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Effect of Direct-current Countershocks on Regional Myocardial Contractility and Perfusion

Experimental Studies

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SUMMARY Very high energy electrical countershocks can cause morphologic damage to the myocardium. In this study we searched for functional correlates of these shock-induced morphologic changes. We used ultrasonic sonomicrometers to measure myocardial contractility and radiolabeled microspheres to assess perfusion. Acute and chronic experiments were conducted in 45 dogs, assessing the effect of both direct (epicardial) and transthoracic shocks on beating and fibrillating hearts. High-energy or rapidly repeated epicardial shocks caused subepicardial contraction abnormalities. This indicates that electrical current delivered to the myocardium in sufficiently high amounts and concentration can cause functional damage. Thus, in open-chest defibrillation during cardiac surgery, low energies (10-20 J) should be used initially and higher energies resorted to only if lower-energy shocks fail. However, single and multiple transthoracic shocks up to 460 J delivered energy caused no detectable contraction abnormalities. Myocardial perfusion did not fall after shocks. Thus, high-energy transthoracic shocks may have no deleterious effects on the contraction and perfusion of normal myocardium.

ALTHOUGH ELECTRICAL DEFIBRILLATION of the heart has been an accepted clinical therapy for many years, some important features of this lifesaving maneuver remain undefined. Very high levels of electrical energy and current can cause gross and microscopic myocardial damage, and this may be an argument for lower-energy defibrillation. However, the electrical doses used to cause such damage are far in excess of the amounts necessary to defibrillate. Lower-energy shocks result in less histologic damage.* The effect of shocks on myocardial function has not been well characterized.

The purpose of these studies was to determine the effects of both low- and high-energy countershocks on two sensitive measurements of myocardial function—regional myocardial contractility and perfusion. We conducted experiments in both open- and closed-chest dogs, using ultrasonic sonomicrometers to register myocardial contractility and radiolabeled microspheres to measure perfusion.

Methods

Forty-five mongrel dogs that weighed 17–45 kg were studied. Dogs undergoing acute experiments were anesthetized with chloralose, 100 mg/kg, plus urethane, 1000 mg/kg i.v., with supplemental amounts as needed. Dogs that underwent sterile surgery for chronic experiments were anesthetized with
i.v. sodium pentobarbital, 25 mg/kg. The dogs were ventilated by a Harvard respirator, using room air and supplementary oxygen via a cuffed endotracheal tube. Tidal volume and respiratory rate were adjusted to maintain arterial Po₂, Pco₂, and pH within a physiologic range.

For acute countershock studies using sonomicrometers, a midsternal thoracotomy was performed, the pericardium incised and the heart exposed. In dogs used for closed-chest shock studies, sonomicrometers were inserted into the myocardium and the sternum and underlying soft tissues were closed in multiple layers. Sonomicrometer wires were brought out at the caudal end of the incision. Defibrillator paddles were then applied to the closed chest. In other dogs, the chest and pericardium were left open to assess the effects of direct (epicardial) shocks from paddles applied directly to the heart. In six dogs that underwent sterile surgery for long-term shock effects, a lateral thoracotomy was used to implant the sonomicrometers. In four dogs who were deliberately fibrillated before countershock, a bipolar pacing catheter was passed via the brachial vein to the right ventricle. Ventricular fibrillation was induced by stimulating the right ventricle with a train of rectangular impulses (60 Hz, 5 V, 5 msec) via the pacing catheter. Polyethylene or polyurethane catheters were placed by cannulation of the left brachial artery and retrograde catheterization of the left ventricle, and (in open-chest dogs) directly into the left atrium. Pressures were measured with Statham P23 strain gauges at midchest level. All recordings were made on a direct-writing polygraph.

