Varying Types of Circus Movement Re-Entry With Both Normal and Dissociated Contralateral Conducting Different Right and Left Atrial Rhythms in Canine Atrial Flutter

Shigeo Yamauchi, MD; John P. Boineau, MD; Richard B. Schuessler, Ph D; James L. Cox, MD

The purpose of this study was to develop an animal model of atrial flutter (AFL) or fibrillation (AFB) and to determine precisely the pathway of atrial activation during arrhythmias induced by programmed stimulation. In 10 dogs, a shunt from the left subclavian artery to the left upper pulmonary vein was created to produce left atrial enlargement. Five months later, using programmed electrical stimulation, it was possible to induce 17 sustained atrial tachycardias in 9 of the 10 dogs, including 9 episodes of AFL caused by circus movement reentry, 6 episodes of focal tachycardia, and 2 episodes of AFB. Short cycle length left atrial tachycardias caused by either circus movement or a focus did not propagate in a uniform 1:1 pattern to the right atrium (RA), resulting in RA dissociation. In these arrhythmias, complex wavefronts from both current and preceding left atrial cycles coexisted in the RA. Circumference movement was associated with a spectrum of different re-entrant pathways with different path lengths. These differences in the path length were determined by various ways in which obstacles such as the superior vena cava and orifice of the right atrial appendage or pulmonary vein orifices were combined by contiguous areas of functional block. (Jpn Circ J 1998; 62: 201–210)

Key Words: Electrophysiology; Activation mapping; Arrhythmias; Atrial flutter; Atrial fibrillation

Atrial flutter (AFL) and atrial fibrillation (AFB) are closely related arrhythmias caused by different forms of intra-atrial re-entry. Precisely how these 2 arrhythmias are related is presently unknown. It is apparent that they represent different degrees of complexity in their activation pattern and mechanisms. Comparative activation data indicate that AFL is less complicated than AFB, as might be expected. It therefore seems logical that, by first working out the factors involved in AFL, a basis for the later understanding of AFB can be developed.

There have been various different experimental models of AFL. Each animal model possesses some unique feature that not only results in the arrhythmia, but also makes it overly specific and limits its applicability in understanding human AFL. AFL can be produced by creating incisional barriers to conduction. AFL can also be induced in animal models with slow atrial conduction, such as that produced by sterile pericarditis. AFL can also be induced in experimental models of right atrial enlargement caused by tricuspid regurgitation and pulmonic stenosis. Each of these experimental models exhibits some of the characteristics of human AFL and others that do not resemble clinical arrhythmia. Naturally occurring AFL closely simulating human arrhythmia has also been studied. Both AFL and AFB frequently occur in patients with left atrial enlargement as a result of mitral disease or secondary to left ventricular dysfunction. Previous experimental models of mitral regurgitation have been used to produce AFL and AFB. These models had a high incidence of acute pulmonary congestion, which limited the number of animals with slowly progressive left atrial enlargement and reproducibly inducible atrial arrhythmias. Thus, we have developed a new experimental model of left atrial overload with a low incidence of acute pulmonary edema and a high incidence of reproducible, sustained atrial arrhythmias at 5 months postoperatively. The model employed a subclavian artery to pulmonary vein shunt and underwent serial evaluation over a 5-month period to determine the optimal state of arrhythmia inducibility before the final activation mapping study. At the time of the mapping studies, a variety of atrial arrhythmias were observed.

Based on a number of activation studies, it has been shown that non-uniform recovery of excitability can lead to intra-atrial re-entry tachycardias. Also, the specific details of how this mechanism contributes to a series of self-initiated re-excitations has been documented. Even so, there continue to be certain unresolved issues. First, it has not been definitively elucidated if and how the atrial anatomy contributes to the re-entrant process in AFL and AFB. A case has been made for anisotropic geometric effects interacting with wavefront orientation and timing,
Fig. 1. The endocardial electrode forms. The left and right atrial electrode forms are viewed posteriorly in their approximate endocardial positions. A total of 252 bipolar electrograms were recorded simultaneously during each arrhythmia. Specially designed computer programs were used to analyze the data automatically and to display the activation sequence maps.

