Participation of Atrial Specialized Conduction Pathways in Atrial Flutter

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SUMMARY The circus movement theory of flutter has been supported by numerous theoretical, experimental, and clinical studies, but there has been little consideration of the possible participation of the specialized conduction pathways of the atria. In the present work we have studied experimental atrial flutter in relation to the behavior of these pathways, in response to elevated potassium and to surgical interruption. Application of trains of stimuli at frequencies appropriate to induce a self-sustained flutter revealed that (1) flutter can be more easily induced by stimuli applied to the left atrial insertion of Bachmann's bundle than to the body of the right atrium, (2) flutter can be initiated by appropriate trains of stimuli without the creation of an artificial obstacle, and (3) flutter exhibits the same characteristic behavior, with or without artificial obstacles. Doses of potassium that elevate the plasma concentration to 9.5 mm/liter cause total inactivation of atrial myocardium without suppressing flutter waves as recorded from the vicinity of the internodal pathways. Section of the middle internodal pathway reduces the frequency of experimental flutter; section of both the middle and posterior pathways prevents the establishment of flutter. The results suggest that the specialized internodal pathways play an important role in the genesis and maintenance of circus movement flutter.

SINCE THE circus movement theory of atrial flutter was proposed by Lewis et al., there have been many theoretical, experimental, and clinical studies in support of the concept. Among the relevant theoretical studies are those of Wiener and Rosenblueth, Rytand, and Stibitz and Rytand, all of whom, in accord with Lewis et al., made the simplifying assumption that the propagation of impulses in the atria occurred at the same velocity in all directions, and selected as the primary condition of mathematical analysis a model with homogeneous properties. Using such a model, Wiener and Rosenblueth demonstrated the necessity for an obstacle around which an impulse could circulate; circulation could be permanent provided the perimeter of the obstacle exceeded the wavelength of the impulse (defined as the distance traveled during a period of time equal to the refractory period). Initiation of the flutter process can be explained by assuming asymmetric recovery of excitability in a zone that includes the perimeter of the obstacle; introduction of a second impulse during the phase of asymmetric excitability then could initiate the one-way conduction that is essential for the self-sustained circus movement.

Most of the published experimental studies, in accord with this hypothesis, suggest that the natural obstacle provided by the caval orifices might suffice, but some investigators, estimating that the perimeter of the natural obstacles would be too small, enlarged the potential circuit path by crushing an area of atrial tissue adjacent to the natural openings. Rosenblueth and Garcia Ramos extended the margin of the inferior caval orifice, a method widely adopted in subsequent studies, and were able to show that the excitation wave in their experimental flutter did indeed sequentially activate the tissue around the obstacle.

Pharmacological evidence reported by Mendez et al. supports the circus movement character of flutter induced by the Rosenblueth and Garcia Ramos technique. Antiarrhythmic agents that increase the wavelength of the excitatory process suppress the flutter; others, like potassium, that are effective in overcoming an aconitine-induced tachyarrhythmia, reduce the wavelength and fail to abolish the circus movement flutter. Clinical confirmation reported by Cardenas et al. suggests that many cases of atrial flutter in man are of circus movement origin. Indirect evidence has also been published by Decherd et al. and by Cabrera and Sodi Pallares, who found that the electrical axis of the atria undergoes a 360° shift with each flutter wave. More recent studies with intracavitary electrodes and with vectorcardiographic and ultrasonic techniques support the hypothesis.

These many studies provide a solid basis for the circus movement theory of flutter, but few have considered the possible role of specialized conduction pathways within the atria, although the functional significance of oriented bundles was considered years ago by Eyster and Meek. The three internodal pathways described by Bachmann probably determine the sequence of atrial activation in a number of species, including man. Struck by the orientation of the internodal pathways, James called attention to the possibility of their participation in circus movement flutter. In some respects the physiological characteristics of atrial specialized fibers resemble those of the ventricular Purkinje tissue: more rapid depolarization during "phase O," faster conduction velocity, a plateau during phase 2, and a greater capacity to undergo spontaneous pacemaker activity than the "ordinary" atrial muscle fibers. Of great
The internodal pathways form closed loops between the sinusal (SA) and atrioventricular (AV) nodes; as preferential pathways, they could play a role in the genesis and maintenance of atrial flutter. To test this hypothesis, we have examined the possibility of producing circus movement flutter by applying rapidly repetitive stimuli to the left atrium, on the assumption that the junction of Bachmann's bundle with the anterior internodal pathway would provide an asymmetric input into the right atrial internodal complex. We have also studied the effects of increasing the plasma potassium concentration on established flutter. Because the loops formed by the three internodal pathways provide three potential circuits of different perimeters, we studied the influence of surgical section of the middle and posterior pathways on the initiation, frequency, and persistence of flutter.

