Catheter-mediated electrical ablation of the posterior septum via the coronary sinus: electrophysiologic and histologic observations in dogs

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ABSTRACT In a series of 12 dogs, the electrophysiologic and histologic effects of a single damped sine-wave shock delivered via standard electrocatheters to the region of the coronary sinus orifice were investigated. Six dogs received 200 J and six received 360 J of stored energy. The shock was delivered to two consecutive proximal poles of a standard quadripo lar catheter positioned at the coronary sinus orifice and connected to the positive output (anode) of a defibrillator. A disc electrode positioned on the anterior chest wall served as the cathode (negative pole). During the shock, voltage and current were recorded. Electrophysiologic testing was done before and 4 weeks after the shock. At 4 weeks, animals were killed and serial sections of the atroventricular groove and conduction system were performed. No significant long-term change in atrioventricular conduction, spontaneous or induced atrial or ventricular arrhythmias was observed. However, transient atrioventricular block was seen in five and idioventricular rhythms in six animals in the short term. No persistent electrocardiographic changes were observed, and no sudden deaths occurred. Microscopically, transmural injury at the anulus proper or basilar ventricular epicardium was inconstant and infrequent. However, transmural atrial injury at the level of the coronary sinus was produced over a 10 ± 5 mm length with the 200 J shock and a 21 ± 6 mm length with the 360 J shock. Neither coronary artery injury nor damage to the conduction system was seen and cardiac tamponade did not occur. However, localized intramural atrial rupture of the coronary sinus wall (on the endocardial aspect only) was observed in each dog, consistent with barotrauma. With the present technique, atrial injury potentially capable of blocking the effects of accessory pathway conduction could be produced without other electrophysiologic alterations or complications. Injury to the anulus proper (and therefore to any accessory pathway per se) is probably unlikely. Barotrauma may play a significant role in the type of injury observed in this study.


DELIVERY of an electrical shock from a defibrillator to the conduction system through an intracardiac catheter has been used to interrupt the atrioventricular (AV) node/His bundle in experimental preparations as well as in human beings.1–11 In the first clinical report of the use of such a technique to ablate the AV node/His bundle,1 a patient was described in whom preexcitation caused by a posterior septal accessory AV connection permanently disappeared after the procedure. Efforts to adapt this technique for intentional interrup-
ventricle, and coronary sinus along the course of the AV groove. An intervention at the os of the coronary sinus may not have the same implications for that applied more distally when the coronary sinus “descends” onto the ventricle. Catheter-mediated ablation interventions should therefore be assessed at a specific anatomic location for gleaning the most information for a particular technique. Another variable is the amount of tissue injury created per shock. Multiple shocks per catheter as done by Brodman and Fisher do not allow for investigation of the effects of energy, voltage, and current on tissue injury. Furthermore, because standard pacing and recording catheters can change dielectric properties after a single shock, and therefore change patterns of current flow, the ability to interpret the effect of several shocks through the same catheter becomes more difficult. In the present study, the intervention was limited to a single damped sine-wave shock delivered at a single site via standard electrocatheters.

**Methods**

Twelve dogs weighing between 23 and 28 kg were anesthetized with sodium pentobarbital (30 mg/kg), intubated with cuffless endotracheal tubing, and ventilated with a Palmer constant volume ventilator pump adjusted to provide a tidal volume of 20 cc/kg at a rate of 20 breaths/min. Additional pentobarbital was given as needed to maintain anesthesia for the duration of the study. No measurements were made for at least 10 min after additional doses of pentobarbital. Electrodes were placed on the four limbs and on the chest for electrocardiographic monitoring.

