Histologic changes and arrhythmogenicity after discharge through transseptal catheter electrode

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ABSTRACT  Ventricular tachycardia commonly arises within the intraventricular septum and successful catheter ablation of septal tachycardia might be enhanced by transseptal electrode placement. We have evaluated the safety of a transseptal ablation procedure. Arrhythmogenicity and histology were examined after high-energy capacitor discharges were delivered to an intracavitary cathode-anode pair placed on opposite sides of the interventricular septum in pentobarbital-anesthetized dogs. After two discharges of 200 or 100 J proved lethal, paired discharges of 30 or 50 J (10 dogs) or a single discharge of 100 J (four dogs) was used to induce 28 lesions. Acute rhythm changes and risk of induction of ventricular tachycardia by programmed stimulation were measures of arrhythmogenicity. Gross and histologic examination of the hearts after 20 min to 28 days allowed characterization of the evolution of lesions. The conduction system in nearby and remote locations was extensively examined in four dogs. Refractory ventricular fibrillation developed with paired shocks at 200 or 100 J. At lower energy levels, acute ventricular fibrillation occurred with 12 of 20 shocks (60%), but defibrillation was consistently achieved. After ablation, no dog had ventricular tachycardia or fibrillation induced with programmed stimulation. Matching anodal and cathodal lesions spanned the septum without perforation in 10 of 16 dogs, and the lesions were of similar histology. Each contained central areas of hemorrhage surrounded by a region of coagulation necrosis merging with normal myocytes peripherally. There was necrosis and edema without inflammation at 20 min, acute inflammatory cell infiltration at 1 to 2 days, and myocyte replacement by granulation tissue after 6 days. Right ventricular apical perforation occurred near the septum without tamponade in one dog after a single 100 J discharge. There was no damage to the atrioventricular node, His bundle, or bundle branches in four dogs. We conclude that low-energy, paired-capacitor discharges can be safely applied through catheter electrodes that straddle the ventricular septum. Permanent cathodal and anodal injury results, without structural damage to the proximal conduction system.


LEFT SEPTAL endocardial resection has been used for control of arrhythmias in patients with ventricular tachycardia arising from the ventricular septum.1–3 For those patients with an intramural septal location of the ventricular tachycardia focus, a more extensive ablative procedure might be required for optimal results. Cheirif et al.,4 for example, found earliest breakthrough over the right septum in patients with septal ventricular tachycardia foci. Recently, catheter electrocautery has been developed as a method for ablation of arrhythmogenic tissue, to avoid the need for thoracotomy in high-risk patients.5,6 Typically, energy is applied between an intracavitary cathode and an anode positioned externally on the chest wall. We have evaluated a new technique for possible ablation of septal ventricular tachycardia in which energy is delivered between intracavitary electrodes contacting the left and right septum. Catheter electrodes are positioned at early tachycardia activation sites on both sides of the ventricular septum. We reasoned that successful localization and ablation might be enhanced if tissue mediating the tachycardia were straddled by electrodes on both sides of the septum. To test the safety of this approach, a study was performed to determine lesion morphology, degree of transseptal injury, and risk of

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septal perforation after this type of ablation. We also assessed the arrhythmogenicity of acutely delivered shocks and the potential for induction of ventricular tachycardia 2 to 28 days after shock-induced damage. Our overall objective was to define an energy level and electrode polarity that produced transseptal damage without perforation or risk of induction of lethal arrhythmias.

Methods

Sixteen adult mongrel dogs weighing 19 to 35 kg were anesthetized with 30 mg/kg intravenous pentobarbital and intubated to maintain airway patency. A jugular vein and carotid artery were isolated aseptically and six French bipolar electrode catheters (United States Catheter and Instrument Corporation, Billerica, MA) containing 5 × 2 mm platinum electrodes separated by 1 cm were positioned on each side of the interventricular septum under direct vision and by simultaneous biplane fluoroscopy. Silk sutures (2-0) fixed the proximal catheter to its vessel, to minimize catheter migration between shocks. In one dog (No. 4), the catheter moved between shocks despite proximal fixation, and visual repositioning under biplane fluoroscopy was required. Direct-current (DC) capacitor discharge was developed with use of a standard defibrillator (Life-Pak 5, Physio-Control, Redmond, WA) with custom-made cables. Cinemagrams of electrode position were obtained on 16 mm film for each dog. Surface electrocardiographic lead I or II and an intracardiac electrogram were recorded on photographic recording paper with an Electronics for Medicine DR-8 oscilloscopic recorder at paper speed 50 mm/sec.