resulting in unidirectional block and conduction. However, these effects remain to be worked out in actual models of AFL and AFB. Additionally, at a macroscopic level, there are data indicating the importance of atrial orifices as the principal obstacles leading to unidirectional circus movement. Re-entrant activation has not been definitively related to gross structural discontinuities, such as the atrial orifices or the orientations of major myocardial bands, etc. Additionally, the relative importance of these anatomic discontinuities (orifices) in comparison with obstacles of functional block has not been clarified. The present study addresses these issues. Using special multielectrode forms, molded from actual casts of canine atria, multiple areas of the convoluted atrial geometry have been recorded from simultaneously. As a result, the role of atrial orifices, functional block, and the interaction between these 2 factors in the formation of re-entrant pathways has been elucidated.

**Methods**

**Left Subclavian Artery to Left Upper Pulmonary Vein Shunt Procedure**

Twelve adult mongrel dogs weighing 21–29 kg (mean 26 kg) were anesthetized with pentothal (20–30 mg/kg intravenously, maintained with 1–2% halothane). Positive-pressure ventilation was maintained via a cuffed endotracheal tube at an inspired oxygen concentration of 50%. The chest was opened through a left thoracotomy via the fourth intercostal space. The subclavian artery was prepared for the shunt procedure. After intravenous heparin (0.5 mg/kg) was administered, the internal thoracic, vertebral, costocervical, and omomochal arteries were ligated and divided in order to obtain a suitable length of the subclavian artery to perform the shunt. The left subclavian artery was anastomosed to the left upper pulmonary vein from end to side at the point where it crosses to enter the pericardium. Immediately following the shunt procedure, acute postoperative electrophysiologic data were obtained using a quadrupolar electrode catheter, which was inserted through the right femoral vein. Electrical stimulation was performed by bursts of rapid atrial pacing for 18 cycles or 30 sec at decreasing cycle lengths from 200 to 10 msec. All simulation protocols were performed at twice diastolic threshold at a stimulus pulse width of 2.0 msec. Two dogs developed acute pulmonary edema on days 3 and 10 postoperatively and were euthanized. The 10 surviving dogs formed the study group.

All animals received humane care in compliance with the Principles of Laboratory Animal Care formulated by the National Society of Medical Research and Guide for the Care and Use of Laboratory Animals prepared by the National Academy of Sciences (NIH publication 80–23, rev 1978). In addition, the study protocol was approved by the Washington University Animal Studies Committee.

**Interim Electrophysiologic Studies**

Forty to 80 days postoperatively, electrophysiologic data, including those from 2 atrial electrograms and the lead II ECG, were obtained and programmed electrical
stimulation was performed using 2 quadripolar catheters that were placed in the right atrium. Programmed electrical stimulation (PES) at a basic cycle length of 300 msec with a single premature stimulus was delivered at progressively shorter coupling intervals (10 msec) until sustained or non-sustained AFL, AFB, or other tachycardias were induced. Subsequently, an S3 and an S4 were delivered if the previous series failed to initiate tachycardia. If PES failed to initiate tachycardia, then a burst of rapid pacing from the high right atrium was begun at a cycle length of 200 msec and progressively decreased in 10-msec decrements until AFB or AFL was produced. The duration of the stimuli was 8 beats, 18 beats, or 30 sec with a pulse width of 2.0 msec at twice the diastolic threshold. We defined sustained AFL or AFB as that lasting longer than 5 min and which required electrical cardioversion to terminate these arrhythmias. Non-sustained AFL and AFB were defined as those arrhythmias that spontaneously terminated within 5 min.

Table 1

<table>
<thead>
<tr>
<th>Dog No</th>
<th>AFL types and contralateral atrial conduction</th>
<th>Focal AFB</th>
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<tr>
<td></td>
<td>IV</td>
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<tr>
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<td>A B</td>
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<tr>
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<td>1</td>
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<tr>
<td>10</td>
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</table>

Subtotals: 2 1 0 1 5 9 6 2

Classes of tachycardia: I, functional block only; II, anatomic obstacle only; III, combination. Contralateral atrial conduction, 1:1 conduction, B, Dissociation.