Shock Technique

Shocks were administered using a Datascopc MD2J damped-sine wave form defibrillator in all but four dogs. Using the Datascopc defibrillator, when an energy level is selected by the operator the energy to be delivered into a 50-Ω resistance is displayed. After discharge of the defibrillator, the peak current (in amperes) that actually flowed between the paddles is displayed and recorded. A pair of paddles consisting of one paddle 8.5 cm in diameter and one paddle 13 cm in diameter was used for transthoracic shocks; the 8.5-cm paddle was placed over the palpable apical impulse on the left chest and the 13-cm paddle at the corresponding level on the right chest. Studies have shown that use of at least one large paddle lowers transthoracic resistance and increases transthoracic current flow⁹ and intracardiac current flow.¹⁰ Transthoracic paddles were coated with Hewlett-Packard Redux paste, a low-resistance interface between paddle and skin.⁹

In the open-chest studies, bare paddles 6 cm in diameter were applied directly to the epicardium, cradling the heart anteriorly and posteriorly (fig. 1). Shocks applied to beating hearts were synchronized so that they were delivered within 20 msec of the onset of the R wave. This preset delay was verified at the end of the study.

Sonomicrometers

We measured regional myocardial function with the sonomicrometer method.¹¹ A sonomicrometer transducer was made by soldering stainless steel wires 0.15 mm in diameter to a 5-MHz piezoelectric crystal 1.5 mm in diameter. This assembly was coated with epoxy for protection and for development of a hemispherical lens for signal broadening. Two transducers were then connected to a dimension gauge. One of the pair was excited by a 0.2-μsec, 200-V pulse, producing a sound wave that travels through tissue at a velocity of 1.5 × 10⁹ mm/sec. The other received the signal and the gauge then converted the transit time into the equivalent distance. The signal received was displayed on an oscilloscope; the data were taken from signals that were accurately tracked on the first pulse received. A pulse repetition rate of 1000 Hz allowed continuous measurement of distance throughout the cardiac cycle. The dimension gauge was calibrated by placing the pair of transducers in saline and measuring the real distance separating the two transducers by a vernier caliper. When implanted in the myocardium, the transducers were calibrated by inserting a 1.0-μsec pulse (equivalent to 1.5 mm) into the gauge.

One sonomicrometer pair was implanted in the subendocardial layer of the left ventricular wall near the left anterior descending coronary artery. In dogs subjected to open-chest shocks, this pair was directly covered by the anterior paddle and so was directly within the current path (fig. 1). Another pair of sonomicrometers was placed in the subendocardium of the lateral wall of the left ventricle; this pair was not covered by a paddle and was, therefore, remote from the current path (fig. 1). In dogs that received subepicardial sonomicrometer pairs, all pairs were inserted so that they were covered directly by the paddles and were, therefore, in the current path. In the closed-chest countershock studies we assumed that all the sonomicrometer crystals were within the current path, especially because one of the paddles was large (13 cm).

The signals were stable and reproducible over the course of the 5-6-hour experiment. Occasional drift was minimized by repeated calibration.

The analog signals were recorded simultaneously and continuously with the ECG and aortic pressure. The largest end-diastolic segment length during the respiratory cycle was recorded at the peak of the R wave of the ECG (fig. 1). End-systolic segment length of the same beat was measured 20 msec before the dicrotic notch of the aortic pressure tracing to correct for the aortic pressure delay. Actual segment lengths ranged from 6-16 mm. To facilitate data evaluation, all segment lengths were normalized: The segment length was divided by the preintervention end-diastolic length, multiplied by 10 and expressed, by convention, in millimeters.¹¹ Thus, all preintervention end-
diastolic lengths are expressed as 10 mm. Normalized segmental change with systole (ΔL) was defined as the normalized end-diastolic length minus the normalized end-systolic length. Therefore, ΔL was expressed as a positive number.

Microsphere Blood Flow Measurements

Regional left ventricular blood flow was measured using microspheres 15 μ in diameter labeled with 141Ce, 85Sr, 45Sc and 95Nb. The spheres were suspended in dextran and vigorously agitated mechanically for 2 minutes; they were examined microscopically to be certain that they were completely dispersed. Blood was withdrawn from the right brachial and femoral arteries at 2.06 ml/min with a Harvard pump, starting 1 minute before and continuing 3 minutes after injection. For each measurement approximately 2 × 10⁶ microspheres were injected over a 10-second period into the left atrium or left ventricle. The cannula was then flushed with 5 ml of saline. This produced an average of approximately 400 spheres per sample. No significant change in hemodynamic variables occurred as a result of microsphere injection.

