Low-energy transvenous ablation of the canine atrioventricular conduction system with a suction electrode catheter

SANJEEL SAKSENA, M.D., PETER P. TARJAN, PH.D., SAROA BHARATI, M.D.,
BIRINDER BOVEJA, B.S., DONALD COHEN, M.S., THOMAS JOUBERT B.S., AND MAURICE LEV, M.D.

ABSTRACT A single suction electrode catheter was used for His bundle electrogram recording, His bundle pacing, and low-energy (20 or 30 J) His bundle ablation in seven dogs. The suction electrode catheter was actively fixed to the atrial endocardium at the His bundle level. Electrophysiologic studies were performed in the control state, immediately after, and late (> 40 days) after His bundle ablation and results were correlated with histologic findings in the conduction system. Unipolar His bundle recording and pacing were successfully performed in all dogs with the suction electrode catheter before and after ablation. Complete heart block developed after a single 20 J shock delivered via the suction electrode catheter in all dogs immediately, but reverted to 1:1 atrioventricular conduction with first-degree atrioventricular block in two dogs in which one or two additional shocks (20 or 30 J) produced complete heart block. Mean ablation energy per shock was 22 ± 4 J. The mean total delivered energy per dog was 31 ± 20 J. Late electrophysiologic study in all dogs showed persistent complete heart block in five dogs and paroxysmal second-degree or third-degree atrioventricular block in two dogs. Gross examination of the ablation site showed a white plaque above the medial tricuspid leaflet (1.4 to 2.0 cm long and 0.4 to 0.6 cm wide). Microscopically, fibrosis of the penetrating and branching His bundle was seen in all dogs, with minimal atrioventricular node and atrial involvement. Significant proximal right bundle branch fibrosis was observed in the two dogs receiving one or two additional shocks. We conclude that the suction electrode catheter permits repeated His bundle recording, pacing, and ablation with a single catheter. Permanent and safe low-energy ablation of the canine His bundle is feasible. Focal injury localized to the target area in the conduction system can be obtained.


TRANSVENOUS CATHETER ABLATION of the atrioventricular (AV) junction is used to interrupt AV conduction experimentally and in patients with refractory supraventricular tachyarrhythmias.1–4 Current experimental and clinical techniques use high-energy (>250 J) direct-current shocks delivered through floating electrode catheters.4,5 These methods can produce permanent AV block, but have limited precision because of the floating catheter used. They provide little opportunity to control the location and extent of ablative effects or reduce energy requirements for successful ablation. Permanent pacemaker implantation is usually necessary, and often hitherto active patients become pacemaker dependent. Major complications are encountered when high-energy shocks are delivered in thin-walled structures such as the coronary sinus. Myocardial depression and serious cardiac arrhythmias have also been reported.6–8 These problems have limited the application of the technique. In this study, we used a specially designed active fixation electrode catheter in an attempt to provide increased precision and reduce energy requirements for ablation of AV conduction. Herein we report the short- and long-term electrophysiologic effects of low-energy (<30 J) direct-current shocks for ablation of the canine AV conduction system with a suction electrode catheter and correlate these with the histologic changes in the AV conduction system.

From the Section of Cardiac Electrophysiology, Division of Cardiology, Newark Beth Israel Medical Center, University of Medicine and Dentistry of New Jersey–New Jersey Medical School, Newark, and the Congenital Heart and Conduction System Laboratory, the Department of Pathology, Deborah Heart and Lung Center, Browns Mill, NJ. Supported in part by grant HL30558-04 from the National Institutes of Health, NHLBI, Bethesda.

Address for correspondence: Sanjeev Sakseya, M.D., Co-Director, Division of Cardiology, Newark Beth Israel Medical Center, 201 Lyons Ave., Newark, NJ 07112.