Terminal 5-Month Mapping Studies

After the 5-month observation period, anesthesia was induced with pentothal as before. The dogs were placed on positive pressure ventilation. A right 5th intercostal space thoracotomy was performed. The heart was cradled in the open pericardium and the shunt was then closed. Intravenous heparin (1 mg/kg) was administered and the right femoral artery and vein were cannulated for cardiopulmonary bypass. Endocardial electrode forms were inserted through the ventriculotomy during normothermic cardiopulmonary bypass. The endocardial systems were constructed from 3-dimensional casts and molds obtained from actual canine atra. These electrode matrices were composed of flexible silastic material that conformed to the irregular, convoluted anatomy of the endocardium, septum, and the inflow and outflow orifices of both atra (Fig 1). The electrode systems contained 252 bipolar electrode sites. Each bipolar site contained a pair of fine silver wire electrodes, separated by an interelectrode distance of 200 μm. Each electrode bipolar pair was separated by a distance of 5–7 mm. The right atrial endocardial template contained 128 bipolar electrode pairs and the left atrial endocardial template contained 124 bipolar electrode pairs. Fig 2 illustrates the atrial anatomy and the location of the endocardial electrodes in relation to the specific atrial structures. In Fig 3A, the encircled electrode locations shown in the anterior and posterior right atrium represent recording sites for the potentials shown in Fig 3B during an example of AFL. Note that the electrograms completely span the entire cycle and that the conduction velocity over the anteromedial aspect of the atrium is faster than over the posterior lateral aspect as indicated by the difference in the slopes of the electrograms. A total of the 256 potentials, consisting of 252 bipolar electrograms, lead II ECG, right atrial reference electrogram, His bundle ECG, and the right ventricular reference ECG, were recorded simultaneously. The bipolar data were recorded at a frequency response of 50–1000 Hz. A computer program was used to determine local activation times from the bipolar tracing. At the end of each study, the dog was euthanized, the heart removed, rinsed with saline, and fixed in 10% formalin. The minimum re-entrant path lengths were determined from the dimensions between the fixed electrode site exhibiting
the lowest successive activation times per unit distance. Typically, these were the sites closest to either the anatomic and/or functional obstacles about which the wavefronts rotated.

Results

Electrophysiologic Data

In 5 of 10 dogs, AFB was induced immediately following the shunt procedure, but terminated spontaneously within a few seconds. Forty to 80 days postoperatively, sustained AFB was induced in 5 dogs and sustained AFL in 1 dog. Left atrial size was also determined by transesophageal echocardiography to look for the development of left atrial enlargement during this study. Dogs with sustained atrial arrhythmias had a larger left atrial diameter than did those dogs with non-sustained atrial arrhythmias. Five months were needed for the animals to recuperate from surgery and develop chronic volume overload and pressure overload for the left atrium. Five months post shunt, 79 episodes of AFL and AFB were induced. All but 1 animal had inducible sustained arrhythmias. Seventeen sustained different tachycardias were induced in 9 animals (Table 1). There were 9 examples of AFL in 6 dogs. AFB was induced in 2 animals. Four animals demonstrated focal tachycardias. Based on the activation sequence maps, the tachycardias were classified as follows. AFL was divided into 3 different varieties: class I consisted of those in which the wavefront circulated about a region of functional block only and orifices did not act to extend the re-entrant circuit (Figs 4 and 5); class II consisted of those in which the circuit wavefront moved about a single orifice only; class III consisted of those examples of AFL in which either a single orifice was combined with a region of functional block to form a somewhat longer re-entrant circuit, or in which 2 orifices were conjoined or linked by an interpolated area of functional block to create an even longer re-entrant path (Figs 6 and 7). Class IV consisted of the focal tachycardias (Fig 8) and AFB is indicated as class V. We defined functional block (function barrier of conduction block) as that decremental conduction leading to propagation and/or differences in conduction properties.