Methods

Young dogs of both sexes, weighing between 10 and 15 kg, were anesthetized with sodium pentobarbital, 35 mg/kg, administered intravenously. Under artificial respiration, the chest was opened in the midline and the heart was suspended in a pericardial cradle attached to the edges of the split sternum.

Electrograms were recorded by means of an ink-writing polygraph (Grass p-7) or an Electronics for Medicine photographic oscillograph. Three pairs of direct leads were attached to the atrial epicardial surface (Fig. 1); one was positioned over the middle internodal pathway near its junction with the SA node (site 8), one was placed over the middle pathway at its passage between the caval orifices (site 10), and the third was attached to the anterior margin of the right atrial appendage (site 11). A fourth channel was used to display a modified lead 2 electrocardiogram, recorded between the superior vena cava and the left hind leg, a lead that enhances the P waves. The distances between atrial recording sites 8 and 10 averaged about 20 mm, and between 8 and 11, 35 mm. Electrode terminals (Grass E2B) were separated by 0.5-1.0 mm and were inserted at a depth of 0.5-1.0 mm in the atrial wall at the specified points. The recording sites were chosen to be representative of events near one of the internodal pathways, in comparison with activity recorded from atrial myocardium. These limited sites do not, of course, permit a map of atrial excitation sequence during flutter, nor was it our objective to record such a map.

Twenty-four experiments, divided in four groups of six, were performed (six additional experiments were used to assess questions raised by the first groups). The first group was used to assess the efficacy of stimuli applied to the left atrium near the distal portion of Bachmann's bundle, compared with similar trains of stimuli applied to the body of the right atrium (site 17). The stimuli, obtained from a Grass S-8 stimulator, were delivered at frequencies of 8, 10 and 15 Hz, in trains lasting 1, 2, 4, 6, 8, and 10 seconds. Stimuli were applied through bipolar electrodes at 0.5-msec duration, and 10% above the diastolic threshold intensity. The characteristics of the resulting flutters, at all combinations of stimulus frequency and duration, were compared, following which the obstacle formed by the inferior vena cava was enlarged by crushing adjacent atrial tissue, and the influence of the site of stimulation was again assessed. The site of the crush, as in the experiments of Rosenblueth and Garcia Ramos, is indicated in Figure 1 by the solid line at position 18. The resulting lesion was about 20 mm in length and 10 mm in breadth, encompassing the full thickness of the atrial wall and the adjacent vena cava. Postmortem examination in three experiments confirmed that the lesion included the posterior internodal pathway (9 in Fig. 1).

In the second group of experiments, flutter was again initiated by stimulation of the intact atria. After an observation period of 30 minutes, a bolus of KCl, 10 mg/kg, was injected into the superior vena cava. Blood samples for potassium analysis by flame photometry were drawn from the brachiocephalic artery before, during, and after the KCl infusion. Electrograms were recorded at a paper speed of 100 mm/sec during the whole period.

In the third set of experiments, KCl was administered during a stable flutter by continuous intracaval infusion at a rate of 10 mg/kg per minute. Blood samples were drawn at intervals of 1 minute for 5 minutes. As before, the electrograms were recorded continuously before, during, and after the KCl infusion.

