Effects of varying electrode configuration with catheter-mediated defibrillator pulses at the coronary sinus orifice in dogs

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ABSTRACT We compared two methods of delivering single bumped sine-wave defibrillator pulses to the coronary sinus orifice in 20 dogs. Ten dogs had "unipolar" (coronary sinus to precordial disc) and 10 had "bipolar" (coronary sinus proximal to coronary sinus distal electrode) discharges. Delivered voltage, current, and energy were recorded during each pulse. Electrophysiologic testing was done before and 4 weeks after the procedure. Histologic examination of the atrioventricular groove was done at 1 mm serial sections. For the unipolar configuration a 200 J defibrillator pulse resulted in a peak voltage of 3370 ± 125 V, a peak current of 21 ± 4 A, and a delivered energy of 253 ± 29 J as compared with 3010 ± 99 V, 70 ± 4 A, and 144 ± 18 J, respectively, for the bipolar configuration (p < .001). Three dogs (two with bipolar, one with unipolar pulses) had gross coronary sinus rupture and died from acute pericardial tamponade. In addition, irrespective of electrode configuration, all dogs showed microscopic rupture of the coronary sinus internal elastic membrane. Transmural atrial scarring occurred in all 10 dogs that received a unipolar pulse but in only two dogs that received a bipolar pulse (p = .0004). Unlike the atrium, injury to the left ventricle was limited in both groups. Similarly, injury to the perianural myocardium was inconsistent and not transmural in either group. No significant electrophysiologic changes were observed. With the present technique, unipolar rather than bipolar catheter-mediated defibrillator pulses result in transmural atrial injury that might prevent accessary pathway conduction. Regardless of electrode configuration, high-energy defibrillator pulses consistently cause some degree of coronary sinus rupture, most likely related to a barotraumatic mechanism.

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PREDICTORS of success or failure for catheter-mediated ablation of atrioventricular (AV) accessory pathways by defibrillator pulses are poorly understood. Many factors may affect the result, including the location of the accessory pathway, the energy used, the catheter structure, the electrode configuration, and other as yet undefined factors. More insight into the essential determinants of tissue injury is needed. The purpose of this investigation was to study the physical, histologic, and electrophysiologic aspects of one of these factors, electrode configuration.

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Methods

Twenty dogs weighing 27 ± 4 kg were randomly divided into two groups of 10 dogs each. Each dog was anesthetized as sodium pentobarbital (30 mg/kg), intubated, and ventilated by means of a Palmer constant-volume respirator. With sterile techniques, the right femoral vein and artery and the right external jugular vein above its bifurcation were exposed. Arterial pressure was monitored via the right femoral artery by a Statham P23Db pressure transducer.

Under fluoroscopic guidance, quadripolar electrode catheters (No. 6F, USCI, Billerica, MA) were positioned in the heart for performance of baseline electrophysiologic studies. A Medtronic 6992A lead was inserted through the jugular vein and positioned in the coronary sinus as the catheter receiving the defibrillator pulse. The Medtronic catheter chosen in this study to carry defibrillator pulses is a No. 9.5F bipolar lead, consisting of two nickel alloy coils insulated with silicone rubber connecting to two ring electrodes (a platinum proximal ring and a platinum-iridium distal ring), separated by 15 mm. Their nominal surface areas are 48 mm² for the proximal ring and 19 mm² for the distal ring. This catheter has high dielectric strength (>5000 V) and is capable of containing high-energy pulses, unlike standard pacing and recording catheters.¹ ²

Before stimulation studies, a baseline electrocardiogram
(ECG) and AV conduction intervals during sinus rhythm were obtained.

Impulse formation and conduction in the atrium and in the ventricle were studied with a pacing and recording technique described previously. 

