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Isolated Atrial Segment Pacing

An Alternative to His Bundle Pacing After Atrioventricular Junctional Ablation

Fei Lü, MD, PHD, FACC,* Paul A. Iaizzo, PHD,† David G. Benditt, MD, FACC,*
Rahul Mehra, PHD,‡ Eduardo N. Warman, PHD,‡ Brian T. McHenry, MSME‡

Minneapolis, Minnesota

Objectives This study was designed to investigate a practical alternative to His bundle pacing after atrioventricular (AV) junctional ablation by pacing a small area of isolated atrial tissue surrounding the AV node.

Background His bundle pacing is preferred after AV junctional ablation in patients with refractory atrial fibrillation. However, it is technically difficult and not clinically useful at the present time.

Methods This study was conducted in an isolated working swine heart model (n = 5), with real-time imaging capabilities. A small area of atrial tissue surrounding the AV node and the His bundle was isolated using sequential radiofrequency ablation lesions.

Results Complete AV block created by segmental atrial isolation was achieved in 5 of 5 experiments. The isolated atrial segment was bordered by the ablation lines, the tricuspid annulus, and the AV node–His bundle. The AV conduction was characterized using a pacing electrode implanted into the isolated atrial segment. Pacing from the atria, the ventricles, and the isolated atrial segment at different rates confirmed complete bidirectional block between the atria and isolated area, whereas antegrade and retrograde AV nodal conduction between the isolated atrial segment and the ventricles remained intact. Pacing from the isolated area produced minimal changes in systolic left ventricular pressure compared with baseline sinus rhythm (mean −2 mm Hg).

Conclusions Isolation of a small area of atrial tissue surrounding the AV node is feasible by transcatheter radiofrequency ablation. This procedure may be a useful alternative to conventional AV junctional ablation because it can create complete AV block, while in effect permitting the equivalent of His bundle pacing after AV junctional ablation. (J Am Coll Cardiol 2007;49:1443–9) © 2007 by the American College of Cardiology Foundation

In the absence of significant His–Purkinje disease, His bundle pacing (1,2) provides a more physiological cardiac stimulation site than does pacing from conventional ventricular positions, especially the right ventricular apex (3,4). Such a more physiological pacing technique may be advantageous for patients who already have severe left ventricular dysfunction, including many individuals with refractory atrial fibrillation undergoing atrioventricular (AV) junctional ablation. Because the His bundle is small, unfortunately, it is technically difficult to achieve stable pacing at this location. On the other hand, we usually have little difficulty implanting a pacemaker lead with good thresholds in atrial muscles.

In this study, we investigated a practical alternative to His bundle pacing by pacing a small region of electrically isolated atrial tissue surrounding the AV node–His bundle region. Electrical isolation was created by application of a series of radiofrequency (RF) ablation lesions in atrial tissue surrounding the AV node. The objective of these experiments was to develop a practicable pacing method equivalent to His bundle pacing for ultimate application in patients with refractory atrial fibrillation who require AV junctional ablation and pacemaker implantation for rate control.

Methods

Preparation of a working heart model. This study was conducted in an isolated working swine heart model. This experimental model was chosen because it offers excellent physiological function with real-time imaging capabilities. The details of this heart model have been published previously (5). This is a modified Langendorff-perfused heart model. In brief, mongrel swine were used after approval from the University of Minnesota Animal Care and Use Committee. Telazol (5 mg/kg; Fort Dodge Animal Health, Fort Dodge, Iowa) was administered intramuscularly. Intravenous access was then obtained through an ear vein for

From the Departments of *Cardiovascular Disease and †Anesthesiology Surgery, University of Minnesota, Minneapolis, Minnesota; and ‡Medtronic, Inc., Minneapolis, Minnesota. This study was funded in part by a grant-in-aid from Medtronic Inc., Minneapolis, Minnesota, and a grant-in-aid from St. Jude Medical, Sylmar, California.

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Abbreviations and Acronyms

AV = atrioventricular
CS = coronary sinus
RF = radiofrequency

fluid administration (Ringers lactate) and delivery of medications as needed. Thiopental, titrated to effect, was administered before intubation. The animal was intubated and ventilated mechanically with 35% to 40% oxygen and 60% to 65% nitrous oxide. General anesthesia was maintained with 1.8% to 2.0% isoflurane.