In the last series, flutter was again initiated in the intact atria, and the effect of a rapid injection of KCl (10 mg/kg) was noted. Thirty minutes later, when the plasma [K] had returned to normal, the middle internodal pathway was sectioned by cutting through the bridge of atrial tissue between the caval orifices (15 in Fig. 1) and immediately ceased.
suturing the incision. Flutter was again initiated, and the electrocardiographic characteristics were compared with those recorded prior to the intervention. The effect of KCl was again observed, and after an additional recovery period of 30 minutes, the posterior tract was sectioned at the point of its passage between the inferior vena cava and the atrioventricular sulcus (site 19). Following this final intervention, attempts to reinitiate flutter were made.

Additional experiments were done to test the effect of elevated plasma [K] on conduction velocity at a stimulation frequency of 7 Hz and to test the effect of section of Bachmann's bundle.

**Results**

**Influence of the Site of Stimulation**

Stimulation of the left atrium proved to be much more effective than stimulation of the right atrium in initiating flutter in the intact atria (Fig. 2).

In the six experiments represented in Figure 2, stimuli were applied at a frequency of 8 Hz in part A, 10 Hz in B, and 15 Hz in C. Open columns indicate the success rate for stimuli applied to the left atrium (Bachmann's bundle), solid columns for right atrium. In no case did stimulation of the body of the right atrium at 8 Hz result in flutter, even with 10 seconds of stimulation, while left atrial stimulation succeeded in one dog at the briefest duration and in 50% at 8 and 10 seconds. The discrepancy is evident also at the higher stimulus frequencies (Fig. 2, B and C). Right atrial stimulation was 100% successful only at the longest duration (10 seconds) and the two highest frequencies, whereas left atrial stimulation was 100% successful in 9 of the 18 possible combinations.

At the lower and upper extremes of the stimulus patterns (low frequency, brief duration; high frequency, long duration), the test procedure does not discriminate between right and left atrial stimulation. With these two extremes eliminated, the data are summarized as a 2 x 2 table in Table 1, which indicates the success rate for right and left atrial stimulation with trains of 1, 2, 4, and 6 seconds at frequencies of 10 and 15 Hz. The exact probability, according to Fisher's bilateral test for 2 x 2 tables, is 4.028 x 10^-10.

Once induced, the frequency of the flutter was the same whether stimulation was applied to the left or to the right atrium, and the frequency was not influenced by crushing tissue adjacent to the inferior vena cava according to the technique of Rosenblueth and Garcia Ramos (Table 2). The greater ease of inducing flutter by left atrial stimulation, apparent in Figure 2, was not influenced by the crushing lesion. In all cases, the flutter persisted until interrupted by a brief burst of rapid stimulation or other intervention.

In three ancillary experiments, crushing lesions were made across the interatrial band. Left atrial stimulation was still effective in inducing flutter in two of these. This result was not unexpected, as the localized interruption would not prevent a bypass and reentry of the specialized tissue beyond the incision. In the third experiment, stimulation on the left atrial side of the lesion was no longer successful, but stimulation of the region of Bachmann's bundle on the right side of the lesion was still effective.

**Effects of Potassium, Single Bolus**

Abrupt elevation of plasma [K] during atrial flutter suppressed electrical activity in atrial muscle (as recorded from the margin of the right atrial appendage), but flutter activity persisted for a brief time in electrograms recorded in proximity to the specialized fibers (Fig. 3). In each of the panels of Figure 3, the two upper traces were recorded from the region of the middle internodal pathway near the sinus node and between the two caval orifices, respectively (sites 8 and 10 in Fig. 1). The third trace displays activity from the atrial myocardium (site 11), and the fourth is the electrocardiogram derived from superior vena cava and left hind leg. Between panels A and B, flutter at a frequency of 8 Hz was established by a brief train of stimuli. All atrial records show the same frequency, also evident in the ECG, in which 2:1 AV block is apparent. The alternating configuration of the electrograms in the atrial records indicates that the flutter was near the maximum frequency permitted by the limiting refractory

**Table 1** Incidence of Flutter Resulting from Stimulation of Right vs. Left Atria with Trains of 1, 2, 4, and 6 Seconds at Frequencies of 10 and 15 Hz

