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PATHOLOGICAL CHANGES IN SKIN AND SKELETAL MUSCLE FOLLOWING ALTERNATING CURRENT AND CAPACITOR DISCHARGE

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Recent advances in instrumentation have led to an increased utilization of electric currents for treating rhythm disorders of the heart. Electric shock constitutes the best method for terminating ventricular fibrillation. Electric pacemakers provide an effective method for sustaining the heart beat in patients with advanced degrees of atrioventricular block or with cardiac standstill.[1] The recent introduction of cardioversion, i.e., programmed direct current transthoracic depolarization of the heart, has further widened the therapeutic application of electricity.[2,3] Using this new method the majority of sustained ectopic tachycardias of atrial, nodal and ventricular origin can be terminated immediately with minimal if any complications. Initial studies on the effects of alternating current and capacitor discharge on the heart were carried out in 1898.[4] Alternating current (AC) defibrillation with 60 cycle current has been extensively studied since its introduction in 1933 by Hooker, Kouwenhoven and Langworthy.[6] The use of direct current (DC) has been widely accepted only within the past few years.[5] To date there has been no critical or systematic investigation of pathologic alterations resulting from various forms of electrical current on the heart or on tissues subjacent to the discharging electrodes. Pathologic studies have been con-

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cerned primarily with death due to lightning or with lesions resulting from accidental contact with power lines.

The present investigation had the following objectives: first, to determine whether electrical currents employed clinically for resuscitation or cardioversion injure tissues between the stimulating electrodes; second, if lesions do occur, to compare the pathologic effects of AC and capacitor discharge (DC) shocks; third, to determine the relation of the lesions to the energy of the electrical discharge, to the number of shocks administered, and to other variables. The 4 limbs of dogs were employed in these studies. This permitted each animal to serve as its own control and as the recipient of the 2 types of electrical discharge. The use of the limbs also permitted characterization of both acute and chronic pathologic changes at the same site.

Material and Methods

These studies were carried out in 43 mongrel dogs of both sexes weighing from 15 to 20 kg. Animals were anesthetized with sodium pentobarbital administered intravenously in a dose of 30 mg per kg. Animals were fixed supine on a nonconducting table and ventilated with room air by means of a Harvard respirator through an endotracheal cannula. The skin over the large muscles of the fore and hind limbs was exposed by close shaving of both inner and outer surfaces. The thorax was shaved to facilitate defibrillation. Electrode paste (Redux, Sanborn Co., Boston, Mass.) was applied to the exposed skin areas of limbs to insure adequate electrode contact and the electrode areas were permanently marked.

The electrode paddles, which measured 8.9 cm and 8.5 cm in diameter for DC and AC units respectively, were applied with pressure on either side of the limb. The paddles remained in position while repeated shocks were delivered at regular intervals. Frequent and at times serious cardiac arrhythmias followed electrical discharge across the limbs. Cardiac rhythm was therefore monitored throughout the experiment on an oscilloscope screen. When ventricular fibrillation was precipitated by the limb shock, the heart was defibrillated transthoracically by means of a DC discharge. Limbs subjected to the control procedure were treated identically by shaving, application of electrode paste and paddles but without administration of electric discharge.

The AC defibrillator employed was a commercial unit (D-72, Electrodyne Manufacturing Company, Norwood, Mass.). This instrument provided 60 cycle alternating current for a duration of 170 milliseconds (0.17 seconds). A setting of 350 volts was used in a majority of the experiments with AC.[5] The DC defibrillator employed was an instrument developed in our laboratory utilizing a 16 microfarad capacitor. An 100 milli-henry inductor in the discharge circuit prolongs the impulse and gives a damped sinusoidal wave form (DC defibrillator, American Optical Company, Chelsea, Mass.). The setting used in these experiments was 70 watt seconds (WS), or a multiple thereof. The 70 WS setting is comparable to 360 volts delivered by the AC unit, since both reverted 65 per cent of episodes of ventricular fibrillation in dogs when the discharge was administered transthoracically.[5]

At selected intervals to be described, biopsy specimens consisting of skin, subcutaneous tissue and muscle were obtained from areas which had been subjected to shock. Sterile technique was employed. A 2 by 1 cm ellipse of skin and subcutaneous tissue and a 1 cc segment of muscle were excised, and immediately placed in 10 per cent buffered formalin. Specimens were assigned random numbers, thus the pathologist

was without information as to the treatment given the biopsy site. The specimens were processed according to conventional histologic techniques. Sections from each specimen were stained with hematoxylin and eosin, Mallory trichrome, Weigert's reticulum and periodic acid-Schiff (PAS) stains. The sections were then examined independently by two pathologists.