At the end of stimulation studies, only a 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, according to fluoroscopic and electrophysiologic criteria defined previously.3

**Energy delivery.** When the proximal ring electrode was at the ostium of the coronary sinus, the standard stainless-steel stylets were inserted into the leads to minimize line resistance (70 Ω without, 4 Ω with stylet). A single stylet was passed to the proximal ring electrode if a “unipolar” discharge was administered. Two stylets, each for either electrode, were inserted, if a “bipolar” discharge was administered. Care was exercised not to dislodge the lead from the previously attained and fluoroscopically confirmed position. A single damped sine-wave pulse (200 J energy setting) was discharged synchronously with the QRS from a Physio-Control LifePak 6A defibrillator. In 10 dogs a unipolar configuration was used to deliver the pulse. In this instance, the proximal ring electrode was the anode (positive pole) and a disc electrode 3.5 cm in diameter (Medtronic, Model 6983) positioned over the anterior chest fluoroscopically opposite the anode served as the cathode (negative pole) (figure 1, top panel). A conductive gel (DEFIB-PADS, Medical Products Division, 3-M) was applied between the skin and the disc electrode. In a second group of 10 dogs, the proximal ring electrode served as the anode and the distal ring electrode served as the cathode (bipolar configuration) (figure 1, bottom panel).

At the time of energy delivery, all four electrodes of the right ventricular catheter and the distal Medtronic ring electrode in the case of unipolar discharges were disconnected to prevent ground loops and unwanted current pathways. During the discharge, surface leads II, III, and aVF were displayed simultaneously with arterial pressure.

**Energy delivery measurements.** The voltage and current waveforms were displayed on an oscilloscope (Tektronix, Model 5111A) equipped with a camera for photographic recording. The voltage was recorded with a resistive voltage divider with a 1000:1 input-to-output ratio interfaced between the defibrillator and a Tektronix 5A14N vertical amplifier. The current was recorded by a Tektronix A6303 current probe connected to a Tektronix AM503 current probe amplifier. The total energy delivered was calculated from the photographic recordings by integrating the voltage and the current waveforms with a computerized digitizer. The experimental schema is shown in figure 1.

**Follow-up.** After the electric discharge, the dogs were monitored for 3 hr. At the end of the study, a repeat ECG was
obtained. Serial 12-lead ECGs were recorded again at 24 hr and 2 and 4 weeks. Rhythm strips were obtained throughout the follow-up period. On each occasion, the dogs were observed for signs of pericardial tamponade, tricuspid regurgitation, or heart failure. Four weeks after delivery of the electric pulse, repeat electrophysiologic testing was performed. At the end of the study, the animals were killed and the hearts were excised and fixed in 10% formalin.

Morphologic studies. After visual inspection for structural damage, serial 5 mm thick transverse sections were made from the apex to within 2 cm from the AV groove, and each section was examined grossly. A tissue block was then removed from the AV groove for light microscopy studies. This block included the AV junction perpendicular to the anulus, spanning 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. The tissue 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. The extent of damage was measured from the histologic sections and expressed as the maximum length, width, and depth in millimeters of necrotic or fibrotic tissue. Verhoeff–van Gieson stains for visualization of elastic tissue were performed on selected sections at points of maximum injury.

Pressure measurements in vitro. To demonstrate that high-pressure shock waves occurred after electric pulses for either configuration, a Piezotronics piezoelectric pressure transducer was used to record shock waves 2 cm from the proximal ring electrode in a saline bath by means of previously described techniques. Pressure tracings were displayed on a Tektronix 7633 oscilloscope and recorded on film for subsequent analysis. Measurements were made 10 times for each configuration and then averaged.

Statistical analysis. Results are expressed as mean ± SD. Differences in measures of electrophysiologic variables before and after the procedure within groups were tested by a paired t test. Mean differences of dimensions of anatomic lesions and of electric variables between the unipolar and the bipolar groups and mean difference of pressure measurements between unipolar and bipolar pulses were tested by a two-sample t test. Differences in atrial transmural injury between the unipolar and the bipolar groups were analyzed with Fisher's exact test. Statistical significance was assumed at p < .05.

Results
Two dogs in the bipolar group (dogs 13 and 19) and one in the unipolar group (dog 8), developed hemodynamic deterioration 10 to 20 min after the discharge and subsequently died within 3 hr (dog 13) and 12 hr (dogs 8 and 19) of pericardial tamponade. Perforation of the coronary sinus at the level of the crux of the heart was demonstrated in each of the three dogs. Another animal (dog 5) showed a marked drop in arterial pressure 10 min after receiving a unipolar pulse spontaneously resolving within 1 hr. This dog had an organized thrombus on the epicardial aspect of the coronary sinus, consistent with a healed rupture of the vessel. The remainder of the dogs did not suffer any complications. No sudden death was observed during the follow-up period.

