The Mechanism of Canine Atrial Flutter

By William G. Hayden, A.B., Edward J. Hurley, M.D., and David A. Rytand, M.D.

ABSTRACT

In anesthetized dogs, electrograms were recorded simultaneously from six atrial epicardial sites and a limb or esophageal lead in order to study the mechanism of atrial flutter induced electrically after an intercaval crush in comparison with atrial tachysystole induced by aconitine. In the former, with limb lead records resembling classical human flutter in form and regularity, activation occupied most of the atrial cycle and progressed in sequence caudally in the right atrium and cranially in the left (counterclockwise flutter); flutter was usually terminated by another crush extending from the initial one to the right atrioventricular junction. The most prominent wave in limb and esophageal leads coincided with left atrial activity. Sometimes activation proceeded clockwise. The two atrial appendages may be activated simultaneously during counterclockwise flutter. These findings support the circus movement hypothesis as the mechanism of pure atrial flutter. In contrast, with aconitine tachysystole, activation proceeded over both atria at once and was so brief that it left most of the slightly irregular atrial cycle electrically silent. Although limb leads after aconitine sometimes resembled those of flutter, the contrast in direction and duration of excitation demonstrates profound physiological differences between the two arrhythmias.

ADDITIONAL KEY WORDS

aconitine atrial tachysystole atrial electrograms experimental flutter

Despite a number of studies, the mechanism of canine atrial flutter remains in doubt. Some workers find evidence for circus movement (1-7); others do not (8-11). The results of this study in the dog strongly support the circus movement hypothesis as the mechanism of pure atrial flutter. In contrast, with aconitine tachysystole, activation proceeded over both atria at once and was so brief that it left most of the slightly irregular atrial cycle electrically silent. Although limb leads after aconitine sometimes resembled those of flutter, the contrast in direction and duration of excitation demonstrates profound physiological differences between the two arrhythmias.

Methods

Acute experiments were performed in 43 mongrel dogs, 16 to 35 kg in weight. Anesthesia was provided by the intravenous administration of pentobarbital sodium (60 mg/kg). Respirations were controlled by a positive pressure ventilator through a cuffed endotracheal tube. A right thoracotomy was performed and the heart fixed in a steady anatomical state by suturing the pericardium to the wound edges. Complete atrioventricular block was produced by crushing the atrioventricular (A-V) node by a clamp introduced through the tip of the atrial appendage or through the stump of the divided azygos vein to minimize or avoid injury to the body of the atria. Bipolar electrodes were placed on three epicardial sites on the right and three on the left atrium. Access to the body of the left atrium was gained by dissection in the interatrial groove and resection of pericardial reflection from the right pulmonary veins. The bipolar electrodes were formed of fine wire, 0.5 mm in diameter with fixed 2-mm interelectrode distances. Recordings of atrial electrical activity were made simultaneously with either aVF or bipolar limb lead II on an eight channel direct-writing pen recorder. In some experiments, activity also was recorded by means of unipolar electrodes at various levels in the esophagus.

From the Departments of Surgery (Cardiovascular Division) and Medicine, Stanford University School of Medicine, Palo Alto, California 94304.

This work was supported by Grants from the Santa Clara County Heart Association and the American Heart Association.

Dr. Hurley is an Established Investigator of the American Heart Association.

Accepted for publication March 23, 1967.
Atrial arrhythmias were induced by low voltage, faradic stimulation (square wave, 8 volts, 0.02 sec duration, 40 cycle/sec) to the body of the right atrium in 29 animals subsequent to the intercaval crush method of Rosenblueth and Garcia Ramos (2), and in 12 by the injection of aconitine, 0.05 ml of a 0.05% solution, into the atrial myocardium. Figure 1 illustrates the site of intercaval crush and the sites of atrial electrode implantation together with the symbols used for the latter in subsequent Figures. Finally, high speed (500 to 3,000 frame/sec) cinematographic studies were made of mechanical contractions of the right atrium during sinus rhythm, atrial flutter, and aconitine tachysystole to compare electrical events with mechanical contractions of the atria.