The 43 dogs used in these experiments were divided into 5 groups. In the first 3 groups, the pathologic effects of AC and DC discharges were compared at equivalent defibrillating levels. In the fourth group the electrical energy dissipated in the limb by each of these 2 methods was determined. In the fifth group a comparison was made of the pathologic changes resulting from the use of AC and DC at similar energy levels.

In group 1, consisting of 12 dogs, the early pathologic effects of a small series of AC and DC shocks were determined. One fore- and one hind limb in each animal served as controls. The remaining two limbs were treated with either AC or DC discharge. One shock was administered to the forelimb while 5 shocks were delivered to the hind limb. Biopsy tissues were taken from each limb 1 and 7 days after the procedure (Table I).

TABLE I

DISTRIBUTION OF BIOPSY SPECIMENS OBTAINED IN 32 DOGS FROM LIMBS SUBJECTED TO EITHER AC OR DC SHOCKS

		Number of biopsies				
			AC		DC	
Group	Dogs	Controls	Fore	Hind	Fore	Hind
1 *	12	48	12	12	12	12
2 †	12	12	6	6	12	12
3 ‡	8	0	16	24	16	23 §
Totals	32	60	34	42	40	47

* Each forelimb received 1 shock while each hind limb received 5 shocks. Half of the biopsies were obtained 1 day after shock, the remainder after 7 days.

† Ten shocks were given to forelimbs and 40 shocks to hind limbs.

‡ Ten shocks given to each limb. Includes biopsies obtained 7 days and 30 days after shock.

§ One site was infected and therefore not subjected to biopsy.

In group 2, also consisting of 12 dogs, the pathologic alterations resulting from a large number of AC and DC shocks were studied. In each animal one limb served as a control. One limb was randomly assigned for AC treatment while the remaining two received DC shocks. Ten shocks were delivered to the forelimbs and 40 to the hind limbs. Biopsy specimens were obtained 7 days after the experiment. DC specimens were taken from areas subjacent to positive and negative electrode sites. (Table I)

In group 3, consisting of 8 dogs, the early and late pathologic changes following 10 electrical discharges were studied. In 4 of these animals AC discharge was delivered to right fore and hind limbs while DC was applied to the left. This order was reversed in the remaining 4 animals. Electrocardiograms were recorded after each shock. Biopsy tissues were obtained at 7 and 30 days. The 30-day hind limb specimens were taken from both sides of the limbs (Table I).

In these 3 groups, a total of 60 control biopsy specimens obtained randomly from right and left fore and hind limbs were compared with 163 specimens obtained from sites receiving either AC or DC treatment. Of the 223 specimens, 48 were obtained within 1 day after the procedure, 128 after 7 days, and 47 after 30 days. The forelimbs received 352 discharges of which 146 were AC and the remaining 206 were DC;

the hind limbs received a total of 940 shocks of which 350 were AC and 590 were DC.

In group 4, consisting of 3 dogs, the energy dissipated by both AC and DC shocks was compared in the same animal by photographing the discharge wave forms on the screen of a calibrated dual beam oscilloscope (Type 565 with type 2A63 preamplifiers, Tektronix Inc., Portland, Ore.) These animals were treated similarly to those in group 2, namely, the forelimbs received 10 and the hind limbs 40 shocks. DC shock was administered to the limbs on the right side, while AC was administered to those on the left side.

For the purpose of measurement the circuits of both defibrillators were modified by the insertion of a one ohm precision resistor in series with one electrode cable. This resistor was connected across one vertical input of the oscilloscope. For each ampere passing through the limb between the electrodes, one volt was applied to this input of the oscilloscope. Since the voltage of the defibrillator discharge could not safely be applied to the oscilloscope input, a voltage dividing network was connected across the defibrillator output. The signal from this circuit (1/100 of the actual discharge voltage) was applied to the second pair of input terminals. The screen thus displayed voltage and current waveforms simultaneously as functions of time. These waveforms, together with a calibration graticule, were photographed by a Polaroid camera mounted on the instrument. From measurements of the curves thus obtained, the resistance of the limb and the energy dissipated in it could be determined. The DC resistance was measured at the first peak of the discharge waveform (Text-fig. IA). The AC resistance was taken as the mean of the initial and final resistances (Text-fig. IB). Values were obtained in this manner from the first, fifth and tenth shocks applied to forelimbs and the first, fifth, tenth, 20th, 30th and 40th shocks applied to the hind limbs.