Electrocardiographic and electrophysiologic findings. Immediately after the synchronized pulse, 17 of the 20 dogs showed episodes of ventricular tachycardia. These lasted between 3 and 30 sec and all terminated spontaneously. In one dog given a unipolar pulse (dog 8), no ventricular arrhythmia followed delivery of the electric discharge. In two dogs given bipolar pulses (dogs 13 and 20), transient episodes of a coronary sinus rhythm (inverted P waves in leads II, III, and aVF) associated with ventricular bigeminy were recorded immediately after the pulse. No AV block occurred in any dog. Short-term PR segment elevation in the inferior leads was observed in all dogs, while ST segment elevation occurred in 11 dogs (seven receiving bipolar and four receiving unipolar pulses). These changes lasted less than 30 min in all dogs, except for the three that developed cardiac tamponade. In these dogs PR and ST segment elevation persisted over the first 3 hr after the discharge and was associated with sinus tachycardia and decreased voltage in all peripheral leads. In the remaining dogs, however, the ECG had returned to normal at the 3 hr recording interval.

At 24 hr, eight of the nine surviving dogs in the unipolar group transiently exhibited an accelerated idioventricular rhythm with a QRS morphology consistent with origin from the posterior ventricular septum. In contrast, only one dog (No. 15) of the eight surviving in the bipolar group exhibited this rhythm at 24 hr electrocardiographic observation. No further episodes of spontaneous atrial or ventricular arrhythmias were observed in subsequent electrocardiographic observations. Later ECGs were unchanged from control tracings.

Electrophysiologic findings are summarized in table 1. No significant changes in AV conduction or induced atrial or ventricular arrhythmias were found from baseline findings.

Gross anatomic and histologic findings. Anatomic changes are summarized in table 2. Gross inspection of the epicardial aspect of the heart was remarkable in the three dogs that had pericardial tamponade. A clot was found to envelope the base and the diaphragmatic surface of the heart. A large subepicardial ecchymotic area extended from the crux of the heart for several centimeters over the left AV groove. The surrounding myocardium was congested and had enlarged venous collaterals in the two animals that survived for a longer period. The coronary sinus showed a tear about 5 to 7 mm long with irregular margins. Viewed from the endocardial aspect, the tear started between 3 and 10 mm within the ostium of the coronary sinus. A large fresh thrombus partly occupied the lumen of the coronary sinus in all three cases.

In another dog (No. 5) a pericardial reaction with a
small organized thrombus was seen on the epicardial aspect of the coronary sinus. This dog had had a transient drop in arterial pressure after a unipolar discharge, consistent with a small laceration of the coronary sinus, promptly sealed by a thrombus. In the remainder of the dogs, gross examination of the epicardial aspect of the heart was unremarkable for changes suggestive of previous inflammation or hemorrhagic effusion.

The endocardial aspect of the right atrium, the tricuspid valve, and the AV node area were normal at gross inspection in all animals. The endocardium of the posterior left atrium occasionally showed a brownish discoloration caused by hemosiderin deposits. The ostium of the coronary sinus was narrowed or occluded by fibrotic tissue in eight of the nine long-term survivors receiving unipolar pulses. In one the coronary sinus was patent. Among the eight long-term survivors receiving "bipolar" pulses, four showed narrowing or complete occlusion of the coronary sinus, up to 1 cm within the orifice, while in the remaining four the coronary sinus was patent, although some endocardial thickening was present around the ostium.

Gross damage to the circumflex coronary artery or changes in the ventricles were not found. Except for evidence in the three dogs that died early, no sign of congestion secondary to coronary sinus thrombosis was seen.

Microscopic examination of the sections taken from the AV groove showed coronary sinus injury in all animals, starting from 2 mm before to 10 mm within the os of the coronary sinus. In general, in the dogs that died early from pericardial tamponade, injury consisted of selective myocardial necrosis associated with contraction bands and areas of hemorrhage interspersed between damaged fibers. In the dogs killed 4 weeks after receiving defibrillator pulses, injury consisted of loss of myocardium, with replacement by fibrous tissue.

