three who had converted to fibrillation. In two other instances the drug was begun following recurrences of atrial flutter in patients who originally had reverted to sinus rhythm.

Table 4—Atrial Flutter and Fibrillation: Electrical Reversion

<table>
<thead>
<tr>
<th></th>
<th>Atrial Flutter</th>
<th>Atrial Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Attempts</td>
<td>Successes</td>
</tr>
<tr>
<td>Initial treatment</td>
<td>8*</td>
<td>8</td>
</tr>
<tr>
<td>During digitalization</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Following failure of digitalis therapy</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Following failure of digitalis therapy, withdrawal</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>21</td>
</tr>
</tbody>
</table>

*One additional successful attempt for ventricular fibrillation during diagnostic catheterization.
following digitalis administration. No more than 1.2 gm daily was administered. The ill-advised administration of 320 mg of quinidine gluconate intravenously to one undigitalized patient resulted in 1:1 A-V conduction requiring emergency countershock (Fig 7).

Subsequent Course

Sinus rhythm returned spontaneously or was restored by therapeutic intervention in 58 of the 71 patients (82 percent) (Table 5). Atrial flutter was noted to recur during hospitalization in six; however, the recurrence was transient in four. A total of 50 patients were discharged in normal sinus rhythm and 6 died with that rhythm during the hospitalization.

Eight patients continued to have atrial flutter. Three of that number died during the hospitalization. Seven patients, five with persistent and two with recurrent atrial flutter, were discharged with this rhythm. Two were known to have chronic flutter and two were suspected of having the sick sinus node syndrome since A-V block sufficient to produce ventricular rates of 40 to 90 per minute were observed even in the absence of digitalis administration. One additional patient refused electrical reversion and two had advanced cor pulmonale. All except the two suspected of having the sick sinus node syndrome were discharged with ventricular rates controlled with glycoside administration. Sinus rhythm had returned at an 18 month and a one-month followup in the two patients with cor pulmonale.

Atrial fibrillation appearing after digitalis administration persisted in five patients. Three died and two were discharged.

Although no systematic attempt was made to obtain longterm followup on these patients, recurrent atrial arrhythmias were documented in 11 of 51 patients discharged in sinus rhythm. In six the recurrent arrhythmia was atrial flutter and in the remainder, atrial fibrillation.

Discussion

The clinical features of the patients in this study are, in general, those reported by others\(^{11-22}\) who
have investigated atrial flutter. Two exceptions are noteworthy. Rheumatic heart disease was less frequent and bronchopulmonary disease rather more common than in earlier reports. The highly selected nature of a Veterans Hospital population probably accounts for these variations. The frequency with which bronchopulmonary disease or pulmonary embolism is associated with this rhythm disturbance was observed by several authors.

The observations presented in this communication underscore the variability of the clinical setting in which atrial flutter appears and reinforce the concept that one must tailor therapy for the individual patient at hand. Certain very practical considerations often dictate the selection of a therapeutic plan. The lack of appropriate equipment or trained personnel may prevent utilization of external countershock or of intra-atrial pacing. On the other hand, a clinical situation characterized by advanced decomposition of the oxygen transport system may dictate their immediate application. The need to obviate an extended hospitalization may also dictate a similar approach. This study emphasizes the need to take into account the variable behavior to be expected from this rhythm disturbance, depending on the setting in which it appears.

In broad terms atrial flutter was observed in two clinical settings. More than half the patients manifested decompensated heart disease; all but a few had moderate or severe biventricular failure. A substantial minority of the series had no manifestations of heart disease or heart disease of only modest degree. In nearly all such instances an acute illness, most commonly bronchopulmonary disease or acute alcoholism, seemed to have precipitated the rhythm disturbance.

Previous authors have distinguished between “transient” and “established” atrial flutter based on the duration of the rhythm disturbance. Hejtmancik observed that transient flutter was frequently associated with a precipitating event. Clearly, a division into groups related to the duration of the arrhythmia is of little value to the clinician who must elect therapy based on the presentation.

Our observations suggest that atrial flutter tends to be “transient” in patients with no more than mild heart disease and “established” when advanced disease is present. Fifty-four patients received no therapeutic intervention, digitalis administration, or digitalis withdrawal during the first five days of hospitalization. Sixteen of 25 (64 percent) without heart failure and 4 of 5 (80 percent) with mild failure resumed sinus rhythm during this period. This may be compared to a return of sinus rhythm in only 4 of the 24 (17 percent) with moderate or severe failure. We concluded that the more advanced the existing heart disease, the less apt is the administration of digitalis and supportive measures to result in sinus rhythm during a relatively brief period of hospitalization. Slow resolution of the acute precipitating process, eg, severe pneumonia, seemed to account for the delay in reversion in some of the more favorable group.

The tendency of patients with little or no heart disease to revert within a few days either spontaneously or with modest amounts of digitalis must be taken into account in evaluating new therapeutic modalities. The reported high rate of successful reversion of atrial flutter with propranolol therapy may be inflated because the drug can only be administered to patients with relatively good myocardial function and those free of bronchospastic disorders. Differences in patient selection may account for the controversy which presently exists regarding the effectiveness of rapid atrial pacing in this arrhythmia.

Digitalis administration to any patients with atrial flutter has been criticized on the grounds that its use tends to delay electrical reversion or force its execution under more hazardous circumstances. In the experience herein described, only three patients were felt to be in urgent need of electrical reversion after an initial decision to administer digitalis. Electrical reversion was safely carried out in all three.

Clearly, atrial flutter can be safely managed in the manner described, but we do not suggest that this is necessarily the best way to manage this problem. We wish only to emphasize other factors which may enter in the selection of a plan of therapy and to suggest that an arbitrary choice of an ideal form of therapy is inappropriate.

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120 LINDSAY, HURST

CHEST, 66: 2, AUGUST, 1974
Franz Liszt—Prodigious Performer and Composer

When Liszt (1811-1886) was only eleven, his first performance created the excitement that only the playing of a fabulously gifted prodigy can do. At his second performance, few days later, after the concert, Beethoven went up to the boy and embraced him. Liszt never forgot this. Beethoven became and remained his god, one that he was to serve well in later years, after Beethoven died and his music still was not accepted as it is today. Liszt was only twelve when he adopted the new piano forte. Through Paganini's influence and example Liszt set himself the goal of becoming a great pianist of his time. It was Liszt who introduced Chopin to the famous woman novelist who called herself George Sand, thereby precipitating another famous romance. Liszt gave up his virtuoso career at the age of thirty-six. He left behind him many changes in piano technique. He also was one of the firsts to turn the piano sideways: he had discovered the music sounded better that way. Liszt composed in enormous quantities. In 1879, he took religious orders, joining the tertiary order of St. Francis in Rome, becoming Abbé Liszt.

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