A medial sternotomy was performed to expose the heart. After heparinization (50,000 U), the heart was arrested, excised, and placed in an iced saline slurry. After removal of excess tissue and isolation of the great vessels, the aorta, the pulmonary artery, the pulmonary veins, the inferior vena cava, and the superior vena cava were cannulated. The heart was then perfused using a modified Krebs perfusate at 37.0°C ± 0.5°C and defibrillated to sinus rhythm using an external defibrillator (model 5358, Medtronic Inc., Minneapolis, Minnesota). A total of 5 animals (body weight 84 ± 12 kg) were studied. The AV node–His bundle isolation targeted for complete AV block was created by application of a series of RF ablation lesions under direct transcatheter video imaging. After the desired AV node–His bundle isolation was achieved in all 5 hearts, 4 of them underwent an additional pacing protocol to characterize the nature of conduction between the atria, the isolated segment, and the ventricles (see Results section for details).

Monitoring, pacing, and ablation. Four intramuscular bipolar electrodes (model 6495, Medtronic Inc.) were placed to the right atrium, the left atrium, the right ventricle, and the left ventricle for recording and pacing. A 5-F microtip catheter (MPC 500, Millar, Houston, Texas) was inserted into the left ventricle through one of the pulmonary veins to monitor left ventricular pressure. All electrical signals and pressure were recorded in a computer using a multiple acquisition system (DI 410 and ATCODAS, Dataq Acquisition Systems, Akron, Ohio) after amplification (Gould, Valley View, Ohio). The filter was set between 10 and 100 Hz for surface electrogram, and 30 and 300 Hz for intramyocardial electrogram.

A 4-mm ablation catheter (RF Marinr MC, Medtronic Inc.) was used for mapping and ablation. Ablation lesions were created using RF energy (Atakr II RF Ablation System, Medtronic Inc.) at 55.0°C for 45 to 60 s. A bipolar lead (SelectSecure, model 3830, Medtronic Inc.) was implanted into the small isolated atrial segment for pacing. A steerable sheath (Attain, model 6226DEF, Medtronic Inc.) was used to facilitate the lead placement or stabilize the ablation catheter, or both. A stimulator (model 5328, Medtronic Inc.) and a pacemaker programmer (model 9790, Medtronic Inc.) were used for pacing. Pacing threshold was measured before ablation was started and after ablation lines were completed.

Direct image of cardiac structure. A transistor-based camera (ILV-C1, Olympus Industrial America, Orangeburg, New York) in conjunction with a 6-mm-diameter flexible videoscope (IV6C6-13, Olympus Industrial America) were used to visualize the Koch triangle area. The videoscope was inserted into the heart through the superior vena cava. Video was recorded on line (UVW-1800 Beta, Sony Inc., Tokyo, Japan) and later digitized at 720 × 540 resolution (Media 100, Optibase, Ltd., Herzlia, Israel).

Statistical analysis. All data are expressed as mean ± SD in this study. Paired Student *t* test was used when appropriate to test for statistical differences. A 2-tailed probability value <0.05 was considered statistically significant.

Results

Baseline measurement. Hearts were allowed to stabilize for 30 min after restoration of sinus rhythm before data collection. At baseline, sinus rate was 106 ± 11 beats/min, generating systolic left ventricular pressure of 85 ± 24 mm Hg. The AH interval was 123 ± 31 ms, HV interval was 53 ± 4 ms, QRS duration was 112 ± 6 ms, and QT was interval 360 ± 32 ms.

Ablation and isolation. All procedures, including mapping, ablation, and lead placement, were performed under direct visualization using the aforementioned imaging system. The location of the His bundle, usually localized at the midinferior atrial septum near the tricuspid annulus, was mapped first (Fig. 1). The AV node was presumably