In the one dog that had gross coronary sinus rupture after a unipolar discharge, the rupture itself consisted of multiple tears toward both the epicardial and the coronary sinus.
endocardial aspect of the coronary sinus and extended for about 20 mm within the ostium. The communication with the epicardium was limited to a length of 5 mm. In the two dogs that had coronary sinus rupture from bipolar pulses, the coronary sinus was ruptured on its superior and endocardial aspects for a length of about 15 mm. The communication with the epicardium extended for 7 mm.

Measurements of histologic tissue injury are listed in table 2. In the atrial wall, the most significant finding was that atrial injury was transmural in all 10 dogs receiving unipolar pulses (figure 2, A) but in only two dogs (Nos. 14 and 19) receiving bipolar pulses (figure 2, B). Maximum length of total atrial injury (not necessarily transmural) was not significantly different in the two groups. Also, maximum height of atrial injury did not differ significantly. Regardless of injury to the atrial wall, however, injury to the periannular myocardium was inconsistent and not transmural (figure 2). The length of total and circumferential coronary sinus injury was not significantly different in the two groups.

Although gross rupture was present in only four dogs, sections stained for demonstration of elastic tissue constantly showed microscopic rupture of the internal elastic membrane of the coronary sinus, regardless of electrode configuration (figures 3 and 4).

Segments of the elastica appeared to have been forcefully displaced toward the endocardial aspect of the atrial wall (figure 4, A), suggesting a role for barotrauma in the genesis of this type of injury. In injury to the left ventricle was limited in all dogs (figure 2). There was no statistically significant difference in maximum length or depth of left ventricular injury in the two groups. However, maximum width of left ventricular injury was significantly greater after unipolar than after bipolar discharges. Transmural ventricular injury was present in only one dog (No. 8, unipolar pulse), although it was limited to a length of 2 mm.

Microscopic examination confirmed the absence of injury to the circumflex coronary artery (figures 2 and 3).

**Energy delivery characteristics.** Values for current, voltage, and delivered energy are summarized in table 2. Delivery of a pulse at a setting of 200 J resulted in a peak voltage of 3370 ± 125 V, a peak current of 21 ± 4 A, and a delivered energy of 253 ± 29 J for the unipolar configuration, as compared with 3010 ± 99 V, 70 ± 4 A, and 144 ± 18 J for bipolar configuration (p < .001 for current, voltage, and delivered energy comparisons). An example of current and voltage waveforms with either electrode configuration is given in figure 5. In both cases it can be seen that current and voltage are out of phase (much more so in the case of a bipolar pulse), with voltage peaking earlier than current, consistent with a nonlinear resistance during energy delivery. The upstroke of the curve is similar for both electrode configurations, until a break occurs about one-third of the way through the discharge. It is conceivable that at this point of the discharge, gas formation occurs around the electrodes, with subsequent plasma arcing. Thereafter, the waveforms smoothly and slowly decline in the case of a unipolar pulse, probably reflecting high tissue resistance encountered by current flow. In the case of a bipolar pulse, however, the voltage drops dramatically, and the current continues to rise until its peak, consistent with an extremely low resistance offered by the blood to the current flow between two electrodes 1.5 cm apart. The abrupt cessation of the pulse after about 4 msec, with some current again flowing shortly thereafter, probably relates to the relay switch built into the defibrillator, which first determines cessation of the pulse and then is subjected to some bounce.

**Pressure results.** Peak pressures observed in saline preparation in vitro for the unipolar pulse were 142 ± 13 atm (108,000 ± 10,000 mm Hg) as compared with 137 ± 8 atm (103,900 ± 6300 mm Hg) for the bipolar
pursued. Unipolar electrode may cause injury to the coronary pathways, it is crucial that delivery of defibrillatory pulses to the coronary sinus yields unpredictable results, while carrying a substantial risk of rupturing the coronary sinus. Several factors, related either to the anatomy of the accessory pathways or to the many vagaries of the currently available catheter technique, may affect the success of the procedure. Better understanding of these factors is essential if electric means of modifying accessory pathway conduction are to be pursued.

