Ventricular Beats Induce Variations in Cycle Length of Rapid (Type II) Atrial Flutter in Humans
Evidence of Leading Circle Reentry
Flavia Ravelli, PhD; Marcello Disertori, MD; Fulvio Cozzi, MD; Renzo Antolini, PhD; Maurits A. Allessie, MD, PhD

Background Slight variation in cycle lengths of common and rapid atrial flutter in humans is an established phenomenon, but its mechanisms have not been completely clarified. In a previous study, we demonstrated that in common atrial flutter the variations in atrial cycle length were due to atrial stretch affecting the revolution time of a reentrant circuit. In the present study, we investigate the nature of atrial cycle length variations in the rapid type of human atrial flutter.

Methods and Results Atrial interval variations of 17 episodes of rapid atrial flutter in 14 patients were investigated by measuring the sequence of atrial intervals from intraesophageal or intra-atrial leads and the onset of QRS complexes from a surface lead (V1). To study whether interval variation in flutter cycle was related to ventricular activity, a phase plot was constructed in which the flutter cycle length was plotted against the time after the previous QRS complex. This showed that the interval fluctuations were strictly coupled to the moment of ventricular activation. After the onset of the QRS complex, the rapid atrial flutter interval gradually decreased by an average of 4.1% (P<.001) and reached a minimum value after 300 to 600 milliseconds. Thereafter, the intervals increased again until the next ventricular beat occurred. In 10 patients developing both common and rapid atrial flutter, two different phase relations were found. Whereas during common atrial flutter the atrial interval increased after the QRS complex, it decreased during rapid atrial flutter. In three patients, intra-atrial pressure was recorded together with the electrical activity during both common and rapid atrial flutter episodes. This showed that variations in atrial flutter cycle length were associated with the rise of atrial pressure during ventricular contraction.

Conclusions These findings indicate a role of contraction-excitation feedback caused by atrial stretch after a ventricular activation. The shortening of the atrial interval after the onset of the QRS complex as found in patients during rapid atrial flutter can be explained by stretch-induced shortening of atrial refractoriness and consequent shortening of the revolution time of a functionally determined intra-atrial circuit. (Circulation. 1994;89:2107-2116.)

Key Words • reentry • arrhythmia • contraction • excitation

Although atrial flutter is considered to be a highly regular rhythm, small variations in cycle length of atrial flutter are observed in relation to the QRS complex. In a previous study, we investigated the mechanisms of these interval fluctuations in common atrial flutter. We found that the variations in atrial flutter cycle length were related to the ventricular activity. The atrial flutter interval increased and reached a maximum value 400 to 500 milliseconds after the onset of the QRS complex. The hypothesis was formulated that these periodic variations in atrial flutter intervals after a ventricular contraction were caused by the influence of stretch of the atrial myocardium on the conduction properties of a circulating impulse in the atrium.

Atrial flutter is not uniquely defined. The Puech classification distinguishes (1) common atrial flutter characterized by biphasic F waves in surface leads D2, D1, and aVF, with a rate ranging from 250 to 320 beats per minute, (2) uncommon atrial flutter characterized by positive F waves in surface leads D2, D1, and aVF and by the same rate as common atrial flutter, and (3) impure flutter or rapid atrial flutter with a rate higher than 320 beats per minute identified as a transitional pattern between pure atrial flutter and atrial fibrillation. Wells et al proposed another classification by dividing atrial flutter into two types: a slower one (type I) and a faster one (type II). The main difference between the two is that rapid atrial pacing from the high right atrium always influenced type I atrial flutter, whereas it did not influence type II atrial flutter. Variations in the cycle length of the rapid type of atrial flutter have been documented by these authors. In the present study, we investigated the mechanisms of fluctuation in cycle length of rapid atrial flutter (type II) in humans.