With sterile technique and fluoroscopic guidance, one standard quadripolar electrode catheter (No. 6F, USCI, Billerica, MA) with 1 cm interelectrode distance was inserted via a right external jugular vein cut-down and positioned in the high right atrium to record an atrial electrogram (proximal two poles) and to pace the atrium (distal two poles). Another standard quadripolar electrode catheter (“shocking” catheter, No. 6F, USCI) with 1 cm interelectrode distance was inserted via the same vein and positioned in the coronary sinus. Only new catheters were used in the coronary sinus position. In addition, before the shock, the coronary sinus catheter had been inspected and tested for electrical continuity to each electrode. For His bundle recording purposes, a No. 6F USCI quadripolar catheter was inserted via a right femoral vein cut-down and advanced across the tricuspid valve. A fluid-filled polyethylene catheter was inserted via a right femoral artery cut-down to the midabdominal level of the aorta and connected to a Statham (Model P23Db) transducer for arterial pressure monitoring. Fluids were maintained with an intravenous infusion of Ringer’s lactate at a rate of 10 ml/kg/hr.

Surface leads I, II, and V1, intracardiac electrograms and arterial pressure were amplified and displayed on a multichannel oscilloscope (Electronics for Medicine, Model VR 12). A filter setting of 30 to 500 Hz and 1 to 500 Hz was used for bipolar and unipolar signals, respectively. Recordings were printed on-line with the use of an eight-channel Siemens-Elema Mingograf (Model 803) ink jet recorder at paper speeds of 50 to 250 mm/sec. Selected portions were recorded with an Electronics for Medicine optical recorder at paper speeds of 50 to 100 mm/sec. Electrical stimulation of the heart was accomplished with a programmable stimulator (Bloom and Associates, Ltd.) that delivered constant-current rectangular pulses of 1 msec duration at twice the diastolic threshold.

**Electrophysiologic studies.** Before the stimulation studies, a control 12-lead electrocardiogram and baseline PA, AH, and HV intervals were obtained. Decremental pacing of the right atrium was performed to ascertain the cycle length at which block occurred in the AV node. Stimulation was ended at a point when atrial fibrillation was observed. After conversion to normal sinus rhythm (spontaneous in all dogs), programmed atrial stimulation with the extrastimulus technique was performed to determine the effective and functional refractory periods of the atrium and AV node and to assess the inducibility of atrial arrhythmias. A maximum of two atrial extrastimuli was used for arrhythmia induction.

On completion of the atrial stimulation protocol, the right atrial catheter was moved to the right ventricular apex and the His bundle catheter to the right ventricular outflow tract. Decremental pacing was performed from both sites at cycle lengths down to 200 msec to assess the inducibility of ventricular arrhythmias. Then, programmed stimulation with one extrastimulus was performed at two basic cycle lengths (500 and 400 msec) from the right ventricular apex and right ventricular outflow tract. If no sustained ventricular arrhythmia was induced with this technique, a second extrastimulus was added to the sequence. If a sustained ventricular arrhythmia was not initiated with double extrastimuli, burst pacing with an eight-beat train of stimuli was performed from both sites, starting at a cycle length of 360 msec and ending at a cycle length of 250 msec by 10 msec steps.

At the end of stimulation studies the right ventricular outflow tract catheter was withdrawn, the right ventricular apical catheter was left in place, and the coronary sinus catheter was positioned with the proximal pole at the orifice of the coronary sinus. Position of the catheter in the coronary sinus was confirmed by the following four criteria: (1) characteristic fluoroscopic appearance in the anteroposterior and lateral views, (2) the presence of negative paced P waves in leads II, III, and aVF, (3) the presence of local bipolar electrograms showing an atrial and a ventricular deflection, with the atrial signal falling between the midportion and the end of the P wave, and (4) the unipolar electrogram from the proximal pole showed a loss of the rapid component of the atrial electrogram indicative of an endocardial potential.

