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A New Animal Model of Atrial Flutter

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A new, simple and reliable model of atrial flutter utilizing postpericardiotomy pericarditis was developed in the dog. Using a sterile technique, the pericardium was opened by way of a right thoracotomy, Teflon-coated, stainless steel wire electrodes were fixed to three selected sites on the atria and exteriorized, the atrial surfaces were generously dusted with talcum powder and a single layer of gauze was placed on the free left and right atrial walls. The dogs were allowed to recover. Subsequently, the inducibility of atrial flutter and selected electrophysiologic properties of the atria were determined by daily programmed atrial stimulation studies with the dogs in the conscious, nonsedated state.

Atrial flutter could be induced in 23 of 25 dogs initially studied. It was sustained (that is, lasting ≥5 min) in 17 of the 23. Neither atrial excitability, intraatrial conduction time nor atrial refractoriness determined by pacing and recording from the three fixed sites predicted the inducibility of atrial flutter. One hundred thirty-nine episodes of atrial flutter induced in these 23 dogs were analyzed. The mean sustained atrial flutter cycle length was 131 ± 20 ms (mean ± SD) (range 100 to 170); the atrial flutter cycle length was 150 ms or more in 23 episodes, between 120 and 150 ms in 64 episodes and 120 ms or less in 52 episodes.

In five dogs, the stability of the atrial flutter cycle length during sustained atrial flutter was studied and shown to be remarkably stable in all five until interrupted by rapid atrial pacing 35 to 95 minutes after its induction. Seventeen dogs were submitted to reoperation for epicardial mapping purposes and atrial flutter could be induced in the open chest state in 12. In conclusion, this sterile pericarditis model of atrial flutter in the canine heart proved to be highly reliable, reproducible and easy to create.

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Atrial flutter was first described more than 7 decades ago (1). Despite numerous experimental and clinical studies since that time (2–14), many aspects of its nature and mechanism remain in doubt. There have been many efforts to study the nature and mechanism of atrial flutter using canine models (1–14). In the first models (10–12), the atrial flutter was quite transient. Subsequent models were largely the result either of creating atrial lesions of one sort or another (for example, intercaval crush) (2–9) or of applying substances (for example, delphinine, aconitine or acetylcholine) to the atria (11–14), making their relevance to clinical atrial flutter uncertain.

Because of the conspicuously frequent occurrence of spontaneous atrial flutter in patients early after open heart surgery (15–18), we postulated that diffuse sterile pericarditis and associated atrial inflammation might cause an increased susceptibility to atrial flutter. We further postulated that if a model of atrial flutter based on creation of sterile pericarditis could be created in the canine heart, it would provide an atrial flutter model closer to that found in the clinical setting. We therefore developed and studied this new model of atrial flutter based on creation of sterile pericarditis in the canine heart. The model proved to be reliable, reproducible and easy to create. In this study, we describe the technique used to create the model and characterize several aspects of the induced atrial flutter.

Methods

Model preparation. Sterile pericarditis was created in 25 adult mongrel dogs weighing 16 to 35 kg. With sterile technique, the dogs were submitted to a right thoracotomy

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under general anesthesia (pentothal, 15 mg/kg body weight intravenously, supplemented with halothane). After a pericardiotomy, the heart was cradled in the pericardium, and three pairs of stainless steel wire electrodes coated with FEP polymer except for the tip (0 Flexon, Davis and Geck) were sutured to selected atrial sites (16–18). One pair was placed high on the sulcus terminalis of the right atrium, one pair on the interatrial band, also known as Bachmann's bundle and one pair on the posteroinferior aspect of the left atrium near the coronary sinus. These electrodes were brought out through the chest wall and exteriorized posteriorly in the neck near the midline. The atrial surfaces were then generously dusted with sterile talcum powder, a single layer of gauze was put on the right and left atrial free walls and the pericardiotomy was repaired. The chest was then closed in standard fashion. Antibiotic and analgesic agents (Innovar) were administered, and the dogs were allowed to recover. Postoperative care included administration of antibiotics and analgesics as needed. All procedures for animal care and experimentation were in accordance with the guidelines provided by this institution.

