Studies in Human Atrial Flutter with the Use of Proximity Electrodes

By YEHEZKIEL KISHON, M.D., AND RALPH E. SMITH, M.D.

SUMMARY

Ten patients with the electrocardiographic diagnosis of atrial flutter were investigated by exploration with electrodes within the esophagus and the right atrium. The records obtained were studied in regard to the timing of the intrinsic deflection and the contour of the atrial waves, as recorded at various sites.

In four patients, atrial activation progressed in sequence cephalad in the left atrial wall and caudal in the right atrium. This process continued through two thirds of the total atrial cycle, favoring “circus movement” as the underlying mechanism. In six other patients, activation was believed to originate low in the left atrium, with simultaneous spread of excitation of both atrial walls in a general cephalad direction and terminating within the first half (usually the first third) of the atrial cycle. This pattern is compatible with, but not conclusive of, an ectopic focal mechanism. Serial records in one patient are presented in which transformation of one type into a second type of atrial flutter is suggested. It is likely that at least two mechanisms are present in human atrial flutter.

Additional Indexing Words:
Circus-movement hypothesis
Ectopic unifocal impulse formation
Right intra-atrial electrocardiogram
Esophageal electrocardiogram
Right intra-atrial esophageal potential-difference curve

ALTHOUGH SEVERAL hypotheses have been proposed for the underlying mechanism in human atrial flutter, they can be grouped broadly into three basic models: (1) the continuous circulating movement of the excitation wave:1-8 (2) the unifocal or multifocal impulse formation4, 5; and (3) the theory of the multiple re-entry.6-8 In spite of extensive studies and the vast amount of information accumulated during the past half century, the gaps between the proponents of each hypothesis are far from being bridged.

This study is an attempt to elucidate the excitation pattern in atrial flutter with the use of electrodes placed within the esophagus and the right atrium.

Excitation in human atrial flutter was first investigated by means of vectorial analysis.9, 10 Using standard limb leads, Sir Thomas Lewis and associates9 in 1921 suggested that circus movement is present around the two cavae, probably directed upward in the left atrial wall and downward in the taenia terminalis of the right atrium. Later, this mechanism was experimentally demonstrated in animals by extensive crushing of tissue between the vena cavae to form an obstacle to the wave progression. With this procedure, circus movement of the wave of excitation could be maintained.5, 6, 11-13 In some studies, interfering with the integrity of the circular pathway stopped the arrhythmia,5, 13, 14 but this technique could not be repeated by others.7, 15, 16 Arrhythmias simulating atrial flutter were produced in animals by the application of such drugs as aconitine5, 12-14, 17, 18 and delphine.19, 20 Circus movement as the underlying mechanism in most of these arrhythmias, was considered unlikely; the relevance of those

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studies to human atrial flutter is questionable. Vectorial analysis in human atrial flutter yielded conflicting results, although the majority of the work showed closed vectorial loops of the atrial waves with counterclockwise rotation in the frontal plane. The introduction of esophageal and later right intra-atrial electrocardiographic electrodes, placing the exploring tool in close proximity to the atrial musculature, permitted a more accurate detection of the process of atrial excitation. Esophageal electrocardiograms in atrial flutter confirmed the caudocephalic direction of the wave in the left atrial wall in most cases, although opposite direction of the wave progression was rarely observed. The morphologic features of atrial flutter in esophageal leads seemed to be almost specific, with the absence of an isoelectric interatrial segment at one or more levels of recording. Electric events in the right atrial wall were detected initially by means of anterior chest leads and later by the use of right intra-atrial electrodes. The relative timing of the intrinsic deflection recorded by proximity leads was used to measure the arrival of excitation at the site of recording. In most studies, atrial activation seemed to occupy a large portion of the atrial cycle, shorter durations of atrial excitation were found less often and were considered to represent atrial tachycardia. Direct exploration of the electric activity in the atrial wall during atrial flutter has been reported only twice to our knowledge. In the first case, the flutter happened to be of the impure type; in both cases, the results were inconclusive.

**Methods**

The study group consisted of 10 patients considered to have the generally accepted electrocardiographic criteria for atrial flutter as described initially by Lewis, who emphasized ceaseless and uniform atrial activity of about 250 to 350 times per minute. General data as well as the electrocardiographic features of the 10 patients are presented in table 1. There were eight men and two women, with an average age of 65.1 years. Five of the patients were receiving maintenance doses of digitalis, whereas the other five did not receive drugs prior to the investigation. Five of the patients did not have heart disease, except for the arrhythmia, but two patients had atrial septal defects. The remaining three patients were considered clinically to have coronary heart disease.