When it was felt that the more proximal of the two shocking electrodes was at the os of the coronary sinus (usually pole 4, except when only three poles could be inserted into the coronary sinus, then pole 3), a single synchronous damped sinusoidal pulse of 200 or 360 J stored energy was delivered to two consecutive poles of the coronary sinus catheter connected to the positive output (anode) of a Physio-Control Life Pak 6a defibrillator. A disc electrode 3.5 cm in diameter (Medtronic Model 6983) was connected to the negative output (cathode) of the defibrillator and positioned on the anterior chest wall opposite the anode as determined by fluoroscopy. A conductive gel (DEFIB-PADS, Medical Products Division, 3-M Co.) was interfaced between the skin and the cathodal electrode. During delivery of the shock, firm pressure was applied to the disc electrode. The two proximal poles (3 and 4) of the coronary sinus catheter were used as the anode in nine dogs, while poles 2 and 3 were used in the remaining three. Poles not used for delivery of shocks and all four electrodes of the right ventricular catheter were disconnected to prevent ground loops and unwanted current pathways. Right ventricular pacing was performed with a temporary external pulse generator when temporary AV block occurred. During the shock, surface leads II, III, and aVF were displayed simultaneously with arterial pressure.
Voltage and current measurements. The waveforms of the voltage across and the current through the electrodes were displayed on an oscilloscope (Tektronix Model 5111A), equipped with a camera for photographic recording. The voltage was recorded in a differential manner by means of a voltage divider with a 1000:1 input to output ratio (Warren Heggen, University Scientific Instruments) interfaced between the defibrillator and a Tektronix 5A14N vertical amplifier. The current was recorded by a Tektronix A6303 current probe that was connected to a Tektronix AM503 current probe amplifier. The current probe was positioned around the cable connecting the positive output of the defibrillator to the two “shock” poles of the coronary sinus electrode catheter. A second, confirmatory reading of the peak current was obtained by a custom circuit which automatically displayed the peak current on the defibrillator (built by Physio-Control). This adjunct to the displayed current waveform confirmed the absence of current leakage outside the catheter. The experimental schema is shown in figure 1.

Follow-up. After the shock, the dogs were monitored for 2 hr, after which a 12-lead electrocardiogram was obtained. Serial 12-lead electrocardiograms were recorded again at 24 hr, 2 week, and 4 week intervals. On each occasion signs of tricuspid insufficiency, pericardial tamponade, and heart failure were sought on physical examination. Four weeks after the shock, repeat electrophysiologic studies were performed as described earlier. Cannulation of the coronary sinus was attempted in all dogs. At the end of the study, the animals were killed and the hearts were removed and fixed in 10% formalin.

Morphologic studies. After gross inspection for structural damage, each heart was sectioned transversely in 0.5 cm slices from the apex to 2 cm from the AV groove, and each slice was examined grossly. The principal areas of interest (i.e., AV groove and AV conduction system) were removed for more detailed examination in two tissue blocks. One block included the AV node/His bundle and the main fascicles. The other block included the AV junction perpendicular to the annulus spanning 4.5 cm from the coronary sinus orifice and including a 2 cm rim of atrial and a 2 cm rim of ventricular myocardium above and below the AV groove. Each block was embedded in paraffin and sectioned serially. At 1 mm intervals, slides were prepared. A section at each interval was stained with Gomori’s trichrome. Verhoeff–Van Gieson stains for visualization of elastic tissue injury were performed on selected sections at points of maximum injury.

Statistics. Results are expressed as mean ± SD. Differences in measures of electrophysiologic parameters before and after the shock were tested by a paired t test. Mean differences of the measurement of transmural atrial injury between the 200 and 360 J groups were tested by a two-sample t test with pooled variance estimate. Statistical significance was assumed at p < .05.

Results

The recovery from the procedure was uneventful in all dogs. No signs of hemodynamic compromise consistent with pericardial tamponade, tricuspid insufficiency, or congestive heart failure were observed. No dog died suddenly during the 4 week follow-up period.