Results

One animal had flutter prior to the onset of the thoracotomy and a spontaneous flutter began in a second animal following minimal mechanical stimulation of the right atrial appendage. In 27 of 29 animals prepared by the intercaval crush method of Rosenblueth-Garcia Ramos, the poststimulatory arrhythmia induced was atrial flutter. With an incomplete intercaval crush, electrical stimulation frequently produced an arrhythmia that was lasting, but too coarse and disorganized to be diagnosed as atrial flutter and was regarded as fibrillation. Only after complete intercaval crush did the atrial arrhythmia assume the regular, organized pattern characteristic of

![Diagram of atrial bodies and appendages, with site of intercaval crush. LA', etc., indicate sites of bipolar electrode implantation; these same symbols are used in subsequent Figures.](image)

![Simultaneously recorded electrograms from 8 atrial sites and lead aVF during sinus rhythm after induction of complete A-V block. Minimal atrial activation duration, 0.025 sec; cycle length, 0.31 sec. "Minimal atrial activation duration" in this and subsequent legends refers to the period between onsets of activation at the earliest and latest sites. In flutter, this measurement is shorter than the cycle duration to an extent which is determined by interelectrode distances. In the original records of this and subsequent Figures, the fine vertical grid lines were 1 mm apart.](image)
Simultaneously recorded electrograms from 6 atrial sites and lead aVF during counterclockwise flutter and complete A-V block. Minimal atrial activation duration, 0.12 sec; cycle length, 0.148 sec. Activation proceeds caudally in the right atrium, cranially in the left. Note coincidence of left atrial activation with prominently inverted wave of lead aVF.

In the 2 dogs with spontaneous flutter and in 16 of the 29 dogs with intercaval crush, the arrhythmia as reflected in lead II or aVF resembled the common classic variety of atrial flutter in man. In these, direct atrial electrograms revealed the wave pathway progressing cephalad in the left atrium and caudad in the right atrium. This is identical to the findings in man with comparable electrocardiographic patterns in lead II or aVF, with the passage of excitation wave summarized elsewhere (13) as being counterclockwise in the sagittal, frontal, and horizontal planes when these are viewed respectively from the left, front, and above. In 11 of these 16 animals, the atrial rates were 430 to 540/min, with a mean of 480. In the remaining 5 animals, with marked atrial dilatation as a result of tricuspid insufficiency subsequent to disruption of the tricuspid valve, the counterclockwise atrial flutter rate varied from 285 to 395/min, with a mean of 350. Figure 2 shows the spread of excitation during normal sinus rhythm; Figure 3 presents the typical results in counterclockwise flutter in our experimental preparation.

In 9 dogs, optical inversion of lead II or aVF was required to make either resemble the common type of classical human atrial flutter. In these, the direction of atrial excitation was also reversed 180°, being clockwise in the sagittal plane when viewed from the left (caudad in the left atrium and cephalad in the right); this is exactly opposite to the excitation pattern in the first group described above with counterclockwise activity. Figure 4 shows a typical example of clockwise flutter. Atrial rates in these animals with clockwise flutter were 385 to 535/min, with a mean of 465.

In 8 dogs, flutter in either direction could be induced by electrical shock, subsequently stopped by electrical stimulation, and then instituted in the opposite direction by a third stimulus. With reversal of the direction, both the atrial electrograms and waves in the limb lead electrocardiogram assumed a 180° shift.
Simultaneously recorded electrograms from 6 atrial sites and lead II during clockwise flutter and complete A-V block. Minimal atrial activation duration, 0.12 sec; cycle length, 0.152 sec. Activation proceeds caudally in the left atrium, cranially in the right. Note coincidence of left atrial activation with prominent upright wave of lead II.

In 7 of 11 attempts, the atrial flutter halted abruptly and sinus rhythm reappeared upon extension of the intercaval crush to the right atrioventricular junction, representing a positive Mines' test for the circulating excitation pattern (1). In the remaining 4 dogs, flutter was terminated by extension of the intercaval crush to the A-V groove in 3, but atrial fibrillation ensued. One may but speculate upon the possible inadequacy of the crush in the single failure to terminate flutter.