Electrophysiologic Studies in the Conscious State

After surgery, electrophysiologic studies were performed daily with the dogs lying quietly in the conscious, nonsedated state. Electrocardiographic leads I, II and III were recorded simultaneously with bipolar atrial electrograms from each of the fixed electrodes. The electrocardiograms were recorded between a band pass of 0.1 and 500 Hz and the electrograms between 12 and 500 Hz. All data were displayed on an Electronics for Medicine DR-12 switched beam oscilloscope, and were recorded on photographic paper at 100 mm/s. The data were also recorded simultaneously on FM tape using a Honeywell 5600C tape recorder for later playback and analysis. All pacing studies were performed using a modified Medtronic 5325 programmable stimulator with a pulse width of 1.8 ms. The following data were obtained during each study:

1. Excitability. At the onset of each study, the threshold of excitability was determined for each site at a pacing rate of 150 beats/min.

2. Conduction time. Using suprathreshold stimuli (20 mA) to assure atrial capture at rapid rates and to permit comparison of conduction times at all rates (19), pacing was instituted in turn at each electrode site at a rate of 200 beats/min. After stable atrial capture was demonstrated, the atrial pacing rate was increased by increments of 10 to 50 beats/min until 2:1 atrial capture was achieved. For each pacing rate at each electrode site, conduction time to each of the other two recording sites was determined by measuring the interval from the stimulus artifact to the first rapid component of the dominant deflection of the atrial electrogram at each of the other two sites.

3. Atrial effective refractory period. This was determined at each of the three electrode sites using stimuli of twice threshold strength. At each site in turn, after a train of eight atrial paced beats (S_1) introduced at a rate of 150 beats/min, a premature atrial beat (S_2) was introduced at coupling intervals decremented by 5 to 10 ms to scan the entire atrial diastolic interval. After the determination of the effective refractory period for a single premature atrial beat, the S_1S_2 interval was fixed at an interval 25 ms greater than the effective refractory period, and a second premature atrial beat was introduced at twice diastolic threshold. Atrial refractoriness of the second premature atrial beat was then determined in the manner just described.

4. Precipitation of atrial flutter. Two methods were used to precipitate atrial flutter: 1) introduction of one or two premature atrial beats after a train of eight paced atrial beats, or 2) rapid atrial pacing for periods of 10 to 30 seconds at rates incrementally faster (by 10 to 50 beats/min) than the spontaneous sinus rate, until either atrial flutter or atrial fibrillation was precipitated, or loss of 1:1 atrial capture was achieved. Thus, the protocol to determine atrial refractoriness and conduction time described earlier also served to induce and permit the characterization of atrial flutter.

Once atrial flutter was precipitated, its rate, regularity and duration were characterized. Also, during each episode of atrial flutter, the configuration of the atrial electrograms recorded from the fixed electrodes and their relative activation sequence were characterized. Only atrial flutter episodes lasting longer than 1 minute were analyzed. We defined sustained atrial flutter as an episode lasting longer than 5 minutes and nonsustained atrial flutter as an episode lasting longer than 1 minute but shorter than 5 minutes. The number of episodes of atrial flutter was tabulated. If atrial flutter was repeatedly induced with a given pacing method, no more than three episodes per induction method were tabulated on a single day to compare inducibility from a standard protocol.

When atrial flutter was sustained, rapid atrial pacing was used to interrupt the flutter (15,18). The pacing was initiated at a rate 10 beats/min faster than the atrial rate and continued at that rate for 10 to 30 seconds. If atrial flutter persisted after cessation of pacing, the pacing rate was then increased by 5 to 10 beats/min increments until finally the atrial flutter was interrupted.

The dogs were then classified into two groups: Group I included the dogs in which three or more episodes of atrial flutter could be induced within the first 4 postoperative days. Group II included the dogs with fewer than three induced episodes. These two groups were compared statistically for each variable separately and by the multivariate Cox regression test. The statistical analysis was performed with the BMDP-PV2 statistical package. The variables studied were 1) threshold of excitability at each pacing site, 2) the atrial effective refractory period at each pacing site, 3) the con-

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duction time from the pacing site to each of the other two electrode sites at different pacing rates, and 4) the weight of each animal.

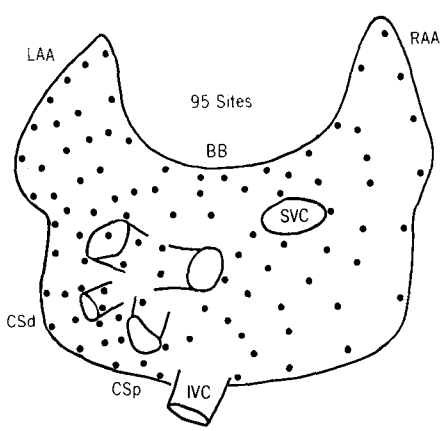
5. Time course of induced sustained atrial flutter. In five conscious dogs in which sustained atrial flutter was induced, atrial flutter was allowed to last for 30 to 90 minutes. The time course of atrial flutter (that is, the configuration of the atrial electrograms, their relative activation sequence, the atrial flutter cycle length and ventricular rate) was noted.