At the end of the experiment the heart was excised and the free wall of the right ventricle, the atria, great vessels, valves and epicardial fat were removed. The ventricle was cut into 96 small segments as previously reported in detail. The average size of each segment was 1.6 × 1.6 × 0.3 cm. Subsequent processing of gamma counts and blood flow calculations were done as previously reported.

Data

Data were analyzed for statistical significance using an analysis of variance to determine if there was a significant difference among the multiple measurements of each variable tested, followed by Duncan's multiple range test to determine which specific variables were significantly different. Data are mean ± SD.

Protocols

The experimental protocols are summarized in table I.

Effect of Direct (Epicardial) Shocks
(Paddles Applied Directly to the Heart)
on Myocardial Contractility and Perfusion

Fourteen open-chest dogs in two groups were studied. Group A consisted of eight dogs with sonomicrometers placed in the subendocardium. Group B consisted of six dogs with sonomicrometers placed in the subepicardium. After hemodynamic and sonomicrometer recordings and (in the group A dogs) microsphere injections in the control state, a series of synchronized shocks was administered to the heart from paddles applied directly to the ventricles (fig. 1). The energy sequence was 20, 40, 75 and 100 J. Continuous hemodynamic and ultrasonic data were recorded. Microsphere injections (in the group A dogs) were done approximately 20 minutes after each shock to allow for stabilization. After the final shock of 100 J and post-shock recordings, the dogs were killed with an i.v. injection of potassium chloride and the heart was removed for perfusion determinations.

Effect of Transthoracic (External) Shocks
on Myocardial Contractility and Perfusion

Acute studies. Twenty-five dogs in five groups (C–G) were studied.

Group C. To evaluate the effects on contraction (measured by subendocardial sonomicrometers) of gradually increasing energy levels, five dogs in regular sinus rhythm received synchronized transthoracic shocks of 100, 200, 300, 400 and 460 J (the maximum energy of the defibrillator). As before, we obtained continuous hemodynamic and ultrasound recordings and injected microspheres after each shock; approximately 20 minutes elapsed between shocks.

Group D. Six dogs in regular sinus rhythm had sonomicrometers placed subepicardially and un-
derwent the same sequence of shocks. Microsphere injections were not done in this group.

**Group E.** To evaluate the effects on contractility of multiple high-energy transthoracic shocks administered rapidly, two dogs with subendocardial sonomicrometers and two dogs with subepicardial sonomicrometers received a series of 10 460-J shocks delivered within a 5-minute period; hemodynamic and ultrasound signals were recorded continuously for 2 hours.

**Group F.** In four dogs we evaluated the effects of progressively higher shock levels delivered to a fibrillating heart. Ventricular fibrillation was initiated with a train of electrical impulses via the right ventricular catheter. After fibrillation was achieved, the electrical current was discontinued. After 10–15 seconds of ventricular fibrillation, defibrillating shocks were given, initially using 100 J. Hemodynamic and ultrasound data were monitored continuously. This procedure was repeated at 15-minute intervals; the second episode of fibrillation was terminated using a shock of 200 J, the third at 300 J and the fourth at 400 J. In this group of dogs only, because of temporary unavailability of theDatascopedefibrillator, we used a Mennen-Greatbatch Cardiosentinal damped-sine wave form defibrillator, with a maximum delivered energy output of 400 J. Two paddles 8 cm in diameter were used with this defibrillator.

**Group G.** In six dogs we evaluated the effects, over a 6-hour period, of multiple high-energy shocks on perfusion. We did no initial thoracotomy in this group and no sonomicrometers were implanted; only arterial and left ventricular catheters were inserted. After control hemodynamic recordings and microsphere injections, nine 400-J shocks were given 1 minute apart, followed by a tenth shock of 460 J. Hemodynamic recordings were made continuously, and left ventricular microsphere injections were repeated at 2, 4 and 6 hours after the shocks.