Methods

Patient Population

We studied 17 episodes of rapid atrial flutter, 5 spontaneous and 12 induced by rapid pacing, in 14 patients, 11 men and 3 women. The mean age was 66±10.2 years. Their clinical data are summarized in Table 1. Associated diseases were coronary
TABLE 1. Clinical Features

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Heart Disease</th>
<th>Type of Flutter</th>
<th>AP</th>
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<tbody>
<tr>
<td>1</td>
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<td>M</td>
<td>CCP</td>
<td>rAF</td>
<td></td>
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<tr>
<td>2</td>
<td>67</td>
<td>M</td>
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<td>↓ cAF</td>
<td></td>
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<tr>
<td>3</td>
<td>79</td>
<td>M</td>
<td>CAD</td>
<td>rAF</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>64</td>
<td>M</td>
<td>MVD</td>
<td>↓ cAF</td>
<td></td>
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<td>65</td>
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</tr>
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<td>60</td>
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<td>rAF</td>
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<tr>
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<td>79</td>
<td>M</td>
<td>CCP</td>
<td>↓ cAF</td>
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<tr>
<td>13</td>
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<td>CM</td>
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<td></td>
</tr>
<tr>
<td>14</td>
<td>79</td>
<td>F</td>
<td>MVD</td>
<td>↓ cAF</td>
<td>Yes</td>
</tr>
</tbody>
</table>

AP indicates atrial pressure recording; CCP, chronic cor pulmonale; CAD, coronary artery disease; MVD, mitral valve disease; CM, cardiomyopathy; HHD, hypertensive heart disease; ND, no detectable heart disease; rAF, rapid atrial flutter; cAF, common atrial flutter; ↓, conversion by rapid pacing; and r, spontaneous conversion.

Electrophysiological Study

After light sedation with diazepam 10 mg IM, a bipolar catheter was advanced into the esophagus for atrial recording and stimulation. The catheter position was chosen to attain a sharp atrial deflection. In five patients, intracardiac atrial recording and stimulation was performed. In this case, the catheter was positioned at the lateral wall of the high right atrium. In two of these patients, the endocardial recording was performed simultaneously with the esophageal recording. At the beginning of the study, 4 patients showed spontaneous rapid atrial flutter, whereas the other 10 patients showed common atrial flutter. After 10 minutes of recording, rapid stimulation was performed to attempt restoration of sinus rhythm. In one case (patient 9) of spontaneous rapid atrial flutter, pacing induced a second episode of rapid atrial flutter characterized by a higher rate and by a different morphology of the atrial electrograms. In 9 of 10 cases of common atrial flutter, atrial pacing induced an episode of rapid atrial flutter. In two of these nine cases, multiple changes from one type of flutter to the other were recorded; in patient 4, the induced rapid atrial flutter converted spontaneously back to common atrial flutter, whereas in patient 11, pacing of the previously induced rapid atrial flutter episode induced a second episode of rapid atrial flutter with a higher rate and different F-wave morphology. In one case (patient 6), pacing of common atrial flutter restored sinus rhythm that lasted only a few minutes until an episode of rapid atrial flutter started spontaneously (see Table 1).

In three patients (patients 12, 13, and 14), intra-atrial pressure was recorded together with the electrical activity. Atrial pressure recordings were obtained by positioning a Swan-Ganz catheter (8F) in the mid right atrium. The atrial electrograms, the body surface ECG (V1) (bandwidth, 30 to 500 Hz), and the atrial pressure signal were recorded on FM magnetic tape (TEAC XR-510).

Data Measurement and Analysis

The signals were digitized at 1 kHz on a personal computer for interval measurements and analysis. A computer program was developed to identify atrial electrogram complexes and measure beat-to-beat atrial (AA) cycle lengths from the esophageal lead and to identify ventricular waves and measure the onset of the QRS complex from the surface ECG (V1).

Atrial interval variability was measured by calculating the mean and standard deviation of the atrial cycle length. A more detailed analysis of the variation of atrial intervals was performed by plotting the sequence of the atrial intervals and by marking those intervals in which a ventricular beat occurred. To study the time relation between flutter intervals and ventricular activity, a phase plot was constructed in which the atrial interval was plotted against the time after the previous QRS complex.

A Student's t test for paired data compared the atrial interval at the QRS complex and at the maximum change during rapid atrial flutter. A value of P < .05 was taken to represent statistical significance.