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Materials and methods

Description of suction electrode catheter (figure 1). The suction electrode catheter used in this study was fabricated by modifying a Cordis torque-controlled No. 7F angiographic catheter that has a central lumen with an end hole (figure 1, A). The catheter was modified according to canine right atrial and ventricular anatomy. Modifications introduced were as follows: (1) A distal ring electrode was placed at the catheter tip that could withstand direct-current discharges up to 100 J. This
electrode was insulated except for the most distal 0.5 mm segment. (2) A proximal ring electrode was placed 13 mm from the catheter tip. (3) The distal end of the catheter was shaped with curves in two perpendicular planes (figure 1, B). This provided easy maneuverability and maintained an orthogonal orientation with the tissue surface from which the recording was to be obtained. Thus, application of suction provided a seal between the tissue surface and the distal electrode ring. Luminal diameter was not altered by these modifications, with the catheter diameter at the distal electrode being 1.5 mm and area being 1.8 mm².

**Experimental technique.** Seven mongrel dogs weighing 12 to 28 (mean 18 ± 5) kg and without preexisting cardiac arrhythmias were used in this study. After induction of anesthesia with sodium pentobarbital, each dog was intubated and ventilated with a mechanical ventilator. The right femoral vein was cannulated by a sterile technique. The suction electrode catheter was flushed and the catheter lumen filled with heparinized saline. The catheter was connected to a manifold via a stopcock and this was linked to a hand-powered suction pump with a vacuum gauge (Metyvac, Neward Enterprises, Cucamonga, CA). The suction catheter was advanced under intermittent fluoroscopic guidance to the right atrium and was positioned just above the septal leaflet of the tricuspid valve. The internal jugular vein was cannulated and bipolar temporary pacing electrode catheters were positioned in the right ventricle and the right ventricular apex. A large R₂ skin-patch electrode (R₂ Corporation, Skokie, IL) was placed over the posterior left thorax over the spine between the scapulae. This was used as the indifferent electrode during unipolar His bundle recording and ablation.

Hard-copy surface electrocardiographic (ECG) and intracardiac recordings were obtained on an eight-channel recorder with variable paper speeds up to 500 mm/sec. Intracardiac recordings were also monitored on a three-channel Tektronix oscilloscope with three differential inputs and sweep rates of 10 to 1000 ms/div. Pacing was performed with a custom-made programmed stimulator (Compensicor, Cordis Corporation, Miami, FL) that permitted stimulation by the extrastimulus and burst pacing techniques as well as recording from the stimulating electrode within a few milliseconds. In addition, a custom-made software programmed stimulator (Cordis Corp, Miami, FL) was used. A standard defibrillator (PhysioControl Model 2852, Lifepak 65) was calibrated to deliver direct-current shocks of 10, 20, 30, and 50 J. The cathodal output of the defibrillator was connected via an adapter to the distal terminal of the suction electrode catheter and the anodal output was connected to the R₂ skin-patch electrode.

Standard lead configurations of two surface ECG recordings were monitored simultaneously with intracardiac recordings. The latter were displayed and typically filtered at 10 to 3000 Hz. Recording sites included the right atrial appendage, the His bundle location, and the right ventricular apex. Initially the suction electrode catheter was manipulated to obtain the largest unipolar His bundle electrogram and suction was applied. A stable His bundle recording was obtained. Programmed atrial stimulation with the extrastimulus and burst pacing technique was performed. Unipolar His bundle pacing from the distal catheter electrode was performed to verify catheter location. Once His bundle pacing was achieved (defined as an identical QRS complex and a stimulus to V interval during His bundle pacing equal to the HV interval in sinus rhythm), the ablation procedure was undertaken.

With the use of the switch box, a 20 J unipolar cathodal shock was delivered through the suction electrode catheter. If second- or third-degree AV block was achieved, the animal was observed for a period of 60 min. Programmed atrial stimulation was repeated. After observation, if persistent second- or third-degree AV block was noted, a permanent transvenous ventricular demand pacemaker was implanted. If persistent high-degree AV block was not achieved, the procedure was repeated and another 20 or 30 J ablation shock was delivered. The animal was allowed to recover from the operation and periodic (bimonthly) surface ECG recordings were obtained over the next 2 months. After 40 to 60 days, the dogs underwent repeat electrophysiologic studies to determine the long-term effects of the ablation on AV conduction. At the end of the procedure, the animals were killed with intravenous potassium chloride. The heart was removed and rinsed. It was examined grossly for tricuspid valvular damage, right atrial dilatation, and right ventricular dilatation. After visual inspection, photographs of the gross anatomy were obtained. In four animals, the approaches to the AV node, the AV node, the AV bundle, and the bundle branches were serially sectioned and every fourthieth section was retained. Alternate sections were stained with hematoxylin-eosin and Weigert–van Gieson stains. In this manner 600, 548, 564, and 524 sections were studied in dogs 4, 5, 6 and 7, respectively. Additional regions analyzed included the aortic and tricuspid valves, the central fibrous body, and the summit of the ventricular septum.