In group 5, consisting of 8 dogs, the effects of AC and DC discharge at similar energy levels were compared. Four dogs received DC discharges at the following energy settings: left forelimb 70 WS; right forelimb 140 WS; right hind limb 140 WS; and left hind limb 280 WS. Each limb received 10 discharges at 1 minute intervals. Oscilloscope photographs were made of the first, second and tenth discharges. The energy measurements thus derived were used to estimate the total energies received by each limb.

The remaining 4 dogs of group 5 received 10 AC shocks to each limb at 1 minute intervals. Frequent energy determinations were made during this procedure. These measurements permitted adjustment of the defibrillator voltage so that the total energy given closely matched that delivered during the DC phase of this experiment. Because the AC unit had only widely separated settings (e.g., 150, 250, 350 volts, etc.), a high capacity variable autotransformer ("Variac" type W20, General Radio Company, Concord, Mass.) was connected to the primary winding of the step-up transformer. This modification permitted continuous variation of the output voltage between 0 and 110 per cent of the voltage indicated on the defibrillator panel. Only output voltage was affected by this circuit change, neither waveform or shock duration was detectably altered.

Group 5 animals had biopsy tissues taken from both sides of the limbs after 7 days. Thus 8 specimens were obtained from each of the 8 animals. Of the 64 specimens half had been subjected to AC and half to DC discharge.

Results

Pathologic Changes Following Shocks at Defibrillating Levels. Skin and subcutaneous tissues subjected to AC or DC discharges were normal in 225 of 227 biopsy samples (Groups 1, 2, 3 and 5). The 2 abnormal specimens were obtained from sites which had developed areas of infec-

tion and ulceration following multiple AC discharges. No pathologic changes were observed in control skeletal muscle tissues or in those specimens obtained from treated sites within 24 hours after shock (Fig. 1). A single shock of either AC or DC resulted in no lesions. No differences were observed between anode or cathode electrode sites.

In contrast to the absence of changes in skin and subcutaneous tissues, the defibrillating currents produced a high incidence of lesions in skeletal muscle. The lesions resulting from AC and DC discharges were morphologically indistinguishable. There were, however, distinct differences in the grade of injury. Lesions varied with respect to the total number of muscle fibers involved and severity of alteration within individual fibers. In the majority of instances the extent of morphologic change was related to the type and number of electrical shocks.

The earliest recognizable lesions consisted of increased numbers of sarcolemmal nuclei within individual muscle fibers (Fig. 2). In advanced lesions this proliferation resulted in agglomeration of large masses of nuclei which exceeded the width of a single fiber (Fig. 3). Additional pathologic changes consisted of basophilic staining of the sarcoplasm, vacuolization, (Fig. 4), focal loss of striations, clumping of sarcoplasm, (Figs. 5 and 6), and frank necrosis with destruction of the investing reticulum of the muscle fiber (Figs. 7 and 8). None of the tissues showed inflammatory cellular infiltrations or vascular lesions. In specimens obtained at 30 days the changes were consistent with those of atrophy, fatty infiltration and regenerative change.

To facilitate comparison of severity of striated muscle lesions, biopsy sections were arbitrarily graded into one of the following categories: grade 0, normal muscle; grade 0.5, focal sarcolemmal proliferation; grade 1, changes found in the previous grade plus focal basophilic staining of muscle fibers; grade 2, previous changes plus focal atrophy, vacuolization of muscle fibers and focal sarcolemmal clumping; grade 3, in addition to the above changes there were also areas of focal necrosis with destruction of investing reticulum and/or atrophy; and grade 4, characterized by diffuse areas of generalized necrosis and/or atrophy.

Biopsies made in limbs 7 days after 5 AC shocks showed definite muscle damage in 4 of 6 specimens. The mean grade of the 4 "positive" sections was 1.3. Sites subjected to the same number of DC shocks were entirely free of lesions (Table II). When the number of shocks to the forelimb was increased to 10, 13 out of 14 specimens obtained from AC treated areas were "positive" with a mean grade of 2.6. Four of these lesions were classified grade 4. By contrast after 10 DC discharges, only 8 of 20 forelimb biopsy specimens were "positive" with a mean grade of 1.4. None of these were classified as grade 4. In hind limbs subjected to

10 shocks, lesions were less severe, but again AC treatment resulted in a higher incidence and greater severity of lesions. After 40 shocks to the hind limb, the incidence of DC induced pathologic changes began to approach that observed in the AC group. However, the average pathologic

