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EXPERIMENTAL STUDIES

Single Site Radiofrequency Catheter Ablation of Atrial Fibrillation: Studies Guided by Simultaneous Multisite Mapping in the Canine Sterile Pericarditis Model

Koichiro Kumagai, MD, Kikuya Uno, MD, Celeen Khrestian, BS, Albert L. Waldo, MD, FACC
Cleveland, Ohio

OBJECTIVES To test the hypothesis that when activation of Bachmann's bundle (BB) is critical to the unstable reentrant circuits that maintain atrial fibrillation (AF) in the sterile pericarditis canine model, a lesion in BB would prevent induction of stable AF.

BACKGROUND One mechanism of induced AF in this model is multiple unstable reentrant circuits, which frequently include BB as part of the reentrant pathway.

METHODS Simultaneous multisite mapping studies during AF and after ablation of BB were performed by recording (384 to 396 electrodes) from both atria and the atrial septum during six induced AF episodes in six dogs with sterile pericarditis. Activation maps of AF (mean duration, 24 ± 28 min) during 12 consecutive 100-ms windows were analyzed.

RESULTS During AF, multiple unstable reentrant circuits (mean, 1.2 ± 0.2 per window; range, 1 to 4) were observed, 68% involving BB. Nonactivation zones (mean duration, 57 ± 16 ms in the right atrium and 53 ± 23 ms in the left atrium) observed during AF were reactivated by a wave front most often coming from the atrial septum via BB (right atrium, 62%; left atrium, 67%). After successful radiofrequency catheter ablation of the midportion of BB, AF >5 s was not induced in all dogs. Mapping studies of transient AF (≤5 s) induced after ablation showed neither reentrant circuits nor wave fronts activating the right atrium via BB.

CONCLUSIONS In this AF model, catheter ablation of BB terminates and prevents the induction of AF by preventing 1) formation of unstable reentrant circuits that involve BB, and 2) activation of the atrial-free walls after a nonactivation period. (J Am Coll Cardiol 2000;36:917–23) © 2000 by the American College of Cardiology

Most previous surgical (1) and catheter ablation techniques (2–6) to prevent or cure atrial fibrillation (AF) have largely been based on the multiple reentrant wavelet hypothesis (7–10), and have required placement of numerous and extensive lesions. Several studies in both patients and animal models suggested that a limited but effective catheter ablation procedure to prevent or cure AF was possible (11–15).

We have recently shown in the sterile pericarditis model that one mechanism of AF is due to one-to-four circulating unstable reentrant circuits of very short cycle length (16). Based on the importance of Bachmann's bundle (BB) as an apparent critical crossroad for the majority of unstable reentrant circuits, we hypothesized that a carefully placed lesion in BB could interrupt or prevent most of the unstable reentrant circuits that generate AF in this model. If that is true, such a lesion should interrupt sustained AF and prevent its subsequent induction. Therefore, in the present study, simultaneous multisite mapping was performed during sustained AF and after epicardial radiofrequency catheter ablation of BB to test the hypotheses that in the canine sterile pericarditis AF model due to multiple unstable reentrant circuits, 1) activation of BB is critical for maintenance of AF and 2) catheter ablation of the midportion BB prevents the induction of AF by preventing formation of the unstable reentrant circuits that otherwise would involve BB, and activation of the atrial-free walls by wave fronts via BB.

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METHODS

All studies were performed in accordance with guidelines specified by our Institutional Animal Care and Use Committee, the American Heart Association Policy on Research Animal Use, and the Public Health Service Policy on Use of Laboratory Animals.

Creation of the sterile pericarditis model. The canine sterile pericarditis model was created as previously described (17) in six adult mongrel dogs weighing 20 to 30 kg. At the time of surgery, pairs of stainless steel wire electrodes coated with FEP polymer (Teflon) except at the tip were sutured on the right atrial appendage in two dogs, Bachmann's bundle in all dogs, and the posterior-inferior left atrium close to the proximal portion of the coronary sinus in all dogs. Another pair was also sutured on the right ventricular apex to be used for ventricular pacing after His bundle

Abbreviations and Acronyms
AF = atrial fibrillation
BB = Bachmann's bundle

ablation as part of subsequent studies in the open chest state.

Open chest studies. On the second postoperative day, an open chest study was performed using standard anesthetic and surgical techniques (16). Before the chest was opened, we produced complete atrioventricular block and initiated ventricular pacing (40 to 100 beats/min), as previously described (16–18).

ATRIAL FIBRILLATION INDUCTION. Atrial fibrillation was induced with rapid atrial pacing for at least 20 beats, as previously described (16). Only episodes of AF lasting >2 min were used for the present study. During these studies, surface ECG lead II and bipolar electrograms obtained from the previously placed atrial epicardial electrodes were monitored on an oscilloscope (VR-16 Electronics-for-Medicine) and also recorded simultaneously on a tape recorder (Honeywell 101 FM). The ECG was recorded between a band pass of 0.1 to 500 Hz and the bipolar electrograms between a band pass of 30 to 500 Hz.