Electrocardiographic findings. Immediately after the synchronized shock, all dogs showed nonsustained, self-terminating episodes of ventricular tachycardia. Upon termination of ventricular tachycardia, third-degree AV block was observed in five dogs (dogs 2, 3, 8, 10, and 12). Complete AV block resolved within 15 min in all but one dog (dog 8), in which it lasted up to 45 min. This animal was the only one to require temporary ventricular pacing because of an extremely slow idioventricular escape rhythm. At the end of the 2 hr monitoring period, normal resting AV conduction had resumed in all dogs.

Other short-term electrocardiographic changes included transient PR and ST segment elevation in all dogs, especially evident in the inferior leads. However, at the 2 hr observation period the 12-lead electrocardiogram did not show any change from the control electrocardiogram.

An idioventricular rhythm competing with sinus rhythm was noted at the 24 hr interval in six animals (dogs 4, 5, 8, 10, 11, and 12). This rhythm showed

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**FIGURE 1.** Schema for catheter-mediated electrical ablation with a defibrillator and a standard electrode catheter. The proximal pole of the electrode catheter was positioned at the coronary sinus orifice. A damped sine-wave shock was delivered to the two consecutive proximal electrodes (3 and 4) by means of a switch box. These two electrodes, coupled together, served as the anode (positive pole), while a 3.5 cm disc electrode served as the cathode (negative pole). Connections to record current (I) and voltage (V) waveforms are illustrated. A current-sensing transformer built into the Physio-Control defibrillator served as an adjunct to the displayed current waveform confirming the absence of current leakage outside the catheter.
periods of acceleration and deceleration, with a QRS morphology of right bundle branch block and left axis deviation, consistent with an automatic focus located along the posterior base of the left ventricle.

Later serial 12-lead electrocardiograms and monitoring strips were unremarkable for changes suggestive of ischemia or conduction defects. No arrhythmic episode was detected after the first 3 days after the shock and no sudden deaths occurred.

**Electrophysiologic findings.** Results are summarized in table 1. Evaluation of PA, AH, and HV intervals 1 month after the shock did not show any significant change from baseline values as a group or in any individual dogs. Decremental atrial pacing studies showed that the longest atrial paced cycle length that produced Wenckebach periodicity in the AV node had not changed significantly. Analysis of the programmed premature atrial stimulation data before and after the procedure failed to show any significant differences in the effective and functional refractory periods of the atrium and the AV node. Ease of induction of atrial fibrillation and duration of the induced arrhythmia remained unchanged in all dogs but one (dog 10, table 1), in which external cardioversion was required to terminate the atrial fibrillation upon 4 week follow-up study.

Differences in the ease of induction of ventricular arrhythmias were not observed. Ventricular fibrillation had been induced with double extrastimuli in the baseline preshock study in two dogs (dogs 1 and 6, table 1). Neither animal had inducible ventricular arrhythmias at the 4 week follow-up study. One dog (dog 5, table 1) had ventricular fibrillation induced with double extrastimuli from the right ventricular outflow tract 4 weeks after the shock. No sustained ventricular tachycardia was induced in any dog before or after the shock.

**Gross anatomic and histologic findings.** Gross inspection of the epicardial aspect of the heart failed to show any sign of healed rupture of the coronary sinus wall. Furthermore there was no pericardial reaction to suggest previous inflammation or hemorrhagic effusion. Examination of the endocardial aspect of the right atrium showed no evidence of damage to the tricuspid valve or to the AV node area.

Scarring was grossly evident around the mouth of the coronary sinus in nine dogs, with narrowing of the coronary sinus orifice in six dogs and complete occlusion in three dogs. In three animals the coronary sinus orifice appeared entirely normal. A brownish discoloration caused by hemosiderin deposits was found occasionally on the endocardium of the posterior left atrium, up to 1 cm above the level of the anulus fibrosus. Gross damage to the circumflex coronary artery was not found in any dog. No congestion or gross changes in the left ventricle secondary to coronary sinus thrombosis were seen.

