In Vitro and In Vivo Effects Within the Coronary Sinus of Nonarcing and Arcing Shocks Using a New System of Low-Energy DC Ablation

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DC shocks within the coronary sinus have been abandoned because of the risk of cardiac rupture and tamponade. Catheter ablation using DC energy to electrodes straddling the ostium of the coronary sinus, when used clinically, has been reported to result in cardiac tamponade in as many as 16% of patients. A new system of energy delivery maximizes voltage while decreasing the undesirable effects caused by barotrauma. This system includes 1) a low-energy ablation power supply with a brief time-constant capacitive discharge that delivers up to 40 J and 3,000 V and 2) a low-energy ablation catheter with a contoured distal electrode. We performed in vitro and in vivo studies of this new system and compared arcing shocks with nonarcing shocks. Ablations were performed using unipolar distal shocks (D) and unipolar shocks to both electrodes made electrically common (P-D). In vitro studies were done in a large tank filled with physiological saline while recording voltage, current, and pressure. High-speed cinematography (32,000 frames/sec) of shocks of 10–40 J permitted detailed analysis of the vapor globe. Anodal shocks of less than 20 J showed no arcing or only minimal vapor globe formation. For D and P-D anodal shocks of 40 J, the diameters of the vapor globe were 31 and 22 mm, respectively, corresponding to pressure recordings of 11 and 4.9 atm. The pressure rise lasted less than 50 μsec. In vivo studies involved 18 dogs that received nonarcing shocks (one to six shocks of 15 J) and 18 dogs that received arcing shocks (one to three shocks of 40 J). Each group was divided between D and P-D shocks; catheter ablation was performed at a mean±SEM distance of 2.94±0.92 cm within the coronary sinus. All dogs tolerated the procedure without cardiac rupture or tamponade. When killed 2–4 days later, the dogs had edema and hyperemia or hemorrhage in the area of the coronary sinus. We compared the effects of multiple (three to six) nonarcing shocks with the effects of one to three arcing shocks. Disruption or rupture of the coronary sinus within the epicardial fat space occurred in two of 12 dogs (17%) with multiple nonarcing shocks but in 13 of 18 dogs (72%) with arcing shocks ($p<0.003$). Occlusion of the coronary sinus occurred in two of 12 dogs (17%) with multiple nonarcing shocks and in nine of 18 dogs (50%) with arcing shocks ($p<0.06$). Sustained ventricular tachycardia occurred only in dogs with arcing shocks. Focal medial necrosis of the circumflex coronary artery occurred in six dogs (33%) in each group and was seen only in dogs that received multiple shocks. In one dog that received three arcing shocks of 40 J, a thrombus was also seen in the lumen of the coronary artery. The mean±SEM volume of myocardial necrosis of the atrial wall and the summit of the left ventricle was 0.41±0.52 and 0.27±0.33 cm^3, respectively, and did not differ significantly between groups. We conclude that DC energy can be given within the coronary sinus using this new system. Compared with arcing shocks, multiple nonarcing shocks cause less damage to the coronary sinus, equal myocardial necrosis, and no sustained ventricular tachycardia. Arcing with barotrauma is primarily responsible for the deleterious effects of catheter ablation; voltage or current density, rather than barotrauma, is responsible for useful tissue injury. Multiple nonarcing shocks within the coronary sinus appear to be safe and may be effective for ablation of left-sided accessory pathways. (*Circulation* 1991;83:279–293)

Catheter ablation in patients with the Wolff-Parkinson-White syndrome has been mainly performed in those with posteroseptal accessory pathways.[1-3] The technique consists of inserting a quadripolar catheter into the coronary sinus while positioning the proximal pair of electrodes to straddle the os of the coronary sinus or to lie slightly outside the ostium.[1] Unipolar shocks with a cumulative energy of up to 1,200 J are delivered through one of the two proximal electrodes or through both electrodes.[1] Although a significant number of patients have had permanent interruption of conduction of the accessory pathway, the technique remains investigational.[4] Indeed, exact positioning of the catheter within the area of the os may be difficult, and in some instances direct positioning of the catheter within the proximal coronary sinus may be necessary.[2] Using this technique, coronary sinus perforation and cardiac tamponade have been reported to occur in as many as 16% of patients.[2,5]

Catheter ablation within the coronary sinus was initially reported to be either unsuccessful (energy of 40–80 J)[6] or to cause coronary sinus rupture and cardiac tamponade (100–150 J).[6] More recently, ablations have been performed using radiofrequency energy,[7-9] either in the unipolar mode from a single catheter in the coronary sinus[7,8] or in the bipolar mode between a catheter in the coronary sinus and a catheter in the left ventricle.[9] Animal studies have shown that these techniques are well tolerated but that the size of the lesions tends to be small.[7-9] One technique involves left heart catheterization,[9] and clinical reports using radiofrequency energy show highly variable success rates.[10,11]

A new low-energy ablation system using DC maximizes energy delivery while minimizing the undesirable effects caused by barotrauma.[12,13] This system consists of 1) a low-energy ablation power supply (Cardiac Recorders, London, U.K.) with a brief time-constant capacitive discharge that delivers up to 40 J and 3,000 V and 2) a low-energy ablation catheter with a contoured distal electrode (Bard-USCI, Tewksbury, Mass.). Our present study was performed to determine the in vitro characteristics of this new system of energy delivery and to compare unipolar distal shocks with unipolar shocks to both electrodes made electrically common. We then evaluated whether low-energy DC ablation, using nonarcing shocks or arcing shocks with the new system, within the coronary sinus of dogs could produce atrial and ventricular necrosis adjacent to the mitral annulus without causing cardiac rupture.

Methods

New System of Energy Delivery

Low-energy ablation power supply. This new ablator[12,13] uses an unmodified capacitive discharge to deliver a peak voltage twofold to threefold that for a given energy and fivefold to sixfold faster than a standard defibrillator, which uses a damped sinusoidal waveform. The capacitor value is 8 μF. The capacitor stores 40 J when charged to 3.0 kV. Peak current varies according to the resistance of the medium and may reach 100 A during in vitro testing in saline. Before shock delivery, the voltage from the capacitor is sensed and regulated to ensure that the set amount of energy will be delivered. During in vivo testing, the delivery of energy is synchronized to the R wave via a connection to an electrocardiogram monitor. The firing circuit is triggered by a 50-msec pulse generated by the detection of the R wave, thus ensuring that the ablation discharge may be delivered only during the safest period of the cardiac cycle.

Low-energy ablation catheter. This new catheter is a 7F bipolar electrode with a contoured distal electrode designed to carry high-voltage energy.[13] Arcing occurs at the point of maximum electric field intensity on the electrode surface.[13] The new catheter, with an oval distal electrode, has a large surface area and a more uniform electric field intensity, allowing for higher voltages to be achieved before arcing occurs.[13] The catheter consists of two platinum electrode circuits separated by Teflon and mounted in a catheter body made from woven Dacron, polyurethane, and an antithrombogenic coating.

Prior testing of this new system of energy delivery. In vitro testing[13] has shown that arcing thresholds are significantly higher with this new system. Compared with a standard defibrillator (Lown shape impulse) coupled with a conventional electrode, the voltage, current, and energy of this new system are increased by 590%, 990%, and 110%, respectively, before arcing occurs.[13] Furthermore, previous work from our laboratory[14] has shown that when arcing occurs with the new system, the vapor globe and pressure wave produced by the shock are significantly smaller than with the conventional system, thus decreasing the barotraumatic effects.