BASELINE MAPPING. For studies of the sequence of right and left atrial activation, specially built electrode arrays containing 372 unipolar electrodes were used to record simultaneously from both atria as previously described (16). The interatrial septum was also mapped simultaneously with a single 12 polar electrode catheter with an interelectrode distance of 2 mm in one dog, or two electrode catheters (20 and 24 polar electrode catheters with an interelectrode distance of 1 mm) in five dogs, as previously described (16).

Acquisition and analysis of data were performed as described previously (16). Atrial electrograms from both atria and the interatrial septum along with ECG lead II were recorded simultaneously during the activation studies. Analysis was based on sequential 100 ms time windows. For each episode, 1.2 s of data (12 consecutive 100 ms time windows) were analyzed, and the activation sequences were depicted by drawing activation maps with 10 ms isochrones. A continuous period of 1.2 s from the middle section of the baseline-induced AF episode was chosen for analysis. After completing each baseline AF mapping study, the epicardial electrode arrays were removed for application of radiofrequency energy.

ABLATION OF BB. After the baseline mapping study, catheter ablation of Bachmann's bundle was performed in all dogs. A cooled tip 9F catheter (Cordis Webster Inc.) with irrigation holes was positioned epicardially on the midportion of Bachmann's bundle. We used this special catheter to produce a full-thickness lesion in this relatively thick part of the atria while preventing an impedance rise, which we found otherwise occurs easily with an epicardial approach. This ablation catheter electrode was irrigated through the catheter lumen with normal saline solution delivered at 20 ml/min using a Harvard infusion pump. Radiofrequency current was provided by a generator (Medtronic Atakr; Medtronic Inc.), and was delivered between the catheter tip electrode and a pad applied to the shaved skin of the lateral chest wall. The radiofrequency power of 50 W from the generator was applied for 60 s. After each application of radiofrequency energy, an attempt was made to reinduce the AF using the same pacing protocol described above. If AF was still inducible, another application of radiofrequency energy was made. This protocol was continued until sustained AF could not be induced. A minimum of 100 attempts at reinduction were attempted.

MAPPING AFTER ABLATION OF BB. After successful ablation of BB, the epicardial electrode arrays were again placed. Although sustained AF, that is, lasting ≥ than 2 min, could no longer be induced, we were nevertheless always able to induce a very short (≤5 s) episode of transient AF. The latter was recorded using the same simultaneous multisite mapping techniques. If the reinduced transient AF lasted >1.2 s, a period of 1.2 s from the middle section of the AF episode was chosen for analysis, and if the reinduced transient AF lasted <1.2 s, the entire period of the transient AF episode was analyzed. Techniques for analysis of electrograms were as previously reported (16,18–21).

Pathologic examination. After the study was completed, triphenyl tetrazolium chloride (2 g/20 ml intravenously) was administered. This dye stains intracellular dehydrogenase and was used to distinguish viable from necrotic tissue. The dog was then euthanized, the heart was removed in standard fashion and the lesion on BB was identified and sectioned. The lesion width and depth were measured.

Definitions. Atrial fibrillation was defined as a rapid atrial rhythm characterized by variability of the beat-to-beat cycle length, polarity, morphology, and/or amplitude of the recorded bipolar atrial electrograms (16,22). Transient AF was defined as AF < 2 min. The mean cycle length of AF was defined as the mean value of all atrial electrogram intervals sampled from a single site in the BB area during the 1.2 s used for analysis. It was measured before the BB ablation, and was the chosen site because of its importance in atrial activation during AF (16). An unstable reentrant circuit was defined as a reentrant circuit in which, from analysis window-to-analysis window, there was change in the length and location of the central area of block, the path of the reentrant circuit, or the cycle length of the circulating wave front (16). A non-activation zone in the atria was defined as the time during a 1.2 s period of analysis in which no depolarization waves were present in either right or left atrial free wall (16).

Table 1. Selected Parameters of Unstable AF Reentrant Circuits

Dog No.	Duration of AF (min)	Mean AF CL (ms)	Total No. of RC/1.2 s	Mean No. of RC/Window	Mean RC CL (ms)	Mean No. of RC Rotations
1	60	118	14	1.2	117	2.1
2	60	119	16	1.3	118	2.7
3	7	109	17	1.4	114	2.4
4	11	105	16	1.3	109	4.1
5	2	126	11	0.9	128	2.9
6	2	110	13	1.1	112	4.7
Mean ± SD	24 ± 28	115 ± 8	15 ± 2	1.2 ± 0.2	116 ± 7	3.2 ± 1.0

AF = atrial fibrillation; CL = cycle length; RC = unstable reentrant circuits.