**Results**

A total of 10 direct-current shocks were delivered in the study group. The mean number of shocks delivered was 1.4 ± 0.8 per dog, and the mean energy per shock was 22 ± 4 J. The mean energy delivered per dog was 31 ± 20 J. All animals tolerated the ablation procedure well without sequela. After ablation, permanent single-chamber ventricular demand pacemakers were implanted in all animals. There was no mortality in the study group during a mean follow-up period of 66 (range 41 to 92) days. No major hemodynamic or embolic complications or symptomatic tachyarrhythmias (e.g., syncope or sudden death) were observed in this period. No arrhythmogenic effects of any of the QRS synchronized shocks were observed. In one dog, an inadvertently delivered asynchronous 20 J shock delivered in the ST segment induced ventricular fibrillation, which was reverted by external defibrillation.

**Short-term electrophysiologic effects of low-energy suction ablation (table 1).** In the control electrophysiologic study, the sinus cycle length in the study group ranged from 320 to 540 (mean 396) msec. The AH intervals ranged from 50 to 70 (mean 60 ± 8) msec and the HV interval varied from 30 to 40 (mean 35 ± 5) msec (table 1). His bundle pacing was achieved in all animals with pacing stimulus to right ventricular electrogram intervals equal to or slightly greater (<10 msec) than the HV interval.

The initial 20 J shock produced immediate complete heart block in all animals. This was localized to the His bundle and infra-His system in all dogs. Five animals remained in complete heart block for the rest of the experiment (>60 min), while 1:1 AV conduction re-
turned in two animals. A new bundle branch block pattern appeared in one of these dogs and first-degree AV block was noted in the other animal. The procedure was repeated with an additional 20 J shock in each dog (Nos. 6 and 7). In both animals, complete heart block followed the second shock and persisted in the latter dog (No. 7) until the end of the experiment. In the former dog (No. 6), 1:1 AV conduction returned with marked HV interval prolongation and right bundle branch block. A third 30 J shock was delivered. This resulted in complete heart block that persisted for the duration of the experiment.

Figure 2 shows the sequence of events in a typical experiment. ECG and intracardiac recordings in dog 5 are shown before, during, and after suction ablation. Before ablation (panel A), sinus rhythm at a sinus cycle length of 345 msec was observed, with a QRS duration of 50 msec. The AH interval was 60 msec and the HV interval was 30 msec, as recorded by the unipolar suction electrode. A single 20 J shock was delivered

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Weight (kg)</th>
<th>Rhythm</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>Ablation</th>
<th>AV conduction after suction ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>17</td>
<td>Sinus</td>
<td>50</td>
<td>40</td>
<td>Yes; 20 shocks</td>
<td>3rd AVB, 3rd AVB</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>Sinus</td>
<td>60</td>
<td>32</td>
<td>Yes; 20 shocks</td>
<td>3rd AVB, 3rd AVB</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>Sinus</td>
<td>50</td>
<td>32</td>
<td>Yes; 20 shocks</td>
<td>3rd AVB, 3rd AVB, Paroxysmal AVB</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
<td>Sinus</td>
<td>65</td>
<td>40</td>
<td>Yes; 20 shocks</td>
<td>3rd AVB, 3rd AVB, Paroxysmal AVB</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>Sinus</td>
<td>60</td>
<td>30</td>
<td>Yes; 20 shocks</td>
<td>3rd AVB, 3rd AVB</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>Sinus</td>
<td>70</td>
<td>30</td>
<td>Yes; 20, 20, 30 shocks</td>
<td>2nd AVB, 1st AVB, Paroxysmal 2nd AVB</td>
</tr>
<tr>
<td>7</td>
<td>16</td>
<td>Sinus</td>
<td>65</td>
<td>40</td>
<td>Yes; 20, 20 shocks</td>
<td>2nd AVB, 1st AVB, Paroxysmal 2nd AVB</td>
</tr>
</tbody>
</table>

AH and HV intervals at baseline were within normal limits. AVB = AV block.