Also indicated in Table 1 is the pattern of contralateral atrial conduction. In the 12 tachycardias caused either by circus movement or by focal origin, contralateral atrial activation was passive and conducted in a 1:1 mode (mode A). In 3 of the tachycardias, 2 caused by circus movement and 1 focal, all in the left atrium, there was contralateral right atrial dissociation (mode B). This typically was characterized by a regularly repeating depolarization cycle in the left atrium and non-uniform conduction to the right atrium varying in local regions.
Fig. 6. Clockwise circus movement re-entry around anatomic obstacles (orifices). Note that the re-entrant pathway encompasses a broad 2-dimensional surface in both anteromedial and posterolateral aspects of the right atrium. Note that the pathway is bound superiorly by an obstacle composed of the orifices of both the SVC and right atrial appendage, which are linked by functional block. The pathway is bound below by a combination of the tricuspid and IVC orifices. Note the longer cycle length of 135 msec as compared with the previous examples of right atrial flutter about obstacles of functional block, and that, in this and other forms of right atrial flutter, the atrium is passively activated via the anterior interatrial band contiguous with the superior atrial septum.

Fig. 7. Counterclockwise circus movement around anatomic obstacles (orifices). Note that, as in the previous example, the principal axis of rotation is about the combined orifices of the SVC and right atrial appendage linked by functional block above and the tricuspid and inferior caval orifices below. However, in this example, the motion is in the opposite direction or counterclockwise.

from 1:1 to higher left to right conduction ratios. Table 2 lists the class, location, cycle length, re-entrant path length, and conduction velocity for the different types of AFL observed in this study.

There were 7 examples of right AFL in 6 dogs and 2 examples of left AFL in 2 different animals (1 of these also demonstrated a form of right AFL). The most frequently recorded form of AFL involved the combined orifices of the superior vena cava (SVC) and right atrial appendage linked by an interposed region of functional block that prevented impulses from traversing this isthmus of tissue (class III, Fig. 6). These examples were associated with the longest re-entrant path lengths.

Several observations can be made from Table 2. First, AFL associated with circus movement about anatomic orifices was generally associated with longer path lengths and cycle lengths. In addition, where the 2 orifices of the SVC and the base of the right atrial appendage were conjoined by an intervening region of functional block, the longest path lengths and cycle lengths were observed. There was also a correlation between path length and conduction velocity: increasing length was associated with an increasing velocity of propagation of the circus wavefront. AFL primarily caused by functional block in which orifices played either no role or secondary roles was associated with shorter path lengths and cycle lengths.

There were 6 different examples of focal tachycardias in 4 dogs (Fig. 8). Two of these were located in the right septum, 1 in proximity to the coronary sinus, and the other adjacent to the fossa ovalis. Other examples of focal tachycardia were: 1 at the junction between the posterior right atrium and the inferior vena cava; 1 in the anterior right atrial appendage; 1 in the left atrial appendage; and 1 located in the lateral left atrium midway between the appendage and left inferior pulmonary veins. There were 2 examples of AFB in 2 animals.

Attral Flutter (Class IIIA) — Right Atrial Circus Movement Around Orifice(s) and Functional Block, 1:1 Left Atrial Conduction

Fig 6 demonstrates an example of the most frequent form of AFL encountered in this series. Note that the re-entrant pathway incorporates the orifices of both the SVC and right atrial appendage, which were linked by an interpositioned region of functional block that prohibited wavefronts from crossing this isthmus. This longer effective re-entrant pathway involved broad 2-dimensional aspects of both the posterolateral and anteromedial right atrium. Additionally, the right atrial septum above the fossa ovalis formed a small portion of the anteromedial pathway. Note that as a result of the longer re-entrant pathway involving 2 orifices, the cycle length of the tachycardia was 135 msec. The direction of the circus wavefront was clockwise. Fig 7 illustrates another example of AFL caused by re-entry involving both the SVC and right atrial appendage orifices. In contrast to the previous example, however, the rotation of the wavefront was in the opposite direction, counterclockwise in this example, clockwise in the previous example. With the exception of the narrow isthmus of activation present in the posterolateral aspect, most of the re-entrant pathway
Fig 8. Focal tachycardia. Four examples of focal tachycardias initiated with either programmed stimulation or rapid atrial pacing are shown. (A) The tachycardia wavefront originates focally from a site at the junction between the inferior vena cava and the body of the right atrium. (B) The wavefront originates from the inferior right atrial septum adjacent to coronary sinus ostium. (C) The focus is located in the lateral posterior left atrium and (D) at the tip of the left atrial appendage anteriorly. Note that although all wavefronts begin focally, they originate from the edges of the atria (see text).