Results

Mean values and standard deviation of atrial intervals of 11 episodes of common atrial flutter and 17 episodes of rapid atrial flutter are presented in Table 2. Mean atrial intervals of rapid atrial flutter ranged from 150 to 197 milliseconds, with an average of 174.8 milliseconds. The average interval of the eight episodes of common atrial flutter was 227.8 milliseconds. All patients showed variations in rapid atrial flutter interval, with a standard deviation ranging from 1.7 to 7.9 milliseconds (mean, 4.3 milliseconds). The mean variation in cycle length of the 11 episodes of common atrial flutter was 4.8 milli-
TABLE 2. Atrial Interval Variations

<table>
<thead>
<tr>
<th>Patient</th>
<th>Common Atrial Flutter</th>
<th>Rapid Atrial Flutter</th>
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<tbody>
<tr>
<td></td>
<td>&lt;AA&gt; SD</td>
<td>&lt;AA&gt; SD</td>
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<tr>
<td>1</td>
<td>... 171 4.2</td>
<td></td>
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<tr>
<td>2</td>
<td>210 5.9 167 4.2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>... 183 2.9</td>
<td></td>
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<tr>
<td>4</td>
<td>244 4.8 176 5.4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>... 179 2.0</td>
<td></td>
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<tr>
<td>6</td>
<td>230 5.4 183 5.8</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>194 4.4 169 3.4</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>235 5.1 197 6.0</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>... 181 7.9</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>220 6.7 192 4.0</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>201 6.4 188 3.2</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>238 2.2 156 2.9</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>241 3.2 183 3.1</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>239 2.6 170 3.5</td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>227.8 4.8 174.6 4.3</td>
<td></td>
</tr>
</tbody>
</table>

<AA> (mean AA interval) and standard deviations are given in milliseconds.

seconds. In those patients in whom both esophageal and intra-atrial recordings were performed, similar variations were found in the atrial flutter cycle length when recording from the two different sites.

In Fig 1 the ECG (V.), the esophageal signal, and the interval plot from an episode of spontaneous rapid atrial flutter are given. Those intervals in which a ventricular beat occurred are indicated by vertical lines. In this example, the rapid atrial flutter was associated with 4:1 AV conduction. It can be seen that the fluctuations in atrial cycle length are related to the ventricular activity with a progressive shortening of flutter intervals after a ventricular beat. The exact time relation between the variation in atrial flutter cycle length and the ventricular activity is given by the phase plot in the lower line plot of Fig 1. After the onset of the QRS complex, the atrial intervals shorten gradually to return suddenly to their original value.

The relation between the atrial flutter cycle length and ventricular activity shows a small variability. This variation has been found in all patients and principally is due to the influence of respiration on the atrial flutter interval. This has been reported previously in humans, but its mechanism was not investigated in the present study.

To better understand the nature of these variations in flutter interval, the cycle length variability of common atrial flutter and the variation in rapid atrial flutter interval have been compared in the nine patients who had both common atrial flutter and rapid atrial flutter episodes during the electrophysiological study. The results of three cases changing in different ways from one type of flutter to the other are presented in Figs 2 and 3. In one case (patient 6), both common and rapid atrial flutter started spontaneously; in the second case (patient 8), the rapid atrial flutter was induced by pacing during the spontaneous common atrial flutter episode; in the last case (patient 4), rapid atrial flutter changed spontaneously into common atrial flutter. The first two cases are presented in Fig 2. The surface ECG (lead V.), the esophageal signal, and the phase plot of both common and rapid atrial flutter are displayed. In the ECG, the difference in morphology of the F waves corresponding to the two types of atrial flutter is evident. In the esophageal signal, the different atrial rates during common and rapid atrial flutter can be seen clearly. The two phase plots recorded in the same patients during the two different types of atrial flutter are strikingly different. In both cases, common atrial flutter shows a prolongation of the atrial cycle length, with a maximum at about 400 milliseconds after the onset of QRS complex followed by a shortening. In contrast, during rapid flutter, the atrial interval decreases after the QRS complex, reaching a minimum
Fig 2. Comparison between the atrial flutter variations during common (A) and rapid (B) atrial flutter in two patients. The ECG (V₁), the esophageal (ESO) signal, and a plot of the atrial interval time aligned to the QRS complex are given. The opposite effect of ventricular activity on the cycle length of common and rapid atrial flutter is evident. Bar indicates 1 second.
value after about 400 milliseconds, then it increases. It should be noted that the two cases displayed in Fig 2 show different degrees of AV block. Patient 6 shows an advanced AV block (range, 2:1 to 7:1) both in common and rapid atrial flutter, whereas patient 8 has a 2:1 AV block in common atrial flutter and a variable AV block (range, 2:1 to 4:1) in rapid atrial flutter.