**Effects of Transthoracic Shocks on Myocardial Contractility: Chronic Studies**

**Group H.** To ascertain whether transthoracic shocks have an effect on myocardial contractility that might not be evident within the first few hours, we studied a group of six dogs that underwent implantation of subendocardial sonomicrometers in the lateral wall of the left ventricle using sterile surgical techniques, via a lateral thoracotomy performed under barbiturate anesthesia. Several days after the initial surgery, after the dogs had fully recovered, they were returned to the laboratory and lay quietly while control ultrasonic recordings were obtained. They were then reanesthetized with pentobarbital and a series of shocks were delivered at 20, 40, 75, 100, 200, 300, 400 and 460 J. The dogs were allowed to recover. They were returned to the laboratory for repeat ultrasonic recordings 1 or 2 days later and again at 4–6 days after shocks.

**Results**

**Effect of Direct Epicardial Shocks on Myocardial Contractility and Perfusion (Groups A and B)**

Eleven dogs completed the entire sequence of shocks, from 20–100 J. Five dogs developed un-
intended ventricular fibrillation after shocks of 20 J (four dogs) and 40 J (one dog); data from the two dogs that were resuscitated and completed the sequence are included. In both of these dogs the period of ventricular fibrillation was less than 30 seconds; arterial blood gases determined after resuscitation remained in a physiologic range.

**Group A (Subendocardial Sonomicrometers)**

The direct shocks of up to 100 J resulted in peak current flows of up to 43 A. The cumulative current flow these dogs received was 80 ± 47 A (range 17–129 A).

There were no significant changes in aortic mean pressure and heart rate.

Perfusion of areas both within and remote from the current path rose slightly, but not significantly (fig. 2). This tendency was somewhat more pronounced in myocardial segments within the current path. Analysis of endocardial and epicardial flows showed a slightly greater tendency of the epicardial flows to rise, with a resultant fall in the endo-epi perfusion ratio. Again, these changes did not achieve statistical significance.

**Sonomicrometers.** There were virtually no detectable changes in subendocardial sonomicrometer-registered contractility immediately or later after the shocks. Normalized end-diastolic length changed from 10 mm at control to 10.1 ± 0.3 mm 20 minutes after 100-J shocks applied directly to the heart. This change was not significant, nor did any of the intermediate shocks cause any significant changes. Normalized end-systolic length changed from 8.6 ± 0.6 mm to 8.9 ± 0.5 mm 20 minutes after 100 J (NS). The ΔL showed no significant changes either in the crystals within the current path: 1.6 ± 0.6 mm control to 1.5 ± 0.4 mm 20 minutes after 100 J (fig. 3), or remote from the path: 1.2 ± 0.5 mm to 1.0 ± 0.6 mm 20 minutes after 100 J.

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**Figure 2.** Effect of direct (epicardial) shocks on myocardial perfusion. Perfusion tended to rise after direct shocks, especially in the area within the current path and in the epicardium. These changes did not achieve statistical significance.

**Figure 3.** Effect of shocks on myocardial contractility (subendocardial sonomicrometers). Systolic shortening (ΔL) did not change significantly after direct (epicardial) and transthoracic shocks.
**Group B (Subepicardial Sonomicrometers)**

The direct shocks of up to 100 J resulted in peak current flows of up to 34 A. Cumulative current flow was 85 ± 24 A (range 45–105 A).

Heart rate and left ventricular end-diastolic pressures did not change. Aortic mean pressure fell after the 100-J shocks, from 81 ± 24 mm Hg to 68 ± 12 mm Hg (100 J) (p < 0.05). Changes after intermediate-strength shocks were not significant.