RESULTS

Characteristics of induced AF. In each dog, multiple episodes (mean, 7 per dog; range, 5 to 10 per dog) of AF were induced. Each of these episodes was mapped, but only one of these episodes of induced AF was chosen for analysis. Of the six episodes (one per dog) analyzed, AF lasted a mean of 24 ± 28 min (range, 2 to 60 min) (Table 1). The mean cycle length of AF sampled from the BB area was 115 ± 8 ms (range, 105 to 126 ms) (Table 1). A representative example of induced AF, in this case induced with rapid atrial pacing, is shown in Figure 1. Electrograms recorded from the previously placed stainless steel wire electrodes on the right atrial appendage, BB and the posterior-inferior left atrium showed a rapid, irregular rhythm with a continuous beat-to-beat variation in electrogram morphology and cycle length.

Analysis of consecutive activation patterns during a representative episode of induced AF. In Figure 2, consecutive activation maps are shown from a representative episode (dog 1) of induced AF in which the mean beat-to-beat cycle length was 118 ms. Twelve separate 100 ms windows of activation at 30 min after onset are demonstrated. As previously described (16), during sustained AF, the atria were activated by one or more unstable reentrant circuits of short cycle length and the daughter wave fronts they generated. Although these reentrant circuits disappeared while others formed, there was always at least one reentrant circuit per 100 ms window (orange, blue or purple). In 9 of the 12 100 ms windows, there were either 2 (windows 1, 3, 4, 5, 6, 7, 9, 10) or 3 (window 2) reentrant circuits per window. Note that 10 of the total of 14 reentrant circuits included BB as a portion of the reentrant circuit.

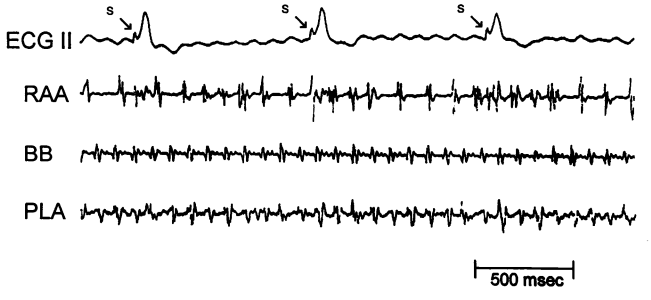

Figure 1. ECG lead II recorded simultaneously with bipolar electrograms from the stainless steel wire electrodes placed on the right atrial appendage (RAA), Bachmann's bundle (BB) and the posterior-inferior left atrium (PLA) demonstrating recording during a representative example of induced atrial fibrillation. The ECG recording shows ventricular paced beats.

NONACTIVATION ZONES IN THE ATRIAL FREE WALLS. In this representative example (Fig. 2), no activation was recorded in the entire right atrial free wall from 290 to 350 ms (zone = 60 ms duration), from 640 to 700 ms (zone = 60 ms duration), from 770 to 820 ms (zone = 50 ms duration), from 980 to 1,030 ms (zone = 50 ms duration), and from 1,120 to 1,160 ms (zone = 40 ms duration). After each disappearance of electrical activity from the right atrial free wall, reactivation occurred via a septal wave front, either breaking through via the Bachmann's bundle area (at 350, 700 and 820 ms) or beneath the inferior vena cava (at 1,030 and 1,160 ms). Similarly, in the left atrial free wall, no activation was observed from 50 to 90 ms, from 160 to 250 ms, from 280 to 370 ms, from 400 to 490 ms, from 530 to 600 ms, from 640 to 710 ms, from 760 to 840 ms, from 890 to 950 ms, from 980 to 1,020 ms and from 1,100 to 1,140 ms. Reactivation of the left atrial free wall occurred primarily via wave fronts coming from the septum via BB or the region of the inferior vena cava. Moreover, no activation was observed in both atrial free walls from 290 to 350 ms, from 640 to 700 ms, from 770 to 820 ms, from 980 to 1,030 ms and from 1,120 to 1,150 ms. Reactivation of both atrial free walls occurred via wave fronts coming from the septum via BB (at 350, 700 and 820 ms) or the region of the peri inferior vena cava (at 1,030 and 1,150 ms).