Studies in the Open Chest State

Seventeen of the 25 dogs initially studied underwent reoperation between postoperative days 2 and 10. The dogs were reanesthetized with pentobarbital (30 mg/kg body weight intravenously), and the chest was opened through a median sternotomy. The parietal pericardium was gently peeled from the adherent epicardium, and a pericardial cradle was again created. The previously placed stainless steel wire electrodes were kept in place for stimulation and recording purposes. For additional exposure of the atrial wall, the inferior pulmonary veins were divided between ligatures. The pericardial reflection was also incised in order to expose the posterior aspect of the left atrium from the sinus of Marshall. Fourteen dogs were reoperated on before the fifth postoperative day to avoid the dense pericardial adhesions encountered with late reoperation and also because of the high inducibility of atrial flutter during the first 4 postoperative days.

Complete epicardial atrial mapping was performed during sinus rhythm to establish the normal sequence of atrial activation in this model. Guided by an anatomic grid, a hand-held, tripolar electrode probe with a 1 mm interelectrode distance was used for sequential recording of 95 predetermined epicardial atrial sites (Fig. 1). Electrograms recorded from the probe and the three pairs of fixed atrial wire electrodes were displayed simultaneously with electrocardiographic lead II using the recording system described earlier. The activation time for each of the 95 sites was determined by measuring the interval between a fixed reference electrogram, usually the electrogram recorded at the Bachmann's bundle site, and the local electrogram recorded from the electrode probe. These activation times were then plotted on the anatomic grid, and 10 ms isochronic lines were manually traced on the grid to display the sequence of atrial activation during each rhythm. Then induction of atrial flutter was attempted as described.

Figure 1. Anatomic grid for atrial epicardial mapping. The recording sites are represented by the 95 **dots** spread on the atrial epicardial surface. BB = Bachmann's bundle or interatrial band; CSd = distal coronary sinus; CSp = proximal coronary sinus; IVC = inferior vena cava; LAA = left atrial appendage; RAA = right atrial appendage; SVC = superior vena cava.



Data analysis. All of the time intervals were measured using a Talos digitizer system from paper records. To obtain the cycle length of atrial flutter, all intervals occurring during a 1 second period were measured and then averaged. All results are shown as mean ± 1 SD. Statistical analysis other than that described earlier was performed using the unpaired Student's *t* test.

Results

Studies in the Conscious (Closed Chest) State

In 23 (92%) of the 25 dogs initially studied, atrial flutter was induced in the conscious, nonsedated state on at least 1 day within the first 4 postoperative days. Sustained atrial flutter was induced in 17 of these 23 dogs, but only nonsustained atrial flutter was induced in the remaining 6. More than one type of atrial flutter (that is, atrial flutter with different flutter cycle lengths or different recorded atrial electrogram configurations, or both) was induced in 13 dogs. We analyzed 139 episodes of atrial flutter induced during the first 4 postoperative days in these 23 dogs.

1. Characteristics of atrial flutter. Of the 139 episodes of atrial flutter, 78 were a sustained rhythm (lasting more than 5 minutes) and 61 were a nonsustained rhythm (lasting more than 1 minute but less than 5 minutes). The mean atrial flutter cycle length was 131 ± 20 ms (range 100 to 170) for sustained episodes, 122 ± 17 ms (range 95 to 160) for nonsustained episodes and 127 ± 19 ms for all episodes. The mean atrial flutter cycle length of the sustained episodes was significantly longer than that of the nonsustained episodes ($p < 0.005$).

Because the overall range of atrial rates during atrial flutter was quite broad, all atrial flutter episodes were classified into three categories (modified from Boyden and Hoffman [20]) according to the atrial rate: 23 episodes were classified as slow (≤400 beats/min), 64 as intermediate (401 to 500 beats/min) and 52 as fast (>500 beats/min). An example of each type of atrial flutter is presented in Figures 2A, B and C, respectively. The polarity of the atrial flutter