In Vitro Study

Experiments were carried out in a rectangular tank measuring 50×30×40 cm with walls made of polymethyl methacrylate. The tank was filled with 30 l physiological saline. The catheter was inserted into the tank and held in place by a plaque attached to the middle of the catheter. The indifferent electrode consisted of a stainless steel defibrillator paddle inserted at the base of the tank (15 cm from the catheter). The catheter was connected to a custom-made connection box that incorporated a high-ten-

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sion relay and was constructed to allow for either cathodal or anodal shocks by coupling this box to the power source described above. A high-frequency hydrophone (Type 8105, Bruel and Kjaer Instruments, Inc., Marlborough, Mass.) was placed in the tank 3 cm from the catheter tip and connected directly to a four-channel, eight-bit digital storage oscilloscope (1604, Gould Inc., Glen Burnie, Md.) to maximize the dynamic range of amplitude and frequency response (0.1 Hz to 100 kHz with a maximum recordable pressure of 100 atm).

Delivered voltage was measured using a precision voltage divider. Delivered current was measured by a current transformer with a ratio of 10 to 1. Voltage, current, and pressure measurements were stored on the oscilloscope.

High-speed cinematography. Actual filming of the shocks was done with a Dynafax Model 350 framing camera (Cordin, Utah). The camera (rotating drum) recorded 224 16-mm frames at rates of up to 32,000 frames/sec. Total recording time was 6.4 msec for 224 frames on 860 mm of 35-mm film. Frames were positioned in two rows, each consisting of 112 frames. An electronic flash unit (Model 359, Cordin) supplied a single square pulse of light; exposure time was 7 msec. The shutter of the camera was left open, and the image was created by the flash that was triggered upon the rise in voltage by a voltage comparator with a very low reference voltage. The trigger time of the flash was 50 μsec. The camera was connected by a C adaptor and a T adaptor to a long-distance microscope (Infinity Photo-optical Co., Boulder, Colo.). This allowed for filming of shocks of 10–40 J by either decreasing or increasing the distance between the camera and the catheter. We used T Max ASA 400 Kodak film (Rochester, N.Y.). The negatives obtained of each shock were carefully analyzed, and the maximum diameter of the vapor globe was recorded. Every second image (31.2 μsec between images) was analyzed until there was either no further increase in diameter of the vapor globe during expansion or disappearance of the vapor globe during implosion.

The effects of modifying the variables related to the delivery of shocks were studied. We compared 1) anodal and cathodal shocks and 2) unipolar shocks using the distal electrode of the catheter and unipolar shocks using both electrodes of the bipolar catheter made electrically common. Shocks of 10, 15, 20, 30, and 40 J were analyzed. Multiple shocks were given for each experiment; the recorded values were highly reproducible.

In Vivo Study

Thirty-six adult mongrel dogs of either sex weighing 14–32 kg were studied. The dogs were anesthetized with pentobarbital and mechanically ventilated. The 7F catheter described above was inserted into the right internal jugular vein by cutdown, advanced under fluoroscopic guidance into the coronary sinus, and positioned as far anteriorly as possible (without further manipulation of the catheter). The location of the catheter in the coronary sinus was confirmed by recordings of the intracardiac electrograms (filtered at 30–500 Hz) with a lead II surface electrocardiogram. All dogs received 1,000 units heparin before the first shock.

Catheter ablation within the coronary sinus was performed using the power source described above. The indifferent electrode consisted of a large posterior chest wall patch. In one group, 18 dogs received nonarcing shocks of 15 J (six dogs received one shock, six dogs received three shocks, and six dogs received six shocks); in the other group of 18 dogs, arcing shocks of 40 J were given (six dogs received one shock, six dogs received two shocks, and six dogs received three shocks). In each group, two subgroups of nine dogs were studied. The first subgroup received unipolar shocks to the distal electrode. In the second subgroup, both electrodes of the bipolar catheter in the coronary sinus were made electrically common and connected to the power source. When more than one shock was given, there was an observation period of 2–10 minutes between shocks. The same catheter was used repeatedly in dogs receiving multiple shocks (later examination did not reveal any gross defects). In all 36 dogs anodal shocks were given; the catheter served as the positive pole and the indifferent electrode as the negative pole. The catheters were removed, and the dogs were returned to the animal quarters where they received analgesics and antibiotics. The dogs were reanesthetized 2–4 days later, and the hearts were excised and fixed in formalin.

During examination of the heart, the pathologist was blinded to the mode of unipolar shocks and to the number of shocks given. The right atrium was opened, and the ostium of the coronary sinus was examined. The left atrium was then opened, and the site of subendocardial hyperemia or hemorrhage was determined. A transverse section at this level was invariably associated with the greatest extent of damage and was estimated to correspond to the distance from the ostium of the coronary sinus where the shocks were given. Transverse blocks of grossly visible lesions were embedded in paraffin, sectioned, and stained with hematoxylin and eosin. The morphology of the ablative lesion was considered to be equivalent to a rotated ellipsoid,[15] and the volume was calculated as $\frac{1}{6}\pi\times\text{depth}\times\text{width}\times\text{length}$. The circumflex coronary artery was closely examined. Transverse cuts were divided into eight segments, and the extent of necrosis was analyzed according to the number of segments involved.

Statistical Analysis

For the in vitro studies, Student's unpaired two-tailed t test was used to compare anodal with cathodal values and to compare unipolar distal shocks with unipolar shocks to both electrodes made electrically common. For the in vivo studies, we compared dogs that received arcing shocks with those that received

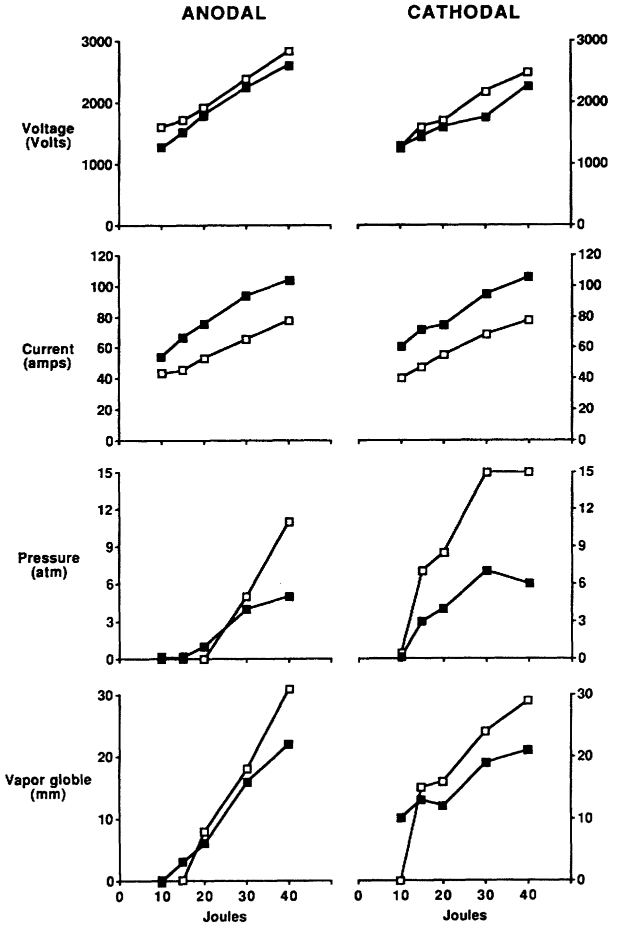


FIGURE 1. *Results of in vitro studies of anodal and cathodal shocks for unipolar distal shocks (□) and unipolar shocks to both electrodes made electrically common (■). There were no significant differences between overall values obtained for voltage, current, pressure, or maximum diameter of the vapor globe.*

nonarcing shocks and dogs that received unipolar distal shocks with those that received unipolar shocks to both electrodes made electrically common. Student's two-tailed *t* test was used to compare the cumulative energy and the extent of hemorrhage and necrosis of the atrium, ventricle, and coronary sinus. The χ^2 test was used to compare the frequencies of pathological abnormalities between groups.