This table shows the clinical and electrophysiologic observations before and after suction ablation.

### FIGURE 2

**PRE-ABLATION**

**30 MINUTES POST-ABLATION**

**60 DAYS POST-ABLATION**

A. Baseline electrocardiogram (standard lead I), right atrial electrogram (RA), and His bundle electrogram (HBE) before ablation in dog 5. The QRS complex is 50 msec in duration. A = atrial electrogram; H = His bundle electrogram; V = ventricular electrogram. B. ECG, RA, and HBE obtained after suction ablation with a single 30 J direct-current shock. Note the development of complete heart block with atrioventricular dissociation. The QRS complex is 70 msec in duration and is minimally altered in its terminal vector. A His bundle deflection follows each atrial depolarization with an AH interval of 60 msec, localizing the level of block to His bundle or at the fascicular level. C. ECG recordings from the same dog after 60 days showing complete heart block with a junctional or high fascicular escape rhythm. The atrial rate is 150 beats/min. and the ventricular rate is 43 beats/min.
through the same electrode. After ablation, His bundle recordings were obtained from the same electrode (panel B). Third-degree AV block and proximal His bundle deflection with an AH interval of 50 msec were noted. The escape QRS complex was 50 msec in duration, which could be consistent with a distal His/fascicular origin. Two months later the animal was in complete heart block with a sinus cycle length of 360 msec and an escape rhythm cycle length of 1140 msec. The QRS duration of the escape rhythm was 70 msec, suggesting an origin at the distal His/fascicular level.

**Long-term chronic electrophysiologic effects of low-energy suction ablation.** Follow-up electrophysiologic evaluation was performed 41 to 92 (mean 66) days after suction ablation. At this time, five animals were in complete heart block with sinus rates ranging from 150 to 210 beats/min and escape rhythms with rates of 16 to 65 beats/min. The escape rhythm was always slower than ventricular pacing, and the animals were completely paced at a demand rate of 72 per minute. In one animal (No. 7), 1:1 AV conduction and first-degree AV block was present at sinus rates below 60/min with paroxysmal high-degree AV block being observed at higher rates. The atrial pacing cycle length producing

**TABLE 2**

**Conduction system**

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Gross</th>
<th>Approaches to AV node</th>
<th>AV node</th>
<th>AV bundle penetrating</th>
<th>AV bundle branching</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Whitish plaque between coronary sinus and TV leaflet, 1.4 × 1.4 cm. Whitish plaque in limbic area, 1 × 0.4 cm, involving middle preferential pathway. At end of anterior preferential pathway, hemorrhagic zone 0.4 cm. Membranous septum thickened at base of right and noncoronary cusps. Summit of VS thickened and whitened.</td>
<td>Fatty infiltration and focal degeneration of myocardium.</td>
<td>Slight-to-moderate fibrosis. Fatty and mononuclear cell infiltration.</td>
<td>Junction of node and bundle converted into granulation tissue. Markedly deformed, compressed and completely destroyed.</td>
<td>Completely destroyed beneath aorta, then normal, then fibrosis.</td>
</tr>
<tr>
<td>5</td>
<td>Thickening and whitening throughout medial leaflet of TV with edema. Thickening extending from coronary sinus to medial leaflet measuring 2 × 0.5 cm.</td>
<td>Endocardial hypertrophy, sclerosis, and chronic inflammation. Atrial musculature to TV showed marked fibrosis with cartilage formation.</td>
<td>Slight to moderate fibrosis.</td>
<td>Surrounded by fat in beginning. Marked fibrosis and fatty infiltration. Cartilage formation in endocardium.</td>
<td>Marked fibrosis and fatty infiltration. Compressed by immense edema.</td>
</tr>
<tr>
<td>6</td>
<td>Whitish thickened areas between coronary sinus and annulus of medial leaflet of TV measuring 1.4 × 0.5 cm. Swelling of valve.</td>
<td>Fatty infiltration, myocarditis, and degeneration of muscle. Nerves involved in inflammation.</td>
<td>Moderate fibrosis. Node surrounded by basophilic fat necrosis.</td>
<td>Surrounded by fat. Increasing fibrosis until complete.</td>
<td>Marked fibrosis.</td>
</tr>
<tr>
<td>7</td>
<td>Whitish plaque from eustachian valve to TV, 2 × 0.6 cm. Hematoma in med. leaflet of TV scar distal and adjacent to right atrial appendage.</td>
<td>Focus of endocardial hypertrophy and sclerosis proximal to tendon of Todaro. Fatty infiltration.</td>
<td>Slight fibrosis in beginning increasing to marked.</td>
<td>Marked fibrosis. Cartilage adjacent and compressed by cartilage. Chronic inflammation.</td>
<td>Marked fibrosis.</td>
</tr>
</tbody>
</table>