**Sonomicrometers.** Normalized end-diastolic length showed no significant changes: 10 mm control to 10.8 ± 1.9 mm 20 minutes after 100-J shocks (NS). However, normalized end-systolic length was significantly increased 20 minutes after the 75-J shock, from 9.4 ± 0.3 mm to 10.6 ± 1.7 mm (p < 0.05 vs control), and showed a further increase to 10.7 ± 2.0 mm (p < 0.05 vs control) 20 minutes after 100 J. The ΔL was 0.6 ± 0.3 mm at control, and decreased to 0.4 ± 0.6 mm (NS) 20 minutes after 75 J and to 0.1 ± 0.6 mm (p < 0.05) 20 minutes after 100 J (fig. 4). In four of the six dogs in this group, one or more subepicardial crystal pairs showed systolic expansion, rather than normal contraction, after direct shocks (figs. 5 and 6). This occurred in one dog after three 40-J shocks were rapidly applied to convert unintended ventricular fibrillation (fig. 5). In the other three dogs, systolic expansion occurred only after shocks of 75 or 100 J were applied to beating hearts; expansion did not occur after lower shock energies (fig. 6). In each of these four dogs, once systolic expansion appeared, it persisted throughout the remainder of the period of recording from the sonomicrometers.

**Effect of Transthoracic Shocks on Myocardial Contractility and Perfusion**

**Acute Studies (Groups C–G)**

**Group C (subendocardial sonomicrometers).** In this group, which received shocks of gradually increasing energy levels, three of the five dogs completed the full shock series (up to 460 J); one dog developed ventricular fibrillation at a shock of 200 J and one dog at 400 J. Neither could be resuscitated; data are included from their lower-energy shocks before ventricular fibrillation occurred. The energy dosage ranged up to a maximum of 20 J/kg for the highest-energy single shock. Peak single-shock current flows ranged up to 67 A (2.6 A/kg); cumulative current flows received were a mean of 281 ± 138 A (range 107–437 A) or 8.8 ± 4.4 A/kg (range 5.8–15.1 A/kg).

There were no significant changes in heart rate or aortic pressures; left ventricular end-diastolic pressure was measured in two dogs and did not vary.

**Sonomicrometers.** There were no significant changes in normalized end-diastolic and end-systolic length and ΔL as a result of the shocks (figs. 3 and 7). Perfusion determinations were obtained in all dogs only for shocks up to 200 J, because only four isotopes

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**Figure 4.** Effect of shocks on myocardial contractility (subepicardial sonomicrometers). Systolic shortening (ΔL) declined significantly after direct (epicardial) shocks of 100 J. The ΔL did not change in dogs that received transthoracic shocks.

**Figure 5.** Subepicardial contraction abnormality induced by direct (epicardial) shocks. Three 40-J shocks were administered rapidly to the epicardium for ventricular fibrillation induced by a 20-J shock. Systolic expansion is present after shocks. Ao = aortic; LV = left ventricular.
were available and because two of the dogs fibrillated before the series of shocks was completed. Ventricular perfusion was $142 \pm 75$ ml/100 g/min at control, $147 \pm 87$ ml/100 g/min after 100 J and $152 \pm 74$ ml/100 g/min after 200 J (NS).

**Group D (subepicardial sonomicrometers).** The energy doses ranged up to 24 J/kg. Peak single-shock current flows ranged up to 74 A (3.4 A/kg); cumulative current flows were $241 \pm 24$ A (range 213–265 A) or $12.1 \pm 1.3$ A/kg (range 10.8–13.9 A/kg).

Heart rate, aortic mean pressure and left ventricular end-diastolic pressure did not change significantly.

**Sonomicrometers.** There were no significant changes in normalized end-diastolic length, end-systolic length or $\Delta L$ (fig. 4). None of the subepicardial sonomicrometer pairs showed systolic expansion in this group of dogs receiving transthoracic shocks.

**Group E (subendocardial or subepicardial sonomicrometers).** In the four dogs that received a series of 10 rapidly delivered 460-J shocks, six pairs of subendocardial and three pairs of subepicardial sonomicrometers were placed. The $\Delta L$ was unchanged 2 hours after the shocks were given.

**Group F (subendocardial sonomicrometers).** In the four dogs that underwent fibrillation-defibrillation sequences using progressively increasing energy levels (up to 400 J) there were transient alterations in left ventricular and aortic pressure and $\Delta L$ (fig. 8). These changes lasted only about 30 seconds after each defibrillation; 10 minutes after each shock, no significant changes appeared in heart rate and aortic pressure and the sonomicrometer measurements.