Results

In Vitro Study

For both modes of shock delivery, voltage and current increased linearly between 10 and 40 J. There was no significant difference between overall values obtained for anodal and cathodal shocks or between values obtained for unipolar distal shocks and unipolar shocks to both electrodes made electrically common (Figure 1). Nonlinear impedance (Figures 2C and 2D) was seen whenever there was vapor globe formation (Figure 3) reflecting isolation of the catheter tip and an electrical arc.[13,14]

Vapor globe formation. During unipolar distal shocks and unipolar shocks to both electrodes made electrically common, the vapor globe was seen for cathodal shocks of 10 J but not for anodal shocks of less than 20 J. The diameter of the vapor globe for shocks of 40 J were similar, ranging between 21 and 31 mm, but cathodal shocks always showed greater increases in pressure. The low-energy ablation catheter was designed to increase the surface area of the distal electrode and carry high-voltage energy. During unipolar distal shocks, the vapor globe formed at both ends of the distal electrode, without midline fusion for nonarcing shocks of less than 20 J (Figure 4) or with midline fusion for arcing shocks, as shown in Figure 3. After collapse of the vapor globe, the pressure increased to 11 atm (Figure 2C) over less than 50 μsec. Small reverberations of pressure followed the initial pressure increase.

For shocks to both electrodes made electrically common, the behavior of the proximal electrode was analogous to that of a standard catheter. The vapor globe formed at the proximal ring of the proximal electrode and readily insulated the electrode. However, flow of current and voltage to the large distal electrode limited the increase in diameter of the vapor globe at both tips. The collapse of the vapor globe (Figure 3B) occurred over 0.56 msec (compared with 0.94 msec in Figure 3A), and the pressure increase reached 4.9 atm (lasting less than 50 μsec). Figure 2 shows that the current fell to 0 A during a significantly longer period of time (1.5 msec) for unipolar distal shocks than for unipolar shocks to both electrodes made electrically common (approximately 0.6 msec) and could reflect more sustained arcing for distal shocks.

In Vivo Study

All 36 dogs tolerated the ablation procedure and showed no signs of abnormal behavior before being killed. Nonsustained or sustained ventricular tachycardia occurred in 14 of 18 dogs (78%) with arcing shocks and in eight of 18 dogs (44%) that had nonarcing shocks ($p<0.04$) (Tables 1 and 2). Lidocaine (50 mg i.v.) was required to terminate ventricular tachycardia in two dogs that had sustained ventricular tachycardia. Nonsustained or sustained ventricular tachycardia terminated spontaneously in the other dogs. Sustained ventricular tachycardia in four dogs occurred only in those that received arcing shocks. In all dogs, the ventricular tachycardia was monomorphic, with a variable cycle length of 280–400 msec. The arrhythmia always occurred within the first minute after the shock.

Gross and histological findings. There was no evidence of blood in the pericardium upon removing the heart, and the coronary sinus never showed external signs of rupture. In one dog (that received an arcing shock), fibrin deposition was present over the coronary sinus, but there was no evidence of perforation. All dogs showed edema, hyperemia, or hemorrhage overlying the area of the coronary sinus in the

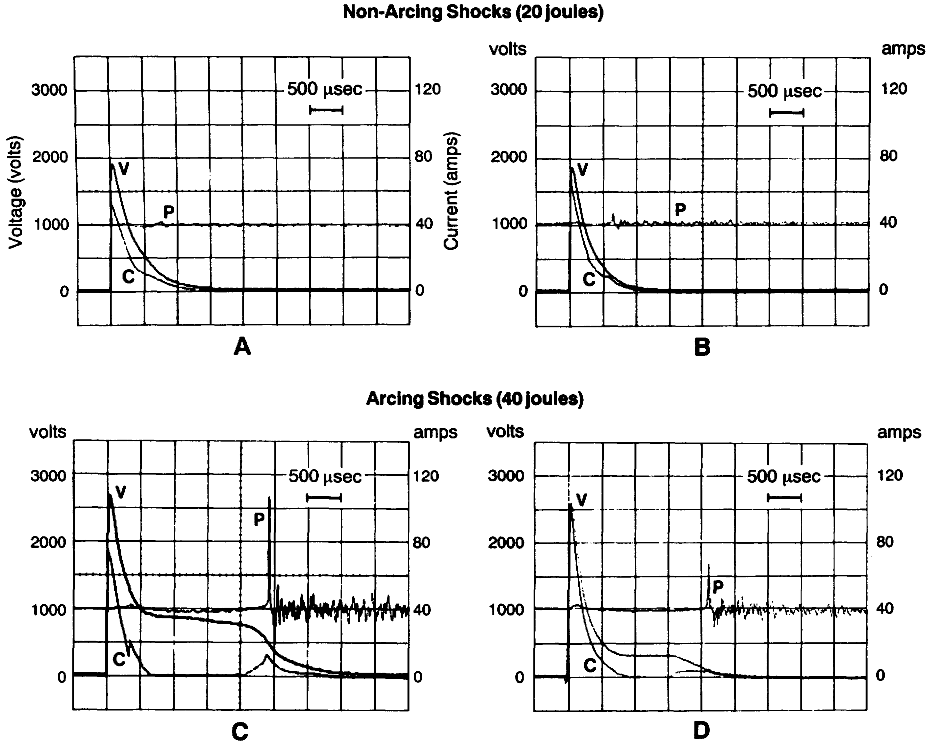


FIGURE 2. *Voltage (V), current (C), and pressure (P) values for in vitro anodal shocks of 15 and 40 J shown in Figures 3 and 4. Nonarcing shocks shown above; in* A, *distal unipolar shock of 15 J (1,900 V, 50 A); and in* B, *unipolar shock of 15 J (1,900 V, 70 A) to both electrodes made electrically common. Both show linear voltage–current curves without any significant increase in pressure. Arcing shocks shown below; in* C, *distal unipolar shock of 40 J (2,700 V, 78 A, 11 atm); and in* D, *unipolar shock of 40 J (3,100 V, 90 A, 4.9 atm) to both electrodes made electrically common. Increase in pressure occurs after implosion of vapor globe, with gas formation around electrode, as shown in Figure 3.*

atrioventricular fat pad; the mean length and width of these changes were 3.5±2.0 and 1.5±0.5 cm, respectively, in dogs with arcing shocks and 2.0±1.5 and 1.0±0.4 cm, respectively, in dogs with nonarcing shocks ($p<0.05$ between groups). Subendocardial hemorrhage or hyperemia of the left atrium was always present in dogs with arcing shocks and occurred in 13 of 18 dogs (77%) with nonarcing shocks. No thrombus was seen in the left atrium.

Myocardial necrosis. Multiple (three to six) nonarcing shocks of 15 J and one to three arcing shocks of 40 J in 30 dogs revealed myocardial necrosis of the atrium and ventricle of 0.41±0.52 and 0.27±0.33 cm^3, respectively, without significant differences between groups (Figure 5 and Table 3). In three dogs that received unipolar shocks to both electrodes made electrically common, subepicardial myocardial necrosis extended to the apex of the left ventricle in one dog that received an arcing shock of 40 J and in one dog that had six nonarcing shocks of 15 J, and the necrosis extended to the right ventricular free wall in another dog that had three arcing shocks of 40 J.

Coronary sinus. In all 36 dogs, the ostium of the coronary sinus was normal. Catheter ablation was performed 2.94±0.92 cm within the coronary sinus. Gross rupture of the coronary sinus, confined to the epicardial fat pad, occurred only in dogs that received arcing shocks and was seen in six of eight dogs that received shocks to the distal pole and in three of nine dogs in the other group ($p<0.02$). Microscopic disruption or rupture of the coronary sinus was seen in all dogs that had distal arcing shocks and in six of nine dogs that had arcing shocks to both electrodes. In dogs that had nonarcing shocks, microscopic rupture of the coronary sinus was seen in three of nine with distal shocks and in none of nine with shocks to both electrodes.

Occlusion of the coronary sinus was seen in three of 18 dogs (17%) with nonarcing shocks and in nine of 18 (50%) with arcing shocks ($p<0.03$). Moderate to extensive hemorrhage within the fat pad was seen in six of 18 dogs (33%) that had nonarcing shocks and in nine of 18 dogs (50%) that had arcing shocks.