Histologic findings are summarized in table 2. Sections of the AV node, His bundle, and proximal bundle branches did not reveal any damage in any dog. Coronary sinus injury, however, was present in all animals. Maximum length of coronary sinus injury was 18 ± 6 mm in the 200 J group and 23 ± 4 mm in the 360 J group. Injury consisted of circumferential fibrosis in all dogs and was associated with loss of coronary sinus intimal muscle. Furthermore, the lumen of the coronary sinus showed varying degrees of stenosis to complete occlusion, consistent with organization of thrombus (figure 2). Finally, although no gross rupture of the coronary sinus was seen in any dog, a constant histologic finding was rupture of the internal elastica of the coronary sinus with displacement of tissue toward the atrial wall on its endocardial aspect as if segments of the coronary sinus had been forcefully disrupted as a result of barotrauma (figure 3). No such injury was observed along the epicardial aspect of the coronary sinus.

In the left atrial wall, fibrous replacement of atrial myocardium extended for a maximum length of 18 ± 5 mm in the 200 J group and 26 ± 4 mm in the 360 J group, reaching a height of 8 ± 3 mm and 11 ± 2 mm for the 200 and 360 J shocks, respectively. Atrial injury was transmural for a length of 10 ± 5 mm in the 200 J group and 21 ± 6 mm in the 360 J group (p < .01). However, transmural fibrosis near the anulus of the mitral valve was inconstant and scanty (figures 2 and 4).

Maximum length of muscle loss and fibrous replacement in the left ventricle paralleled that of the left atrium, being 15 ± 5 mm long in the 200 J group and 19 ± 5 mm long in the 360 J group. However, the depth of involvement of the left ventricular wall was never greater than 5 mm. Additionally, injury width was at most 5 mm at the point of maximum left ventricular injury, except in one dog (dog 12, table 2). Transmural ventricular injury was present only in this one dog and was limited to a length of 3 mm.

Microscopic findings confirmed the absence of injury to the circumflex coronary artery (figures 2 and 4). An occasional finding was the presence of a foreign body reaction with giant cell formation in the areas of maximum injury associated with platinum deposition in the tissue (documented by spectroscopy) (figure 5). This is likely the result of erosion of the electrode after the passage of current.
TABLE 1
Electrophysiologic findings before and after shock

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>PA (b)</th>
<th>AH (b)</th>
<th>HV (b)</th>
<th>RA DEC P (Wenckebach)</th>
<th>AVN ERP</th>
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<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>65</td>
<td>35</td>
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<td>410</td>
<td>300</td>
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</tr>
<tr>
<td>Mean</td>
<td>32</td>
<td>60</td>
<td>36</td>
<td>267</td>
<td>225</td>
</tr>
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</table>

± SD  ±5 ±7 ±24 ±13 ±3 ±3 ±73 ±31 ±54 ±35

Data expressed as milliseconds.

a = after; AF = atrial fibrillation; AH = atrio-Hisian interval; AVN = atrioventricular node; b = before; DEC P = decremental pacing; ERP = effective refractory period; FRP = functional refractory period; HV = Hisventricular interval; NSu = nonsustained; PA = surface P wave to local AVN atrial electrogram interval; RA = right atrium; Su = sustained; VAR = ventricular arrhythmias; VF = ventricular fibrillation.

Voltage and current during energy delivery. Delivery of 200 J stored energy resulted in a peak current of 18 ± 2 A and a peak voltage of 3300 ± 100 V. At a 360 J setting, peak current and voltage were 29 ± 3 A and 4150 ± 152 V. Values for peak current as calculated on the oscilloscope screen were always in agreement with readings obtained on the defibrillator display, which therefore excluded any leakage of current outside the catheter.

Current and voltage waveforms were never smooth, like their counterpart obtained with 50 Ω dummy load. They showed a different time course of voltage and current, consistent with a nonlinear resistance or complex impedance during delivery of the shock. An example of current and voltage waveforms recorded during delivery of a 200 J shock is shown in figure 6.