During counterclockwise flutter in 3 dogs, unipolar electrograms from the esophagus were identical in form to those found in man with classical flutter; that is, they were negative in the more caudal sites, positive in the more cranial ones, and biphasic in the intermediate positions (Fig. 5). Such waves
Simultaneously recorded electrograms from 6 atrial sites and lead aVF in dog with complete A-V block, showing arrival of activation at right and left atrial appendages (RAA and LAA, respectively). During sinus rhythm (left segment), activation of RAA precedes that of LAA by 0.04 sec; during counterclockwise flutter (central segment), activation of RAA and LAA are virtually synchronous; during clockwise flutter (right segment), activation of RAA precedes that of LAA by 0.056 sec (or follows by 0.116 sec). Only during clockwise flutter does activation of RAA follow that of RA1.

from the esophagus were synchronous with left atrial activation as detected by simultaneously obtained direct atrial leads. During clockwise flutter in 3 dogs, esophageal electrograms resembled those of uncommon flutter in man; they were negative in the cranial sites and positive in the more caudal ones. As with counterclockwise flutter, they were synchronous with left atrial excitation and did not reflect activation of the right atrium.

High speed motion pictures confirmed the direction of the wave of activation in the right atrium in counterclockwise and clockwise flutter. Technical difficulties prevented success in attempts made to photograph the atrial appendages simultaneously. However, it was found in one experiment that the two atrial appendages were activated simultaneously during counterclockwise flutter, despite the demonstration of asynchrony of electrical activity in the atrial bodies. The two atrial appendages were not activated simultaneously.
FIGURE 7
Simultaneously recorded electrograms from 6 atrial sites and lead aVF during a later phase of atrial tachysystole induced by injection of aconitine near site RA3 in a dog with complete A-V block. Atrial activation duration, 0.028 sec; atrial cycle length about 0.16 sec. Note irregular atrial rhythm, especially during central portion of segment recorded at 25 mm/sec and associated with altered wave form in aVF.

In the one experiment in which pericaval electrograms demonstrated clockwise progression. Figure 6 illustrates these results.

In 12 experiments after aconitine injection, the pattern of activation was entirely different from those just described in classic Rosenblueth-Garcia Ramos or spontaneously occurring flutter. In contrast to the latter, aconitine-induced atrial tachysystole was unstable, often irregular in rhythm, and largely unaffected by further electrical stimulation. Following the application of aconitine, a gradual takeover or dominance by the ectopic site at the injection region occurred. In one instance the increases in rate were episodic, as repetitive sequels to atrial premature beats which in turn immediately followed ventricular excitation. When the site of application of aconitine was at the caudal end of the right atrium, near the inferior vena cava, there was reversal in polarity of activity recorded in the direct atrial leads as the wave of depolarization spread cephalad simultaneously in both atria rather than in the customary caudad direction. There occasionally occurred a brief transitory period of aconitine tachysystole during which the ectopic site fired at a rate sufficiently rapid to produce a rather narrow but flutter-like wave in the limb lead, but the epicardial wave pattern was quite unstable or irregular (Fig. 7). In these studies the excitation wave spread in a single direction over both atria virtually simultaneously, with demonstrable electrical activation occurring in only 10 to 15% of the atrial cycle and the remainder being electrically silent (Fig. 7). In general, aconitine-induced arrhythmias were completely disorganized, resembling fibrillation.

In 1 dog, a narrow transverse crush of the
right atrial myocardium, just cephalad to an aconitine focus in the caudal part of that atrium, resulted in the following modification. From the focus, the wave progressed cephalad in the left atrium, then caudad in the right until it was halted by the transatrial crush. Although 0.13 sec of the cycle seemed electrically silent prior to the crush, the cycle length increased from 0.160 to 0.176 sec before to 0.21 sec after the crush as the duration of excitation rose from 0.03 to 0.06 sec.

High speed motion pictures of the atria following aconitine injection revealed disorganized, aperiodic contraction waves.

**Discussion**

A detailed review of experimental atrial flutter in relation to flutter in man and to the circus movement hypothesis has been presented elsewhere (14). Certain important points, however, deserve emphasis here.