<table>
<thead>
<tr>
<th>Experiment</th>
<th>R.A. (Hz)</th>
<th>L.A. (Hz)</th>
<th>R.A. (Hz)</th>
<th>L.A. (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.0</td>
<td>8.0</td>
<td>8.0</td>
<td>7.8</td>
</tr>
<tr>
<td>2</td>
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<tr>
<td>6</td>
<td>8.9</td>
<td>9.0</td>
<td>8.7</td>
<td>8.6</td>
</tr>
</tbody>
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R.A. = right atrium; L.A. = left atrium.
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FIGURE 3
Initiation of flutter and its persistence in specialized atrial tissue (upper two traces in each panel) after inactivation of atrial myocardial activity (3rd trace) by single bolus of KCl. Further explanation in text.

period of the tissue in the circuit pathway. In panel C, recorded 11 seconds after the bolus of KCl, the atrial frequency was retarded to 6.7 Hz, and activity at that frequency appeared in both of the upper traces. The electrograms recorded from the region of the middle pathway at this stage are more variable, in spite of the slower frequency, as a result of the K-induced slowing of conduction (see below). Activity in the atrial muscle was reduced to approximately half the flutter frequency, but flutter with 2:1 AV block was still apparent in the electrocardiogram. Panel D, recorded 2 seconds later, shows the last three flutter waves recorded near the middle pathway and a complete absence of electrical activity in the ordinary myocardium. Upon restoration of sinus rhythm (last half of panel D), the ECG showed ventricular complexes without P waves, presumptive evidence for a sinoventricular rhythm. Activity was still present in the region of the middle pathway, but the total mass of participating atrial tissue was obviously too small to generate manifest P waves, even in the modified lead 2 recording. Plasma K concentration reached 8 mM/liter in the 1st minute and returned to normal within the following 5 minutes. Similar events were consistently observed in all six experiments of this series.

Several conclusions may be drawn from this set of experiments. First, activity may be recorded from the vicinity of the internodal pathways at a time when areas of atrial myocardium are inexcitable. Second, the frequency of the flutter movement diminishes as the concentration of K in the plasma increases. Third, conduction in the flutter pathway becomes compromised, as indicated by the varying configuration of electrograms recorded in the region of the internodal pathway.

It is known that elevated [K], beyond about 7 mm, will depress conduction in cardiac tissues even at "normal" frequencies of stimulation, but accessory experiments were conducted to assess the effect of elevated K on conduction at a driving frequency comparable to the flutter frequency. In three experiments, conduction intervals were measured between sites 8 and 10, as representative of conduction in the specialized pathways, and between 5 and 11 (Fig. 1), as representative of conduction in atrial muscle. Stimuli were applied at a site close to site 8, at a frequency of 7 Hz, as KCl was infused at a rate of 7.0 mg/kg per minute. The data are listed in Table 3 as apparent velocities (i.e., interelectrode distance divided by the time interval) as a function of plasma K concentration. In the control condition (K = 4.25 mM), the apparent velocity was approximately 60% faster in the region of the middle internodal pathway than in the direction of the path in atrial myocardium. As the K concentration increased, conduction was depressed in both routes; 2:1 block appeared in the atrial muscle at K = 8.9 mM, and total block at an average of about 10 mM. At this level, conduction velocity as estimated in the internodal pathway was depressed to a degree compatible with the observed depression of frequency of the flutter movement recorded following K injection in the series of experiments represented by Figure 3. The last two rows of figures in the table were recorded during recovery from K infusion.

Potassium Infusion

When potassium was injected as a bolus, as in Figure 3, dissociation between the specialized pathways and the atrial myocardium was limited to a very brief period just prior to arrest of the flutter, and precise estimation of the plasma [K] at the critical moment could not be achieved; accordingly, six additional experiments were conducted in which KCl was infused at a rate of 10 mg/kg per minute. In three of these, the flutter was induced by left atrial stimulation in intact atria; in the other three, the method