In this study, the intervention was limited to the region of the coronary sinus orifice, based on the consideration that a catheter technique would be most valuable for posterior septal accessory connections. Interventions at the more lateral portion of the coronary sinus currently seem inadvisable, in consideration of the proximity of the left circumflex coronary artery and of a greater likelihood of rupture of the coronary sinus.

Certain technical aspects should also be emphasized. One important variable of catheter-mediated electric ablation is the amount of tissue injury created per pulse. Therefore the intervention was limited to a single pulse at a single site. The choice of the catheter is also crucial. If the effects of the delivered energy on tissue injury are to be investigated, the use of a catheter able to carry a given current becomes imperative. Previous work on catheter dielectric strength has shown that catheters currently used for electric ablation purposes may easily misdirect currents, possibly resulting in

**TABLE 2**

Anatomic changes after a 200 J defibrillator pulse

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<th>Voltage (V)</th>
<th>Delivered energy (J)</th>
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<th>Elastic rupture</th>
<th>Max. length (mm)</th>
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± SD | ± 4 | ± 125 | ± 29 | ± 6 | ± 6 | ± 3 | ± 5 | ± 4 | ± 8 | ± 2 | ± 1 |

| p < .001 | p < .001 | p < .001 | p = NS | p = NS | p = NS | p = NS | p = .0004 | p = NS | p = NS | p = NS | p < .02 |

**Discussion**

Since the first reports of patients treated by a catheter technique for electric ablation of their accessory pathways, it has become clear that delivery of defibrillatory pulses to the coronary sinus yields unpredictable results, while carrying a substantial risk of rupturing the coronary sinus. Several factors, related either to the anatomy of the accessory pathways or to the many vagaries of the currently available catheter technique, may affect the success of the procedure. Better understanding of these factors is essential if electric means of modifying accessory pathway conduction are to be pursued.

In this study, the intervention was limited to the region of the coronary sinus orifice, based on the consideration that a catheter technique would be most valuable for posterior septal accessory connections. Interventions at the more lateral portion of the coronary sinus currently seem inadvisable, in consideration of the proximity of the left circumflex coronary artery and of a greater likelihood of rupture of the coronary sinus.

Certain technical aspects should also be emphasized. One important variable of catheter-mediated electric ablation is the amount of tissue injury created per pulse. Therefore the intervention was limited to a single pulse at a single site. The choice of the catheter is also crucial. If the effects of the delivered energy on tissue injury are to be investigated, the use of a catheter able to carry a given current becomes imperative. Previous work on catheter dielectric strength has shown that catheters currently used for electric ablation purposes may easily misdirect currents, possibly resulting in...
FIGURE 2. Cross sections of the left atrioventricular groove taken at the site of maximum injury and prepared with Gomori's trichrome stain. Section A was taken 9 mm within the coronary sinus orifice from a dog (No. 10) that received a unipolar pulse. Section B was taken 12 mm within the coronary sinus orifice from a dog (No. 12) that received a bipolar pulse. The lumen of the coronary sinus is occluded by an organized thrombus, and the wall of the coronary sinus shows loss of muscle and replacement by circumferential fibrosis in both sections. However, fibrosis of the left atrial wall is transmural (arrow, section A) only in the unipolar configuration. Note that neither configuration resulted in fibrosis adjacent to the annulus of the mitral valve. The left ventricular wall at the level of the coronary sulcus was not injured by the unipolar discharge, but it shows a very limited area of fibrosis as a result of a bipolar discharge (arrowhead, section B). The circumflex coronary artery is patent and no coronary artery wall abnormalities are present in either section. (Original magnification × 5.2 for section A, × 5.7 for section B.) CCA = circumflex coronary artery; CS = coronary sinus; ENDO = endocardium; EPI = epicardium; LA = left atrium; LV = left ventricle; MV = mitral valve.

in failure of the technique or unintended damage of remote tissue. In the experimental setting, this also would make difficult the interpretation of the effects of energy on tissue injury. Therefore a catheter that had high dielectric strength and was capable of transferring high energy to the tissue was needed. The leads used in this study have dielectric strength in excess of 5000 V, but they present the inconvenience of a very high resistance, which would dissipate most of the delivered energy in the catheter. The use of stainless-steel stylets solves this problem yielding values for peak current and voltage comparable to those observed with a USCI No. 6F catheter yet not encountering problems with a high lead resistance.