Characteristics and location of unstable reentrant circuits during induced AF in all episodes before ablation. Selected parameters of the unstable reentrant circuits during AF in all episodes are summarized in Table 1. The total number of unstable reentrant circuits during the 1.2 s of AF analyzed were 15 ± 2 (mean number, 1.2 ± 0.2 per window) with a range of 11 to 17. They had very short cycle lengths (mean, 116 ± 7 ms; range, 109 to 128 ms) and 1 to 11 (mean, 3.2 ± 1.0) consecutive rotations per reentrant circuit. Episodes 5 and 6 of AF had a relatively short duration (2 min) associated with a lower number of unstable reentrant circuits during the 1.2 s of analysis and, consequently, a lower number of reentrant circuits per 100-ms window compared with the

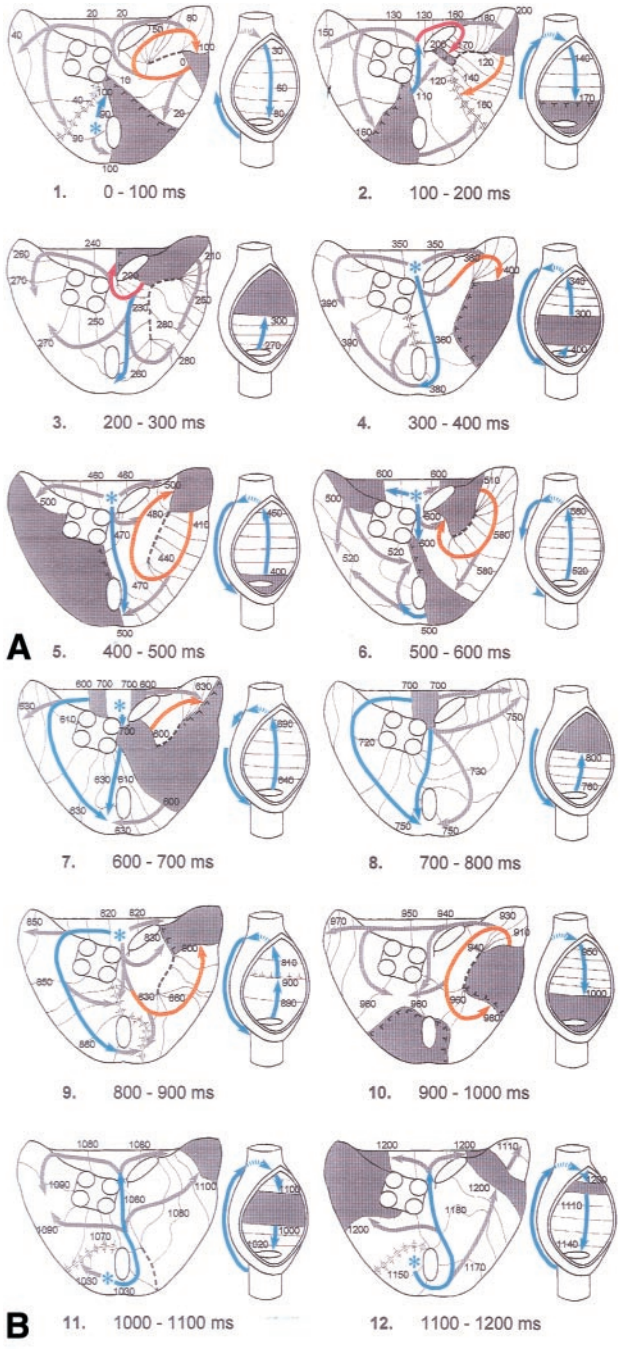

Figure 2. Representative example of the sequence of activation maps during 12 consecutive 100 ms windows (total of 1.2 s of analysis) from an episode (dog 1) of atrial fibrillation. The **colored lines** indicate the location of unstable reentrant circuits. The **gray arrows** indicate the activation wave fronts that are not part of a reentrant circuit. **Thin lines** represent isochrones at 10 ms intervals. The **thick dashed lines** represent lines of functional block. The **asterisk** indicates the epicardial breakthrough point of the wave front coming from the septum. The **dark gray regions** indicate the atrial areas that were not activated during a 100 ms window. BB = Bachmann's bundle area; CS = coronary sinus; IVC = inferior vena cava; LAA = left atrial appendage; PV = pulmonary vein; RAA = right atrial appendage; SVC = superior vena cava. See text for discussion.

other episodes (No. 1 to 4) of AF with a longer duration. This suggests that a larger number of reentrant circuits is required to sustain AF for longer periods. However, the role of activation of BB as a part the reentrant circuits was similar in all six episodes.

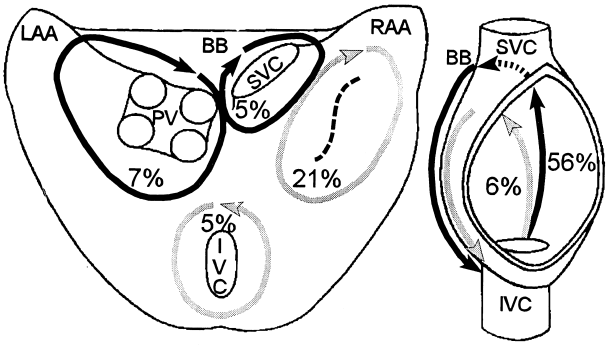

Figure 3. The locations of unstable reentrant circuits are shown. The unstable reentrant circuits were observed 1) involving the septum and the atrial epicardium via BB (56%) and not via BB (6%), and 2) in the right atrial free wall (21%), and were sometimes observed 3) around the pulmonary veins (7%), 4) the inferior vena cava (5%), and 5) the superior vena cava (5%). Three (#1, 3, 5; in total; 68%) of these five location reentrant circuits involved the BB (denoted by **black lines**). See text for discussion.