A major difficulty has been that of the definition of flutter. Many laboratory investigators continue to use the word in the old and nonspecific sense of MacWilliam as a term for his visual observation of regular contractions, at a rapid rate, appearing to spread over canine atria from a point of stimulation (15). On the other hand, most clinicians now regard flutter as the specific electrocardiogram pattern first reported as such by Jolly and Ritchie (16) and best described by Lewis (12):

**The auricular complexes in electrocardiograms.**—The beating of the auricles usually displays itself characteristically in these curves. At each cycle in leads II and III the curve ascends sharply to a blunt summit and returns more gradually. . . . The gentle downsweep is often notched. In lead I the complexes are usually diminutive. . . . Another important feature to which I have drawn attention is that the complexes are contiguous: the string is moving constantly and rests for no measurable period on a baseline; as soon as one complex is complete the next starts, and this action is continued even throughout ventricular systole. . . . Each complex is a duplicate of the last in form, though this is disguised when ventricular complexes fall with them; the length of the complexes is wonderfully uniform. . . . Another striking feature is that the forms of the auricular complexes in curves taken from different patients usually present a curious and often remarkable resemblance. . . .

In man, practical difficulties in diagnosis continue to arise, especially at slow atrial rates when the "gentle downsweep" of Lewis may superficially resemble an isoelectric line (17). In the dog with open chest and right atrium exposed, leads II, III, or aVF reflect events especially of the posterior, left atrium even more than they do in intact man, with consequent loss of some of the classical appearance of flutter owing to diminished contributions from the right atrium. Kato et al. called attention to this, and to a certain extent were able to restore the right atrial contribution by instilling saline into the pericardial sac to imitate the normal volume conductor relationship (4). It should not be surprising, then, that the limb lead records of dogs with Rosenblueth-Garcia Ramos flutter are not exactly like those of human flutter or that they may resemble those of aconitine tachysystole. The latter differentiation proved to be difficult at times in our experiments despite the prior induction of complete A-V block, produced to allow optimum examination of atrial activity.

In man, activation of the atria in flutter progresses from site to site throughout most or all of the cycle (14). Our studies, confirming previous ones (3, 4, 7), indicate that this occurs also in canine flutter of the Rosenblueth-Garcia Ramos type. We submit that this finding, so different from observations in sinus rhythm or in atrial tachysystole induced by either aconitine or electrical stimulus (3, 4, 7, 18), constitutes a physiological definition of atrial flutter; we further suggest that aconitine and other tachysystoles in which atrial activation is brief should not be designated as flutter. Even when a crushing injury after aconitine prolonged atrial excitation somewhat by redirecting the wave's path, activation remained but a brief part of the cycle length.

In some of our dogs with counterclockwise (13) flutter, extremity leads bore a close resemblance to those of human flutter of the classical type (e.g., aVF in Fig. 3). With clockwise flutter, the appearance of extremity leads was inverted, as it rarely is in man. In both types of experiments, the lengthy duration of atrial activation would seem to
EXPERIMENTAL ATRIAL FLUTTER

preclude any designations of a beginning or an end of the cycle. Without records from the atrial epicardium, especially lacking those from the right atrium, it is easy to see how one might erroneously regard the "P" waves as representing those of nodal rhythm (counterclockwise flutter) or of tachycardia from a high atrial focus (clockwise flutter). As noted by others (5), esophageal leads were of no great assistance in this regard; they, like extremity leads, yielded clear evidence of left but not right atrial activation. Of course, records from multiple levels in the esophagus did show direction of excitation in the left atrium.

The appearance of many of our records suggests that the velocity of the wave front may have been greater in the left atrium than in the right; some of this could be artefactual, inasmuch as the left atrial electrodes were more closely placed (Fig. 1) for technical reasons. However, measurements in 1 dog did show that the velocity was greater on the left both during sinus rhythm (1.95 m/sec left, 0.65 m/sec right) and flutter (1.3 m/sec left, 0.6 m/sec right counterclockwise; 2.2 m/sec left, 0.46 m/sec right clockwise). Takayasu et al. (7) also remarked on greater velocity on the left in flutter. Calculations based on measurements from the illustrations published by Kimura et al. (3) indicate that the velocity is slower on the right in both counterclockwise and clockwise flutter (right velocity 37% and 51% of left, their Figs. 4b and 5b, respectively); the reverse, however, was true in another instance of clockwise flutter (left velocity 69% of right, their Fig. 6). In aconitine tachysystole, velocities were equal and greater than in flutter (their Fig. 3b). It has been shown for man that right, but not left, atrial dilatation is associated with slower than usual flutter rates (14).