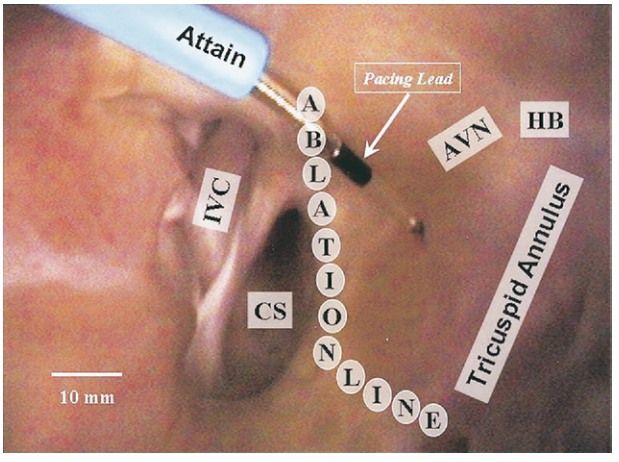

Figure 1 Ablation Lesions and Lead Placement

Isolation of small atrial segment surrounding the atrioventricular node and the His bundle was achieved by sequential radiofrequency ablation. The structures around the Koch triangle in the swine heart are shown here. The relative location of the CS and the IVC is different from that in humans. Please see text for details. The location of the tricuspid annulus is labeled. In this study, the His bundle is mapped first. The atrioventricular node is presumably just proximal and inferior to the His bundle. The small atrial segment surrounding the atrioventricular node and the His bundle is isolated using a series of radiofrequency ablation lesions. The isolation is started with a line crossing the CS–tricuspid isthmus, which is extended superiorly along the anterior edge of the CS. A pacing lead is inserted into the isolated atrial segment surrounded by the ablation lines, the tricuspid annulus, and the atrioventricular node–His bundle. The results of this study are not dependent on the location of the lead implantation (either closer to the AV node or closer to the CS–tricuspid isthmus). The **circles** labeled as A.B.L.A.T.I.O.N.L.I.N.E. represent the linear ablation lesions. Attain = steerable guiding sheath; AVN = location of the atrioventricular node; CS = coronary sinus; HB = the location of the His bundle; IVC = inferior vena cava.

localized immediately proximal to the His bundle. The Koch triangle was the target for isolation in this study. It was noted that the anatomical structure near the Koch triangle in the swine heart was similar to that of a human heart except for the relative positions of the ostium of the coronary sinus (CS) and the inferior vena cava. The ostium of the CS was more lateral and inferior in the swine heart (Fig. 1). The inferior vena cava was medial and superior to the ostium of the CS. These 2 structures were very close to each other.

A small atrial tissue surrounding the His bundle and AV node was isolated as in Figure 1 using RF energy. The lesion formation was confirmed by direct visualization. It usually took 10 to 15 s to form a discernible lesion. If needed, additional RF applications were added to ensure a complete lesion formation. The isolation procedure was initially performed in the CS–tricuspid isthmus starting from the tricuspid annulus (Fig. 1). As used for ablation of typical atrial flutter, a linear ablation (usually requiring approximately 4 to 5 lesions in a series) was created across the CS–tricuspid isthmus at approximately 6 o'clock to the tricuspid annulus. Another ablation line (usually requiring approximately 5 to 6 lesions in a series) was created from the ostium of the CS to a point just superior to the inferior vena cava, connecting the previous ablation line across the CS–tricuspid isthmus. When the second ablation line was extended beyond the ostium of the inferior vena cava, complete AV block emerged in all 5 hearts (Fig. 2). The DDD pacing at 86 beats/min (700 ms) was immediately started at this time using a pacemaker programmer (model 9790, Medtronic Inc.). There were 2 interesting points noted during the isolation process. First, we initially thought that a complete isolation line surrounding (i.e., inferior, posterior, and superior to) the AV node was necessary to achieve complete AV block. However, it was consistently shown that such ablation lesions in the region superior to the AV node were not necessarily required to achieve complete AV block. Second, it took more efforts in ablating the region corresponding to the fast pathway to create complete AV block.