### Table 1

*General Data and Electrocardiographic Features of 10 Patients With Atrial Flutter*

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex and age (yr)</th>
<th>Duration of flutter</th>
<th>Atrial rate (beats/min)</th>
<th>Degree of A-V block</th>
<th>Etiologic heart disease</th>
<th>Drugs prior to investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 61</td>
<td>Several months</td>
<td>250</td>
<td>2:1</td>
<td>Unknown</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>M, 46</td>
<td>Unknown</td>
<td>240</td>
<td>2:1-4:1</td>
<td>Atrial septal defect</td>
<td>Digitalis</td>
</tr>
<tr>
<td>3</td>
<td>M, 81</td>
<td>6 yr</td>
<td>280</td>
<td>2:1-4:1</td>
<td>Atherosclerosis</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>M, 78</td>
<td>Several months</td>
<td>325</td>
<td>3:1-2:1</td>
<td>Unknown</td>
<td>Digitalis</td>
</tr>
<tr>
<td>5</td>
<td>M, 72</td>
<td>Unknown</td>
<td>255</td>
<td>3:1-4:1</td>
<td>Atherosclerotic heart disease</td>
<td>Digitalis</td>
</tr>
<tr>
<td>6</td>
<td>M, 75</td>
<td>5 wk</td>
<td>315</td>
<td>Complete</td>
<td>Atherosclerotic heart disease</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>F, 58</td>
<td>1 yr</td>
<td>300</td>
<td>3:1-6:1</td>
<td>Unknown</td>
<td>Digitalis</td>
</tr>
<tr>
<td>8</td>
<td>M, 58</td>
<td>6 mo</td>
<td>285</td>
<td>2:1</td>
<td>Unknown</td>
<td>Digitalis</td>
</tr>
<tr>
<td>9</td>
<td>F, 57</td>
<td>4 mo</td>
<td>240</td>
<td>2:1</td>
<td>Postclosure of atrial septal defect</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>M, 66</td>
<td>1 yr</td>
<td>260</td>
<td>3:1</td>
<td>Unknown</td>
<td>None</td>
</tr>
</tbody>
</table>
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disease. None of the patients had evidence of congestive heart failure.

For obtaining intra-atrial recording, an 80-cm length of fine polyethylene tubing (PE60) containing an electrode wire was introduced, under sterile conditions, into an antecubital vein, and was advanced under continuous electrocardiographic monitoring into the right ventricle. Details of this procedure have been described previously.\(^3\) Isolation amplifiers with high impedance were used in the recording system to minimize electric hazards.\(^3\) Immediately subsequent to obtaining right ventricular complexes, the intracardiac electrode was withdrawn through the right atrium back into the superior vena cava. Simultaneous recordings of esophageal leads and unipolar chest (V\(_1\)) leads were obtained. Then, with the endocardial electrode advanced to the right atrium, simultaneous esophageal (at various levels), right intra-atrial and unipolar chest (V\(_1\)) tracings were recorded. Finally, a potential-difference curve between the esophageal and the right intra-atrial electrodes was obtained by methods previously described.

Two different aspects of the records were considered in detail: First, the contour of the atrial waves both in the esophageal and intra-atrial tracings was studied in order to estimate the general direction of the spread of atrial excitation. Whenever a major positive deflection was recorded at high level and a major negative deflection at low level, the wave front of excitation was assumed to progress superiorly. Whenever a major positive deflection was recorded at low level and a major negative deflection at high level, the wave front of excitation was assumed to progress inferiorly.\(^2\), \(^3\), \(^4\) Second, the relative timing of the intrinsic deflection was used to map the time sequence of the wave-front arrival at the site of recording. In all cases, the lower part of the left atrium was chosen arbitrarily as the zero point.

Results

Different patterns of electric behavior of the atria were observed from the analysis of our 10 patients; these patterns might be best illustrated by presenting the results in two patients (cases 1 and 3).