The location of all the unstable reentrant circuits is summarized in Figure 3. The unstable reentrant circuits were observed as follows: 1) involving the septum and the atrial epicardium a) via BB 56% of the time, and b) not via BB 6% of the time; 2) in the right atrial free wall 21% of the time; 3) around the pulmonary veins 7% of the time; 4) around the inferior vena cava 5% of the time; and 5) around the superior vena cava 5% of the time. As seen in Figure 3, three of these five reentrant circuit locations included BB as part of the reentrant circuit 68% of the time.

Nonactivation zones in the atrial free walls and their reactivation. During the 1.2 s of analysis of all episodes of AF, the mean duration of nonactivation zones was 57 ± 16 ms (range, 40 to 90 ms) in the right atrial free wall, and 53 ± 23 ms (range, 20 to 100 ms) in the left atrial free wall. The site from which reactivation of these nonactivation zones occurred after a period of nonactivation in the right atrium, the left atrium and both atria in all six dogs is summarized as follows. In the right atrium, the nonactivation zone was reactivated 91% of the time by a wave front coming from the septum (21/23) and 9% of the time by a wave front coming from the left atrial free wall (2/23). The reactivation from the septum occurred via three sites: 1) Bachmann's bundle (13/21; 62%); 2) the posterior-inferior aspect of the superior vena caval region (5/21; 24%); and 3) the peri-inferior vena caval region (3/21; 14%). In the left atrium, the nonactivation zone was reactivated 78% of the time by a wave front coming from the septum (43/55) and 22% of the time by a wave front from the right atrial free wall (12/55). The reactivation from the septum occurred via three sites: 1) BB (29/43; 67%); 2) the intercaval region (11/43; 26%); and 3) the peri-inferior vena caval region (3/43; 7%). Nine instances of a nonactivation zone present simultaneously in both atria were observed in three dogs.

Their reactivation occurred from the septum via BB in seven (78%) and the peri-inferior vena caval region in two (22%).

Thus, a wave front coming from the septum via the BB area makes the greatest contribution to the reactivation of the right and left atrial free walls following a nonactivation period during AF. The presence of these nonactivation zones strongly suggests that neither the right atrium alone nor the left atrium alone is responsible for maintenance of AF in this model. In addition, the fact that the reactivation results most often from an exiting wave front from a septal component of the reentrant circuit demonstrates that a reentrant circuit involving the septum is a major factor for the maintenance of AF in this model.

Catheter ablation of BB. Catheter ablation of BB was performed during AF which persisted for at least 1 h in two dogs (No. 1 and 2), and during sinus rhythm in the other four dogs (No. 3 to 6). In the former two dogs (No. 1 and 2), creation of the lesion in BB interrupted the AF. Following several applications of the radiofrequency energy to the ablation site in the latter four dogs, and following interruption of AF in the former two dogs, an attempt to reinduce AF with rapid atrial pacing was made. If AF could be induced, radiofrequency energy was again applied to the BB site and attempts at reinduction of AF were repeated. In all dogs, following a mean of 7.3 radiofrequency applications (range, 5 to 8), AF >5 s was no longer inducible despite rapid atrial pacing over and over again (a minimum of 100 times). The lower 95% confidence limit on the probability of success was 0.605 using the exact binomial test. A brief episode of transient AF (mean duration, 1.9 s; range, 0.5 to 5 s) was always induced.

Analysis of consecutive activation patterns during a representative episode of induced transient AF after catheter ablation. In Figure 4, consecutive activation maps are shown from a representative episode (dog 1) of induced transient AF after radiofrequency ablation of a site in the mid portion of BB. This AF episode lasted a total of 650 ms after cessation of rapid pacing. Six separate 100 ms windows of activation through termination of AF are demonstrated. The black area in Bachmann's bundle indicates the linear lesion created by the radiofrequency catheter ablation. In this 0.6 s portion of the transient AF episode, there were no reentrant circuits of short cycle length in any window, and depending on how one defines a reentrant circuit, perhaps no reentrant circuits at all. By following the activation wave front depicted by the black line, perhaps a reentrant circuit did exist from window 1 to window 2 (cycle length = 140 ms). However, after window 2, when this wave front breaks through to the atrial epicardium, its course is meandering until it returns to the atrial septum in window 4 (cycle length = 170 ms). Its subsequent course includes epicardial breakthrough at 370 ms followed by additional meandering around the right atrial free wall and then briefly in the atrial septum to emerge again at the atrial free wall (cycle length, 140 ms). Even if this continuous activation depicted by the black wave fronts represents a reentrant