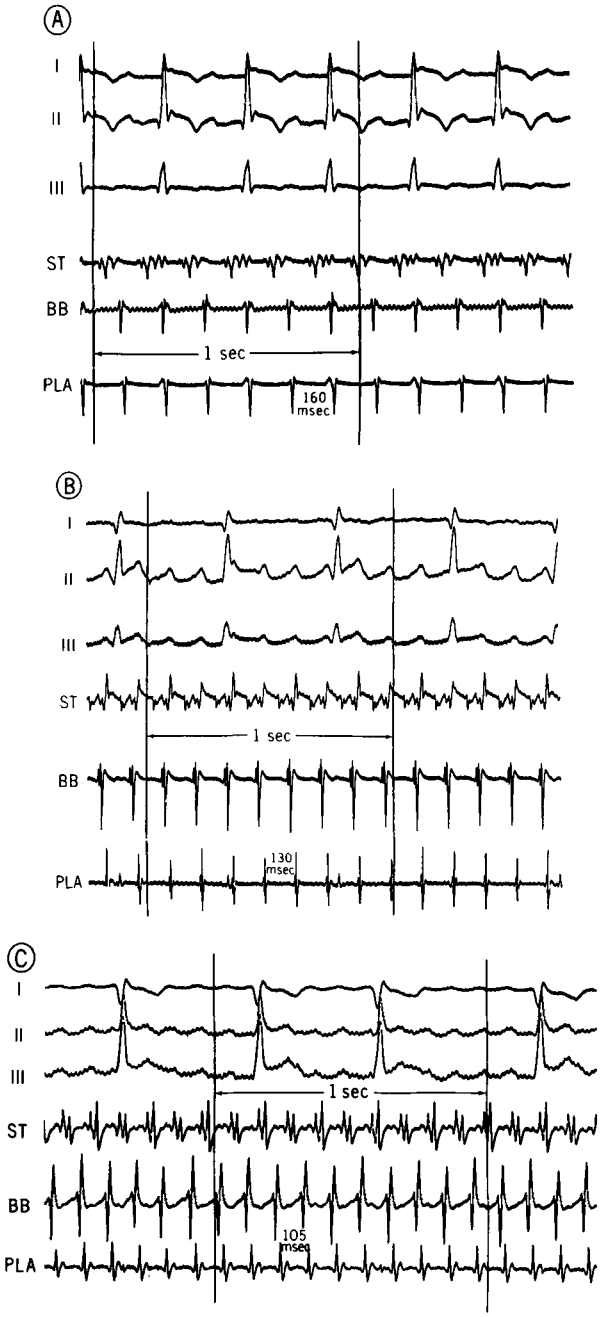


Figure 2. Examples of atrial flutter induced in the conscious, nonsedated state. **A**, Slow type. **B**, Intermediate type. **C**, Fast type. Electrocardiographic leads I, II and III are recorded simultaneously with bipolar electrograms at the sulcus terminalis (ST), Bachmann's bundle (BB) and posteroinferior left atrium (PLA). The flutter cycle length is indicated in each panel.

wave (atrial complex) identified in surface lead II was positive in 94 episodes (68%) (20 of slow type, 41 of intermediate type and 33 of fast type), was flat or slightly positive in 39 (28%) and was negative in 6 (4%). Regardless of type, all episodes of atrial flutter were characterized by a remarkably constant beat to beat cycle length, configuration and polarity of the recorded bipolar atrial electrograms (Fig. 2) (17,18). In all but three dogs, the electrograms recorded from the sulcus terminalis site during atrial flutter were markedly abnormal (Fig. 2). These abnormal electrograms either resembled fractionated signals or showed double potentials. During sinus rhythm, the signal recorded from this site was always normal (Fig. 3).

2. Mode of induction. The site and technique of atrial stimulation associated with the induction of atrial flutter were assessed (Table 1). Nineteen (14%) of the 139 atrial flutter episodes were precipitated by the extrastimulus technique and 120 (86%) by the rapid pacing technique. The posteroinferior left atrium was the induction site for 69 episodes, the Bachmann's bundle was the site for 45 episodes and the sulcus terminalis was the site for 25 episodes. The extrastimulus technique never induced atrial flutter from the sulcus terminalis site. When atrial flutter was induced with extrastimuli from left atrial sites, it was either a slow or an intermediate type. When rapid pacing was used, the induced episodes were more likely to be of the intermediate or fast type. However, because the incidence of atrial flutter induction was greatest with rapid pacing, the slow type of atrial flutter was induced more often by rapid pacing (16 of 23 episodes) than with the extrastimulus method. The intermediate type of atrial flutter was usually provoked by rapid pacing from the posteroinferior left atrial site. The rapid pacing rate that precipitated atrial flutter ranged between 450 and 600 beats/min. Overall, the best method for induction of atrial flutter in this model was rapid pacing from the Bachmann's bundle site or posteroinferior left atrial site. Of note, regardless of the pacing method used, a brief period of atrial fibrillation preceded the onset of atrial flutter in most instances (Fig. 4).