The energy setting of 200 J was chosen because of previous experiments indicating that pulses of this magnitude delivered in a unipolar fashion results in a desirable extent of transmural atrial injury.³

The results of this study show that electrode configuration is an important factor in determining the extent and the distribution of injury. This is reflected by the inconsistency of transmural atrial injury obtained with a bipolar configuration, as compared with the constancy of such findings after delivery of unipolar pulses. The extent of tissue injury observed in this study with unipolar discharges confirms the results obtained in a previous study.³ In contrast, Brodman et al.¹⁵ did not find a difference in tissue injury between unipolar and bipolar discharges in their series of dogs. Furthermore, in a later clinical report of a series of patients undergo-
ing catheter-mediated ablation of their accessory pathways, these authors expressed the opinion that the same distribution of injury can be a result of either electrode configuration. Unfortunately, their observations cannot be used for comparison in that those authors used multiple shocks at different locations in the coronary sinus in their canine studies with catheters unlikely to direct the current appropriately. Westveer et al. on the other hand, found a significantly greater extent of necrosis after unipolar than bipolar discharges in the canine left ventricle.

We have shown the histologic effects of barotrauma resulting from catheter-mediated defibrillator discharges in the canine coronary sinus. In that study, even at energies as high as 360 J no gross rupture of the coronary sinus occurred, possibly because of the use of two electrodes in parallel as the anode, which avoided concentration of electric energy to a very small area. However, rupture of the coronary sinus elastic membrane within the atrial wall was a constant histologic finding in that study. In the present study, not only was intramural rupture of the coronary sinus elastica present in all animals irrespective of electrode configuration, but also gross epicardial rupture occurred in two dogs of each group. It is conceivable that the higher current densities achieved with the use of a single anodal electrode are responsible for such an occurrence. Brodman et al. observed coronary sinus perforation with bipolar 240 J discharges. Our results show that barotraumatic rupture of the coronary sinus can occur after delivery of pulses at a setting of 200 J with either electrode configuration. However, although unipolar pulses constantly produced atrial transmural injury, this was noted in only two of the 10 dogs receiving bipolar discharges. This latter observation helps substantiate that barotrauma is more of a deleterious phenomenon that does not result in useful tissue injury. With either electrode configuration, pressure rises of similar magnitude were recorded in vitro. Therefore barotrauma is not the sole means of

FIGURE 3. The same sections as in figure 2 are shown after a Verhoeff-van Gieson stain for elastic tissue. Section A (unipolar pulse) and section B (bipolar pulse) correspond to sections A and B of figure 2. Note that, regardless of electrode configuration, the internal elastic membrane of the coronary sinus is ruptured (arrowheads) on the endocardial (section A) and on the endocardial and superior aspect (section B) of the coronary sinus. Also, a segment of the elastica is detached from the rest of the membrane in section A. The kind of disruption observed with both electrode configurations is consistent with a barotraumatic mechanism as the cause of coronary sinus elastica rupture. (Original magnification × 5.2 for section A, × 5.7 for section B.) EM = elastic membrane of the coronary sinus. Other abbreviations are as in figure 2.
FIGURE 4. Higher-power magnifications of the sections in figure 3, showing details of the rupture of the coronary sinus elastica (arrows) after a unipolar pulse (section A) and bipolar pulse (section B). In section A, the arrowhead points to a segment of the elastica that appears to have been forcefully disrupted and displaced within the atrial wall. (Verhoeff–van Gieson stain; original magnification × 12 for section A, × 13.8 for section B.) Abbreviations are as in figures 2 and 3.
FIGURE 5. Representative examples of the voltage and current waveforms obtained for a unipolar (dog 10) (top panel) and a bipolar (dog 15) (bottom panel) discharge. The voltage waveform is displayed above the current waveform. The defibrillator was set at 200 J and for the unipolar pulse resulted in a peak voltage of 3.3 kV, a peak current of 24 A, and a delivered energy of 249 J. For the bipolar pulse, there was a peak voltage of 3.0 kV, a peak current of 75 A, and a delivered energy of 174 J. Note that the upstroke of the curve is similar for both electrode configurations, until a break occurs. It is believed that at this point of the discharge a bubble of electrically nonconductive gas envelops and insulates the electrode, thereby resulting in a sudden rise in impedance. The voltage, on the other hand, continues to rise, triggering an electron “avalanche,” which eventually transforms the previously insulating gas into an ionized conductive gas. In the presence of a conductive medium, current flow can be reestablished, resulting in a spark. The spark, in turn, results in large pressure surges (see figure 6), thus explaining the coronary sinus elastica rupture observed in figures 3 and 4. Thereafter, the waveforms smoothly and slowly decline. In the unipolar pulse, this slow decline probably reflects high tissue resistance to current flow. In the bipolar pulse, the voltage drops dramatically as the current rises, consistent with a very low resistance to current flow between two electrodes situated in blood and only 15 mm apart. The abrupt cessation of current flow observed with the bipolar pulse after approximately 4 msec in the waveform is automatically determined by a mechanical relay switch built into the defibrillator. This relay switch is subject to some bounce, allowing for some current to flow again briefly.