LBB = left bundle branch; RBB = right bundle branch; VS = ventricular septum; CFB = central fibrous body; TV = tricuspid valve.
AV Wenckebach block in this animal was 800 msec. In the one remaining dog (4) that had developed complete heart block after a single 20 J shock; 1:1 AV conduction resumed with a moderate increase in the AV interval (33 msec) and PR interval (20 msec) from preablation values. The atrial pacing cycle length producing AV Wenckebach block was 300 msec. Right bundle branch aberrancy that had been absent before ablation developed during atrial pacing. Bradycardia-dependent second-degree AV block was intermittently observed.

Pathology of low-energy suction ablation. In four animals (Nos. 4 to 7), detailed pathologic examination of the canine AV conduction system and adjoining tissues was performed. On gross examination, a whitish plaque was observed situated between the coronary sinus ostium and the tricuspid valve in all animals (table 2). The plaque typically extended to the tricuspid annulus and medial leaflet and ranged from 1.4 to 2.0 cm in length and from 0.4 to 0.5 cm in width. Figure 3 shows the lesion in dog 5, whose electrophysiologic findings are shown in figure 2. Note the focal lesion above the tricuspid leaflet. The detailed microscopic findings in each dog are shown in table 2. In

<table>
<thead>
<tr>
<th>LBB</th>
<th>RBB</th>
<th>Summit of VS</th>
<th>CFB</th>
<th>Aortic valve</th>
<th>Tricuspid valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completely destroyed at base, then normal, then fibrosis.</td>
<td>In beginning, slight fibrosis. Then normal.</td>
<td>Tremendous fibrosis on left side with zones of calcification. Entire summit replaced by old connective tissue. Right side more distally showed tremendous fibrosis with destruction of myocardium.</td>
<td>Marked fatty infiltration.</td>
<td>Marked increase and vacuolization of spongiosa.</td>
<td>Marked fatty infiltration with increase in spongiosa. Eventually, tremendous swelling and vacuolization of entire valve.</td>
</tr>
</tbody>
</table>
FIGURE 3. Gross appearance of lesion. Note the area of thickening and whitening of the medial leaflet of the tricuspid valve that extends to the area of the AV node and His bundle. RA = right atrium; TV = tricuspid valve; RV = right ventricle; C = coronary sinus. Arrows point to fibrosed area.

Discussion

Experimental and clinical studies have established the efficacy of catheter ablation of the AV conduction system. In general, the approaches to the AV node showed minimal focal degeneration or fatty infiltration. The AV node showed slight fibrosis in the beginning, which increased progressively. In all animals the penetrating and branching portions of the bundle of His were almost completely or completely destroyed and replaced by fibrous tissue (figure 4), the proximal segments of the left bundle branch showed marked fibrosis, and the proximal segments of the right bundle branch showed mild fibrosis. The proximal segment of the right bundle branch showed severe fibrosis in dogs 6 and 7. The peripheral bundle branches were normal in all animals, but there was marked fatty infiltration or degeneration of the central fibrous body. The base of the tricuspid and aortic valves showed an increase in amount and vacuolization of the spongiosa (figure 4), but the integrity of the valve rings and leaflets was preserved. Calcification or cartilage formation was seen in all dogs. Fibrosis of the summit of the ventricular septum was likewise present in all dogs.