TABLE II

INCIDENCE AND SEVERITY OF PATHOLOGIC CHANGES ONE WEEK FOLLOWING AC AND DC SHOCKS TO FORE AND HIND LIMBS

No. shocks	Limb	Type shock	Biopsies no.	Positive no.	Mean grade
1	Fore	AC	6	0	0
		DC	6	0	0
5	Hind	AC	6	4	1.3
		DC	6	0	0
10	Fore	AC	14	13	2.6
		DC	20	8	1.4
	Hind	AC	8	5	1.6
		DC	8	3	1.3
40	Hind	AC	6	5	2.7
		DC	12	8	1.4

Mean grade was determined from biopsies exhibiting pathologic changes which were assigned numerical values as indicated in text.

grade for the latter was nearly twice as severe as for the sites subjected to DC shocks. At 30 days all lesions had regressed in severity. Pathologic changes still present were minor and inconsistent. AC and DC specimens showed no difference.

In summary, when defibrillating level settings were used, twice as many DC as AC shocks were required to produce pathologic lesions. DC lesions were always less severe regardless of site or number of shocks. DC lesions remained of the same grade whether 10 or 40 shocks were given, while AC lesions progressed in severity.

Comparative Energy Dissipations of AC and DC. In the above studies comparison between AC and DC was based on energy settings required to defibrillate the dog heart transthoracically. To determine the actual amount of electrical energy dissipated within the tissue by each of the two methods, simultaneous voltage and current discharge waveforms were photographed. These studies were conducted in 3 animals (Group 4). Energy dissipation and limb resistance were found to be functions of the waveforms used (Table III). The resistance found with AC was greater than that obtained with DC. Forelimbs offered less resistance than hind limbs. The ratio of AC to DC energy delivered was 11 to 1 for forelimbs and 7 to 1 for hind limbs (Table III). The discharge waveforms are illustrated in Text-figure 1.

Pathologic Changes Following AC and DC Shocks of Similar Energies. In an attempt to define the roles of voltage and energy in the production of pathologic lesions, nearly identical energies of AC and DC were administered to limbs of an additional 8 animals (Group 5). Each of the 4 limbs received 10 shocks. The DC energy settings employed

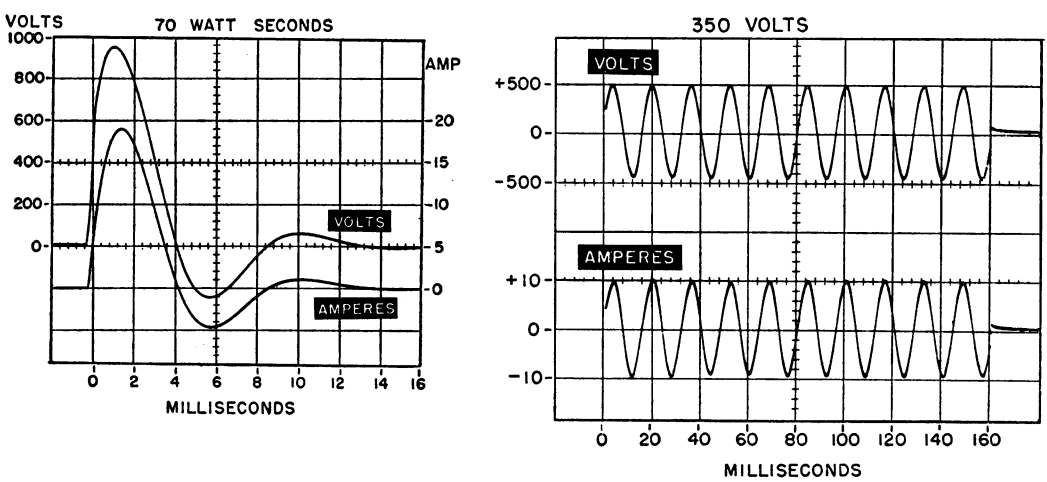
TABLE III

RESISTANCE AND ENERGY MEASUREMENTS WITH DC (70 WATT SECONDS) AND AC (350 VOLTS) DISCHARGES IN FORE- AND HIND LIMBS †

	DC	AC
Forelimb		
Number of measurements	30	30
Resistance of limb * (ohms)	40 ± 6	51 ± 19
Mean energy per shock * (watt sec)	36 ± 5	393 ± 76
Hind Limb		
Number of measurements	120	120
Resistance of limb * (ohms)	48 ± 5	90 ± 25
Mean energy per shock * (watt sec)	37 ± 1	252 ± 51

* Mean and standard deviation.