Placement of pacemaker lead. After complete AV block was confirmed, the previously described positive-fixation pacing lead (SelectSecure, model 3830, Medtronic Inc.) was placed in the center of the isolated atrial segment surrounded by the 2 ablation lines, the tricuspid annulus, and the His bundle and the AV node (Fig. 1). A steerable sheath (Attain, model 6226DEF, Medtronic Inc.) was used to guide lead placement. It took approximately 3 clockwise turns to secure the screw-in fixation of the lead into the myocardium. The pacing threshold was tested (<1.5 mA at 2.0 ms, which was not significantly different compared with that measured before RF ablation). At maximal output of 28 mA at 2.0 ms from the pacing lead implanted in the isolated atrial segment, no direct ventricular capture was observed in all of our experiments. This is in contrast to direct His bundle pacing, in which direct ventricular capture is usually seen at a high stimulation output. The above observations were consistent no matter where the location was within the isolated atrial segment (closer to the His bundle or closer to the ablation line in the CS–tricuspid isthmus).

Pacing protocols. Pacing from the isolated atrial segment at rates of 86, 100, 120, 150, and 200 beats/min (or up to development of Wenckebach AV conduction) was consistently followed by ventricular activation with a fixed interval between the pacing spike and the ventricular activation at each pacing rate. Activation of the isolated atrial segment and the ventricles was dissociated from the rest of the right atrium and the left atrium (Fig. 3).

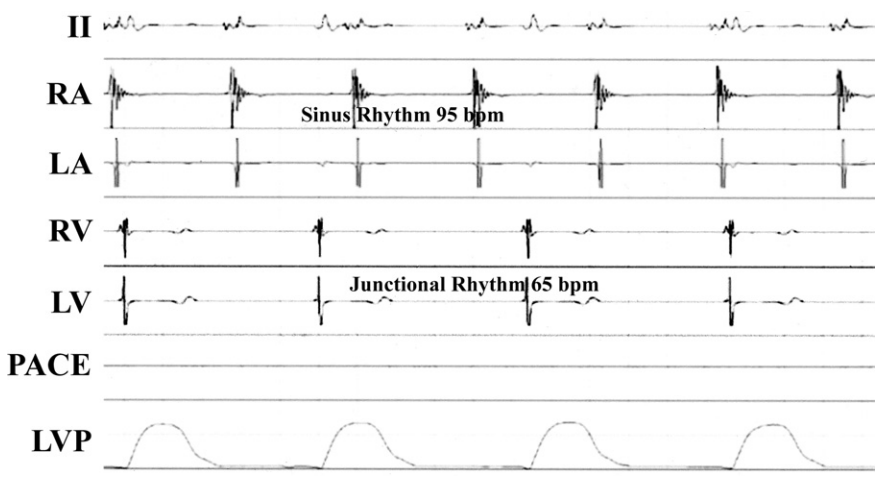

Figure 2 **Creation of Complete Atrioventricular Block**

Complete atrioventricular block created by isolation of a small atrial segment surrounding the atrioventricular node and the His bundle. Complete atrioventricular block emerged on completion of the linear ablation with junctional rhythm at 65 beats/min in the ventricles and sinus rhythm at 95 beats/min in the atria. The recordings from top to bottom are a modified lead II surface electrocardiogram (II), right atrial electrogram (RA), left atrial electrogram (LA), right ventricular electrogram (RV), left ventricular electrogram (LV), recording from pacing lead inserted into the small isolated atrial segment (PACE), and left ventricular pressure (LVP).