FIGURE 6. Pressure tracings obtained in a saline bath by means of a piezoelectric transducer placed 2 cm from the proximal ring electrode of a Medtronic 6992A lead. A defibrillator pulse was discharged at a setting of 200 J in a unipolar fashion (proximal electrode as the anode to disc electrode as the cathode) (top panel) or in bipolar fashion (proximal electrode as the anode to distal electrode as the cathode) (bottom panel). Pressure rises of comparable magnitudes were recorded with both electrode configurations, measuring 150 atm (114,000 mm Hg) for the unipolar pulse and 132 atm (100,200 mm Hg) for the bipolar pulse. Although these marked pressure surges can explain the barotraumatic injury of the coronary sinus observed with both electrode configurations, the similarity in pressure rises would not account for the difference in transmural atrial injury seen between unipolar and bipolar pulses. This suggests that barotrauma is not the only mechanism of injury and that a different distribution of electric factors alone may play an important role in determining the extent of tissue injury seen with the two electrode configurations.

tissue injury, as evidenced by the atrial transmural injury seen with the unipolar pulse. These observations suggest that electric factors alone could result in appropriate tissue injury.

Some features of acute tissue injury observed in this study (selective myocardial necrosis with contraction bands) have previously been reported as consequences
of external or internal defibrillator discharges.\textsuperscript{17-26} However, they cannot be considered specific “markers” for electric injury, since a number of different insults have been shown to lead to subcellular derangements that eventually cause the pattern of injury observed in this study.\textsuperscript{27, 28} Even though there are quite a few published studies on the histologic effects of defibrillator pulses on the myocardium,\textsuperscript{17-23} none of those studies has been able to address the question of what component of an electric pulse results in cell injury. Using a different approach, Jones et al.,\textsuperscript{29, 30} in their work on the effects of electric pulses applied to cultured chick myocardial cell layers, have suggested that electric field strength can cause cellular dysfunction. Dielectric breakdown of cell membrane has been shown to occur in cells subjected to high-intensity electric fields by defibrillator pulses.\textsuperscript{31} It is conceivable that a similar process takes place during catheter-mediated defibrillator pulses in the myocardial cells subjected to the highest current densities, like the ones that are in the pathway of the electric current flow. A similar distribution of myocardial damage at the site of electrode application was found in studies on cardiac damage produced by epicardial “countershocks.”\textsuperscript{17, 32} A direct relationship between strength of the electric field stimulation and cell dysfunction has been reported by Jones et al.\textsuperscript{29} This could explain why myocardial cells at a greater distance from the electrode site or those not traversed by the current flow could suffer only transient damage. Thus the difference in the distribution of injury observed in this study between unipolar and bipolar discharges could be viewed in terms of differences in electric field strength reached in the target tissue.

The waveforms obtained for current and voltage suggest the occurrence of a spark discharge with gas formation at the electrode receiving electric energy.\textsuperscript{4} Both curves show a break in their initial portion, which likely occurs when current flow stops transiently, due to formation of insulating gas around the electrode. The defibrillator inductor, however, continues to deliver current to the insulated electrode, generating an overvoltage that ionizes the insulting gas. After formation of an ionized gas (“plasma”),\textsuperscript{33} current starts flowing again, since the plasma is a good conductor, resulting in an arc or spark. The spark discharge, in turn, results in a shock wave,\textsuperscript{4, 34-39} which probably accounts for the coronary sinus rupture observed in this study. Our pressure measurements in vitro are consistent with such an occurrence, showing that high pressure surges may indeed occur during defibrillator pulses at a 200 J energy setting.