<table>
<thead>
<tr>
<th>Plasma K+ concentration (mm/liter)</th>
<th>Apparent conduction velocity (m/sec)</th>
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<tr>
<td></td>
<td>Middle pathway</td>
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<tr>
<td>4.25</td>
<td>1.3</td>
</tr>
<tr>
<td>5.05</td>
<td>1.1</td>
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<td>6.05</td>
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<tr>
<td>8.9</td>
<td>0.8</td>
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<tr>
<td>10.5</td>
<td>0.7</td>
</tr>
<tr>
<td>11.8</td>
<td>0.5</td>
</tr>
<tr>
<td>9.8</td>
<td>1.1</td>
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<tr>
<td>6.8</td>
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The figures represent the average of three experiments. The lower two rows were recorded during recovery from infusion of KCl.
of Rosenblueth and Garcia Ramos was used. In the first two experiments with intact atria, the flutter ceased within a few seconds after electrical activity in the atrial myocardium was suppressed. In the third experiment, the infusion of KCl was stopped at the first sign of inactivation of the atrial muscle. Examples of the record obtained from this experiment are shown in Figure 4. In each panel the upper tracing was recorded from the intercaval portion of the middle pathway, the second tracing from the right atrial appendage, and the lower trace from the modified lead 2. As in Figure 3, panel A was recorded during sinus rhythm and panel B after initiation of flutter at 8.7 Hz, again with 2:1 AV block. Panel C was obtained 80 seconds after the KCl infusion was begun. Flutter frequency had diminished to 7.3 Hz, with considerable irregularity of the configuration of the electrograms, and some degree of block was apparent in the myocardial record. Plasma [K] at that time had risen from 3.5 to 7.5 mM/liter, and the infusion was stopped. Five seconds later (panel D) the atrial electrogram was completely silent, but activity continued at a frequency of about 6 Hz in the middle pathway, as shown in the upper tracing. At that time, the plasma [K] had reached 9 mM/liter. Suppression of atrial myocardial activity, with continuing activity in the vicinity of the middle pathway, persisted until plasma [K] dropped below 7.0 mM/liter. Recovery of activity in the muscle is shown in panel E, recorded 2 minutes after the end of the infusion. Recovery to the initial flutter pattern in all leads was complete within the following 2 minutes.

In the three experiments in which the caval orifice was extended by crushing, the KCl infusion was stopped at the first sign of inactivation of the atrial muscle. In each of these the sequence of events was similar to that in Figure 4. The results from one of these are graphed in Figure 5. KCl was infused for 75 seconds beginning at time 0. The flutter frequency, initially 500/min, dropped progressively to 170/min, but was still maintained in the region of the internodal pathway (open circles) while atrial muscle (crosses) was completely inactive. Plasma [K] reached a peak value of 9.3 mM/liter (triangles); when it had fallen to 7.6 at the 3rd minute, the flutter frequency began to increase, and activity returned in the atrial muscle. During the subsequent 5 minutes, both the [K] and the flutter frequency approached the initial conditions. The principal conclusion of these experiments is that, while activity in atrial muscle was completely suppressed, activity (although slower and irregular in contour) persisted in the region of the internodal pathway, and that the initial flutter frequency was resumed when the elevated K receded.

Influence of Section of the Internodal Pathways

In six experiments, the middle internodal pathway was sectioned between the venae cavae (site 15, Fig. 1). Invariably, this intervention reduced the flutter frequency (Fig. 6). In the example shown in the figure, electrograms were recorded from the middle internodal pathway near the SA node and at its intercaval passage (upper two traces; 8 and 10, respectively, in Figure 1), from atrial muscle of the right atrial appendage (3rd trace), and from the epicardial surface of the ventricle. Panel A was recorded during sinus rhythm, and B was recorded 30 minutes after flutter had been established at a frequency of 8 Hz. At this frequency, alternating configuration and cycle lengths occurred in all three atrial records. Between B and C, a rapid injection of KCl, as in previous experiments, caused the development of 2:1 block between the specialized tissue and atrial muscle (last half of panel C), progressing to complete block in D. Soon after, the flutter was replaced by sinus rhythm. When plasma [K] had returned to its original level, the middle pathway was sectioned, and flutter was reestablished, but at a frequency

![Figure 5](http://circres.ahajournals.org/)

**Figure 5** Frequency of flutter in specialized atrial fibers (circles) and in atrial muscle (crosses) in relation to changes in plasma potassium concentration (triangles).
of 7 Hz (panel E). At the slower frequency, all of the atrial electrograms recorded regular rhythmic activity. Again, KCI injection terminated activity in the atrial muscle, while flutter waves persisted in the region of the middle pathway (panel F). In panels D and F, it is clear that the persistent activity in the upper two traces is flutter, not fibrillation. In each panel, however, the frequency was reduced at the higher potassium concentrations.