Table 2

<table>
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<tr>
<th>Dog No</th>
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<th>CL (msec)</th>
<th>PL (cm)</th>
<th>CV (m/sec)</th>
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Focal Tachycardia

1
2
3
4
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6
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SVCO, orifice of superior vena cava; RAAO, orifice of right atrial appendage; PVO, orifice of pulmonary veins; RA, right atrium; LA, left atrium; —denotes functional block either as the principal reentrant obstacle in an atrium (ie, —RA) or in apposition to an orifice (ie, SVCO—-) or in linking two orifices into one large obstacle (ie, SVCO—RAAO).

Atrial Flutter (Class II-B) — Left Atrial Circus Movement Around Orifices With Complex Dissociation of Conduction in the Contralateral Right Atrium

In contrast to all forms of right AFL with 1:1 left atrial conduction, the examples of rapid left atrial tachycardia as a result of short cycle length circus movement or focal origin demonstrated a complex form of passive right atrial dissociation. That this contralateral dissociation was completely passive and not caused by separate self-sustaining or reinitiating re-entrant circuits in the right atrium could be documented by demonstrating its dependence on the repeating, reinitiating event in the left atrium. Fig 9 demonstrates 2 cycles of left atrial re-entry tachycardia in which the flutter wave circles about the confluent orifices of the left inferior pulmonary veins. Activation maps were made for 2 time windows corresponding to successive left atrial re-entrant cycles occurring from the middle of a sustained run of tachycardia. Both maps are referenced to the 2 successive left atrial re-entrant cycles. The wavefronts are marked coded to correspond to the specific re-entrant cycle (and source) from which they originate, stippled wavefronts from the first re-entrant cycle and cross-hatched wavefronts from the prior cycle (not shown). This assists in following wavefronts that originate in the first re-entrant cycle in the left atrium but persist in the right atrium after the beginning of the second re-entrant cycle. Note that the re-entrant cycle length is 115 msec. In cycle 1, the anteromedial (septal) aspect of the right atrium was activated passively from the left atrial re-entrant circuit, with earliest activity developing in the right septum adjacent to the coronary sinus. This wavefront propagated throughout the right septum and inferior-anteromedial right atrium to the tricuspid valve annulus, but terminated along a boundary of functional block beginning at the posterior SVC and extending anteriorly to the tricuspid valve annulus. This same wavefront
moved inferiorly between the IVC and tricuspid annulus on the anteromedial wall to gain access to the posterior — inferior right atrium at 30 msec. Subsequently, the posterior right atrial wavefront spread superiority, activating the SVC between 50 and 70 msec, and the tip of the right atrial appendix at 100 msec. This same wavefront, upon reaching the crest of the right atrium at the SVC-RA junction, propagated on to the anterosuperior right atrium and collided with the same boundary of functional block in the mid-anterosuperior right atrium above the septum. This boundary was maintained in a constant state of repolarization owing to the phase differences between the early and late components of the passively depolarizing right atrial wavefront. The same wavefront also blocked along an extended boundary in the posterolateral right atrium and atrial appendage. This boundary of block resulted from a delayed portion of the right atrial wavefront originating from the preceding left atrial re-entrant cross-hatched area at 10—50 msec in site D). In the second cycle (116—230 msec), the pattern of left atrial activation was unchanged. However, activation of the right atrium was quite different from the pattern demonstrated in the previous cycle. Earliest activation reached the right atrium adjacent to the coronary sinus and septum as before. This wavefront propagated anterolaterally and activated a small portion of the tricuspid annulus. However, the wavefront blocked both in its inferior and superior directions. Note that the stippled wavefront at 116 msec in the anterior edge of the right atrial appendage represents activation from the preceding cycle. This same wavefront continued on to the posterolateral surface of the right atrium, activating it between 135 and 175 msec (site D) Note that the infero-posterior aspect of the right atrium is activated twice by this wavefront (stippled), which originated with the first cycle and continued into the second. However, the postero-lateral surface of the right atrium and appendage were activated only once by this wavefront, as this area was unexcitable during the first cycle owing to prior activation of the area by a wavefront from the immediately preceding re-entrant cycle (cross-hatched). This initial activation and subsequent reactivation by the same wavefront represents a form of partial or limited re-entry of the right atrium. Thus, the wavefront, which originated from the left atrial re-entrant circuit and activated a majority of the right atrium in the first cycle (stippled), did not completely terminate during that cycle and continued through a partial re-entrant loop to re-excite a limited portion of the posterior right atrium between 185 and 230 msec in the second cycle. However, this return wavefront did not complete the entire circuit in the right atrium during the second cycle or generate a third cycle, and thus represents an incomplete or abortive form of re-entry. Between 116 and 230 msec of the second cycle, the large areas of the anterior and posterior upper right atrium, which appear blank in the maps, were not activated during this time window. Therefore, wavefronts were not represented in these regions for this interval of time.