Case 1

The conventional electrocardiographic findings showed typical waves of atrial flutter, predominantly negative in leads II, III, and aVF (fig. 1). Simultaneously recorded right intra-atrial and esophageal tracings, as described previously, showed that the excitatory wave was progressing superiorly in the left atrial wall (as disclosed by the esophageal tracing obtained at various levels), and inferiorly in the right atrial wall (fig. 2). The

Figure 1

Case 1. Atrial flutter with 2:1 atrioventricular conduction. Atrial rate, 255/min; ventricular rate, 127/min.
timing of the appearance of the intrinsic deflections at the various sites is depicted in figure 3. It is shown that the sequence of the intrinsic deflection is directed superiorly in the left atrium and proceeds inferiorly in the right atrium, occupying altogether about 67% of the entire atrial cycle. Simultaneous recording of right intra-atrial (RA) and esophageal (E49) electrodes (fig. 4) at a chart speed of 50 mm per second showed clearly a difference of about 100 msec between onsets of the intrinsic deflections at the two sites. Potential-difference curves (fig. 4, bottom) between the right atrial and the esophageal sites verified that this interval was physiologic, by excluding technical factors. In these curves, two separate intrinsic deflections appeared during each cycle, representing superimposition on one tracing of the intrinsic deflections present in the esophageal (fE) and right intra-atrial (fRA) recordings.

Case 3

Tracings were obtained from this patient and analyzed as previously described (fig. 5). It seemed justifiable to assume from the wave configurations and the timing of the intrinsic deflections that the process of activation originated low in the left atrium, progressing caudoccephalad in both atrial walls, and terminating within the first two fifths of the atrial cycle. During the latter portion of the atrial cycle, no electric activity to indicate wave arrival could be demonstrated at any site of recording. A right atrial-esophageal potential-difference curve (fig. 6, bottom) revealed only a single intrinsic deflection; it was followed by a small incisura, indicating that the arrival of excitation in both sites (left and right atrial wall) was almost simultaneous.

All 10 patients were subjected to the same investigation as the patients in the illustrative cases were. The data were analyzed and are summarized schematically in figure 7. The timing of intrinsic deflection was accurately determined at the various levels within both atria. In addition, the general direction of the atrial wave was estimated (fig. 7, arrows within the atria), based on the configuration of the wave, as mentioned earlier. Two different patterns of the sequence of excitation within the atria could be distinguished as...
Case 1. Schematic illustration of the assumed sequence of atrial excitation. Esophageal and right intra-atrial tracings were obtained in order to determine precisely the timing of the intrinsic deflections at various sites. Numbers in this figure correspond to the time (in seconds) of the appearance of intrinsic deflections. Left atrial base has been arbitrarily chosen as zero point. A counterclockwise sequence of excitation is clearly obvious, compatible with the circus-movement hypothesis. Abbreviations: RA and LA = right and left atria, respectively; VCS and VCI = superior and inferior venae cavae, respectively; LV and RV = left and right ventricles, respectively; LPV and RPV = left and right pulmonary veins, respectively.

previously illustrated by cases 1 and 3 (fig. 8). Accordingly, the patients could be separated into two categories.

Group A (Patients 1, 2, 5, and 10)

In the patients of this group, the sequence of excitation, as indicated by the wave form and timing of the intrinsic deflection, progressed caudocephalad in the left atrium, and cephalocaudad in the right atrium. The inscription of positive atrial wave in V₁ (fig. 8, top) probably reflected an anterolateral progression of the wave within the atrial roof after the completion of left atrial activation and prior to right atrial activation. Potential-difference curves, between the proximity electrodes, characteristically showed two separate and distinct intrinsic deflections in timing far apart for each atrial cycle (fig. 4, bottom). Two intrinsic deflections were occasionally seen also in unipolar right atrial tracings (fig. 3, recording from middle of right atrium, and fig. 9). By time-correlation, we considered only one (f₁) to be the intrinsic deflection resulting from the arrival of excitation at the right atrium, while the second (f₂) represented movement of excitation within the left atrial wall and was better shown in the esophageal tracing.
Figure 4
Case 1. Simultaneous recording with esophageal electrode 40 cm from nares (E₁₀), right atrial electrode (RA), and precordial lead (V₁). (Bottom) Potential-difference curve (RA-E₁₀) between the above esophageal and intra-atrial electrodes. Superimposition of right atrial (f_RA) and left atrial (f_E) waves, with their respective intrinsic deflections, is apparent. The two deflections are separated by an interval of about one half of an atrial cycle.

Group B (Patients 2-4, 6-8, and 9)
Activation of the left atrium was similar to that found in group A, with caudocephalad sequence. The activation of the right atrium, however, started and terminated much earlier in the atrial cycle, part of it occurring simultaneously with left atrial activation, and the general direction was the same as in the left atrial wall (illustrated by case 3 in figure 8). Excitation in any site of the right atrium was detected within 20 msec after the latest activation of the left atrium; it occurred always within the first half of the atrial cycle, and usually within the first third of the atrial cycle. Only case 3 (figs. 5 through 8) showed distinct caudocephalad orientation of the excitation sequence in the right atrium; in the other cases, it seemed that activation of the right atrial wall was accomplished by lateralward progression (fig. 7).