Correlation of electrophysiologic and histologic effects (table 3). In these animals, His bundle recordings were obtained after ablation. Intra-Hisian block demonstrated as a proximal His deflection without a distal recording was present in three dogs. In the fourth animal, differentiation from infra-His block was not reliably possible. In all animals severe His bundle deterioration and fibrosis of the penetrating and branching portions was observed, correlating with the electrophysiologic findings. In dog 6, spontaneous right bundle branch block was observed after ablation, and in dog 7, atrial pacing elicited right bundle branch aberrancy. In both animals, severe fibrosis of the proximal right and left bundle branches was observed. Since more than one ablation shock was delivered to achieve AV block in these two dogs, multiple sites of conduction system damage would be expected. However, histologic findings in dogs resuming intermittent AV conduction (4 and 7) and those failing to do so (5 and 6) were similar.
FIGURE 4. Left, Low-power view of the fibrosed penetrating His bundle and cartilage in the anulus of the tricuspid valve, the small fibrotic scar on the summit of the ventricular septum, and the swelling of the spongiosa of the tricuspid and aortic valves. Weigert-van Gieson stain x 14.25 original magnification. B = Bundle of His; TV = tricuspid valve; AV = aortic valve; V = ventricle; C = cartilage. Arrows point to the fibrotic lesion in the ventricular septum. Right, Enlargement of left. Weigert-van Gieson stain, original magnification x 46.5.

TABLE 3
Correlation between electrophysiologic (EP) and histologic findings after low-energy suction ablation

<table>
<thead>
<tr>
<th>EP observations</th>
<th>Severity of chronic AV block</th>
<th>Histologic findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog No. Location of AV block</td>
<td>P 2° or 3° AVB</td>
<td>Slight-to-moderate fibrosis with fatty and mononuclear cell infiltration of AV node. Complete destruction of AV bundle and beginning of LBB. Slight fibrosis of beginning of RBB.</td>
</tr>
<tr>
<td>4 Intra and/or infra-His</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Intra-His</td>
<td>3° AVB</td>
<td>Slight-to-moderate fibrosis of AV node. Marked fibrosis and fatty infiltration of AV bundle. Destruction of beginning of LBB and of RBB.</td>
</tr>
<tr>
<td>6 Intra- and infra-His, RBBB</td>
<td>3° AVB</td>
<td>Moderate fibrosis of AV node. Marked fibrosis of AV bundle and LBB and RBB.</td>
</tr>
<tr>
<td>7 AV node and intra-His</td>
<td>1° and P 2° AVB</td>
<td>Marked fibrosis of AV node, bundle, and LBB; moderate fibrosis and fatty changes of RBB.</td>
</tr>
</tbody>
</table>

Note the location of the injury correlated directly with the level of AV block or bundle branch block induced by the shocks.

P = paroxysmal; RBBB = RBB block; others as in table 2.
system with high-energy direct-current shocks.\textsuperscript{1-5, 10} This technique employs a floating electrode catheter for energy delivery. In a significant proportion of patients, multiple high-energy shocks are required to achieve adequate ablation.\textsuperscript{10} The incidence of complications and adverse reactions increases rapidly when repeated high-energy direct-current shocks are delivered, particularly in patients with compromised left ventricular function.\textsuperscript{6,7,10,11} There is little control of the extent of electrophysiologic effects and even less precision in limiting the injury to the target structures. The high-energy ablation technique is not applicable to ablation attempts in thin-walled cardiac structures such as the coronary sinus because of risk of rupture and bleeding.\textsuperscript{6} Serious myocardial depression and cardiac arrhythmias have been induced after multiple high-energy shocks, resulting in fatalities.\textsuperscript{8,10,11} This has severely limited the use of the technique for accessory pathway ablation in locations other than the posteroseptal right ventricle due to safety concerns. This is particularly unfortunate since myocardial ablation is more readily achieved in the normal rather than in the diseased ventricle.\textsuperscript{12,13} These adverse effects are probably related to the high-pressure shock wave generated by the high-energy direct-current shock. In addition to the shock wave, a light emission and a rise in temperature at the electrode tip have been described.\textsuperscript{14,15} Temperatures in excess of 1700\degree C have been reported, which can result in the disruption of platinum electrodes. In some instances, arcing between electrodes with temperatures above 5000\degree C has been suggested.