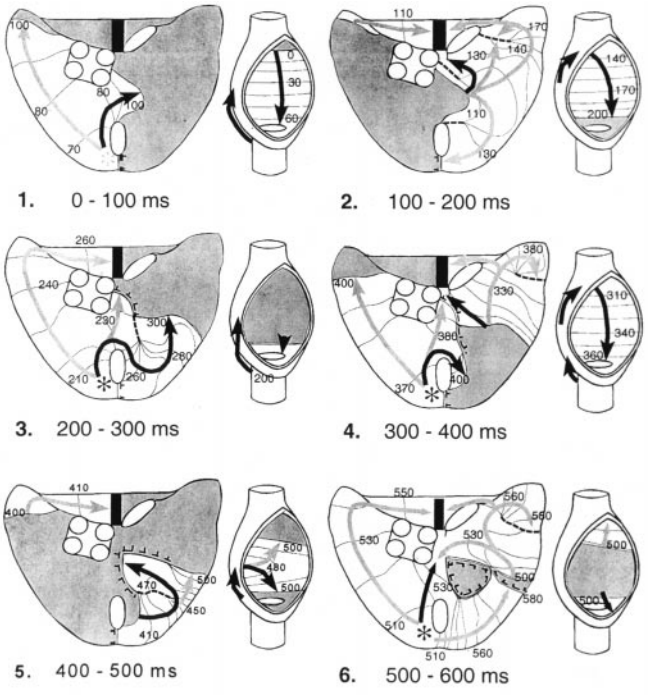

Figure 4. Sequence of activation maps during the six consecutive 100 ms windows just before termination of transient AF after ablation in the same episode (dog 1) as shown in Figure 2. The **black area** in BB indicates a linear lesion created by radiofrequency catheter ablation. Note the absence of reentrant circuits of short cycle length in any window. The **black line with arrows** indicates the meandering course of activation along the atrial free wall. The right atrium was primarily activated by the wave fronts from the left atrium via the intercaval region. See text for discussion.

circuit, its cycle length is relatively long (140 to 170 ms). But, importantly, without activation of BB, no reentrant circuits of the type described earlier which seemed responsible for maintaining AF were formed. Note also that the right atrium was primarily activated by wave fronts from the left atrium via the intercaval region. These observations additionally serve to confirm that the BB region was clearly ablated. In fact, mapping after ablation in the other five dogs also demonstrated conduction block across BB. No reentrant circuit involving Bachmann's bundle was seen after successful ablation in any transient (≤5 s) episode. Thus, catheter ablation of BB prevented the induction of AF by preventing 1) formation of unstable reentrant circuits with short cycle length and 2) activation of the atrial free walls by wave fronts via the BB.

Pathologic examination. A transmural linear lesion in the mid portion of BB was created in all dogs using the ablation technique described. The lesion was irregular at its edges, but in general had a rectangular shape on the epicardial surface. The mean dimensions of the lesion were 2.5 ± 1.3 cm perpendicular to the long axis of BB, 1.5 ± 1.2 cm parallel to the long axis of BB and 0.6 ± 0.5 cm in depth (Fig. 5). The depth of the lesion was determined by measuring the depth from at least two sections cut through the middle of the lesion. These observations additionally served to confirm that the midportion of BB was completely ablated.

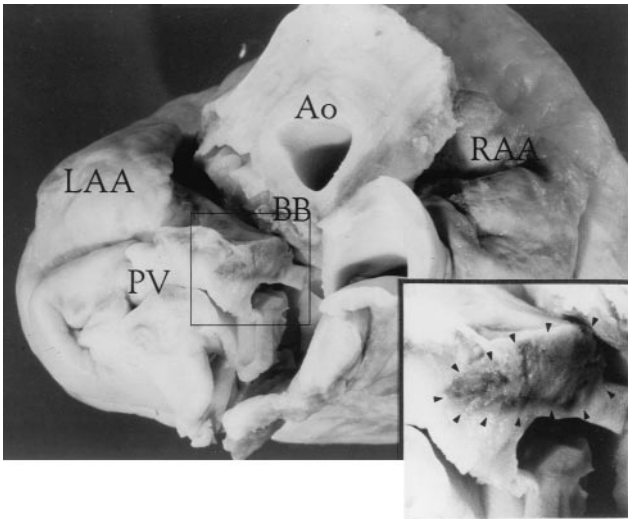

Figure 5. This figure is a picture of a cross section of the lesion in the mid portion of BB created by the radiofrequency catheter ablation. The **arrow** points to the lesion. Note that the lesion is transmural.