Discussion
The purpose of the present investigation was to determine the pathway and the mechanism of atrial excitation in patients with clinically typical atrial flutter. Occasionally, the general direction of the atrial excitation could be estimated by the morphologic features of the “f” waves, recorded by esophageal leads, and infrequently by right intra-atrial leads. Far more informative was the timing of the atrial intrinsic deflection, as recorded at the various sites by the proximity leads, which enabled us to map the approximate sequence of excitation in the atrial wall. Our basic assumption that the intrinsic deflection coincides with the arrival of excitation of the wave under the exploring electrode has been repeatedly challenged in the past, but is supported by recent experimental work with esophageal and intracardiac electrodes. The location along the vertical axis behind the left atrium of the esophageal electrode tip could be estimated during the recording. A similar determination of the position of the right intra-atrial electrode was not feasible without the use of chest fluoroscopy or roentgenograms, as done by others. Instead, continuous recording was performed while the electrode tip was withdrawn at a constant speed from the right ventricular cavity into the superior vena cava. This sequence was repeated in all cases to ensure reproducibility. We assumed that the withdrawal course from the tricuspid valve was linear, with a superior and slightly rightward course in the frontal plane (fig. 3). This assumption was based on anatomic considerations and fluoroscopic observations during cardiac catheterization. It is true that the electrode tip may bear variable spatial relationship within the right atrial cavity. This seems to be of secondary importance as long as the relative sequence of the excitation of the two atria is being considered.
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Whether the tip is recording potentials from the anterior, posterior, or right wall of the right atrium, adequate information about the excitation sequence along the cephalocaudal axis may be obtained.

In four of our patients (group A), observations supported circus movement as the underlying mechanism. Intrinsic deflections appeared at various atrial sites throughout almost two thirds of the atrial cycle. This finding is in agreement with others. Exploration of the excitatory pathway is incomplete with this technic; those regions near the conjunction of the atria are not explored. The direction of the wave of atrial activation was counterclockwise in the frontal plane in all four patients, which was compatible with similar observations in the "common" type of atrial flutter. An easy understanding of the wave form recorded at any point near a circus movement is provided by the simple model presented in figure 10. The principles of the "solid angle" theory, by which electric potential induced by depolarization and repolarization of electrically charged membranes can be calculated, have been simplified and reduced from their spatial geometry into two dimensions. Using this model, hypothetical atrial waves have been constructed for proximity (esophageal and right intra-atrial) and remote (aV_R) leads (fig. 10), which bear surprising similarity to those recorded in our cases. The hypothetically constructed potential-difference curves between the esophagus and the right atrium demonstrate two separate intrinsic deflections for each atrial cycle. This phenomenon was seen when the esophageal electrode was placed behind the midleft atrium (about

Figure 5
Case 3. Atrial flutter with atrial rate of 250/min and ventricular rate of 40 to 100/min. The time interval between the intrinsic deflections as recorded simultaneously at various sites by esophageal and right intra-atrial electrodes never exceeded 0.09 sec, and the general direction of the excitation sequence was caudocephalad in both atria.
Case 3. Six standard electrocardiographic leads provide accepted criteria for classic atrial flutter. (Bottom) Esophageal and right atrial potential-difference curve. Only one main intrinsic deflection is seen in each atrial cycle, which is shortly followed by small notching. This is probably due to the fact that waves of excitation arrive almost simultaneously at both atria. This pattern is compatible with, but not conclusive of, unifocal activation.

37 cm from the anterior nasal nares) and the endocardial electrode was moved from the right ventricle into the superior vena cava. Prinzmetal and associates\(^4\) required the presence of two intrinsic deflections in an esophageal tracing as evidence for circus movement and considered their absence as an argument against this theory. We could not detect this M-shaped atrial wave in any of the esophageal tracings of group A, but it was seen in all potential-difference curves between esophageal and right atrial positions and also in intratrial tracings, as shown in figure 3 (recorded from midright atrium) and figure 9. In those tracings, each deflection of the pair corresponded to separate activation of each of the two atria.