3. Time course of inducibility. Figure 5 indicates the proportion of dogs in which atrial flutter could be induced on each postoperative day. The incidence ranged from 56

Figure 3. Electrocardiographic lead II recorded simultaneously with bipolar electrograms recorded at the sulcus terminalis (ST), Bachmann's bundle (BB) and posteroinferior left atrium (PLA). Note that during sinus rhythm (**A**), the electrogram at the sulcus terminalis site is normal whereas during atrial flutter (**B**), it is abnormal (fractionated).

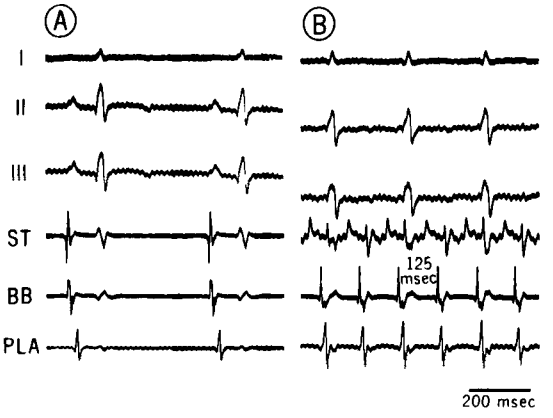


Table 1. Mode of Induction of Atrial Flutter

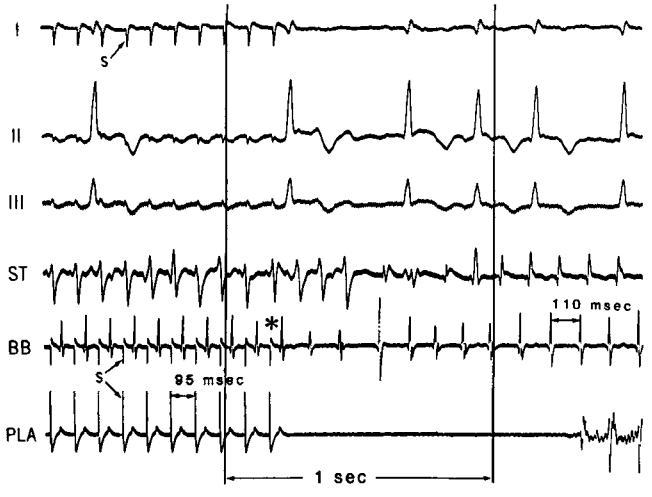
Flutter Type	No. of Episodes	Extrastimuli				Rapid Pacing			
		ST	BB	PLA	All Sites	ST	BB	PLA	All Sites
Slow	23	0	2	5	7	4	5	7	16
Intermediate	64	0	6	6	12	9	12	31	52
Fast	52	0	0	0	0	12	20	20	52
Total	139	0	8	11	19	25	37	58	120

BB = Bachmann's bundle or interatrial band recording/pacing site; PLA = posteroinferior left atrial recording/pacing site; ST = sulcus terminalis recording/pacing site.

to 71% for the first 4 days. It fell below 50% after the fourth postoperative day. However, the difference in the number of episodes induced before and after the fourth postoperative day, both per day and per dog, did not reach statistical signficance (Table 2). The same type of atrial flutter was inducible on more than 1 consecutive day in 14 dogs.

4. Pacing during atrial flutter. Rapid atrial pacing (stepwise incremental continuous pacing for 10 to 30 seconds) was attempted 6 minutes after initiation of atrial flutter. All episodes could be interrupted by rapid pacing, with either sinus rhythm or transient atrial fibrillation present immediately after cessation of pacing. Sinus rhythm typically was present after the cessation of rapid pacing of the slower types of atrial flutter, but after cessation of rapid pacing of the fast type, a brief period of atrial fibrillation was present before sinus rhythm returned.

Figure 4. Electrocardiographic leads I, II and III recorded simultaneously with bipolar electrograms recorded at the sulcus terminalis (ST), Bachmann's bundle (BB) and posteroinferior left atrium (PLA) during initiation of atrial flutter. Rapid atrial pacing at 630 beats/min was performed at the posteroinferior left atrial site. The **asterisk** represents the last stimulus (s) in the Bachmann's bundle recording. Note that with abrupt termination of atrial pacing, an irregular atrial rhythm of eight spontaneous atrial beats appeared before atrial flutter with the typically constant beat to beat atrial electrogram amplitude, polarity, configuration and cycle length.