Coronary arteries. There was never evidence of occlusion or disruption of the circumflex coronary artery. However, focal medial necrosis was seen in six dogs (33%) that had nonarcing shocks and in six dogs (33%) that had arcing shocks. As shown in Tables 1 and 2, lesions to the circumflex coronary artery were only seen in dogs that received multiple nonarcing or arcing shocks. In one dog that received three arcing shocks of 40 J, a thrombus was also seen in the lumen of the coronary artery.

Discussion

This study shows that using a new system of energy delivery, low-energy DC ablation within the coronary sinus may produce significant necrosis of the atrium and summit of the left ventricle without causing cardiac rupture and tamponade. The areas of involvement with coagulation necrosis suggest that ablation of an accessory pathway could be accomplished with this technique. However, remote lesions in the ventricle were seen in three dogs that had unipolar shocks to both electrodes made electrically common. Circumflex coronary artery lesions were also documented using this new system of low-energy DC ablation.

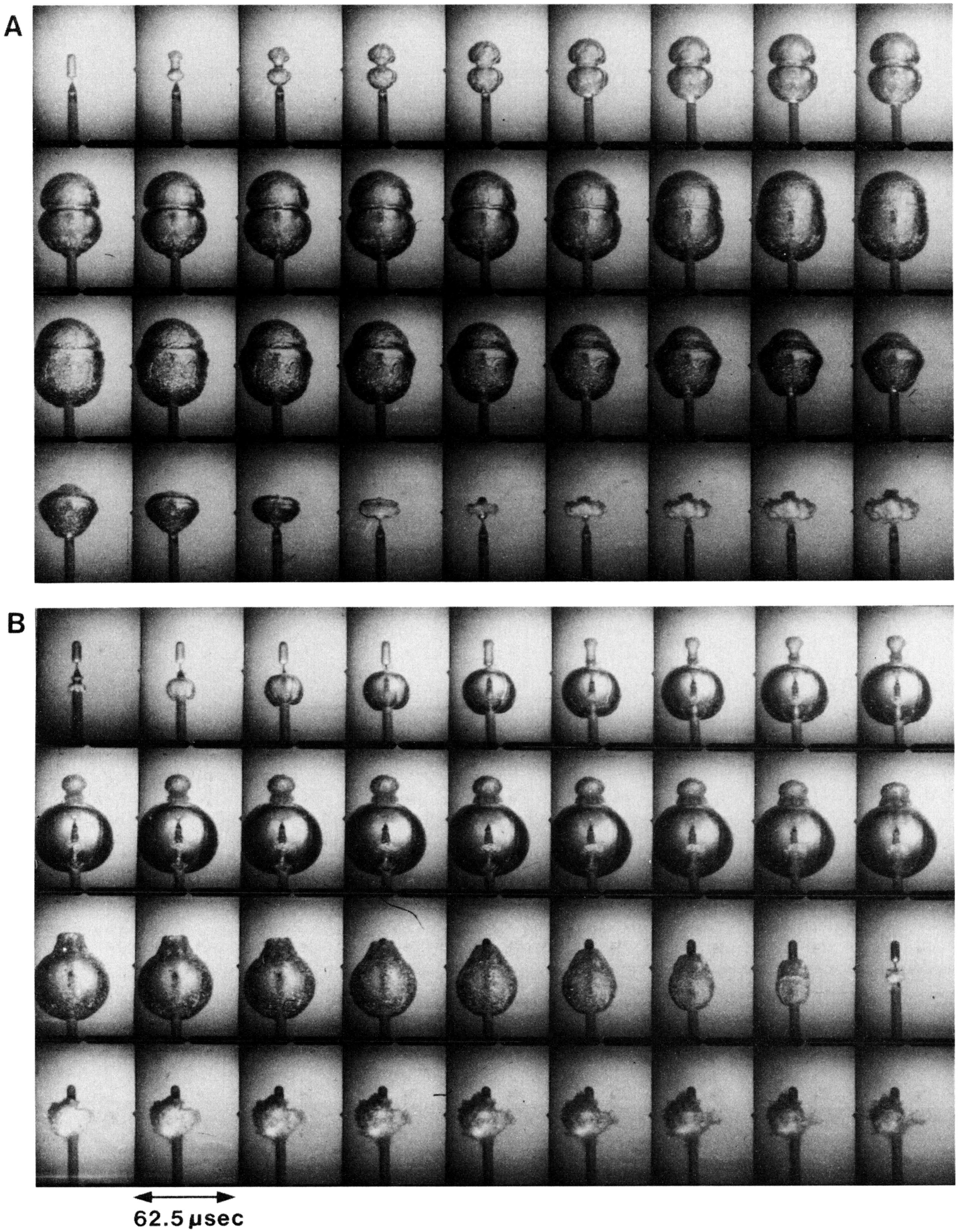


FIGURE 3. *High-speed cinematography of in vitro anodal arcing shocks.* A: *Unipolar distal shock of 40 J;* B: *Unipolar shock of 40 J to both electrodes made electrically common. A and B correspond to voltage, current, and pressure curves shown in Figures 2C and 2D, respectively. Time between frames is 62.5 µsec. Sequence follows from left to right, through rows 1–4. Two frames at end of each row have been eliminated for space arrangement.*

TABLE 1. Low-Energy DC Ablation: Pathological Findings Induced by Nonarcing Shocks Within Coronary Sinus in 18 Dogs

Dog	Heart weight (g)	Shocks (*n*)	E (total)	V (total)	C (total)	Coronary sinus: Os (max cm)	Site of shock† (max cm)	Ext (H/edema) L (max cm)	Ext (H/edema) W (max cm)	Int	Rupture G	Rupture M	EFP H	TM	Left atrial necrosis: LAH (max cm)	L (max cm)	Ht (max cm)	Left ventricular necrosis: L (max cm)	D (max cm)	W (max cm)	VT	CxCA
Unipolar distal shocks																						
1	180	1	15	1,873	8	0.5	4.0	1.5	0.7	N	No	No	...	No	0	0.8	0.6	0	0	0	No	N
2	160	1	15	2,170	4	0.6	3.0	1.4	0.8	N	No	Yes	++	Yes	0.8	1.2	1.0	0.8	0.7	0.3	No	N
3	180	1	15	2,275	8	0.6	4.5	3.0	1.2	0	No	No	++	No	0.3	1.2	0.4	0	0	0	No*	N
4	120	3	45	6,021	27	0.5	5.5	1.0	1.0	N	No	No	...	No	0	0	0	1.0	0.4	0.9	No	N
5	90	3	45	5,828	20	0.3	2.8	2.0	1.2	0	No	Yes	...	Yes	0.8	1.4	1.1	1.2	0.2	0.6	No	FMN(⅛)
6	150	3	45	6,039	26	0.4	2.8	2.0	1.1	N	No	No	+	Yes	1.5	1.6	1.4	0.8	0.8	0.2	NS	N
7	150	6	90	11,730	44	0.6	2.5	1.0	1.0	N	No	Yes	+	Yes	0.8	1.2	1.2	1.6	0.7	0.2	NS	FMN(⅛)
8	180	6	90	11,690	34	0.5	4.2	7.0	2.0	0	No	No	+++	Yes	3.5	5.6	1.7	1.8	1.2	0.4	NS	FMN(¼)
9	130	6	90	11,380	54	0.6	3.5	2.5	1.5	N	No	No	+	Yes	2.5	1.6	2.0	1.6	1.0	0.5	NS	N
Total	149±31			6,556±4,138	25±17	0.5±0.1	3.6±0.9	2.4±1.9	1.2±0.4						1.1±1.2	1.6±1.5	1.0±0.6	1.0±0.7	0.6±0.4	0.3±0.3		
Unipolar shocks to both electrodes made electrically common																						
10	130	1	15	1,590	6	0.4	3.5	4.0	0.5	N	No	No	...	No	1.5	0	0	0.8	0.4	0.1	No	N
11	140	1	15	1,610	10	0.4	3.0	0	0	T	No	No	+	No	0	0.8	0.8	0	0	0	NS	N
12	140	1	15	2,030	7	0.4	2.8	1.5	0.8	N	No	No	++	Yes	0.4	1.2	1.3	0.6	0.5	0.3	No	N
13	230	3	45	3,935	22	0.8	2.5	1.5	1.0	T	No	No	...	Yes	1.0	1.5	1.0	0	0	0	No	N
14	320	3	45	5,630	30	0.8	3.5	2.0	1.0	N	No	No	++	Yes	0	1.6	1.2	0	0	0	No‡	N
15	160	3	45	5,940	36	0.4	3.0	1.0	0.6	T	No	No	...	Yes	0	1.8	1.2	1.8	1.0	0.2	NS	FMN(⅛)
16	130	6	90	11,660	31	0.3	2.6	2.0	1.0	N	No	No	+	Yes	1.3	0.8	0.6	0.6	0.5	0.2	NS	N
17	190	6	90	11,160	29	0.5	3.0	2.0	1.0	T	No	No	+	Yes	1.0	1.2	1.8	0	0	0	No	FMN(¼)
18	190	6	90	11,570	31	0.6	2.0	1.5	0.8	N	No	No	++	Yes	1.0	0.4	0.3	1.2	0.7	0.2	NS	FMN(¼)
Total	181±62			6,125±4,307	22±11	0.5±0.2	2.8±0.5	1.7±1.1	0.7±0.3						0.7±0.6	1.0±0.6	1.7±2.4	0.6±0.6	0.7±1.3	0.1±0.1		
Mean	165±50			6,341±4,103	24±14	0.5±0.1	3.3±0.8	2.0±1.5	1.0±0.4						0.9±0.9	1.3±1.2	1.4±1.7	0.8±0.7	0.6±0.9	0.2±0.2		