To minimize these undesirable effects, we examined the feasibility of low-energy transvenous ablation of the AV conduction system using an active fixation catheter. However, we sought to maintain the ability to record and pace from the ablation site without local damage due to the active fixation process. This eliminated the need for a screw-in lead, as has been suggested.\textsuperscript{8,15,16} Suction electrodes have been used for recording intracardiac electrograms and monophasic action potentials in vitro and in vivo\textsuperscript{17,18} and His bundle pacing has been performed with a floating electrode catheter. Thus, use of a suction electrode for His bundle recording and pacing appeared feasible. In this study, the suction electrode was devised with two curves to remain perpendicular to the recording surface.\textsuperscript{9} This configuration permitted stable His bundle recordings and pacing in dogs.

Unipolar recordings are usually preferred for localization of the ablation site, and with this suction electrode unipolar electrogram recordings can be obtained directly from a potential ablation site. In addition, verification of electrode location can be obtained by pacing the potential ablation site, e.g., the His bundle. On removal of suction, permanent damage to the underlying myocardium is avoided, since AV conduction is preserved before, during, and after application of suction. Thus, repeated attempts at localization with electrogram recordings for identification of a potential ablation site is feasible. In this respect, reversible active fixation techniques, such as suction may be preferable to screw-in electrodes, such as Helixfix electrodes.\textsuperscript{15} However, a larger experience will be necessary to evaluate these issues.

This study has demonstrated that low-energy direct-current shock ablation of the His bundle region is feasible with a suction electrode catheter. In our experience with other ablation energies we noted that low energies (< 100 J) produce myocardial ablation of a magnitude that could be adequate for His bundle ablation.\textsuperscript{12} In this report, energies of 30 J or less have been used to achieve ablation. Previous studies with active fixation electrodes for His bundle ablation have used high-energy direct-current shocks, usually in excess of 100 J.\textsuperscript{19} A recent clinical study used medium-energy shocks and floating electrode catheters for AV junctional ablation. In this study, several shocks with total delivered energy ranging from 50 to 170 J were required for AV junctional ablation.\textsuperscript{20} Furthermore, detailed pathologic correlation has been established.

The lesion size in our experiments was of a magnitude that was suitable for His bundle and accessory pathway ablation. The lesion obtained was focal with destruction localized to the target tissues only. This limited the damage to surrounding structures and permits sparing of ventricular and valvular function. The long-term pathologic effects of the lesion were established in this study. No evidence of thrombosis, widespread ventricular or valvular damage, or myocardial rupture was noted. The lesion healed well and produced a strong and organized scar by the time of sacrifice. The short-term arrhythmogenic effect observed in one dog can be ascribed to improper shock timing. While chronic arrhythmogenicity was not systematically examined, no sudden clinical arrhythmic event was recorded. It is useful to compare the effects on the conduction system of the methods used in this experiment with the effects of electric shock.\textsuperscript{2,5,16} In general, in this experiment the effects were more localized to the conduction system than those in previous studies. In addition, the myocardium close to the conduction system was less involved in the present experiments. For purposes of modification of AV conduction, use of still lower energy shocks or ablation of other sites in
the specialized conduction system such as the AV node should be examined.

Potential role of suction ablation. The successful development of catheter ablation techniques is dependent on three major factors: (1) precise control of the ablation energy, (2) the catheter delivery system, and (3) the nature of the anatomic substrate to be ablated. A variety of different energy sources could be suitable for the ablation procedure, but comparative evaluation of these energy sources is currently limited. This report addresses improvements in the catheter energy delivery system. Localization of ablation energy delivery to the target tissues will permit greater precision in catheter ablation techniques and limit adverse effects. The low-energy transvenous suction ablation technique has been applied to the His bundle region in this study. This technique permits repeated recording, pacing, and low-energy shock delivery. It could also be particularly valuable if modification rather than interruption of conduction is sought. We have reported that this is clinically feasible with low-energy pulsed laser energy delivery techniques. We have reported modification of AV nodal conduction by an asynchronous high-energy direct-current shock or pulsed argon laser discharges in patients with intranodal reentrant tachycardia, which obviated the need for permanent pacemaker implantation during follow-up. Combination with alternative energy sources, such as laser energy or radiofrequency current could also be feasible with the suction ablation concept. Further evaluation of this approach is warranted.

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