5. Correlation of electrophysiologic data with incidence of atrial flutter inducibility. There were 18 dogs in Group I (three or more episodes of atrial flutter induced during the first 4 postoperative days) and 7 dogs in Group II (none or fewer than three episodes of atrial flutter induced). A stepwise logistic regression analysis found no statistically signficant difference between Group I and Group II dogs for the four variables studied (Table 3).

The pacing threshold increased from 1.9 ± 0.3 to 3.5 ± 2 mA from day 1 to day 4, but there was no correlation with atrial flutter inducibility. Furthermore, this change may be entirely due to the characteristics of the stainless steel wire electrodes (18,21).

The atrial effective refractory period ranged from 119 to 215 ms. There was no statistically significant difference between various atrial sites or between the postoperative day on which the atrial effective refractory period was determined. Also, the inducibility of atrial flutter did not correlate with any of these aspects of atrial refractoriness.

The intraatrial conduction time measured during rapid pacing from the sulcus terminalis site at two different pacing rates is shown in Figure 6. There was no statistically significant difference in conduction times between Group I and Group II dogs at either rate and no correlation with inducibility of atrial flutter.

6. Time course of induced atrial flutter (studies in five

Figure 5. Susceptibility to atrial flutter in the conscious, nonsedated state. The proportion of dogs in which atrial flutter was induced is shown for each postoperative day. See text for discussion.

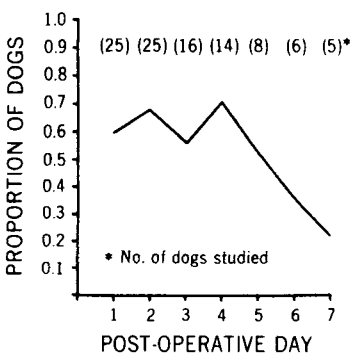


Table 2. Incidence of Induced Atrial Flutter in 25 Dogs

	Days 1 to 4	Days 4 to 10	p Value*
EPI/dog	5.5 ± 4.3	2.7 ± 3.6	0.09 (NS)
EPI/day	3.2 ± 0.9	3.6 ± 3.0	0.71 (NS)
EPI/day per dog	1.9 ± 1.6	0.8 ± 1.3	0.08 (NS)

*Nonpaired Student's *t* test. EPI = mean number of atrial flutter episodes induced during the study period; NS = not significant.

dogs with sustained atrial flutter). In five dogs, sustained atrial flutter was not interrupted by rapid atrial pacing for a period of at least 35 minutes (36 minutes in three dogs and 70 and 90 minutes, respectively in the remaining two dogs) in order to characterize its time course. The mean atrial flutter cycle length was 136 ± 29 ms (range 103 to 169). Figure 7 shows one example of the time course of induced atrial flutter. The configuration of all atrial electrograms, their relative activation sequence, the atrial flutter cycle length and ventricular rate were remarkably stable throughout the 90 minute period in this dog. The atrial flutter induced in the other four dogs demonstrated similar characteristics. The coefficient of variation of the artial flutter cycle length was 0.7 to 2.7% (mean 1.6) for all five dogs. Thus, sustained atrial flutter induced in the present model was found to be remarkably stable over time, just as was its clinical counterpart (17,18).

Studies in the Open Chest State

Reoperation was performed in 17 of the original 25 dogs in this series between postoperative day 2 and 5. Induction of sustained atrial flutter was attempted in all 17 dogs, but could be induced in only 12. A detailed sequence of atrial activation mapping during sustained atrial flutter will be reported separately in another series of dogs with pericarditis and inducible atrial flutter.

Of the 17 dogs in this series that underwent reoperation, epicardial activation maps during sinus rhythm were performed in 9: 7 with previously inducible atrial flutter and 2 with no previously inducible atrial flutter. The total atrial activation time during sinus rhythm did not differ in these two groups of dogs: 54 ± 6 versus 53 ± 4 ms, respectively. Patterns of activation found (Fig. 8) confirmed previous descriptions of the normal sequence of atrial activation during sinus rhythm (7,22). Neither fractionated electrograms