E, energy (J); V, voltage (V); C, current (A); Ext, external; H, hemorrhage; Int, internal; EFP, epicardial fat pad; TM, transmural; LAH, hemorrhage or hyperemia of subendocardial aspect of left atrium; L, length; Ht, height; D, depth; W, width; VT, ventricular tachycardia; Cx CA, circumflex coronary artery; G, gross; M, microscopic; N, normal; O, occlusion; T, thrombus; +, slight; ++, moderate; +++, extensive; NS, nonsustained; FMN, focal medial necrosis (area of involvement in circumference).

Coronary sinus ostium was normal in appearance in all dogs.

p=NS between mean values in each group. Nonsustained ventricular tachycardia terminated spontaneously in all dogs.

*Nonsustained atrial fibrillation

†Site of shock, in cm from ostium of coronary sinus.

‡Sustained atrial fibrillation, but reverted to sinus rhythm in <12 hours.

TABLE 2. Low-Energy DC Ablation: Pathological Findings Induced by Arcing Shocks Within Coronary Sinus in 18 Dogs

						Coronary sinus								Left atrial necrosis				Left ventricular necrosis				
							Site of	Ext (H/edema)		Int	Rupture		EFP H									
Dog	Heart weight (g)	Shocks (n)	E (total)	V (total)	C (total)	Os (max cm)	shock† (max cm)	L (max cm)	W (max cm)		G	M		TM	LAH (max cm)	L (max cm)	Ht (max cm)	L (max cm)	D (max cm)	W (max cm)	VT	CxCA
Unipolar distal shocks																						
1	130	1	40	3,598	11	0.4	1.5	7.0	2.0	T	Yes	Yes	+	Yes	1.0	1.8	1.1	1.3	1.5	1.2	NS	N
2	250	1	40	3,338	15	0.3	2.3	2.1	1.1	0	No	Yes	...	Yes	1.2	1.6	1.0	1.6	0.3	0.5	No	N
3	180	1	40	3,000	14	0.5	4.0	3.0	1.0	0	No	Yes	+	Yes	0.2	0.8	1.0	0.5	0.2	0.4	NS	N
4	180	2	80	6,624	37	0.4	2.5	3.0	2.0	T	Yes	Yes	...	Yes	1.2	1.6	0.7	0.3	0.3	0.4	S	N
5	130	2	80	5,710	27	0.4	2.8	8.5	2.2	T	Yes	Yes	+++	Yes	1.5	2.0	0.9	2.0	0.4	0.9	No	N
6	170	2	80	6,726	32	0.4	3.2	4.5	1.2	N	Yes	Yes	+	Yes	1.0	1.6	1.0	0.8	0.3	0.8	No	N
7	130	3	120	8,450	44	0.3	1.8	3.0	1.5	0	Yes	Yes	+++	Yes	1.0	1.6	1.0	0.8	0.2	0.3	NS	N
8	140	3	120	9,309	43	0.3	2.2	3.0	1.3	0	No	Yes	+++	Yes	2.5	1.6	1.6	1.6	0.2	0.5	S*	FMN(⅛)
9	140	3	120	8,866	59	0.4	2.5	3.0	1.5	0	Yes	Yes	++	Yes	1.3	1.5	1.2	1.5	0.6	1.2	NS	FMN(⅛)
Total	161±39			6,180±2,443	31±16	0.4±0.1	2.5±0.7	4.1±2.2	1.5±0.4						1.2±0.6	1.6±0.3	1.1±0.2	1.2±0.6	0.4±0.4	0.7±0.3		
Unipolar shocks to both electrodes made electrically common																						
10	160	1	40	3,129	14	0.4	3.2	5.0	1.5	0	Yes	Yes	++	Yes	3.5	4	2.8	3.5	0.4	0.7	NS	N
11	120	1	40	3,156	14	0.4	2.5	1.5	1.0	T	No	No	...	Yes	0.8	1.0	0.9	1.5	0.3	0.7	NS	N
12	140	1	40	3,181	15	0.4	1.8	2.0	0.8	T	No	Yes	++	Yes	0.5	1.6	1.0	0.8	0.3	0.6	S	N
13	110	2	80	5,659	41	0.4	2.0	1.5	1.0	T	No	No	...	Yes	1.4	1.0	1.5	1.0	0.3	0.8	NS	N
14	120	2	80	5,763	40	0.4	5.0	2.5	1.4	0	Yes	Yes	+++	Yes	1.0	1.0	0.8	1.2	0.5	1.0	No	N
15	160	2	80	5,476	40	0.5	1.8	2.0	0.8	T	No	No	++	Yes	0.8	0.8	1.4	0.9	0.2	0.6	NS	FMN(⅛)
16	180	3	120	8,111	50	0.3	2.0	1.5	1.2	T	No	No	...	Yes	1.5	2.0	1.3	2.0	0.7	1.4	NS	FMN(¼)
17	170	3	120	8,944	42	0.5	2.5	4.0	2.5	0	Yes	Yes	...	Yes	1.4	1.5	1.4	1.5	0.4	1.1	NS	FMN(½)
18	210	3	120	8,032	61	0.6	3.5	6.0	2.2	0	No	No	+++	Yes	2.0	2.5	1.5	2.5	0.7	1.2	S*	FMN(¼)‡
Total	152±33			5,717±2,271	35±17	0.4±0.1	2.7±1.0	2.9±1.7	1.4±0.6						1.4±0.9	1.7±1.0	1.4±0.6	1.7±0.9	0.4±0.2	0.9±0.3		
Mean	157±35			5,948±2,300	33±16	0.4±0.1	2.6±0.9	3.5±2.0	1.5±0.5						1.3±0.7	1.6±0.7	1.2±0.5	1.4±0.8	0.4±0.3	0.8±0.3		

E, energy (J); V, voltage (V); C, current (A); Ext, external; H, hemorrhage; Int, internal; EFP, epicardial fat pad; TM, transmural; LAH, hemorrhage or hyperemia of subendocardial aspect of left atrium; L, length; Ht, height; D, depth; W, width; VT, ventricular tachycardia; Cx CA, circumflex coronary artery; G, gross; M, microscopic; N, normal; O, occlusion; T, thrombus; +, slight; ++, moderate; +++, extensive; NS, nonsustained; FMN, focal medial necrosis (area of involvement in circumference).