Fig 3 shows the sudden spontaneous conversion from rapid to common atrial flutter (see arrow). The rapid atrial flutter was associated with a 4:1 AV conduction, whereas the common atrial flutter showed a 2:1 to 3:1 AV conduction. The sudden deceleration of the atrial rhythm is well visible both in the ECG and in the esophageal signal. The beat-to-beat variation of the flutter interval before and after the transition is displayed by the interval plot. The initial cycle length of common atrial flutter was 275 milliseconds. During the first 40 cycles, the interval gradually decreased to reach an average value of 254 milliseconds. The transformation from one type of flutter into the other was associated with a change in the effect of the ventricular beats on the variation in atrial flutter cycle length. Whereas during rapid atrial flutter the cycle length was shortened after the QRS, it prolonged during common atrial flutter (see Fig 3).

In three patients, two different episodes of rapid atrial flutter were recorded. In Fig 4, the phase plots of patient 9 corresponding to two episodes of rapid atrial flutter, one spontaneous and the other induced by rapid pacing, are given. The spontaneous episode showed a 4:1 AV conduction, whereas the induced episode was associated with an advanced AV block (range, 4:1 to 8:1). In both episodes of rapid atrial flutter, a shortening of the interval after the ventricular contraction was found.

In Fig 5, the results of all cases are summarized. In the upper phase plot, the average beat-to-beat cycle length changes of all 17 episodes of rapid atrial flutter are superimposed. Each phase plot has been normalized to the mean atrial interval. On the average, a shortening of the atrial cycle length of 4.1% \( (P<.001) \) occurred after the QRS complex. The flutter cycle reached a minimal value between 300 and 600 milliseconds after the onset of the QRS complex. In the lower phase plot, the average changes in atrial cycle length of the 11 episodes of common atrial flutter are plotted. In this type of flutter, the atrial cycle length prolonged after each ventricular beat, then shortened.

To test whether a relation exists between variations in atrial flutter cycle length and changes in atrial pressure, in three patients intra-atrial pressure was recorded together with the electrical activity during both common and rapid atrial flutter episodes. Of the three episodes of common atrial flutter, two (patients 12 and 13) were associated with a 2:1 AV conduction and one (patient 14) with a 3:1 to 4:1 AV conduction. The episodes of
rapid atrial flutter were associated with a 4:1 AV conduction in one case (patient 12) and with an advanced AV block (range, 3:1 to 6:1) in the other two cases (patients 13 and 14).

In Fig 6, representative samples of atrial pressure are plotted together with the atrial electrogram and ECG during both common and rapid atrial flutter. A rise of the atrial pressure after each ventricular beat was observed. Mean atrial pressures and the amount of atrial pressure increase after the ventricular beats of the three analyzed cases are given in Table 3. Mean increase of right atrial pressure after the QRS complex ranged from 2 to 5.5 mm Hg. No substantial difference exists between atrial pressure values during common and rapid atrial flutter in the same patient.

In Fig 7, the variation of atrial flutter cycle length after the QRS onset was compared with the variation in atrial pressure at the same time scale for both common and rapid atrial flutter (patient 14). During common atrial flutter, the prolongation of the atrial interval was in close relation to the rise in atrial pressure. During rapid atrial flutter, the rise in atrial pressure coincides with the shortening of the flutter cycle length. This correlation between atrial pressure rising and a change in atrial flutter interval was found in all three analyzed cases.

Discussion

The existence of small fluctuations in cycle length of both classic and rapid atrial flutter was first described by Wells et al. In type I and type II atrial flutter, they measured a mean beat-to-beat variation of 4.3 milliseconds and 6.4 milliseconds, respectively. However, the mechanisms of these variations remained unclear.

In a previous study, we found that in common atrial flutter the interval fluctuations were coupled to the QRS complex, flutter intervals after a ventricular beat being consistently prolonged. The hypothesis was formulated that these variations in flutter interval were caused by atrial stretch as a result of ventricular contraction. In case of an anatomically defined reentrant circuit, stretch of the atrial wall might increase the conduction time of the impulse, thereby prolonging the return time of the circus movement.