If circus movement truly exists and if the exploring electrode is incidentally placed symmetrically in regard to the excitation pathway, one may expect a pattern like that hypothetically drawn in figure 10, left. Indeed, a similar pattern has been seen by one of us (Y.K.) and has been previously published (fig. 11, bottom). This arrhythmia started with clearly defined intrinsic deflections (fig. 11, top) and was transformed during the intravenous administration of lidocaine into the peculiar sinusoidal-like pattern. A minimal but important change in rate accompanied this change of configuration. This seemed to exclude positional change as the cause for the change in wave configuration.

Whereas circus movement seems the most likely mechanism in the first group, the interpretation of the results in the second group...
is more difficult. Activation of the left atrial wall appears to be oriented superiorly, as in group A, lasting for a shorter duration. In this group, as indeed in the first group, pathways of atrial excitation are not completely revealed by these technics. However, it has been shown that whereas no two sites in the two atria, respectively, were activated simultaneously in group A, there was considerable overlap of time between the activation of both atria in the second group, best illustrated by case 3 in figure 8. These facts, although inconclusive, support the presence of a single discharging focus, low in the left atrial wall with impulses radially propagated to both atria. Similar conclusions were reached by Mirowski and Alkan, using vectorial analysis. However, this pattern could still result from an impulse re-entering a small circular pathway located low in the left atrium. Activation could then spread to both atria in a radial fashion. It is questionable whether group B is different from the well-defined clinical entity of atrial tachycardia originating from a low atrial focus. The undulatory pattern of the base line in this group probably results from alternate depolarization and repolarization in contrast to the undulatory pattern in group A, which is most likely caused by continuing waves of depolarization. Transformation of one type into another has been demonstrated experimentally and has been suggested as a possible spontaneous event in the human atrium. This subject has been reviewed recently, and the case presented in

Figure 7

Schematic illustration of the assumed sequence of atrial excitation in 10 patients with atrial flutter. Numbers within the heart silhouette represent the timing of the intrinsic deflections (see figure 3 and text) as obtained by simultaneous recordings with esophageal and right intra-atrial electrodes. The duration of atrial cycle (f-f) is given at the top of each case. Cases 1, 2, 5, and 10 show patterns compatible with the circus-movement hypothesis. The rest may present a unifocal pattern of excitation. Solid arrows represent atrial wave direction, derived from the configuration of the wave, regardless of the sequence of the intrinsic deflection. Open arrows indicate that direction could not be determined with certainty by analysis of wave forms; direction was assumed by the sequence of the intrinsic deflections.
Figure 8

Two different excitation sequence patterns were observed in our 10 patients with atrial flutter. They are represented in the diagram by cases 1 and 3, where the relationship between the course of the "f" waves (recorded in V1) and the arrival of the wave of excitation at various sites of the right atrium (RA) and left atrium (E) is given. Atrial activity in case 1 continues during two thirds of the whole atrial cycle, caudocephalad in left atrial wall and cephalocaudad in the right atrium. In case 3, activation of the atria occurs during a brief interval and progresses simultaneously in both atria, with general cephalad sequence.

References

Case 2. Atrial flutter, with varying degrees of atrioventricular block. Atrial rate of 235/min and ventricular rate of about 60/min. (Top) Simultaneous recording with precordial (V₁) and right intra-atrial (RA) electrodes. While atrial waves in lead V₁ seem to be of short duration with prolonged iso-electric segment intervening, the atrial activity (as recorded in the right atrium) is prolonged and shows two distinct deflections for each atrial cycle. The first deflection (fₑ) coincides with the atrial wave in lead V₁ and probably represents left atrial activity, whereas the second deflection (fᵦ) occurs during the silent period of the precordial recording, probably representing right atrial activity. (Bottom) Standard bipolar electrocardiogram of same patient.

12. BROWN, B. B., AND ACHESON, G. H.: Aconitine-induced auricular arrhythmias and their rela-
A hypothetical model for easy prediction of the morphologic features of waves resulting from circus movement. Elliptic (or circular) pathway of the excitatory wave may result in different wave forms, depending on the site of recording. Curves were constructed by geometric synthesis, the instantaneous amplitude being directly related to the angle (tθ) magnitude at which the wave front is seen at the point of recording. A typical saw-tooth appearance was obtained at a remote point (aVR). Both esophageal and intra-atrial electrodes, as well as potential-difference curves between those electrodes, show striking similarity to the curves obtained in case 1. In the rare situations in which the pathway is circular or the electrode is placed symmetrically in regard to an elliptic pathway, the constructed curve (left) resembles the flutter waves seen in figure 11, bottom.


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