Following recovery from elevated plasma [K], two additional surgical interventions were made. The anterior pathway was cut near the sinus node (site 12) and the posterior pathway where it passed between the inferior vena cava and the AV groove (site 19). Both incisions were scalpel cuts of about 10 mm in length, immediately sutured. Following these procedures, it was no longer possible to induce a persistent flutter.

When the initial flutter frequency in the intact atria is high, the electrograms are often irregular in contour. In one experiment, illustrated in Figure 7, it was impossible to induce a stable flutter before surgical intervention. Brief periods of irregular activity at a variable frequency of 7.3-7.6 Hz, and lasting less than 2 minutes, followed repeated attempts (Fig. 7, panel B). With the objective of increasing the potential path length, the intercaval portion of the middle pathway was crushed. A stable flutter at a frequency of 6.3 Hz followed the first burst of stimuli applied to the left atrium (panel C) and persisted for 30 minutes until terminated by an injection of quinidine.

Discussion

The results described above provide one more piece of evidence in support of the circus movement theory proposed by Lewis, but there are some important differences between results of these studies and the many previous reports. In earlier studies, in accord with the concepts of Lewis et al., the atrial myocardium was considered as a physiologically homogeneous system, despite accumulating physiological and anatomic evidence to the contrary. The nonhomogeneous nature of intra-atrial conduction forms the basis of our experimental approach.

Influence of the Site of Stimulation

The very significantly greater ease with which stimulation of the left atrium can induce flutter, compared with the right, can be explained in terms of the physiological properties and anatomic relations of Bachmann’s bundle. The interatrial band and a branch of the anterior pathway share a common origin, and they run a parallel course to their divergence at the level of the interatrial septum. Bachmann’s bundle continues along the superior aspect of the left atrium, giving rise to branches that are distributed to the appendage and body of the left atrium. Given the greater conduction velocity of the bundle, an impulse initiated in the left atrium should be conducted quickly to the junction of Bachmann’s bundle and the anterior pathway, a site at which the necessary asymmetry postulated by Wiener and Rosenblueth might permit the initiation of one-way conduction. This would be less likely to occur for impulses initiated in the body of the right atrium.

The frequency of flutter induced in the intact atria, and that produced in the same heart after a crushing lesion adjacent to the inferior vena cava, were invariably the same. This suggests that the circuit pathway could not have been limited to the natural obstacle of the caval orifice, for enlargement of that obstacle should have diminished the flutter frequency. Admittedly, the observation of identical frequencies does not deny the possibility of another loop not involving the inferior vena cava,
but it is wholly compatible with the trajectory provided by circuits involving the specialized fibers.

The emphasis on internodal pathways as “loops” providing a preferential circuit pathway does not, of course, imply that the internodal bands are insulated from the ordinary atrial muscle with which they are intermingled. If a bundle of fibers with a higher conduction velocity is enmeshed in an otherwise homogeneous matrix, the “wake” of a wavefront will describe an angle with the axis of propagation that depends on the relative velocities; the transit time of a loop will still be determined by the intrinsic velocity in the “specialized” bundle.

Elevated Potassium Concentrations

The effect of elevated [K] provides the strongest evidence for the participation of the specialized circuits, namely, the persistence of flutter waves in electrograms recorded from the vicinity of the internodal pathways at a time when no activity could be recorded from the atrial myocardium. The greater resistance of the specialized fibers to potassium is perhaps the strongest physiological evidence for their fundamental difference from atrial muscle.36-37

Block between the specialized fibers and atrial muscle induced by increased [K] is enhanced by rapid driving rates.37 The high frequency of flutter must in itself increase the chance of block and, thus, facilitate demonstration of the persistence of flutter after cessation of atrial myocardial activity.