Table 3. Comparison of Variables of Atrial Flutter in Groups I and II*

		Group I (n = 18)		Group II (n = 7)	
Variable	Day	Mean ± SD	n	Mean ± SD	n
Threshold (mA) ST site	1	1.9 ± 1.1	18	1.9 ± 0.9	7
	2	1.8 ± 0.6	18	1.6 ± 0.3	7
	3	3.9 ± 2.5	11	3.9 ± 2.9	4
	4	4.3 ± 2.4	10	3.8 ± 0.4	2
ERP (ms) ST site	1	140 ± 18	18	140 ± 19	6
	2	162 ± 29	18	157 ± 27	7
	3	150 ± 26	11	181 ± 29	4
	4	150 ± 32	10	155 ± 16	2
BB site	1	123 ± 18	18	131 ± 27	7
	2	136 ± 31	18	132 ± 33	7
	3	122 ± 43	11	148 ± 41	4
	4	151 ± 42	10	172 ± 21	2
PLA site	1	119 ± 14	18	114 ± 17	7
	2	126 ± 17	18	122 ± 6.2	7
	3	135 ± 25	11	131 ± 35	4
	4	151 ± 42	10	171 ± 21	2
Weight (kg)		20 ± 4	18	20 ± 5	7
CT (Day 1) (ms) at CL 200 ms		40.6 ± 11.2	18	37.4 ± 13	7
(ST-PLA) (ms) at CL 450 ms		42.8 ± 12	18	39.5 ± 13.4	7

*Group I = preparations in which three or more episodes of atrial flutter were induced. Group II = preparations in which fewer than three episodes of atrial flutter were induced. No variables were significant as a contributing factor (p = 0.05) from the multivariate analysis. BB, ST, PLA = pacing and recording sites at Bachmann's bundle, sulcus terminalis and posteroinferior left atrium, respectively; CL = cycle length of rapid atrial pacing; CT = conduction time from the sulcus terminalis to the posteroinferior left atrium when pacing from the sulcus terminalis. ERP = effective refractory period; n = number of dogs.

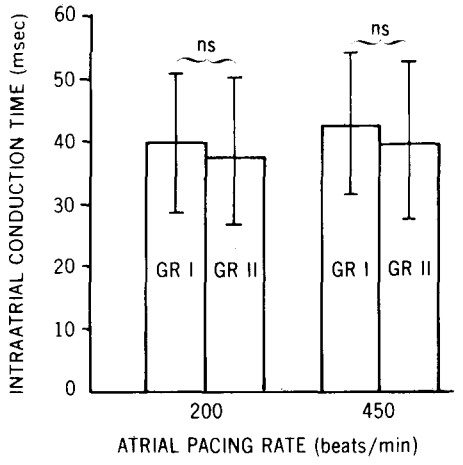


Figure 6. Intraatrial conduction time measured from the stimulus artifact to the electrogram at the posteroinferior left atrium during rapid pacing from the sulcus terminalis site at two pacing rates (200 and 450 beats/min). There is no statistically significant difference between the two groups of dogs at either of the two pacing rates. GR I = Group I (≥3 episodes of flutter in first 4 postoperative days); GR II = Group II (≤3 episodes of flutter).

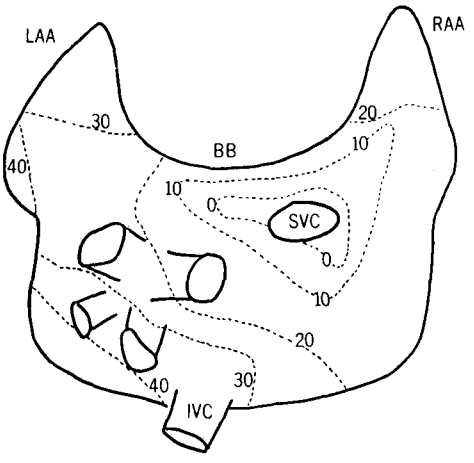


Figure 8. A representative example of an activation map during sinus rhythm. The **isochronic lines** are drawn at 10 ms intervals. The excitation started in the sinus node area high in the right atrium. Abbreviations as in Figure 1. See text for discussion.

nor electrograms showing double potentials were demonstrated during sinus rhythm, although bipolar electrograms recorded from sites where talcum powder was still adherent to the atrial epicardium usually showed low amplitude potentials.

Discussion

Characteristics of atrial flutter in the present animal model. This study demonstrates that it is possible to induce sustained atrial flutter in otherwise healthy dogs within 4 days of the creation of sterile pericarditis. Furthermore, this preparation readily permits the investigation of atrial flutter both during the conscious state (closed chest) and under general anesthesia (open chest).

This model is noteworthy not only for its ease of creation, but also for its resemblance to a clinical counterpart. In fact, in several respects it closely resembles atrial flutter in patients, particularly that observed after open heart surgery (17,18): 1) the beat to beat regularity of the atrial cycle length; 2) constant configuration and polarity of recorded atrial electrograms; 3) easy interruption with rapid atrial pacing; and 4) time course after surgically producing sterile pericarditis.