Coronary sinus ostium was normal in appearance in all dogs.

p=NS between mean values in each group.

*Sustained VT terminated by intravenous lidocaine (50 mg). Nonsustained or sustained VT terminated spontaneously in other dogs.

†Site of shock, in cm from ostium of coronary sinus.

‡Thrombus within the lumen of circumflex coronary artery.

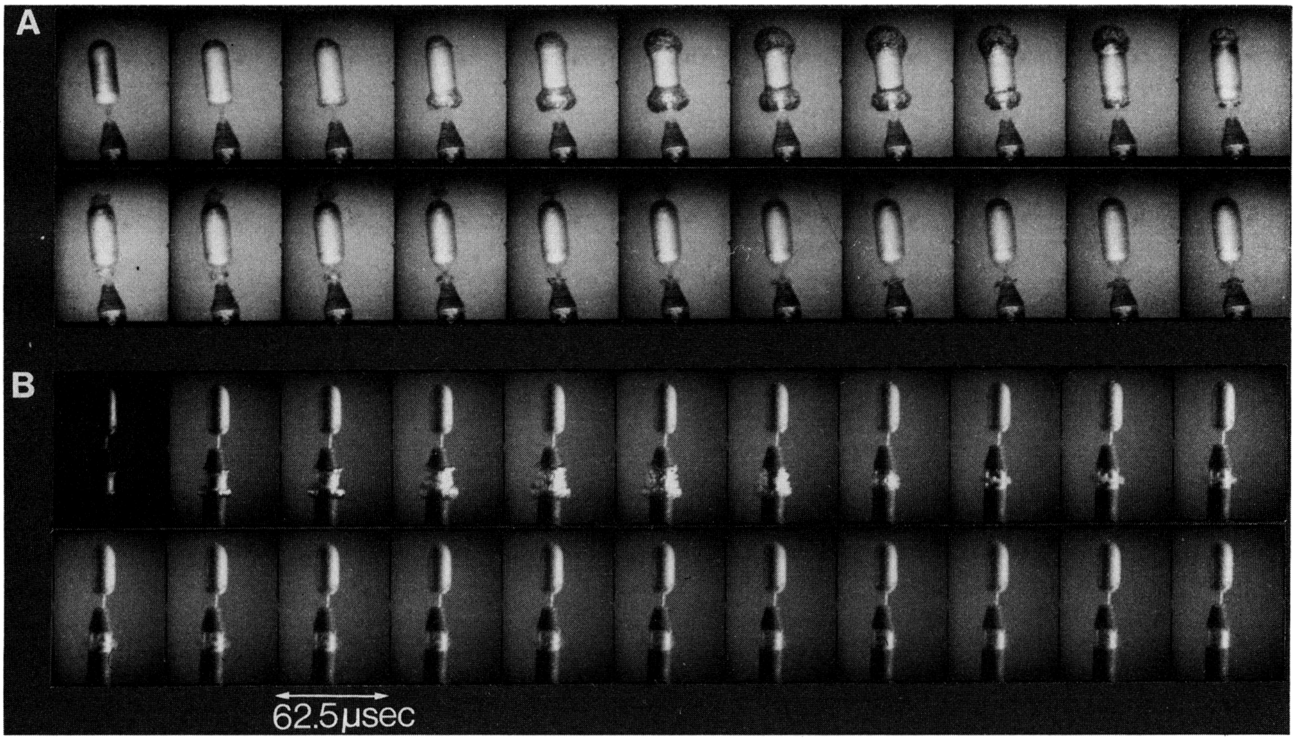


FIGURE 4. *High-speed cinematography of in vitro anodal nonarcing shocks.* A: *Unipolar distal shock of 15 J;* B: *Unipolar shock of 15 J to both electrodes made electrically common. Vapor globe does not fully isolate electrode. A and B correspond to voltage, current, and pressure curves shown in Figures 2A and 2B, respectively. Time between frames is 62.5 µsec. Sequence of events follows from left to right, through rows 1 and 2.*

Several reasons account for the recent interest in catheter ablation techniques within the coronary sinus.[6–9,16] First, most patients with the Wolff-Parkinson-White syndrome have a left-sided accessory pathway[17] and thus the technique could benefit many patients. Second, surgical[18] and pathological[19,20] studies have shown that, in contrast to right-sided accessory pathways that are located subendocardially, left-sided pathways are present in the subepicardial fat pad area, close to the coronary sinus. Insertion of the accessory pathway within the wall of the coronary sinus itself has also been suggested.[21] Finally, coronary sinus catheterization would make this technique simpler and more accessible than the endocardial approach (transeptal or retrograde aortic catheterization).[22,23]

Low-Energy Power Source and the Contoured Catheter

This new system of energy delivery provides greater arcing thresholds.[12–14] Clinical studies have shown that this technique is highly successful without complications.[24–27] In four patients with ventricular tachycardia, two to 24 (mean, eight) shocks of 3–40 J were delivered with a cumulative energy of 50–390 (mean, 175) J. None of the patients had recurrence of tachycardia, and there were no complications.[24] In a report of 14 patients with atrioventricular node ablation, the mean cumulative energy for successful ablation was 38 (range, 4–98) J.[26] In patients with supraventricular tachycardia, our success rate using the new system of low-energy ablation was 86%.[27] However, because nonarcing shocks may not always be effective,[23–27] we opted to study whether both low- (nonarcing) and maximal- (arcing) energy delivery with this new system could be achieved without causing cardiac rupture. Anodal shocks were given because during the in vitro study at low energies, they caused less vapor globe and pressure wave increases than cathodal shocks.

Characteristics of the Vapor Globe and Pressure Wave Changes

These are more pronounced for distal unipolar shocks than for unipolar shocks given to both electrodes made electrically common. Anodal shocks of 20 J or less do not cause arcing, and this was confirmed by high-speed cinematography during in vitro testing and by recording linear voltage–current curves during in vivo ablations. Significant increases in the diameter of the vapor globe occur with shocks of more than 20 J. The increases in pressure with greater energies follow the changes seen in vapor globe formation. Therefore, barotraumatic shocks were delivered in 18 dogs, and the in vitro results suggest that the shocks were more intense in the dogs that received distal unipolar shocks. This may explain why significantly more dogs with arcing distal unipolar shocks had gross rupture of the coronary sinus. However, all 36 dogs tolerated several shocks of 15 or

TABLE 3. Summary of Pathological Findings Induced by Nonarcing Shocks Compared With Arcing Shocks Within Coronary Sinus Using Low-Energy DC Ablation

	Nonarcing shocks (15 J each)		Arcing shocks (40 J each)
Shocks (*n*)	1–6	3–6	1–3
Dogs (*n*)	18	12	18
Coronary sinus			
Site of shock (mean distance in cm from ostium)	3.3±0.85*	3.2±0.9	2.6±0.9
External aspect (hemorrhage, hyperemia, or edema)			
Length	2.0±1.5*	2.1±1.6*	3.5±0.2
Height	0.96±0.4*	1.1±0.3*	1.5±0.5
Internal aspect			
Occlusion	3 (17)*	2 (17)	9 (50)
Rupture			
Gross	0*	0*	9 (50)
Microscopic	3 (17)*	2 (17)*	13 (72)
Epicardial fat pad hemorrhage (moderate to severe)	6 (33)	4 (33)	9 (50)
Ventricular tachycardia			
Nonsustained	8 (44)	7 (58)	10 (55)
Sustained	0*	0	4 (22)
Circumflex coronary artery			
Focal medial necrosis†	6 (33)	6 (50)	6 (33)
1/8	3	3	3
1/4	3	3	2
1/2	0	0	1
Left atrial necrosis			
Length (cm)	1.3±1.2	1.6±1.4	1.6±0.7
Height (cm)	1.4±1.7	1.1±0.6	1.2±0.5
Volume (cm^3)	0.4±0.5	0.4±0.5	0.5±0.5
Left ventricular necrosis			
Length (cm)	0.8±0.7*	1.0±0.7	1.4±0.8
Depth (cm)	0.6±0.9	0.5±0.4	0.4±0.3
Width (cm)	0.2±0.2*	0.3±0.3	0.8±0.3
Volume (cm^3)	0.1±0.1*	0.1±0.1	0.3±0.4

*$p<0.05$ between nonarcing and arcing shocks.
†Area of involvement (circumference).
Values in parentheses indicate percentages.