After duplicate 200 J and two 100 J shocks caused lethal ventricular fibrillation in two dogs (see Results), duplicate transseptal discharges of 30 or 50 J were given to the next 10 dogs. To evaluate the histology and risk of perforation after higher energy shocks, the last four of 16 dogs received a single 100 J discharge. To discourage acute ventricular fibrillation, each of these four dogs was treated with 10 mg/kg lidocaine before transseptal ablation. DC discharges were delivered during sinus rhythm, and were synchronized to fall during QRS inscription. If postshock ventricular fibrillation occurred, transhoracic rescue defibrillation (200 J) was used to restore a hemodynamically stable rhythm. Duplicate shocks were separated by 30 min. The influence of electrode polarity was also studied. The distal electrode of the left ventricular catheter was connected to the cathodal output of the defibrillator and the distal right ventricular electrode was connected to the anodal sink of the defibrillator in 10 dogs. In six other dogs polarity was reversed; i ventricular cathode and left ventricular anode were used.

Arrhythmogenicity. Arrhythmogenicity of high-energy discharges was assessed in two ways: (1) acute rhythm response to the DC shock, and (2) latent vulnerability to inducible ventricular tachycardia or fibrillation. The acute rhythm response was recorded as nonsustained ventricular tachycardia (≥6 repetitive complexes of less than 30 sec in duration), sustained tachycardia (≥30 sec), or ventricular fibrillation.

Latent vulnerability to ventricular tachycardia was assessed with the use of programmed extrastimulation of the left ventricle. No attempt was made to pace the actual ablation site. Inducibility of tachycardia before shock was compared with inducibility 2 to 28 days after DC shock with the use of an electrode catheter (USCI), a programmable stimulator with an isolated constant current source (DTU-101, Bloom and Associates, Redding, PA), and standard pacing techniques. Programmed stimulation was performed only in the 10 dogs that received 30 to 50 J duplicate shocks and survived at least 2 days. Since inducible ventricular tachycardia does not develop less than 2 days after ischemic injury in the canine heart,6,9 we reasoned that a similar low yield might occur 1 to 2 days after electrocautery. Thus, in six of 16 dogs killed after less than 2 days (two dogs), receiving a single 100 J transseptal shock (two dogs), or both (two dogs), programmed stimulation was not performed.

After the dogs were killed, the heart of each was removed and fixed in phosphate-buffered formalin (pH 7.0). In four dogs in which ablation catheters were fluoroscopically positioned at the mid-to-high septum, the specialized ventricular conduction system was carefully examined for involvement as a result of these shocks.

Morphologic examination. Ablation lesions were carefully examined in 10 hearts* and the conduction system was examined in four hearts. During gross examination, each heart was serially sectioned in 3 mm slices proceeding from the apex to the base of the ventricles. The sections were visually examined and photographed. Left ventricular and right ventricular septal lesions were identified and measurements were made directly from the fixed lesions. The area of injury in each section was determined by planimetry counting the number of "hit points" on a superimposed grid of squares (figure 1). The area of each square was calculated as (A) = (s − 1),2 where s = length of each side of a square.10 Lesion volume was then calculated as the number of 3 mm thick sections with visible injury multiplied by the sum of injured areas:

\[ V = \sum_{i=1}^{N} A_i \]

The diameter of each lesion in the visibly largest section was also measured.11 Representative samples from 2-, 4-, and 8-day-old lesions were embedded in paraffin, and sections were cut and stained with hematoxylin and eosin, Masson’s trichrome for collagen, periodic acid Schiff (PAS) for glycogen or polysaccharides, and von Kossa chloranilic acid to detect calcium12 during light microscopic examination. Ultrastructural injury was examined in one dog after a single transseptal discharge of 100 J. One hour after the injury, the left coronary artery was injected with 2.5% 0.2M cacodylate-buffered glutaraldehyde (pH 7.4) and the entire heart was submerged in the same fixative overnight (4°C). The heart was sectioned as described above and tissue from an injured region and a visibly noninvolved area was selected. Selected areas were stained with uranyl acetate and lead citrate before examination with a JEDL 100S electron microscope.