Additional observations on the nature and mechanism of atrial flutter in this pericarditis model will be presented in subsequent reports. However, several comments about this model should be offered on the basis of data from the present study. First, it is clear that active pericarditis is critical to the ability to initiate and sustain atrial flutter. In fact, this observation supports the notion that sterile pericarditis is an important factor in the relatively high incidence of spontaneous atrial flutter that occurs in adult patients soon after open heart surgery regardless of the type of operation or underlying heart disease (16,18). Second, although no relation between the effective refractory period at the three selected atrial sites and the inducibility of atrial flutter was found, this may not reflect a true picture. Clearly, systematic pacing from very few sites was performed, and others (7) have found that inducibility of atrial flutter and, to a certain

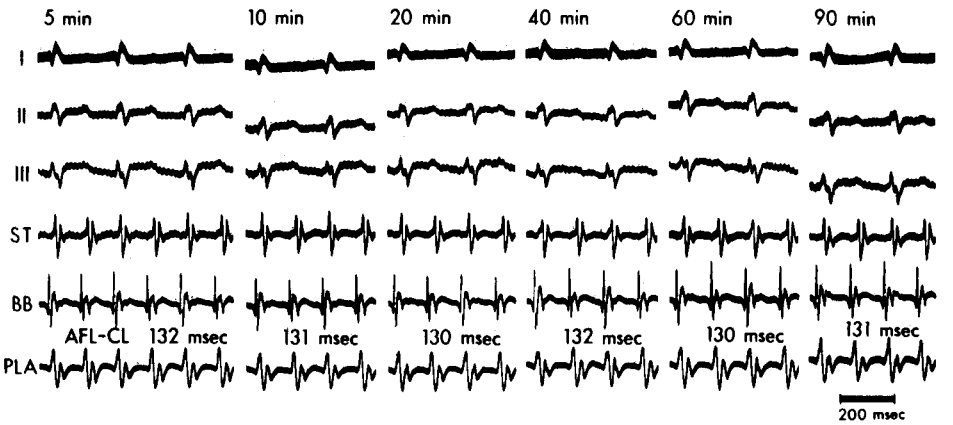


Figure 7. Electrocardiographic leads I, II and III recorded simultaneously with bipolar electrograms at the sulcus terminalis (ST), Bachmann's bundle (BB) and posteroinferior left atrium (PLA) at 5, 10, 20, 40, 60 and 90 minutes after the initiation of atrial flutter. Note the remarkably stable time course of atrial flutter. AFL-CL = atrial flutter cycle length.

extent, the rate of atrial flutter are determined by the inhomogeneity of atrial refractoriness. Third, during atrial flutter, fractionated electrograms and double potentials were recorded at some atrial sites but were not recorded from the same sites during sinus rhythm. Although it is not the purpose of the present report to discuss the exact significance of this finding, it could reflect local reentry (23,24), collision of wave fronts in the center of a reentry circuit (14) or localized fibrillation in this part of the atrium with atrial flutter in the remainder of the atria (25). These phenomena observed in our model need further investigation. Finally, the separation of atrial flutter into three types by rate criteria was simply for the convenience of analysis. There is no evidence, at least from this study, that the different rates of atrial flutter seen in this model reflect anything other than a continuum of atrial flutter due to one mechanism.

Comparison with other models of atrial flutter. Much earlier in this century, Sir Thomas Lewis and coworkers (10) were limited in their studies of atrial flutter by the unstable nature of the arrhythmia provoked by rapid pacing. To obtain a stable form of atrial flutter, subsequent experimental models utilized localized destruction of atrial tissue (cutting or clamping) or application of substances (for example, delphinine, aconitine or acetylcholine) to the atrial myocardium (2–14), making their resemblance to a clinical situation unlikely. The more recently described technique (20,26) that utilizes pulmonary banding and surgical induction of tricuspid insufficiency is another promising method for studying atrial arrhythmias (atrial flutter and fibrillation) and seems much closer to the clinical state than previously described models. An advantage of this latter model is that the time course of inducibility of atrial flutter does not appear limited. Several weeks (up to 3 months) of long-term care of the dogs, however, are needed before the atrial flutter becomes inducible. Thus, the ease in creation of our model of atrial flutter, its similarity to a clinical counterpart and its reliability and reproducibility compare most favorably with previous models.

Conclusions. This pericarditis model of atrial flutter in the canine heart is reliable, reproducible and easy to create. It can be used to study atrial flutter in the closed or open chest state. All these characteristics, along with its similarities to a clinical counterpart, indicate that this model should be most useful for the study of atrial flutter in any of its aspects.

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