The persisting activity in the area of the internodal pathway often became irregular in the presence of elevated [K], but the basic frequency is still readily estimated. The irregular contour might resemble “fibrillation,” but it is almost surely not fibrillation for the following reasons. (1) Fibrillation, even when all of the atrial tissue mass is available, can be induced and sustained in the normal dog heart only when gross dispersion of atrial refractory periods is induced by vagal stimulation.39 So induced, the frequency, when it can be measured at all, is considerably greater than that of circus movement flutter. (2) When fibrillation terminates (with cessation of vagal stimulation), it is almost invariably followed by resumption of sinus rhythm. In the experiments described here, the frequency of recorded activity diminished gradually as the potassium concentration increased, and increased gradually as the potassium level receded. (3) Fibrillation, defined in a mechanistic sense (i.e., not merely irregular contours of extracellular electrical records), cannot be maintained in a narrow channel; this probably accounts for experimental and clinical observations of dissimilar rhythms in the two atria.

Elevation of plasma [K] to levels that suppressed activity in atrial muscle often terminated the flutter (Fig. 3 and Fig. 6D), and invariably decreased the frequency. Extracellular [K] levels that suppress activity in atrial muscle will reduce the conduction velocity in the specialized fibers; the fundamental difference between the tissues is a quantitative one, not qualitative (Table 3). It is not, however, justified to say that a reduction of 60% in the frequency represents a 60% reduction of conduction velocity, for it is quite possible that a “short circuit” through atrial muscle within the specialized loop provides a somewhat briefer cycle at normal [K] but is suppressed at the higher concentrations, forcing the flutter wave to traverse a longer pathway composed solely of specialized fibers. Nevertheless, the apparent depression of conduction shown in Table 3 (at [K] = 10 mM) is approximately equivalent to the observed reduction in flutter frequency.

The remaining possibility, that the flutter is the expression of a rapidly firing ectopic focus, is incompatible with the demonstrated action of elevated potassium on the abnormal pacemaker activity generated by aconitine36,38 or digitalis.39

Section of the Internodal Tracts

The three internodal pathways have different lengths, providing three possible loops of different circumferences (anteromedial, anteroposterior, posteromedial). If we assume that the conduction velocity is the same in each, then the flutter must circulate at a frequency determined by the shortest loop. Anatomically, the shortest loop is that provided by the anterior and median bundles, and this is probably the circuit pathway whether flutter is established in the intact atria, or following the technique of Rosenblueth and Garcia Ramos. The crushing lesion at site 18 interrupts the posterior pathway but does not alter the flutter frequency (Table 2). When the medial pathway is sectioned, the only remaining option is the loop formed by the anterior and posterior bundles, which must necessitate a lower flutter frequency. This was the invariable result. Persistence of the slower flutter in the presence of elevated potassium (Fig. 6) again attests to the participation of the specialized fibers, as does the failure to establish a stable flutter after section of the anterior and posterior tracts.

Are Obstacles Needed?

Although there are anatomic obstacles present within the postulated loops (venae cavae, membranous septum), it should be apparent that such obstacles are not a prerequisite. The “obstacle” can be a functional one, determined by the duration of refractoriness relative to the conduction velocity. This was apparent in a mathematical model of atrial fibrillation37 and was recently elegantly demonstrated in isolated rabbit atrial tissue by Allessie et al.36,37

Finally, we note the analogy between the topography of the circuits36,37 and the trajectory of the flutter waves recorded in man.36-38

Conclusions

These studies, based on different premises than previous investigations of circus movement flutter, permit the following conclusions. (1) The internodal pathways form loops through which flutter waves may circulate. (2) The greater conduction velocity in the loops of specialized tissue than in atrial muscle makes a flutter movement possible without the necessity for a physical obstacle. (3) The insertion of Bachmann’s bundle into the loops via its
junction with the anterior pathway facilitates the initiation of flutter by impulses initiated in the left atrium. (4) The greater resistance of the specialized fibers to hyperkalemia suggests the need to examine possible differences in the response to other substances, with the objective of improving our understanding of the mechanism of action of antiarrhythmic agents in the treatment of flutter.

Acknowledgments

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