40 J with a mean cumulative voltage of approximately 6,000 V without perforation of the epicardial fat space (all dogs received 1,000 units heparin before ablation). The limited barotraumatic effects of this new system during arcing shocks could be related to the extremely short duration (<50 μsec) of the pressure increase after collapse of the vapor globe.

Atrial and Ventricular Injury

The mean lengths of necrosis of the atrial wall and summit of the left ventricle were 16 and 14 mm, respectively, for arcing shocks and 16 and 10 mm, respectively, for multiple nonarcing shocks. Accessory pathways are believed to course over a distance of 10–15 mm[28,29]; the extent of necrosis of the atrium, the ventricle, and the epicardial fat space suggests that conduction over an accessory pathway could be interrupted after such lesions. The degree of necrosis is greater than with radiofrequency energy. Jackman et al[9] used bipolar radiofrequency energy between a catheter in the coronary sinus and another in the left ventricle beneath the mitral valve high against the annulus. They found that the median diameter of the lesions was 4 mm and that the median atrial and ventricular necrosis extended 2.5 and 2.6 mm, respectively. Combining two other studies using radiofrequency energy,[8,9] atrial necrosis was seen in 58% of energy deliveries and necrosis at the summit of the left ventricle was seen in 34%. Thus, the limited extent of coagulation necrosis produced by radiofrequency energy may not be enough for ablation of accessory pathways. In a group of 14 patients with the

Wolff-Parkinson-White syndrome,[12] radiofrequency energy (up to 13 pulses of energy delivery per patient) caused interruption of accessory pathway conduction in only 43%. Using the coronary sinus to left ventricle radiofrequency technique, Kuck et al[10] produced permanent accessory pathway block in only two of eight patients (25%).

Coronary Sinus

The coronary sinus is the most constant feature of the cardiac veins and measures up to 16 mm in diameter over its 40-mm length.[30,31] Previous studies have shown that DC shocks either at the ostium[1,2,32] or within the coronary sinus[6] may cause cardiac rupture and tamponade. This has been associated with a Lown shape impulse (conventional defibrillators), with energy deliveries of 150 J or more. The vapor globe produced with such energies is greater than 40 mm, and the pressure is sustained and may exceed 10 atm.[3,14,33,34] The longer duration (6 msec) and the greater energy delivery appear to explain why DC discharges with the conventional defibrillator cause significantly more barotrauma than with the low-energy power source.[14,33–35] In our study, moderate to extensive hemorrhage within the epicardial fat space was seen in 33% of dogs that had nonarcing shocks and in 50% of those that received arcing shocks, but fat within the coronary sulcus acted as a natural barrier to future damage.

Occlusion of the coronary sinus after low-energy DC ablation occurred in 33% of our dogs. Brodman and Fisher[16] have reported an incidence of coronary sinus occlusion of 50% in dogs that received one to four shocks of 35–45 J. Langberg et al[8] have reported a 20% incidence of coronary sinus occlusion using radiofrequency energy delivery. The clinical consequences of coronary sinus thrombosis, if any, appear very limited. In one study, asymptomatic coronary sinus occlusion was seen in two patients who underwent surgical ablation of their accessory pathways 8 weeks after catheter ablation. Other reports of coronary sinus occlusion after cardiac catheterization also appear to have occurred without sequelae.[36–40]

Circumflex Coronary Artery

Thrombus within the lumen of the coronary artery was seen in one dog that received three arcing shocks of 40 J. A total of 12 dogs (33%) had focal medial necrosis of the circumflex coronary artery, occurring only in those that had multiple shocks. In six of 12 dogs, lesions were estimated to correspond to one eighth or less of the circumference of the artery. In five other dogs, necrosis of the media was one fourth or less of the circumference. In one dog that received three shocks of 40 J, one half of the circumference of the artery was involved by focal medial necrosis. Necrotizing arteritis was seen in two of 20 dogs that had radiofrequency energy in the coronary sinus in the study by Huang et al,[7] in one of 15 dogs studied by Jackman et al,[9] and in one of 16 dogs reported by Langberg et al.[8] Clinical studies have shown that catheter ablation may cause coronary artery spasm[41] or myocardial infarction.[22] The long-term consequences of the focal abnormalities of the circumflex artery reported with DC shocks[16] or radiofrequency[7–9] are unknown. Clinical studies have not reported, so far, any long-term sequelae,[22,23] but these coronary artery lesions need to be further evaluated by long-term studies. The equal frequency of focal medial necrosis seen with nonarcing and arcing shocks suggests that these lesions may be voltage mediated or related to current density.

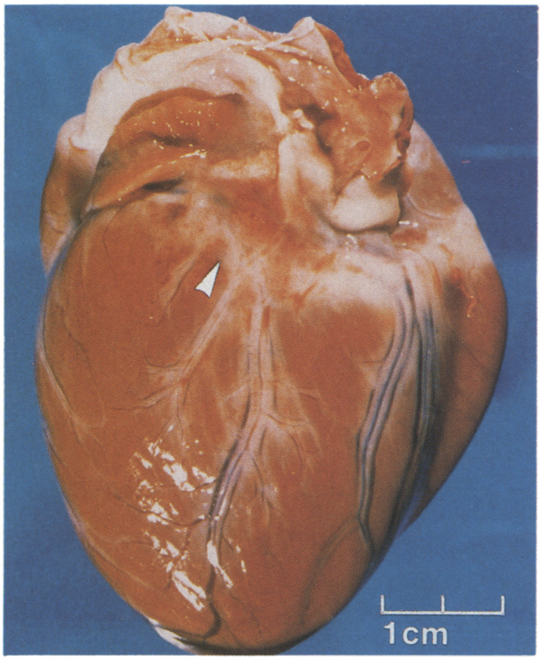
Unipolar Mode of Energy Delivery

Unipolar shocks to both electrodes made electrically common have been reported to possibly cause less barotrauma[4,37] and have been used clinically for ablation of posteroseptal accessory pathways.[1] However, cardiac tamponade has been reported to occur with such a mode of energy delivery.[1] Our in vitro results suggest less barotrauma, which is confirmed by the lower incidence of rupture of the coronary sinus during nonarcing or arcing shocks. However, catheter ablation using both electrodes made electrically common appears to have been associated with more untoward effects. Our in vivo results revealed myocardial necrosis extending to the apex of the left ventricle in two dogs, and involving the right ventricle in another. Current flow may have been channeled by blood flow, causing remote lesions. Thrombus within the circumflex coronary artery also occurred in one dog that received an arcing shock to both electrodes. Focal necrosis of the circumflex artery, occlusion of the coronary sinus, and hemorrhage within the epicardial fat pad occurred with equal frequency in dogs that received distal shocks and those that received shocks to both electrodes, although there was no microscopic evidence of coronary sinus rupture when shocks were given to both electrodes. Both types of shocks caused comparable amounts of coagulation necrosis of the atrium and ventricle. Therefore, our results do not support a significant advantage of unipolar shocks to both electrodes made electrically common compared with unipolar distal shocks. In fact, shocks to both electrodes could be associated with more complications.