**Group G (myocardial perfusion).** In the six dogs that underwent perfusion determinations at 2, 4 and 6 hours after 10 shocks of 400 and 460 J, without thoracotomy, myocardial perfusion, endo-epi perfusion ratio, heart rate and blood pressure did not change significantly.

**Group H (subendocardial sonomicrometers, chronic studies).** The effects of shocks on myocardial contractility up to 6 days after the shocks were studied in six dogs. The $\Delta L$ was $1.6 \pm 0.3$ mm during the control period and $1.8 \pm 0.5$ mm (NS) at the final recording (4–6 days after shocks). Heart rate and aortic pressure did not change significantly.
Discussion

The main finding of this study was that electrical countershocks caused contraction abnormalities in subepicardial zones when the shocks were given directly to the epicardium. However, even high-energy transthoracic shocks caused no demonstrable contraction abnormalities. Neither epicardial nor transthoracic shocks caused demonstrable reductions in myocardial perfusion.

Alterations in ventricular performance after cardioversion have been reported clinically and have been ascribed to depression of myocardial contractility by the shocks. Experimental studies have evaluated the effects of countershocks on the myocardium, using histology, electrocardiographic precordial mapping, enzymatic analysis and scintigraphic methods. These studies have demonstrated that myocardial damage can be produced by high-energy countershocks. However, the functional effect of such damage is unclear. This study is the first to report shock-induced abnormalities of regional myocardial contraction.

The morphologic abnormalities after shocks reported by others have been patchy and localized to the epicardium at lower energies, becoming confluent and transmural only with very high-energy shocks. Experimental studies of open-chest defibrillation with electrodes applied directly to the heart, have emphasized this subepicardial distribution of damage. Our finding of subepicardial contraction abnormalities in open-chest dogs that received direct epicardial shocks is consistent with these histologic reports, as was the occurrence of such abnormalities only after very high energy shocks or rapidly repeated multiple shocks. We did not, however, see subendocardial or subepicardial contraction abnormalities in dogs that received transthoracic shocks. Presumably, during transthoracic shocks the more diffuse intracardiac current caused less necrosis and/or a more patchy distribution of damage. It is very unlikely that the lack of contraction abnormalities after transthoracic shocks was the result of sonomicrometers being out of the current path; the paddles were large and positioned on the chest overlying the heart, and multiple sonomicrometers in various locations were used. In a separate study using similar paddle size and placement and intracardiac electrodes, we showed substantial intracardiac current flow from transthoracic shocks. Of course, in the dogs that received direct epicardial shocks all the subepicardial sonomicrometer crystals were covered by the paddles and were known to be directly in the current path.

We saw major subepicardial contraction abnormalities in one dog after epicardial shocks of 40 J and in several after 75 J. Such energy doses are occasionally used clinically in the operating room. Patients who receive epicardial shocks of 40 J or higher may sustain myocardial damage from the shocks. Ten to 20 J are generally effective in surgical defibrillation. Such low energy should be used initially for open-chest defibrillation, and higher energy resorted to only when lower-energy shocks fail.

Other investigators have suggested that shock-induced cardiac damage is best correlated with electrical current flow (amperes). Geddes et al. showed in animals that a current dose of 1 A/kg body weight is the threshold level for defibrillation. Tacker et al. showed that animals that received transthoracic shocks of 1 A/kg had no demonstrable histologic changes, whereas dogs that received 3-4.8 A/kg showed damage. Our dogs received single-shock peak transthoracic currents up to 3.4 A/kg, and their cumulative transthoracic current flows were far higher (cumulative currents received ranged from 5.8-15.1 A/kg). Thus, the electrical current should have been sufficient to produce damage from transthoracic shocks, according to previous studies. Despite these high peak and cumulative current levels, we saw no changes in sonomicrometer-registered contractility in dogs that received transthoracic shocks. Still higher current or energy levels might cause damage from transthoracic shocks, but defibrillators that deliver...
more energy or current than used in this study are not generally available for clinical use.