In the present study, cycle length variations of common atrial flutter and rapid atrial flutter were compared. The existence of spontaneous fluctuations in cycle length during rapid atrial flutter, as observed previously by Wells et al., was confirmed. The mean beat-to-beat variation of 4.3 milliseconds as found in our study is in good agreement with the average variation of 6.4 milliseconds as reported by Wells et al. Our results further demonstrated that the variations in atrial cycle length were coupled to ventricular activity. After the QRS complex, the cycle length of rapid atrial flutter decreased to reach a minimum value after 300 to 600 milliseconds. The effects of ventricular beats on atrial cycle length in patients with rapid flutter were quite different compared with patients with common atrial
flutter. Whereas during common atrial flutter the ventricular beat causes a prolongation of the flutter interval followed by a shortening, during rapid atrial flutter the atrial cycle first decreases, then increases. This result is particularly evident in the 10 patients who during the electrophysiological study exhibited episodes of both common atrial flutter and rapid atrial flutter.

Intra-atrial pressure recordings during both common and rapid atrial flutter episodes have shown that atrial pressure increases during ventricular contraction. Moreover, echocardiographic studies during atrial flutter also report an increase of the atrial volume during ventricular systole. The comparison between changes in atrial flutter cycle length and changes in atrial pressure after the QRS complex indicate that the variations in atrial flutter interval coincide with changes in atrial pressure.

**Table 3. Changes in Right Atrial Pressure During Atrial Flutter**

<table>
<thead>
<tr>
<th>Patient</th>
<th>RAP, mm Hg</th>
<th>ΔRAP, mm Hg</th>
</tr>
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<tbody>
<tr>
<td>12</td>
<td>14</td>
<td>5.5</td>
</tr>
<tr>
<td>13</td>
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<td>2</td>
</tr>
<tr>
<td>14</td>
<td>13</td>
<td>5</td>
</tr>
</tbody>
</table>

RAP indicates mean right atrial pressure; ΔRAP, mean changes in RAP after the ventricular beats.
The synchronism between changes in flutter cycle length and variations in atrial pressure indicates that variations in atrial flutter cycle length could be directly caused by stretch of the atrial wall. A reflex mechanism would be improbable because the change in flutter cycle length had already started at about 50 milliseconds after the ventricular contraction\(^3\) and reached a maximum between 300 and 600 milliseconds. Since this time delay of 50 milliseconds is too short for a nervous reflex, the most plausible explanation is to assume a direct effect of various degrees of atrial stretch on the mechanism responsible for types I and II atrial flutter.

Studies in isolated heart muscle and in intact hearts have shown that myocardial stretch can alter the electrophysiological properties of cardiac tissue by means of a contraction-excitation feedback mechanism.\(^{10}\) There is good evidence that myocardial stretch or dilation decreases the refractory period, whereas quick release of the fibers causes an increase of refractoriness.\(^{10-19}\) This phenomenon has been demonstrated to operate on a beat-to-beat basis\(^{10-13,18}\) and to occur rapidly with a time lag of only 10 to 20 milliseconds.\(^{11}\) The long-term effects of myocardial stretch are less clear.\(^{20,21}\) The effects of stretch on conduction velocity appear to be more complex. Conduction velocity increases\(^{22,23}\) or remains constant\(^{24}\) as the tissue is stretched to the length at which it achieves peak active force development; with further stretch, conduction velocity decreases.\(^{23,24}\) However, even in the case in which the conduction velocity increases with stretch, such increase is less than the increase in the path length or is nearly equal. This implies that the time required for conduction from one point to another should increase with stretch. This result was confirmed in a recent study in which an increase of the intra-atrial conduction time was found after dilatation of the atrium.\(^{16}\) A decrease of the resting potential\(^{24}\) and induction of afterdepolarizations\(^{13,15}\) after stretch also have been reported.

**Effects of Atrial Stretch on the Electrophysiological Mechanism of Type II Atrial Flutter**

The current view, supported by many experimental and clinical studies, points to a reentrant mechanism underlying human atrial flutter.\(^{6,25-32}\) The characteristics of the reentrant circuit, the dimension of the excitable gap, and the presence in the circuit of an area of slow conduction are under investigation.\(^{33}\) Moreover, it is still unclear whether the various types of flutter observed in humans are based on different types of reentrant mechanisms. Common atrial flutter has been
widely investigated, and the most probable mechanism appears to be a large reentrant circuit in the right atrium based on an anatomic substrate with an area of slow conduction.6,28,34,35 Less information exists about the underlying mechanism of rapid atrial flutter in humans. The observation of Wells et al5 that type II flutter cannot be interrupted by rapid pacing may point to a functionally determined intra-atrial circuit (leading circle).