Nonarcing and Arcing Shocks

The extent of coagulation necrosis of the atrium and ventricle was not significantly different between dogs that received multiple (three to six) nonarcing shocks of 15 J and those that received one to three arcing shocks of 40 J. The absence of vapor globe formation and pressure wave increases during nonarcing shocks (as shown during in vitro testing and by recording linear voltage–current curves during in vivo ablations) and the extent of coagulation necrosis after nonarcing shocks confirm that voltage or current density, rather than barotrauma, is responsible for useful tissue injury. In the 18 dogs that received one to three arcing shocks of 40 J, barotrauma appears responsible for the greater extent of hemorrhage of the epicardial fat pad, of

A



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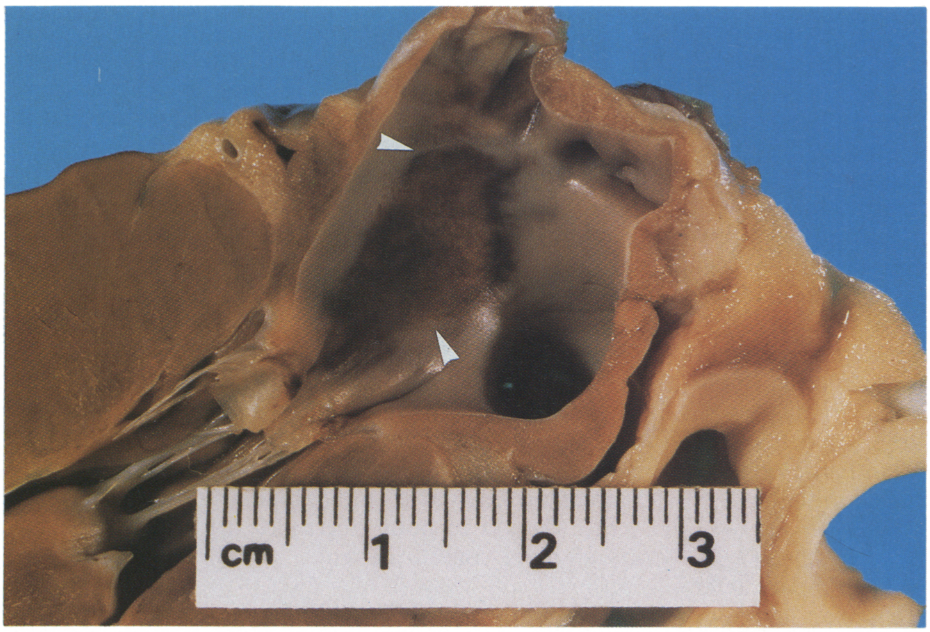

FIGURE 5. *Low-energy DC ablation of six nonarcing unipolar distal shocks of 15 J 3.5 cm within the coronary sinus in dog 9 (Table 1).* A: *Photograph of heart just after being excised (viewed posteriorly). There is hemorrhage and edema (arrowhead) in area of atrioventricular groove.* B: *Sagittal cut of heart showing (arrows) subendocardial hemorrhage of left atrium overlying area of ablation within coronary sinus.* C: *Six transverse sections of coronary sinus, summit of left ventricle, and left atrium (most proximal section, near coronary sinus, situated far left, and most distal lesion, far right); all sections show patency of coronary sinus and circumflex coronary artery without thrombus, disruption, or rupture. Arrows show coagulation necrosis of left atrium and left ventricle. There is mild hemorrhage in epicardial fat pad (EFP).* D: *Close-up view of section 5 (counting from left to right).* E: *Close-up of section 4, hematoxylin and eosin stain; arrowheads show coagulation necrosis and an inflammatory infiltrate. In C, D, and E, epicardium is on right and endocardium (EN) is on left and shows mitral valve (MV) leaflet insertion. Left atrium (LA) is above, and left ventricle (LV) is below. Coronary sinus (arrow) is superior and medial to coronary artery.*

rupture of the coronary sinus, of coronary sinus occlusion, and of sustained ventricular tachycardia. However, the absence of tamponade or cardiac rupture, as previously demonstrated with the conventional system of DC catheter ablation,[1,2,5,6] introduces the concept of limited arcing and low-level barotrauma. This concept could represent a new standard for DC catheter ablation; if continued success is demonstrated in all areas of the heart,[24–27] this new system should eliminate the use of high-energy barotraumatic shocks. However, coronary sinus disruption and coronary arterial lesions may occur even with nonarcing shocks.

Clinical Implications

The results of this study suggest that the low-energy power source coupled with the ablation catheter could cause sufficient necrosis for successful ablation of accessory pathways. The relative safety of this new system within the coronary sinus, especially with nonarcing shocks, provides support for its use in the area of the ostium for ablation of posteroseptal accessory pathways. Further studies using low-energy catheter ablation will show whether DC shocks within the coronary sinus, a technique that has been abandoned, can be rehabilitated.

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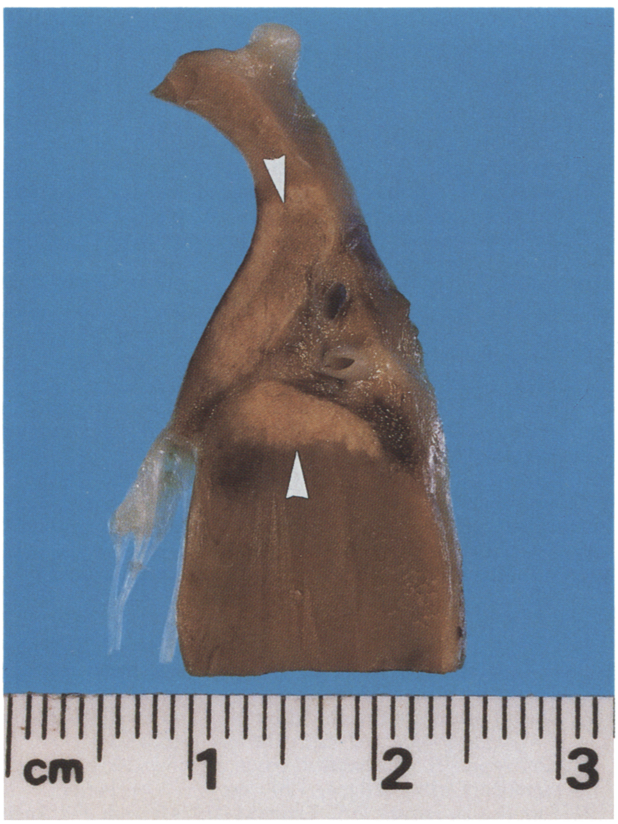

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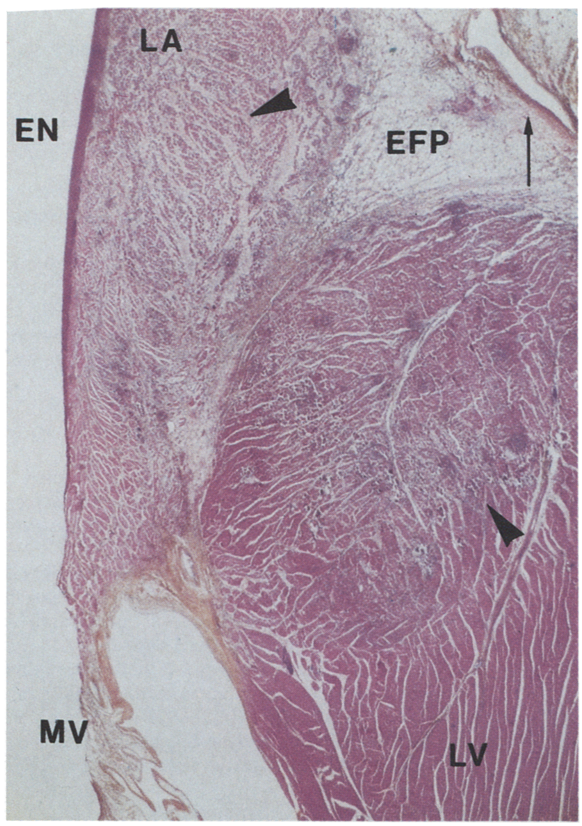

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KEY WORDS • catheter ablation • coronary sinus • arrhythmias • Wolff-Parkinson-White syndrome