DISCUSSION

The data presented in this study again show that in the canine sterile pericarditis model, one mechanism of AF is due to multiple, unstable, reentrant circuits that cyclically disappear while others reform. Further support for this mechanism is that inability to form these reentrant circuits after making a full-thickness lesion in BB was associated with inability to reinduce sustained AF. Furthermore, as we had hypothesized, the development of these unstable reentrant circuits depends importantly on activation of BB, either as part of the actual reentrant circuit or as a path for conduction of wave fronts which then formed these unstable reentrant circuits. In short, activation of BB appears critical for the maintenance of this mechanism of AF in this model.

Relation to previous studies. There have been several ablation studies to treat AF in experimental animal models and humans (1–6,11–15), many of which have explicitly or implicitly assumed that the AF is due to the Moe-Allessie concept of multiple, simultaneously circulating reentrant wave fronts (7–10). While that assumption may sometimes or often be correct, we do know that there are other mechanisms of AF. That a single focus firing rapidly can generate AF has been shown in animal models (7,13,23–26) and in patients (12,15,27,28). In fact, mapping identification of a single focus firing rapidly has led to successful radiofrequency ablation of AF (12,15). In addition, type II atrial flutter in patients (29) and a microreentrant circuit of very short cycle length in the canine right atrium (30) also have been shown to generate AF. Furthermore, limited endocardial application of radiofrequency catheter ablation lesions has resulted in prevention of AF in some instances without really understanding why (11,13,14). In sum, it seems reasonable to suggest that because there may be many mechanisms of AF, there may also be many potentially effective types of ablative procedures to prevent AF. Our study certainly supports this concept. Furthermore, from this discussion, it seems clear that one should not expect that an ablative lesion placed in BB would be effective unless conditions similar to those shown during mapping of AF in this study are present. Interestingly enough, Abi-Samra et al. (31) have recently reported successful prevention of AF induction in a pig model of AF following ablation of a portion of BB.

Study limitations. As we have previously stated (16), a limitation of the mapping data obtained in this study is the limited recording resolution when mapping from the atrial septum. Clearly, recording from 12 to 24 electrodes from the atrial septum is less than optimal. Of note, apart from our studies in the canine sterile pericarditis model, we are aware of only one previous study in an animal model (9,10) in which simultaneous, multisite atrial mapping included atrial septal mapping during AF. Nevertheless, our mapping data from this study are consistent with our previous data (16). Furthermore, the role of BB activation in the maintenance of AF in these studies was strongly supported by the effects of the radiofrequency ablation.

Finally, we do not know if other types of ablation lesions would also prove effective in preventing induction of AF in this model. We only showed that understanding the nature of atrial activation during induced AF in this model led us to predict and then demonstrate a vulnerable target to ablate, and thereby prevent AF reinduction.

CONCLUSIONS

In this model of AF, radiofrequency catheter ablation of BB terminated AF and prevented the following: 1) the formation of the unstable reentrant circuits that involve BB; 2) the reactivation of the atrial free walls after a nonactivation period by wave front via the BB; and 3) the maintenance of AF.

Reprint requests and correspondence: Dr. Albert L. Waldo, Division of Cardiology, University Hospitals of Cleveland, 11100 Euclid Avenue, Cleveland, Ohio 44106-5038.

REFERENCES

1. Cox JL, Schuessler RB, D'Agostino JH, et al. The surgical treatment of atrial fibrillation. III: development of a definitive surgical procedure. J Thorac Cardiovasc Surg 1991;101:569–83.
2. Elvan A, Pride HP, Zipes DP. Radiofrequency catheter ablation of the atria reduced inducibility and duration of atrial fibrillation in dogs. Circulation 1995;91:2235–44.
3. Haissaguerre M, Jais P, Shah DC, et al. Right and left atrial radiofrequency catheter therapy of paroxysmal atrial fibrillation. J Cardiovasc Electrophysiol 1996;7:1132–44.
4. Elvan A, Huang X-d, Pressler ML, Zipes DP. Radiofrequency catheter ablation of the atria eliminated pacing-induced sustained atrial fibrillation and reduces connexin 43 in dogs. Circulation 1997; 96:1675–85.
5. Mitchell MA, McRury ID, Haines DE. Linear atrial ablations in a canine model of chronic atrial fibrillation: morphological and electrophysiological observations. Circulation 1998;97:1176–85.
6. Gaita F, Riccardi R, Calò L, et al. Atrial mapping and radiofrequency catheter ablation in patients with idiopathic atrial fibrillation: electro-