Cobb et al. studied DC shocks in intact, nonfibrillating dogs and in isolated hearts. They demonstrated sinus arrest and increases in aortic pressure and epicardial strain gauge-measured contractile force in intact hearts, and suggested that the shocks excited intracardiac adrenergic and cholinergic nerves. The inotropic effects were positive and transient; shocks of up to 200 J produced changes that lasted less than 1 minute. Pansegrau and Abboud noted that countershocks administered without a preceding period of ventricular fibrillation caused minimal and inconsistent hemodynamic changes compared with those after defibrillation. Our studies confirmed this latter observation, as can be seen by comparing figures 7 and 8: a high-energy transthoracic countershock applied to a dog in sinus rhythm caused virtually no change (fig. 7), whereas a defibrillating shock (fig. 8) was followed by transient sinus bradycardia and small declines in left ventricular and aortic pressures. These responses were very brief, lasting no more than 30 seconds. There were virtually no changes in sonomicrometer-registered length and contractility.

Doherty et al. suggested that delivered energy above 21 J applied directly to the heart caused reduction in regional myocardial blood flow, assessed 6 hours later using indium-113 macroaggregate. Cohn et al. used localized high-intensity current to produce myocardial lesions resembling those of ischemia. These observations raise the question of whether myocardial damage after shocks might be the result of shock-induced vascular damage and reduced myocardial perfusion. Although Dicka et al. showed no change in regional myocardial blood flow after countershock in dogs, they assessed flow 24 hours after the shocks. This does not exclude the possibility that an earlier flow reduction might have occurred with a temporary period of ischemia. Therefore, we used radiolabeled microspheres to assess myocardial perfusion 20 minutes to 2 hours after high-energy direct epicardial shocks and up to 6 hours after transthoracic shocks. Perfusion did not fall; in fact, it tended to rise after the shocks, especially in the areas of the heart that (in the open-chest studies) could be identified as directly in the current path. We have not, however, excluded the possibility that a transient myocardial blood flow reduction might have occurred immediately after the shock but before the first 20-minute microsphere injection.

In some dogs we studied the effect of synchronized countershocks on beating nonfibrillating hearts. This was done to focus on the effects of the shocks themselves, as distinct from the deleterious effects of repeated episodes of ventricular fibrillation. These experiments are analogous to the clinical situation of elective cardioversion, where shocks are given to nonfibrillating and often nonischemic hearts. In other dogs, we deliberately fibrillated the heart first and then applied defibrillating shocks; the brief period (10–15 seconds) of ventricular fibrillation we induced is clinically analogous to witnessed ventricular fibrillation in coronary care units. Depression of myocardial contractility after defibrillation after longer periods (several minutes) of ventricular fibrillation may occur, but is probably related to metabolic abnormalities and myocardial ischemia accompanying prolonged ventricular fibrillation rather than to the defibrillatory shocks.

During our experiments several of the open-chest shocks and transthoracic shocks induced ventricular fibrillation despite the use of a synchronized discharge 20 msec after the onset of the R wave. This has been reported in humans. Lepeschkin et al. collected 85 cases of shock-induced ventricular fibrillation that occurred during properly synchronized cardioversion. These deleterious electrophysiologic effects from countershocks may be related to activation of autonomic reflexes. The methods used in this study are sensitive and precise. The theoretical resolution of the sonomicrometers is a small fraction of the wave length of the sonomicrometer signal (<0.05 mm), and the gauge is capable of detecting very small changes in ventricular function. The left ventricle is divided into 96 myocardial segments, and each segment is only 3 mm, so even small declines in myocardial perfusion would have been detected had they occurred.

In summary, single and multiple transthoracic shocks up to 460 J applied to 17–45-kg dogs, caused no detectable abnormalities of myocardial contraction or perfusion. However, high-energy or rapidly repeated direct (epicardial) shocks did cause subepicardial contraction abnormalities. This indicates that electrical current delivered to the myocardium in sufficiently high amounts and concentrations can cause functional damage. Direct (epicardial) shocks during cardiac surgery should be initiated at 10–20 J to avoid cardiac damage, resorting to higher energies only when lower-energy shocks fail. However, the lack of demonstrable contraction or perfusion abnormalities from high-energy transthoracic shocks suggests that transthoracic shocks may have no deleterious effects on the contraction and perfusion of normal myocardium.

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