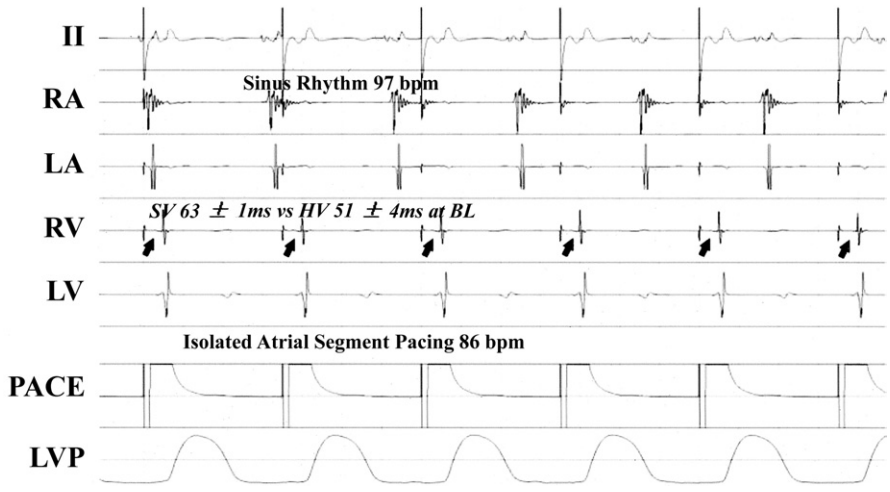

Figure 3 **Dissociation of the Isolated Segment From the Atrium**

After complete atrioventricular block was confirmed, pacing from the isolated atrial segment at a cycle length of 700 ms (86 beats/min) activated both ventricles with a fixed interval between the pacing spike and the ventricular activation. The isolated atrial segment and ventricles were dissociated with both the atria, which were in sinus rhythm at 97 beats/min. The recording format is the same as in Figure 2. HV = interval between His potential and ventricular signal; SV = interval between pacing spike and ventricular signal; other abbreviations as in Figure 2.

Traditionally, His bundle pacing is confirmed by comparison of the interval from the pacing spike to the onset of the earliest ventricular activation (SV) to HV interval in addition to changes in QRS morphology (3). No significant changes in surface QRS morphology were noted during pacing at different rates from the isolated atrial segment in this study. During pacing from the isolated atrial segment at 120 beats/min, the SV interval was slightly longer than the HV interval at baseline (63 ± 1 ms vs. 51 ± 4 ms, $p < 0.01$). During ventricular pacing at each pacing cycle length, activation of the isolated atrial segment was consistently associated with each ventricular activation with a fixed interval between the signals recorded from these 2 locations (Fig. 4). These observations suggest that the antegrade AV conduction from the isolated atrial segment to the ventricles and the retrograde ventriculoatrial (VA) conduction from the ventricles to the isolated atrial segment remained intact despite the presence of complete AV block from the ablation lines. Wenckebach AV conduction from the isolated atrial segment and the ventricles was also observed during pacing from the isolated atrial segment (Fig. 5).

Furthermore, it could be shown that the isolated atrial segment–ventricles and the atria activated independently from each other. Although the isolated atrial segment was paced at 86 beats/min, pacing from either the right atrial appendage or the left atrium at 120, 150, 180, 200, 300, 400, and 600 beats/min (or up to refractoriness) did not change ventricular rate (Fig. 6). Finally, in an attempt to mimic atrial fibrillation in the excluded atrial tissue, pacing at 50 Hz from the right atrial appendage or the left atrium had no effect on ventricular rate either. These findings suggest that there was a complete block from the rest of the

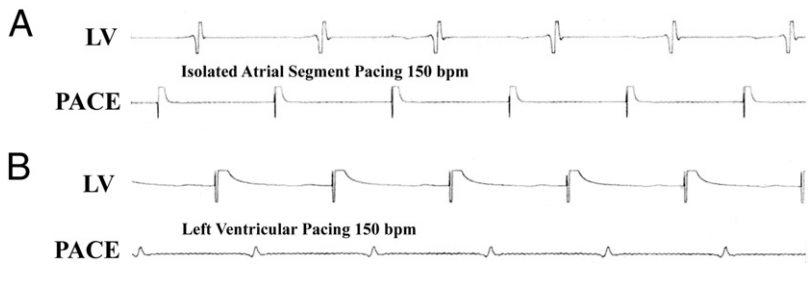

Figure 4 **Antegrade and Retrograde AV Conduction After Isolation**

The antegrade atrioventricular (AV) conduction and retrograde ventriculoatrial (VA) conduction remained intact after the small atrial segment was isolated. **(A)** Pacing from the isolated atrial segment at 150 beats/min subsequently activated the ventricles with 1:1 AV conduction. **(B)** Pacing the left ventricle at 150 beats/min resulted in 1:1 VA conduction. LV = recording from the left ventricle; PACE = recording from pacing lead inserted into the isolated atrial segment; other abbreviations as in Figure 2.