physiological findings and ablation results. Circulation 1998;97:2136–45.
7. Moe GK, Abildskov JA. Atrial fibrillation as a self-sustaining mechanism independent of focal discharge. Am Heart J 1959;587:59–70.
8. Moe GK, Rheinboldt WC, Abildskov JA. A computer model of atrial fibrillation. Am Heart J 1964;67:200–20.
9. Allessie MA, Lammers WJEP, Smeets JRLM, Bonke FIM, Hollen J. Total mapping of atrial excitation during acetylcholine-induced atrial flutter and fibrillation in the isolated canine heart. In: Kulbertus HE, Olsson SB, Schlepper M, eds. Atrial Fibrillation. Molndal, Sweden: Lindgren and Soner; 1982:44–62.
10. Allessie MA, Lammers WJEP, Bonke FIM, Hollen J. Experimental evaluation of Moe's multiple wavelet hypothesis of atrial fibrillation. In: Zipes DP, Jalife J, eds. Cardiac Arrhythmias. New York: Grune & Stratton; 1985:265–76.
11. Haissaguerre M, Gencel L, Fischer B, et al. Successful catheter ablation of atrial fibrillation. J Cardiovasc Electrophysiol 1994;5:1045–52.
12. Jais P, Haissaguerre M, Shah DC, et al. A focal source of atrial fibrillation treated by discrete radiofrequency ablation. Circulation 1997;95:572–6.
13. Morillo CA, Klein GJ, Jones DL, Guiraudon CM. Chronic rapid atrial pacing-structural, functional, and electrophysiological characteristics of a new model of sustained atrial fibrillation. Circulation 1995;91:1588–95.
14. Tondo C, Scherlag BJ, Otomo K, et al. Critical atrial site for ablation of pacing-induced atrial fibrillation in the normal dog heart. J Cardiovasc Electrophysiol 1997;8:1255–65.
15. Haissaguerre M, Jais P, Shah D, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med 1998;339:659–66.
16. Kumagai K, Khrestian C, Waldo AL. Simultaneous multisite mapping studies during induced atrial fibrillation in the sterile pericarditis model: insights into the mechanism of its maintenance. Circulation 1997;95:511–21.
17. Pagé P, Plumb VJ, Okumura K, Waldo AL. A new model of atrial flutter. J Am Coll Cardiol 1986;8:872–9.
18. Ortiz J, Niwano S, Abe H, et al. Mapping the conversion of atrial flutter to atrial fibrillation and atrial fibrillation to atrial flutter: insights into mechanism. Circ Res 1994;74:882–94.
19. Olshansky B, Okumura K, Henthorn RW, Waldo AL. Characterization of double potentials in human atrial flutter: studies during transient entrainment. J Am Coll Cardiol 1990;15:833–41.
20. Feld GK, Shahandeh-Rad F. Mechanism of double potentials recorded during sustained atrial flutter in the canine right atrial crush-injury model. Circulation 1992;86:628–41.
21. Shimizu A, Nozaki A, Rudy Y, Waldo AL. Characterization of double potentials in a functionally determined reentrant circuit: multiplexing studies during interruption of atrial flutter in the canine pericarditis model. J Am Coll Cardiol 1993;22:2022–32.
22. Wells JL, Karp RB, Kouchoukos NT, MacLean WAH, James TN, Waldo AL. Characterization of atrial fibrillation in man: studies following open heart surgery. PACE 1978;1:426–38.
23. Scherf D. Studies on auricular tachycardia caused by aconitine administration. Proc Exp Biol Med 1947;64:233–9.
24. Scherf D, Roman JF, Terranova R. Experimental studies on auricular flutter and auricular fibrillation. Am Heat J 1958;36:241–51.
25. Scherf D, Terranova R. Mechanism of auricular flutter and fibrillation. Am J Physiol 1949;159:137–42.
26. Wijffels MCEF, Kirchhof CJHJ, Dorland R, Allessie MA. Atrial fibrillation begets atrial fibrillation: a study in awake chronically instrumented goats. Circulation 1995;92:1954–68.
27. Waldo AL, MacLean WAH, Karp RB, Kouchoukos NT, James TN. Continuous rapid atrial pacing to control recurrent or sustained supraventricular tachycardias following open heart surgery. Circulation 1976;54:245–50.
28. Moreira DAR, Shepard RB, Waldo AL. Chronic rapid atrial pacing to maintain atrial fibrillation: use to permit control of ventricular rate in order to treat tachycardia induced cardiomyopathy. PACE 1989;12:761–75.
29. Waldo AL, Cooper TC. Spontaneous onset of type I atrial flutter in patients. J Am Coll Cardiol 1996;28:707–12.
30. Schuessler RB, Kawamoto T, Hand DE, et al. Simultaneous epicardial and endocardial activation sequence mapping in the isolated canine atrium. Circulation 1993;88:250–63.
31. Abi-Samra F, Feng Z, Mobarek SK. Radiofrequency ablation of Bachmann's bundle terminates and suppresses atrial fibrillation in pigs (abstr). J Am Coll Cardiol 1998;31 suppl A:368A.