Electrograms recorded from 5 different locations in both the left and right atrium during this arrhythmia are also shown. The locations of these electrograms correspond to the sites indicated on the maps. Note the repeatedly consistent left atrial cycle length indicated by the electrogram recorded from the left atrial appendage (site A). Also, note the corresponding consistency of the electrogram recorded from site B in the right atrial septum. By contrast, note the increasing, then decreasing, cycle lengths in several of the right atrial electrograms resembling a Wenckebach periodicity. Thus, although there was a 1:1 correspondence between the left atrial re-entrant circuit and the right septum (115 msec), there was a 7:6 or 7:5 conduction ratio between these 2 sites and other portions of the right atrium (140 msec). This right atrial dissociation was entirely passive, and a consequence of the right atrial delay in which wavefronts continued to propagate in the right atrium long after completion of their re-entrant origin in the left atrium. Because of the marked delay in the right atrial activation, with wavefronts lasting longer than the left atrial re-entrant cycle time, the right atrial wavefronts were repeatedly shifted into subsequent left atrial re-entrant cycles. The result was that during any left atrial cycle there were always wavefronts from the current and preceding cycles.
present in the right atrium during the same interval.

Discussion

The Canine Model

The advantage of the subclavian artery to pulmonary vein shunt is slowly developing left atrial enlargement. By avoiding direct pulmonary recirculation, there is a considerably lower incidence of acute left heart failure and pulmonary edema. In addition, and more relevant to this study, there is a high yield of inducible, sustained, and reproducible atrial arrhythmias. The latter occurring in this substrate would be expected to resemble, both clinically and in their mechanism, atrial arrhythmias occurring in patients as a result of atrial overload syndrome. The model does not necessitate the creation of atrial incisions, which can themselves be substrates for re-entrant atrial arrhythmias. Disadvantages of this model are that there were no spontaneous atrial arrhythmias, and that a 5-month period is required for the atrium to reach a state permitting repeatedly inducible sustained arrhythmias.

Types of Atrial Flutter

Previously, electrode systems used to record from the atrium were limited by either too few electrodes, inconsistent location of electrodes, or inconsistent contact between electrodes, and different areas of complex geometry. The system of electrodes for use in these studies solves some, but not all, of these problems. Because the electrode forms were constructed from molds made from actual atria, there was good contact and definition between each recording site and each anatomical region, including the appendage, A-V annuli, irregular surfaces, and specific regions of interest such as the crista, limbus, fossa, and coronary sinus. Thus, the relative importance of the inflow and outflow orifices as obstacles, the major rapid conduction pathways such as the crista terminalis, precaval and intercaval bands, tricuspid annulus, coronary sinus, and atrial septum and their relative roles in contributing to the circus motion re-entry could be evaluated. The findings indicate that both atrial orifices and functional block are important and essential factors in AFL. The venous inflow orifices, particularly the SVC and the right atrial appendage orifice, together formed an important obstacle that was involved in combination with a contiguous region of functional block that effectively lengthened the re-entrant pathway, or more frequently was linked by functional block to the right atrial appendage orifice, resulting in a long circumferential path length and a stable sustained arrhythmia. These forms of AFL of long path length were associated with the most rapid conduction velocities.