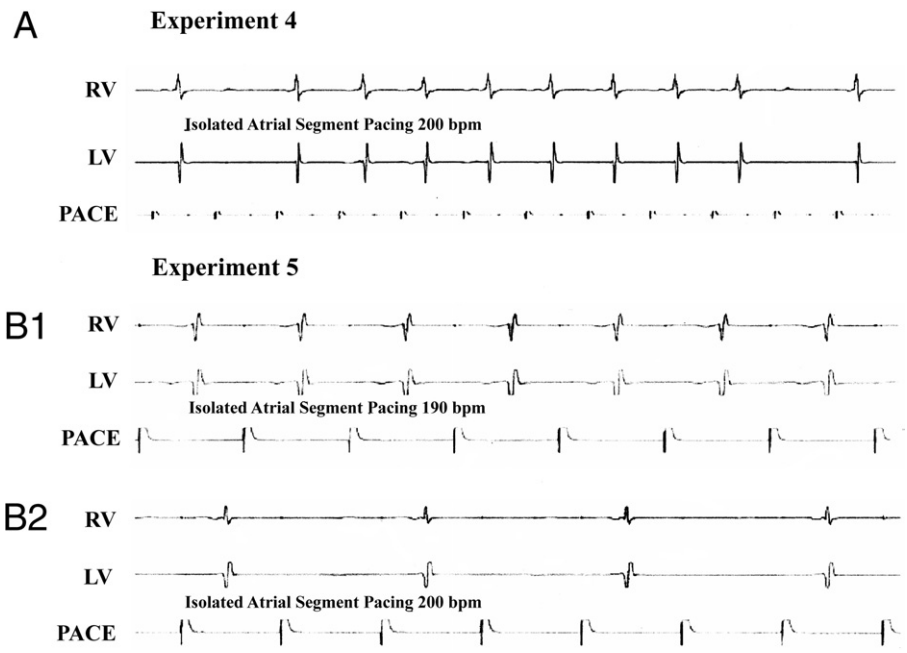

Figure 5 **Wenckebach AV Conduction After Isolation**

Wenckebach atrioventricular (AV) nodal physiology was reserved after the small atrial segment was isolated. **(A)** In experiment 4, pacing from the isolated atrial segment at 200 beats/min resulted in Wenckebach AV conduction. **(B1)** In experiment 5, pacing from the isolated atrial segment at 190 beats/min resulted in constant 1:1 AV conduction. **(B2)** In the same experiment as B1, pacing from the isolated atrial segment at 200 beats/min resulted in 2:1 AV conduction. LV = recording from the left ventricle; PACE = recording from pacing lead inserted into the isolated atrial segment; RV = recording from the right ventricle.

right atrium and the left atrium to the small isolated atrial segment. On the other hand, pacing from the isolated atrial segment had no influence on atrial rate (not shown). This suggests that there was a complete block from the small isolated atrial segment to the rest of the right atrium and the left atrium.

Effects on left ventricular pressure. The difference in systolic left ventricular pressure during pacing from the

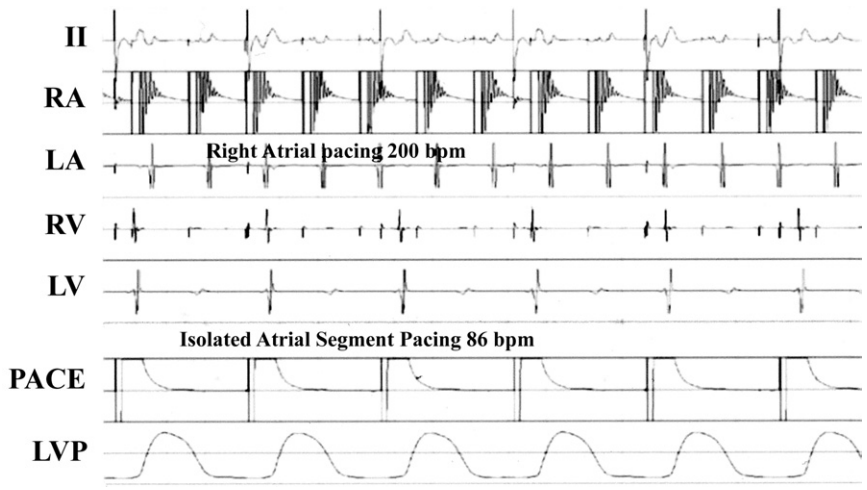

Figure 6 **Fast Atrial Pacing After Isolation**

During pacing at 700 ms (86 beats/min) from the isolated atrial segment, the ventricles were activated after each pacing, with a fixed interval between the pacing spike and the ventricular activation, despite the fact that the right atrium was paced at 200 beats/min (which activated the left atrium as well). This suggests that there was complete conduction block from the right–left atria to the isolated tissue–ventricles. The recording format is the same as Figure 1. LVP = left ventricular pressure; other abbreviations as in Figure 5.

isolated atrial segment at a rate of 120 beats/min compared with that during sinus rhythm at baseline before ablation was minimal (85 ± 24 mm Hg vs. 83 ± 23 mm Hg, not significant).