Discussion

Catheter-mediated electrical ablation techniques are receiving increasing attention for a variety of both supraventricular and ventricular arrhythmias. This technique has almost supplanted surgery for arrhythmias requiring AV node/His bundle interruption. The relative safety and effectiveness in producing AV block by catheter technique suggests the possibility that accessory AV pathways also could be ablated without the use of open heart surgery.

The experimental work of Brodman and Fisher supported the feasibility of a catheter technique to ablate left-sided bypass tracts. However, subsequent application of transvenous catheter-mediated electrical ablation procedures to patients with left-sided or posterior septal accessory connections has yielded failures and complications that demand reassessment of the validity and utility of this technique. It is clear from preliminary work that success of the technique is variable and less constant than that observed with surgical approaches. More work is necessary to refine the procedure before it can approach AV node/His bundle interruption in safety and efficacy.

In this study, we focused on the electrophysiologic and anatomic effects of delivery of a single electrical shock to the posterior septum via the coronary sinus. Surgical experience with posterior septal accessory pathways has revealed the complexity of the approach necessary to divide them, especially if one is to avoid the AV node/His bundle in the dissection. The approach to the posterior septal region with external cryoablative procedures may avoid the problem of AV block, although experience with this approach re-
remains limited and it still requires a sternotomy. Therefore we addressed the specific question of whether a catheter technique can result in damage potentially able to prevent conduction through such posterior septal pathways without affecting the anatomy and the functional characteristics of the normal AV conduction system. We also desired to garner information on the mechanism of catheter-induced electrical injury.

The choice of the present technique was dictated by the following theoretical considerations. If any vectorial component related to delivery of electrical energy is involved in producing the desirable effects, then the current flow should be able to traverse the target area, resulting in adequate current density at that point. Positioning of the cathode on the anterior chest wall opposite to the anode should allow adequate current density

<p>|TABLE 2| Anatomic changes after the shocka |
|---|---|---|---|---|---|---|---|---|</p>
<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Coronary sinus injury</th>
<th>Atrial injury</th>
<th>Left ventricular injury</th>
<th>Conduction system injury</th>
<th>Circumflex arterial injury</th>
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<tbody>
<tr>
<td>Dog No.</td>
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<td>Elastie rupture</td>
<td>Length (mm)</td>
<td>Max. length (mm)</td>
<td>Transmural injury (mm)</td>
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<td>Mean±SD</td>
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<td>20</td>
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<tr>
<td>Mean±SD</td>
<td>23±4</td>
<td>26±4</td>
<td>21±6</td>
<td>11±2</td>
<td>19±5</td>
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</table>

aDogs 1 to 6 received 200 J shocks, and dogs 7 to 12 received 360 J shocks.
to flow across the area where damage is desirable. Another consideration relates to electrically induced myocardial damage. Cardiac defibrillation studies have shown that the use of very small paddles results in high transthoracic resistance, with a reduced total current flow. On the other hand, intracardiac current increases with larger paddles, although the distribution of this current covers a larger cross-sectional area. Therefore the use of smaller paddles should result in less transthoracic current flow, with points of higher current density, which can be useful in producing the desired damage. Additionally, because of the variability of locations of the accessory connections from endocardium to epicardium, an ideal injury should cover the entire thickness of the atrial wall near the anulus.

Based on these considerations, we used high-energy shocks and a small (3.5 cm) paddle positioned on the anterior chest wall, opposite the coronary sinus catheter.

The decision to couple together two consecutive poles of the coronary sinus catheter as the anode was based on the consideration that accessory pathways may cover, morphologically or functionally, a zone of myocardium larger than simply the punctate site, which might be expected from catheter mapping. Early surgical experience with posterior septal and left-sided accessory pathways, seems to confirm the impression that accessory AV fibers are often not discrete and can occur over several centimeters. As a consequence, an injury able to prevent accessory pathway conduction ideally should involve a wider area of the atrioventricular sulcus or atrial wall above the anulus fibrosus other than the precise area of earliest activation. Furthermore, the decision to use a pair of electrodes as the anode was taken in view of previous experience indicating that standard catheters may not tolerate high-energy shocks, which can result in failure of the internal wire or the insulation, with subsequent delivery of the shock to poles other than the ones intended. Coupling of two electrodes of equal resistance in parallel would cause total current flow to divide in half through either of them, possibly preventing at least failure of the internal wire. On the other hand, separate shocks of less energy on either pole might not obtain the same result, the first shock possibly weakening the dielectric strength of the catheter.