The extent and distribution of injury observed with bipolar discharges are clearly inadequate to interrupt an accessory pathway reliably or even to prevent conduction through it. The only exception could be an accessory pathway with its atrial end inserting directly into the coronary sinus muscle.\textsuperscript{40, 41} Also, the kind of injury resulting from unipolar discharges is far from ideal. An ideal injury should cover the entire thickness of the AV groove at the level of the anulus fibrosus and should, at the same time, extend far enough along the AV groove to leave a good margin for the lack of precision of catheter mapping. Surgical experience indicates that accessory AV connections can occur over a wider area of the AV groove other than the precise area of earliest activation.\textsuperscript{42-44} Our results confirm previously reported results\textsuperscript{1} showing that unipolar pulses can result in transmural atrial injury above the AV groove but not at the level of the anulus fibrosus. Transmural atrial injury typically started about 5 mm above the anulus of the mitral valve, sparing the periannular myocardium. The periannular area showed, at most, tiny ramifications of interstitial fibrosis. It is unknown at present whether varying the position of the chest plate can result in more periannular damage. However, if that is the case, concern should be raised about the possibility of creating more extensive ventricular damage. Theoretically, a boundary of fibrotic tissue above the atrial insertion of an accessory pathway should prevent or delay the arrival to or the exit from the bypass tract of a depolarization wavefront. Also, a change in the refractoriness of the myocardium at the area of insertion might modify the characteristics of the tachycardia, making it less frequent, slower, or more easily controllable by previously ineffective antiarrhythmic agents. In fact, the latter possibility has been reported in some patients undergoing attempted electric ablation of their accessory pathways.\textsuperscript{8, 10-12}

No significant electrophysiologic alterations were observed in this study, regardless of electrode configuration. The almost constant observation of brief episodes of ventricular tachycardia immediately after the discharge is not surprising. Previous studies in the canine left ventricle have documented the arrhythmogenicity of endocardial catheter-mediated electric discharges.\textsuperscript{16, 25-26, 45} Although in those studies actual damage of the ventricular myocardium may have been an important factor in the development of arrhythmias, the brief runs of ventricular tachycardia observed in our study would be more consistent with transient depolarization of cells subjected to a high-intensity electric field\textsuperscript{29} given the absence of a clearly defined anatomic substrate. Very little ventricular myocardial
injury was produced in our study with either electrode configuration.

Similarly, no short or long-term impairment of AV conduction was noted in this study. This might have been surmised from previous results obtained by catheter techniques. Unless the discharging electrode is in very close contact with the AV node/His bundle area, production of permanent AV block is unlikely.56-58

In conclusion, catheter-mediated electric ablation techniques applied to the coronary sinus orifice may have better chances of success if a unipolar electrode configuration is used. With bipolar discharges, the deleterious effects of the technique seem to prevail, while preventing factors associated with useful tissue damage from exercising their effects. However, neither configuration prevents rupture of the coronary sinus. The possibility of barotraumatic rupture and several other limitations imposed on the technique by the source of electric energy and by the electrodes used should be carefully weighed before applying this technique in human subjects.

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References

23. Lepeschkin E, Jones JL, Rush S, Jones RE: Analysis of cardiac damage following elective cardiac defibrillation. In Geddes LA, Tacker WA, editors: Cardiac Defibrillation Conference. Purdue University, 1975, p 103
28. Davis JS, Lie JT, Bentinck DC, Titus JL, Tacker WA, Geddes LA: Cardiac damage due to electric current and energy. In Geddes LA, Tacker WA, editors: Cardiac Defibrillation Conference. Purdue University, 1975, p 27
Effects of varying electrode configuration with catheter-mediated defibrillator pulses at the coronary sinus orifice in dogs.
F Coltorti, G H Bardy, D Reichenbach, H L Greene, R Thomas, D G Breazeale and T D Ivey

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