Discussion

Main findings. This pilot study in the swine working heart model was designed to test the feasibility of a new practicable concept to achieve the equivalent of His bundle pacing. We showed that it is feasible to isolate a small zone of atrial tissue surrounding the AV node and the His bundle and thereby create a complete AV block. This isolation served 2 purposes. Firstly, it created complete AV block, which could be used to achieve rate control in refractory atrial fibrillation. Secondly, the reserved small atrial segment around the AV node and the His bundle was usable for easy implantation of a pacemaker lead. Pacing this isolated atrial segment subsequently activated the AV node and the His–Purkinje system and served as potentially more easily effected alternative to direct His bundle pacing. These preliminary data showed that this new concept is a feasible and effective way to achieve an equivalent to His bundle pacing after AV junctional ablation.

Proposed mechanisms. This pilot study was designed to test the feasibility of a new practical concept to achieve the equivalent of His bundle pacing. It was not our intention to examine other related issues, such as atrial inputs to the AV node or AV nodal electrophysiology, or to provide a detailed technical investigation.

There is evidence showing that there may be 3 distinct preferential atrial inputs to the AV node (lateral, medial, and superior atrionodal bundles) (6). It is our hypothesis that the ablation line across the CS–tricuspid isthmus blocked the lateral atrionodal muscle bundle, and the other ablation line from the ostium of the CS to the inferior vena cava blocked the medial and superior atrionodal muscle bundles. It seems that ablation or block within the relatively large area superior to the AV node and the His bundle was not required to create complete AV block in this heart model. This latter observation is probably caused by lack of direct electrical connection between the muscle fibers in that region and the specialized conduction fibers in the AV junction (whether this is true or not in humans remains unknown), or the superior atrionodal bundle had already been ablated during ablation in the region of the fast pathway and nearby area. It is our impression that the isolated atrial segment targeted for pacing lead implantation corresponded with the so-called slow pathway area, which contained the lateral atrionodal bundle. This may partially explain the relatively short interval from the pacing spike to the ventricles during pacing from the isolated atrial segment. From a clinical standpoint in ablation of AV nodal re-entry tachycardia, we simply might have ablated the slow and fast pathway of the AV node. A previous study in patients with paroxysmal atrial fibrillation showed that AV conduction persisted after slow and fast pathway ablation in approximately two-thirds of these patients (7). In addition to the difficulties in completely blocking the slow and fast pathways using the current ablation techniques in daily practice, multiple atrial inputs to the AV node may be responsible for the failure to achieve compete AV block after ablation of the slow and fast pathways.

Clinical significance. Although important advances have been made in nonpharmacological management of atrial fibrillation (in particular atrial fibrillation ablation and AV junction ablation), currently available techniques in this field are far from ideal (8). Atrial fibrillation ablation, albeit promising, is limited in its application by moderate success rates and potential severe complications. In many patients with atrial fibrillation, rate control remains the mainstay of treatment. Rhythm or rate control by pharmacological agents is largely limited by their proarrhythmic and negative inotropic effects, adverse side effects, and potential increase in mortality rate. One option for avoiding such disadvantages of antiarrhythmic drugs for rate control is to ablate the AV junction with a backup ventricular pacemaker. However, ventricular pacing, particularly from the right ventricular apex, has been consistently shown to be associated with deleterious hemodynamic changes (1–4). This latter liability is particularly important when patients already have significant cardiac dysfunction, a circumstance often present in patients with atrial fibrillation. Although left ventricle-based pacing has been shown to be associated with improved hemodynamics compared with right ventricular apical pacing, His bundle pacing may be preferred, especially in patients without intraventricular conduction delay. Unfortunately, as discussed earlier, stable long-term His bundle pacing is technically difficult to achieve.