The conduction system was examined extensively in four dogs. Blocks containing the sinoatrial and atrioventricular node and the bundle and branches up to the level of the moderator band were serially sectioned and every twentieth section was retained. These were stained with hematoxylin and eosin and Weigert-van Gieson stains and in this manner, a total of 1252 sections from dog 5, 1628 sections from dog 6, 1516 sections from dog 7, and 1820 sections from dog 8 were obtained for study.

Statistical methods. Comparisons of quantitative measurements were made with Student’s t test for paired observations. Fisher’s exact test was used to compare the incidence of ventricular fibrillation at 30 and 50 J and after the initial and second shock of a pair. Data are expressed as the group mean ± SD. A

*Those from dogs 1 and 9 were examined grossly but technical problems in processing prevented estimation of the volume of damaged tissue.
The incidence of tachyarrhythmias immediately followed transseptal capacitor discharge in every dog (table 1). Severity of arrhythmia ranged from isolated ventricular ectopy to ventricular fibrillation (figure 2). Ventricular fibrillation was lethal in two initial dogs given duplicate transseptal shocks at 200 and 100 J (16 and 5.7 J/kg, respectively). In both dogs, the second shock resulted in ventricular fibrillation, difficult defibrillation, and subsequent electromechanical dissociation during ventricular pacing. For this reason, each of the next 10 dogs were given two lower energy shocks of either 30 or 50 J stored energy. Ventricular fibrillation occurred with 12 of these 20 shocks (60%) and defibrillation was consistently achieved. Defibrillation was required after two of six (33%) shocks at 30 J and 11 of 14 shocks (79%) at 50 J (p = NS). In the final four dogs given a single 100 J shock after lidocaine pretreatment (10 mg/kg), ventricular fibrillation occurred after one of the four shocks. The incidence of ventricular fibrillation was similar for the first (10/12; 83%) and second (6/12; 50%) shock (p = NS; table 1). Ventricular fibrillation occurred with the initial shock in 10 of 12 dogs (83%) and with the second shock in six of 12 (50%; p = NS) dogs (table 1). Similarly, polarity did not influence the incidence of postshock ventricular fibrillation. Defibrillation was required after 12 of 16 shocks (75%) delivered by a left ventricular cathode and six of eight shocks (75%) from a left ventricular anode (p = NS). After defibrillation or spontaneously resolving ventricular tachycardia, the incidence of ventricular ectopy gradually waned over 30 sec to 20 min. Transient atrial fibrillation lasting 30 to 120 sec was observed after six of the nine shocks but did not require defibrillation.

**Latent arrhythmogenicity.** Twelve dogs were returned to the postanesthesia recovery area in sinus rhythm. No dogs died in their cages. During control (preshock) pacing, three extrastimuli induced ventricular fibrillation in one dog. Two through 28 days after transseptal shock, no dog had inducible repetitive activity exceeding two complexes.

**Morphologic examination.** There was no qualitative difference between the anodal and cathodal lesions. Grossly, the lesions contained a red-brown to black central discoloration and a surrounding region of pale, firm tissue merging with normal-appearing myocardium (figure 3). Paired anodal and cathodal lesions extended across the intraventricular septum in eight of 10 dogs, but septal perforation did not occur (table 2).
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TABLE 1

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Energy dose (J)</th>
<th>Rhythm after shock 1/2</th>
<th>Cage death</th>
<th>Inducible VT</th>
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<tr>
<td>1</td>
<td>200 x 2</td>
<td>VF/VF, EMD</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>100 x 2</td>
<td>VF/VF, EMD</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>50 x 2</td>
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<td>No</td>
</tr>
<tr>
<td>4</td>
<td>30 x 2</td>
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<td>No</td>
</tr>
<tr>
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</tr>
<tr>
<td>6</td>
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<td>No</td>
</tr>
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<td>No</td>
</tr>
<tr>
<td>9 (&lt;LV&lt;sub&gt;AN&lt;/sub&gt;)</td>
<td>50 x 2</td>
<td>VF/VT&lt;sub&gt;ss&lt;/sub&gt;</td>
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<td>No</td>
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<tr>
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<td>VF/VF</td>
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</tr>
<tr>
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<td>50 x 2</td>
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<td>No</td>
</tr>
<tr>
<td>12 (&lt;LV&lt;sub&gt;AN&lt;/sub&gt;)</td>
<td>50 x 2</td>
<td>VF/VF</td>
<td>No</td>
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</tr>
</tbody>
</table>

LV<sub>AN</sub> = left ventricular anode; VF = ventricular fibrillation; VT<sub>ss</sub> = nonsustained ventricular tachycardia; VE = isolated ventricular ectopics or ventricular couplets; EMD = electromechanical dissociation; AF = atrial fibrillation.