If stretch of the atrial wall during ventricular systole is directly responsible for the shortening of the atrial flutter cycle length, this may give some clues to the underlying mechanism of type II atrial flutter. The increase in flutter rate by stretch cannot be explained by the existence of an anatomically determined circuit with a fully excitable gap. In such a circuit, shortening of the refractory period by stretch will not lead to an increase in conduction velocity, and the increase in path length would lead instead to a decrease in the rate of the arrhythmia.

The present observation that in type II flutter the cycle length shortens after the QRS complex is consistent with leading circle reentry as a mechanism of type II atrial flutter.36 In contrast to reentry in an anatomically determined circuit, the cycle length of reentry around a functional area of conduction block is not directly dependent on conduction velocity because a change in conduction velocity can be immediately neutralized by a change in the length of the circuitous pathway. It has been shown that the leading circle cycle length is primarily proportional to the duration of the refractory period.30-38 Thus, the shortening of the atrial interval after the onset of the QRS complex as found in patients during rapid atrial flutter can be explained by stretch-induced shortening of atrial refractoriness and consequent shortening of the revolution time of leading circle reentry.

A shortening of the refractory period after stretch also would alter conduction velocity and cycle length of an anatomically defined pathway in which the impulse conducts through partially refractory tissue.30,39 In such a circuit, shortening of the refractory period would prolong the excitable gap, accelerate conduction velocity, and consequently decrease the flutter cycle length. However, because the rate of this form of reentry is also dependent on path length, the response to stretch will depend on the balance between increase in conduction velocity and lengthening of the reentrant pathway. The net result will largely depend on the size and the properties of the excitable gap. The smaller the excitable gap and the less recovery of its excitability, the more likely it is that stretch will accelerate the flutter rate.

The effect of stretch on atrial flutter interval may differ according to the type of reentrant mechanism underlying the arrhythmia. The two different reactions of atrial flutter cycle length after a ventricular contraction during common and rapid atrial flutter could be ascribed to the presence of two different types of reentry: reentry governed mainly by the conduction time (path length/conduction velocity) and reentry determined by the refractory period. By increasing the conduction time or by shortening the refractory period, atrial stretch will either produce slowing of flutter in case of anatomical reentry with a large excitable gap or will accelerate the flutter rate when caused by a leading circle mechanism.

**Limitations of the Study**

This study hypothesized that changes in electrophysiological variables (refractory period, conduction velocity, and reentrant path length) of the atria secondary to pressure and/or volume changes were responsible for the changes in atrial flutter cycle length. The detection of conduction velocity and reentrant path changes is very difficult to perform in humans during an electrophysiological study. Changes in the refractory period on a beat-to-beat basis could be detected by monophasic action potential recordings. However, in the present study, such a technique has not been used. Thus, the electrophysiological effects of atrial stretch on conduction and refactororiness were derived from the literature and were not directly measured during atrial flutter in our study group.

**Conclusions**

Our study demonstrates that the fluctuations in atrial flutter cycle length depend on the ventricular contractions, indicating that a contraction-excitation feedback mechanism may exist in atrial muscle and influence the electrophysiological properties of the supraventricular arrhythmia. Furthermore, the detailed analysis of variations in flutter cycle length might be a useful tool to study the underlying mechanisms of atrial arrhythmias. The two different phase relations between atrial cycle length and ventricular activity found during type I and type II atrial flutter point to different reentrant mechanisms in these two forms of flutter. The identification of different pathophysiological mechanisms in patients with atrial flutter may have important clinical implications as far as prevention and treatment of flutter are concerned.40

**Acknowledgments**

This investigation was supported by Istituto Trentino di Cultura, Consiglio Nazionale delle Ricerche (CNR-GNCR), and Ministero per l'Università e la Ricerca Scientifica e Tecnologica (40% Biofisica).

**References**


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Circulation. 1994;89:2107-2116
doi: 10.1161/01.CIR.89.5.2107

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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World Wide Web at:
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