The technique described in the present study may provide a practical alternative to His bundle pacing after AV junctional ablation. By achieving the effective equivalent to His bundle pacing, this technique avoids the increasingly recognized detrimental aspects of right ventricular pacing and may thereby improve our management of atrial fibrillation in large numbers of patients being considered for AV junctional ablation and permanent pacing.

Study limitations. This study was performed in sinus rhythm rather than in atrial fibrillation. We tried to induce atrial fibrillation but failed to do so in this experiment model. However, the finding that the His bundle pacing equivalent remained effective during high-rate atrial pacing mimicking atrial fibrillation suggests that the technique of this study should be valid during atrial fibrillation. It is well known that a large area of muscle tissue is needed to sustain electrical fibrillation in the heart. We believe that a small isolated atrial segment such as has been constructed by this method should not be susceptible to fibrillation, and therefore could be used as a stable pacing site in such patients. This hypothesis is supported by the finding in the present study that the electrical activity of the small isolated atrial segment was completely independent of that of the remain-

ing right atrium and the left atrium during high-rate atrial pacing. However, future studies in the setting of atrial fibrillation are needed to further verify this observation.

It is possible that the isolated atrial tissue may not be mechanically viable after longstanding atrial fibrillation. For the same reason, the pacing threshold may be higher in the isolated atrial segment than in normal healthy tissue, but it is less likely to be electrically inactive. We believe that our technique should be applicable in patients with chronic atrial fibrillation.

Atrioventricular block long has been noted in some patients who undergo atrial flutter ablation when an ablation line is created between the CS ostium and inferior vena cava in addition to an ablation line across the cavotricuspid isthmus. Extensive ablation as performed in this study may damage perinodal blood supply. Whether perinodal blood supply can be damaged by the ablation lesions in our study warrants a long-term study.

This study was designed to pace the targeted isolated atrial segments. We cannot completely rule out the possibility that we might have paced the AV node directly (or more likely the proximal part of the AV node in the slow pathway region) rather than perinodal atrial muscular tissues, especially when the pacing lead was implanted near the AV node. The observation that similar results were obtained by pacing from different sites within the isolated area suggests that it is likely that we paced perinodal atrial tissue rather than the AV node directly. Further, the observation of Wenckebach AV conduction during pacing from the isolated atrial segment suggests that direct His bundle pacing was unlikely in the present experiments. In either case (capturing perinodal tissue vs. capturing the AV node directly), the clinical significance of our study remains the same.

All of the procedures in this study were performed under direct video imaging visualization of all the related cardiac structures. Although there were no difficulties in isolating the tissue surrounding the AV node and the His bundle, and in implanting a pacemaker lead into the isolated tissue in the heart model, it may nevertheless prove to be challenging to achieve such isolation and implantation clinically. Fortunately, 3-dimensional mapping systems are now widely available and can be used to facilitate such a procedure. There are some differences in the anatomical structure and specialized conduction system in humans compared with the swine heart, and these may also contribute to difficulty in performing such procedures in humans. However, our initial experience in humans suggests that the anatomical differences are relatively minor and leads us to believe that this technique for effecting a physiological pacing site is feasible and practical.

Other potential clinical difficulties may include: 1) the need for ablation of the anterosuperior input to the AV node; 2) the need for ablation of the potential left-sided AV nodal inputs; and 3) concern about the mid-term and long-term persistence of both AV block and isolated atrial segment viability for pacing. These issues need to be clarified in humans, although they seem not the case in this acute animal study.

Conclusions

This study was performed in an isolated working heart that has been shown to be an excellent model for studying both electrical and mechanical physiology similar to that in vivo (5). Despite the small number of animals studied, we believe that the findings validate the feasibility of the concept; namely, that a small segment of atrial tissue adjacent to the AV junction can be electrically isolated from the remainder of the atria and serve as a novel site for providing stable physiological pacing for patients undergoing AV junction ablation as part of the treatment strategy for refractory atrial fibrillation.

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Reprint requests and correspondence: Dr. Fei Lü, Cardiac Electrophysiology Laboratories, Cardiovascular Division, MMC 508, University of Minnesota, 420 Delaware Street SE, Minneapolis, Minnesota 55455. E-mail: luxxx074@umn.edu.

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