Table 2

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Anodal (cm&lt;sup&gt;3&lt;/sup&gt;)</th>
<th>Cathodal (cm&lt;sup&gt;3&lt;/sup&gt;)</th>
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<td>0.7</td>
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</tr>
<tr>
<td>16</td>
<td>2.8</td>
<td>2.9</td>
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</table>

Transseptal extension occurred in three of the four dogs given a single 100 J shock. In one dog given a single 100 J transseptal shock, right ventricular (anodal) free wall perforation occurred near its junction with the apical septum, without hemopericardium or tamponade. Before shock, the catheter had been firmly wedged between the septum and free wall.

The anodal lesions were larger than cathodal lesions in eight of 10 dogs (table 2). This trend persisted whether the anode was placed in the left or right ventricle. Group mean areas were 4.9 ± 3.8 cm<sup>3</sup> for anodal and 2.8 ± 3.5 cm<sup>3</sup> for cathodal lesions, but the difference was not statistically significant. The largest diameters of the lesions were 1.38 ± 0.39 cm for the anodal and 1.19 ± 0.5 cm for the cathodal (p = NS). The four dogs given paired 50 J shocks had larger lesions than four dogs given a single 100 J shock. Group mean areas were 6.4 ± 3.2 cm<sup>3</sup> for anodal and 5.9 ± 3.5 cm<sup>3</sup> for cathodal lesions after paired 50 J shocks, and 2.4 ± 0.6 cm<sup>3</sup> and 1.0 ± 1.3 cm<sup>3</sup> after a single 100 J shock.

Histologic evolution was followed by examining anodal and cathodal lesions at different times 0 to 28 days after septal shock. Histologic change was evident

FIGURE 2. Acute ventricular fibrillation immediately followed 50 J of capacitor energy delivered between catheter electrodes placed on each side of the intraventricular septum. Transthoracic countershock promptly restored sinus rhythm.
FIGURE 3. Ablation of interventricular septal endomyocardium. The heart was fixed 30 min after duplicate 100 J discharges were delivered to the apical septum. Serial sections are in 3 mm slices moving from apex to base. The right ventricular lesion (anodal) first appears in the second section and the smaller left septal lesion (cathodal) appears in the fifth section. Each contains central dark discoloration and a surrounding halo of firm coagulated tissue merging with normal myocardium. No lesion is present above the eighth section.

within 20 to 30 min after discharge of energy. Injured myocytes had a glossy appearance, indistinct striations, and pyknotic nuclei. There was no inflammatory cell infiltration at this time and the reticular framework remained largely intact. By 2 days, there was intense infiltration of acutely inflammatory cells into areas of necrosis. Early organization was evident by day 3. After 5 to 8 days, there was nearly complete replacement of the necrotic myocardium by granulation tissue, lymphocytes, and macrophages. When electron micrographs from injured and uninjured areas were compared, sarcomere degeneration and mitochondrial intercristal densities distinguished the injured areas (figure 4). The Z bands were variably thickened and degenerated myofibrils contained minimal to absent filamentous framework. Densely clustered mitochondria contained electron-dense clumps in the intercristal spaces. Swelling of intercristal spaces was not consistently seen. Noninjured areas contained normally striated sarcomeres and mitochondria with no intercristal clumping of electron-dense material.

Conduction system. The proximal conduction system, including the sinoatrial and atroventricular node, penetrating and branching bundle, and the beginning of

FIGURE 4. Electron micrographs from uninjured and injured regions. A. Normally oriented sarcomeres with abundant mitochondria and a central nucleus. In contrast, B, from an injured area, shows significantly altered sarcomeres with variably thickened Z bands, degenerated myofibrils, and numerous mitochondria that contain electron-dense intercristal clumping. Bars = 5 μm.
the bundle branches (figure 5), were spared in all four dogs. However, the periphery of the left and right bundle branches revealed chronic inflammatory cell infiltration in three of four dogs.

Discussion

Arrhythmogenicity

Acute arrhythmogenicity. Risk of acute ventricular tachycardia or fibrillation after intense stimulation has been reported by others\textsuperscript{11, 13, 14} and appears related to the magnitude of applied electrical energy. We found that acute ventricular arrhythmias were common and that high-energy paired shocks (i.e., 200 J) were associated with lethal ventricular arrhythmias.