Although inflow orifices and functional block were documented as independent principal factors, most often these 2 factors were interactive. Functional block was most consistently observed in the upper posterolateral right atrium at its junction with the SVC. Thus, there was frequent opportunity for contiguity or apposition between an anatomic and functional obstacle at this site. Previous studies have demonstrated a gradient of atrial repolarization progressing from left atrium to right atrium, and from medial to lateral, and inferiorly to superiorly in the right atrium, with longest recovery times in association with the upper SVC — right atrial junction. Thus, this association represents a commonly encountered substrate and perhaps explains the increased frequency of right AFL caused by circus movement involving this region as compared with the IVC and left atrial structures.

Focal Tachycardia

Tachycardias of focal origin described in this study were unexpected and were not seen in previous studies of AFL. It seems likely that these arrhythmias also represent some form of re-entry in which the circus pathway could not be properly defined owing either to the location of the circuit or to the resolution of the maps. In each case, the arrhythmias were initiated by programmed or rapid atrial stimulation. Although this does not rule out triggered activity, the cycle lengths were similar to those associated obviously with re-entrant circus movement about obstacles of functional block. Additionally, the sites, although focal, were almost always near the atrial margins, where electrodes were either sparse, or located adjacent to an anatomic edge, in which the tissue folded on itself or joined orifice (Fig. 8). Although a competing focal tachycardia at the identical cycle length and simultaneously initiated by programmed stimulation could not be ruled out, logic suggests otherwise. As these tachycardias were initiated by programmed stimulation or rapid pacing and had cycle lengths similar to, or shorter than, the circus movement variety, we presume that the focality is the result of a change to a more convoluted 3-dimensional re-entrant circuit, which could not be resolved accurately by the mapping techniques limited to the endocardial surfaces.

Contralateral Atrial Dissociation

Previous studies have reported dissimilar rhythms in the right and left atria. There have been prior observations based on recordings from small numbers of atrial electrodes suggesting different rhythms or arrhythmias in the 2 atria. The observations obtained in this study indicate that such rhythms could be due to uniform AFL in 1 atrium with passive dissociation in the contralateral chamber. Specifically, short cycle length tachycardias in the left atrium did not propagate uniformly throughout the right atrium at equivalent cycle lengths. As indicated in Table 1, there were 3 examples of contralateral right atrial dissociation owing to short cycle length tachycardias originating in the left atrium, 2 owing to re-entry and 1 focal, and probably also the result of a re-entrant mechanism. In the examples shown in Fig. 9, wavefronts from current and prior left atrial re-entrant cycles coexisted in the right atrium simultaneously. Thus, the right atrial dissociation was characterized by some areas conducting 1:1, other areas conducting in a progressiveneckebach-like delay and ending in unidirectional block with activation from another or opposite direction by either a subsequent left atrial cycle or by the same right atrial wavefront arriving after a considerable delay over a longer pathway. The right atrial wavefronts could be followed in the map from their entry via the inferoseptal-coronary sinus pathway to their extinguishment. The right atrial wavefronts were extinguished by several different mechanisms. First, they were extinguished by arriving at a terminal boundary, having completed the pathway from the entry site to the right atrial border or tricuspid annulus. Second, they were extinguished between sites of entry and termination by
merger or collision with simultaneous wavefronts from other directions. Third, they were extinguished between entry and termination sites by blocking on a boundary of refractory myocardium. In addition, there was no evidence in the maps of re-initiated right atrial activation or the formation of a complete re-entrant pathway leading to either sustained or non-sustained repetition, independent of the left atrial process.