It is important to emphasize, with respect to ablation techniques, that accessory AV pathways usually skirt the anulus fibrosus in close approximation to it. However, our results show that delivery of high-energy shocks in proximity to the coronary sinus orifice principally produces damage to the wall of the coronary sinus and to left atrial myocardium above the AV groove but not to the anulus proper. Nevertheless, an area of delay could be created so that the accessory connection would become functionally silent. This kind of injury may be all that is necessary or indeed safely possible from a clinical perspective. In our series of dogs, even at high energies, inconsistent injury was produced at the anulus fibrosus. This is consistent with anatomic observations in dogs and in man that the coronary sinus occupies a relatively high position in the pyramidal space with respect to the anulus.

Risk of permanent AV block associated with the surgical approach of posterior septal accessory pathways is a well-known possibility. With the use of the technique described in this study, no functional or
morphologic damage was produced to the conduction system. Impairment of AV conduction was observed only transiently in our series of dogs, probably as a consequence of the barotrauma associated with the electrical shock. Furthermore, the arrhythmogenic potential of this particular technique appears to be minimal, as judged from the results of atrial and ventricular stimulation studies and from the long-term absence of spontaneous arrhythmias. The episodes of induced ventricular fibrillation in the preshock studies are of uncertain significance and probably reflect hypersensitivity of dogs to ventricular programmed electrical stimulation. The failure to demonstrate any cardiac abnormalities in these dogs other than that induced after the shock suggests that inducible ventricular fibrillation was a false-positive event. The one dog that had ventricular fibrillation induced after the shock was similar to the other two dogs in that no ventricular abnormality was found except for a region at the shock site corresponding to a 0.27 cm³ volume of damaged muscle. Although not proved, it is unlikely that this ventricular injury resulted in inducible ventricular fibrillation.

Preservation of normal anatomy of the left circumflex coronary artery was observed in this study, confirming previously reported findings. However, the anatomic relationship of the coronary sinus orifice to the circumflex coronary artery is optimal at the posterior or septum. Coronary arterial injury at this location is less likely (assuming no significant right coronary artery extension branch). In the lateral AV groove, on the other hand, such a technique may be more likely to cause coronary arterial injury.

No evidence of gross rupture of the coronary sinus was found in any of our dogs, even when energies as high as 360 J were used. This could have resulted from the use of two electrodes coupled together as the anode, which avoided concentration of electrical energy to a very small area and in essence divided the energy between two electrodes. An interesting finding, however, was the presence of intramural rupture of the coronary sinus in all animals. The morphologic features of this damage would be rather difficult to explain on the basis of a pure electrical or thermal injury. The observation that parts of the internal elastic membrane of the coronary sinus were displaced literally...
higher energy shocks or multiple shocks more likely to lead to gross coronary sinus rupture. Conceivably, rupture might occur more easily in a small coronary sinus. Therefore adequate angiographic visualization of the coronary sinus should be recommended in potential candidates for catheter-mediated shocks in this locale. Caution should be exercised before proceeding with currently available catheter techniques in children, in patients with a small coronary sinus, and in the more lateral portion of the coronary sinus where the vessel tapers considerably as it becomes the great cardiac vein.

That internal elastic membrane rupture into the atrial wall can result from a barotraumatic mechanism is supported by previous work in underwater pulse technology.34 Experiments in vitro conducted to assess the reliability of electrode catheters to be used for electrical ablation32,35 suggest that high pressures could be generated during delivery of high-energy endocavitary shocks. To which extent arcing through ionic gases formed from electrolysis by high-energy shocks may be one mechanism responsible for barotrauma is unknown.