Lethal ventricular arrhythmias 1 to 2 days after free wall ventricular endocardial ablation in dogs has been reported by Lerman et al.\textsuperscript{15} Sudden death occurred 18 to 36 hr after DC shock in eight of 11 dogs given 100 J or more. When shock intensity was reduced to 50 J, nine of 10 dogs survived, but nonlethal ventricular tachycardia was seen during electrocardiographic monitoring in six dogs. The time and electrocardiographic appearance of this ventricular ectopy are similar to those of the ventricular arrhythmia that develops 24

FIGURE 5. Photomicrograph of the undamaged branching bundle from dog 8. Weigert-Van Gieson stain; original magnification \( \times 35 \). VS = ventricular septum; B = branching bundle.
hr after coronary ligation in dogs.\textsuperscript{16-18} No dogs died in their cages after transseptal shock intensities of 30 to 50 J. Thus, postablation survival was similar to the experience of Lerman et al.,\textsuperscript{15} who used a single 50 J discharge, and to that of Kempf et al.\textsuperscript{19} and Westveer et al.,\textsuperscript{11} who used even larger shocks.

\textit{Latent arrhythmogenicity.} Inducible arrhythmias were not observed days after transseptal electroshock injury. The relatively small size and homogeneous fibrous replacement of injured areas after transseptal ablation would not be expected to foster inducible tachycardia. Inducible ventricular tachycardia occurring after ischemic canine infarction usually requires damage to 20\% to 35\% of the left ventricular mass\textsuperscript{20,21} and a patchy infarct architecture with islands of viable myocardium interspersed within necrotic areas. While we found no evidence of latent arrhythmogenicity in these animals, extrapolation of these encouraging data to man seems premature.

\textit{Morphology.} Electroshock appears to create localized, permanent injury with replacement of myocytes by scar. Unlike ischemic infarction, myocardial injury was detected within 30 min of the application of electroshock, well before any inflammatory reaction. Electron micrographic findings of contraction bands, increased mitochondrial numbers, and electron-dense mitochondrial inclusions are common in descriptions of ischemic\textsuperscript{22,23} and electrical myocardial injury,\textsuperscript{24,25} suggesting these may be nonspecific myocardial responses to severe injury. The intense local inflammatory reaction evolved to organization and replacement by scar in every dog examined. Postinflammatory restoration of normal myocardial structure was never seen.

A duplicate discharge protocol was used in an attempt to increase total energy and discourage intractable postshock arrhythmias in these dogs, and because two or more shocks are commonly used at each ablation site during ventricular tachycardia ablation in man.\textsuperscript{26,27} While two 50 J discharges produced larger lesions compared with a single 100 J shock in four dogs, the relatively small number of lesions studies does not allow for definitive conclusions. We did not compare transcatheter shocks with shocks delivered from catheter to chest patch; however, Kempf et al.,\textsuperscript{19} using an intracardiac cathode and external anode, produced 0.87 ± 0.1 cm\textsuperscript{3} lesions with 100 J shocks, similar to the 1.0 ± 1.3 cm\textsuperscript{3} cathodal lesions we obtained with single 100 J shocks.

Transient periods of sinus arrest or atrioventricular conduction disturbances were common after delivery of the shock. These effects may be in part due to the strong electrical field or to concussive forces generated by the shock.\textsuperscript{13,28} Histologic examination of the sinoatrial node as well as the conduction system revealed no changes that could be ascribed to the shocks. This was true even when shocks were delivered to the higher ventricular septum.

\textit{Clinical implications.} Duplicate 30 or 50 J shocks consistently produced discrete septal lesions, without injury to conduction tissue. In normal canine myocardium, the discrete anodal and cathodal lesions averaged 1.0 to 1.5 cm in diameter and the paired lesions extended transeptally in 10 of 16 dogs (63\%), without septal perforation. This pattern of ablation injury may prove reasonable for application to patients with septal ventricular tachycardias, perhaps the focus can be accurately mapped. Further study is needed to determine if previously scarred myocardium is less sensitive to electroshock injury than normal muscle.\textsuperscript{29}

Based on observations from this study, we are encouraged that a technique for transseptal ablation of ventricular tachycardia can be safely developed for use in man. Our experience with transseptal catheter discharge in the normal canine heart indicates that total energy levels equal to or less than 5 J/kg body weight produce localized endomyocardial cautery without perforation or irreversible arrhythmias.

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