Mechanisms of Right Atrial Delay
The question of why the right atrial activation was prolonged can be answered in part from the maps. After entry into the right septum, the wavefronts had to traverse the additional length of the right atrial pathways beyond the left atrial re-entrant circuit. The lengths of the right atrial pathways exhibiting the greatest delay were considerably longer than the longest left atrial pathways outside the re-entrant circuit. The complex patterns of dissociation in the right atrium cannot be accounted for from the data in this study alone. However, from previous studies, it is likely that the longer repolarization time of the right atrium as compared with the left, as well as the inhomogeneity of repolarization in the right atrium, were important factors. Several studies have demonstrated the shortest effective refractory periods in the left atrium and the longest in the right atrium. Therefore, the left atrium is more able to sustain short cycle and path length re-entry with uniform circus motion activation than the right atrium, which may respond with dissociation. It is of some interest that it is this same long effective refractory period distribution that contributes to both the longer cycle length right AFL and the right atrial dissociation with fast left AFL.

Differences Between Atrial Flutter and Fibrillation
As the multiple wavelet hypothesis was originally proposed for AFL, the concept has been interpreted to mean chaotic activity consistent with multiple changing re-entrant circuits. However, Moc and Moe et al. stated that multiple wavefronts could also result from a single rapidly re-exciting re-entrant circuit. Inspection of the electrograms in Fig 9 would ordinarily suggest AFL and, based on previous concepts, one might assume multiple changing re-entrant circuits. However, in an example, a single stable re-entrant circuit produced these patterns. It is likely that AFL involves multiple separate re-entrant circuits. However, the present study demonstrates that, although multiple simultaneous wavelets may be present, they may originate from a single circuit. This situation develops if the propagation time of waveforms departing the re-entrant circuits is longer than the re-entrant cycle time. Thus, wavefronts from prior cycles can be propagating distally at the same time new ones are being formed by the next re-entrant cycle. Ideker et al first demonstrated this phenomenon in activation maps obtained during ventricular fibrillation in dogs. In the present study, the greatest disparity between re-entrant cycle time and the duration of passive depolarization was due to the long circuitous pathways in the right atrium resulting from unidirectional functional block. However, functional block was not the only factor causing a disparity between the durations of re-entry and passive depolarization.

To distinguish between the 2 arrhythmias, we have defined AFL as a re-entrant process confined to a single circuit, and AFL as 2 or more independent re-entrant circuits. Based on these definitions, we have referred to the complex forms of re-entry described in this report as AFL with passive contralateral atrial dissociation. How can these arrhythmias be differentiated from AFL? They may not be distinguishable electrocardiographically, and may only be distinguishable by the kind of activation mapping described here. The ECG pattern of ventricular response may or may not be useful. Although the right septal surface was activated 1:1 from the left atrial circuit in an example illustrated in Fig 9, A-V conduction differed. In the example (Fig 9), there was a progressive Wenckebach-like delay in the R-R interval over 5 cycles with the interval increasing from 211 to 227 msec. This corresponded to an approximate 2:1 conduction ratio. Thus, in the presence of apparently chaotic atrial activation, either a regular or a progressively increasing R-R interval would suggest a flutter-like regular repetition of atrial activation involving the right septum (Fig 9). However, the ventricular response could be quite variable as it is highly dependent upon the flutter cycle length, the interval, and regularity of the wavefront in the septal input pathway interacting with the subject’s particular A-V nodal conduction characteristics and refractory period. Extending these observations to atrial fibrillation, it might be anticipated that the complex irregular cycle lengths seen over the atra are due to both: (a) some small number of independent re-entrant circuits; and (b) multiple areas of passive dissociation such as those shown in this study. Following this logic, certain questions arise. (1) How much of the complexity seen in atrial fibrillation is active or due to multiple re-entrant circuits and how much is due to complex passive atrial dissociaion? (2) How often is an arrhythmia thought to represent atrial fibrillation clinically, actually a form of fast flutter with dissociation? These questions are relevant to the control of these arrhythmias as there may be relatively few active re-entrant circuits and many more regions of passive dissociation, rather than the opposite situation.

References