The role barotrauma has in producing the desired damage rather than creating complications remains to be established. Certainly, barotrauma by itself cannot explain other features of the injury. That discharge of transthoracic or epicardial shocks can result in myocardial injury is a well-known fact.27,28,36,37 Myocyte necrosis with contraction bands, mineralization of necrotic tissue with subsequent macrophage infiltration, and later fibrosis have all been described in the setting of myocardial electrical injury induced by extracardiac countershocks.36,38,39 Similar findings have been reported recently in canine studies, where the effects of intracardiac shocks delivered through endocardial catheter electrodes were evaluated.40,41 Our study design did not allow us to evaluate sequential ultrastructural changes of this kind, even though the observation in our study of areas of macrophage infiltration with giant cell formation is in keeping with the results of those studies40,41 and with previous observations made in dogs undergoing transvenous ablation of the AV node.4 However, all these changes could merely represent a pattern of response of the myocardial cell to injury42 rather than being specific for an electrical type of injury. Therefore such changes seem insufficient to clarify the mechanism by which endocavitary shocks produce their effects.

With the use of this technique, transmural atrial injury potentially capable of preventing accessory pathway conduction in the posterior septum was con-

**FIGURE 4.** Cross section of the left atrioventricular groove, taken 18 mm within the coronary sinus orifice. This dog (No. 4) received 200 J. The left ventricular wall and the coronary artery do not show any damage. Coronary sinus injury is limited. However, transmural atrial fibrosis is present in the atrial wall facing the endocardial aspect of coronary sinus (arrows). Note undamaged atrial muscle just above the mitral valve. This injury could still leave a bypass tract anatomically intact. Conceivably, however, such a boundary of transmural fibrotic tissue could prevent depolarization wavefronts from entering or exiting the accessory pathway. See text for discussion. (Gomori’s trichrome; original magnification ×8.7.) Abbreviations as in figure 2.

into the atrial wall would suggest that the pressure increase associated with the electrical discharge was of such a magnitude as to create damage by means of barotrauma. In support of this mechanism, one would expect that close contact between the electrodes of a No. 6F catheter and the coronary sinus wall at the moment of the shock is unlikely to occur because of a disproportion between their respective sizes. This would prevent current directly from contacting the coronary sinus wall, allowing, on the contrary, for some current to be dissipated in the blood. This would result in electrolysis of the blood with plasma (ionic gas) formation.34,35 Arcing of the current through the plasma would lead to a shock wave and the barotrauma that gives rise to rupture of the coronary sinus elastica. Coronary sinus rupture with tamponade therefore would be dependent on the degree of barotrauma, with
FIGURE 5. High-power photomicrograph from the area of maximum injury in dog 10. A foreign body reaction with giant cell formation is present in the coronary sinus associated with platinum deposition in the tissue (arrows). See text for discussion. (Gomori's trichrome; ×300.)

FIGURE 6. Current and voltage waveforms recorded during delivery of 200 J in dog 4. The shock resulted in a peak voltage of 3300 V (top) and a peak current of 23 A (bottom). Note that current and voltage show a different time course, consistent with a nonlinear resistance during delivery of the shock.

Consistently produced with a single shock. No AV block, electrophysiologic alterations, pericardial tamponade, or other complications were seen. Atrial intramural rupture of the coronary sinus was observed in each dog, the histologic features of which are suggestive at least in part of a barotraumatic mechanism. The evidence of barotrauma brings into question the controllability of the technique. Changes in the procedure will therefore be necessary for safe application. Successful catheter modification via the coronary sinus of tachycardias using posterior septal accessory pathways may depend on the ability of transmural atrial injury above the anulus proper to effectively block depolarization wavefronts from entering or exiting the accessory pathway, which is likely to persist after the shock.

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