Fujiwara also observed occasionally that the atrial cycle duration in aconitine tachysystole may lengthen after ligations designed to interrupt specialized conduction tracts in the atria (18). He more consistently induced block between the atria by such methods, and confirmed Scherf’s earlier observations that aconitine rarely caused fibrillation when injected at a distance from the sinus node; he implicated special pathways for aconitine tachysystole.

In the absence of more direct observations, one may but speculate upon an analogy between the mode of onset of aconitine-induced atrial tachysystole as described above on the one hand, and reexcitation of the atrium (echo phenomenon) as observed even with complete A-V block in dogs during vagal stimulation (20) on the other.

Of great interest is the confirmation (Fig. 6) of the findings by Kimura et al. (3) and in part by Takayasu et al. (7) that the two atrial appendages are excited simultaneously in counterclockwise, but not in clockwise, flutter. Prinzmetal et al. (19) demonstrated simultaneous contraction of the two appendages by means of high speed cinematography in man with counterclockwise flutter of classic appearance. They concluded that such a demonstration was “entirely incompatible with the circus movement theory,” but the experimental results just cited seem to negate such a conclusion.

Finally, we succeeded in halting flutter in 10 of 11 attempts by a crush from the intercaval one to the right atrioventricular junction, in full confirmation of three other such studies (2, 3, 7); as Mines wrote, this represents “the best test for a circulating excitation” (14).

Conclusions

In 2 animals with spontaneous atrial flutter and in 16 with flutter induced by the technique of Rosenblueth and García Ramos, all with records of atrial activity unmasked by complete A-V block, limb leads resembled the electrocardiograms of man with atrial flutter as described by Lewis. In such experiments, direct epicardial atrial electrograms revealed the path of activation to be directed cranially in the left atrium, then caudally in the right, and so on in contiguous cycles (counterclockwise flutter). Left atrial activation, recorded both directly by epicardial bipolar leads and indirectly from esophageal electrodes, coincided with the prominently inverted waves of limb lead II or aVF.
In 9 comparable animals, excitation proceeded in the opposite direction (clockwise flutter), whereupon left atrial activation was synchronous with a prominent upward wave in lead II or aVF. These limb lead tracings resembled those of classical flutter in man only upon their inversion optically. Flutter in both directions, counterclockwise and clockwise, was recorded in 8 dogs. In all such experiments, activation occurred somewhere in the atria throughout almost the entire atrial flutter cycle. This appears to represent an important criterion toward a more physiological definition of flutter.

Extension of a crushing injury from the intercaval region to the right atrioventricular junction terminated flutter promptly in 10 of 11 trials.

In 12 animals with atrial tachysystole induced by aconitine, activation progressed away from the site of injection over both atria simultaneously and briefly; most of the cycle was electrically silent. In limb leads, corresponding atrial waves were often more narrow than in Rosenblueth-Garcia Ramos flutter and at times were irregular in rhythm. In one experiment, a crushing injury caused the excitation wave to reach the atria in sequence; duration of excitation remained brief but lengthened somewhat, and the atrial cycle also increased.

The atrial appendages may be activated synchronously even though epicardial leads simultaneously demonstrate counterclockwise flutter.

The results confirm the presence of a circulating excitation (circus movement) about an obstacle in canine atrial flutter of the Rosenblueth-Garcia Ramos type. The findings after aconitine application are compatible with an ectopic focus mechanism for a paroxysmal atrial tachysystole. The two arrhythmias are physiologically different.

Acknowledgment

We are grateful to William M. Palmer and Steven Kohl for surgical assistance in some of the reported experiments.

References

18. Fujikawa, M.: Experimental study on auricular flutter and fibrillation induced by aconitine.

Circulation Research, Vol. XX, May 1967
EXPERIMENTAL ATRIAL FLUTTER


The Mechanism of Canine Atrial Flutter
WILLIAM G. HAYDEN, EDWARD J. HURLEY and DAVID A. RYTAND

Circ Res. 1967;20:496-505
doi: 10.1161/01.RES.20.5.496

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1967 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circres.ahajournals.org/content/20/5/496

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in
Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further information
about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/