† Resistance was measured by dividing the peak voltage by the peak current. It is equal to 50 ohms in both discharges. In each case the energy dissipated may be closely approximated by the sum of RMS voltage, current, and time products for each half cycle. (RMS = peak × 0.71) Using this approach the energies are found to be 41 watt seconds and 365 watt seconds for DC and AC respectively. The difference between DC setting and energy dissipation is due to losses within defibrillator circuit elements.



TEXT-FIGS. 1A and B. Discharge waveforms of the two defibrillators employed in the present study, A) DC or capacitor discharge at a setting of 70 WS, and B) AC at setting of 350 V. Both of these settings represent levels effective for transthoracic defibrillation of the dog heart.

were 70 and 140 WS in the forelimbs and 140 and 280 WS in the hind limbs. With DC the measured energy dissipated was less than the setting because of losses in the defibrillation and monitoring circuits. These losses increased with higher energy settings which caused decreased limb resistance. Thus the per cent of energy delivered varied from 60 per cent at 70 WS to 40 per cent at 280 WS (Table IV). The AC voltage was adjusted to give equal total energy dissipation in the same number of shocks, and ranged from 105 to 210 volts. Biopsies were made on both

TABLE IV

INCIDENCE AND SEVERITY OF PATHOLOGIC CHANGES 7 DAYS AFTER 10 AC OR DC DISCHARGES OF NEARLY IDENTICAL ENERGY‡

		Mean energy (watt seconds)†	RMS§ Volts	RMS§ Amperes	Positive biopsies* (no.)	Mean grade
Forelimb						
Left	AC	367 ± 17	149	1.5	0	0
	DC	400 ± 18	600	12.0	5	1.6
Right	AC	738 ± 52	230	2.0	5	2.0
	DC	757 ± 16	850	17.0	7	3.1
Hind Limb						
Right	AC	603 ± 14	181	2.0	1	1.0
	DC	676 ± 26	850	17.0	6	2.8
Left	AC	1114 ± 25	260	2.6	4	2.5
	DC	1116 ± 63	1200	25.0	5	3.3

* There were 8 biopsies obtained for each category.
† Mean and standard deviation.
‡ The duration of the AC discharge was 170 msec and of DC, 2.5 msec.
§ Root mean square.

sides of the limbs after 7 days. No changes were noted in skin and subcutaneous tissues. Muscle lesions were both more frequent and more severe following DC shock than after AC shock. Lesions were seen in 30 of the 40 DC biopsy specimens, but in only 14 of 40 AC tissues. At each energy setting the grade of lesions was more severe when DC had been administered.

These findings suggest that when employing similar AC and DC energies the occurrence and severity of muscle damage is related to voltage or current. The difference between the two types of discharge was most marked when 40 WS was delivered in a single shock. However, when the energy per shock exceeded 100 WS, AC and DC discharges caused nearly similar pathologic changes despite wide disparity in voltage and current values (Table IV).

Arrhythmias Following AC and DC Limb Shocks. An incidental se-

ries of observations concerned the occurrence of arrhythmias following AC and DC shock in the 8 animals of group 3 (Table V). Shocks to the hind limb did not result in arrhythmias. When the forelimbs were employed, either atrial or ventricular fibrillation followed 38 of the 160 shocks. Of the 10 episodes of atrial fibrillation, 5 followed DC shock to the right forelimb and 5 followed AC shock to the left forelimb. Of the 28 episodes of ventricular fibrillation 27 followed the use of AC (generally after shocks to the left forelimb).

Discussion

In the present study lesions in skeletal muscles were compared after alternating current (AC) and direct current (DC) shock. AC defibrilla-

Table V

COMPARISON OF ARRHYTHMIAS FOLLOWING AC OR DC SHOCKS ACROSS LIMBS IN 8 ANIMALS *

Type of Shock	Limb Location		No.	AF	VF
AC	Front	Right	40	0	6
		Left	40	5	21
	Hind		80	0	0
DC	Front	Right	40	5	1
		Left	40	0	0
	Hind		80	0	0

AF—Atrial fibrillation.
VF—Ventricular fibrillation.
Right and left hind limbs are combined since no arrhythmias were observed.
* Group 3.

tion with 60 cycle current has been extensively investigated since its introduction in 1933 by Hooker and co-workers.[6] The use of DC or capacitor discharge, has been widely accepted only within the past few years.[7-9] Capacitor discharge may be made to assume a wide variety of waveforms. Determination of optimal waveform for clinical use will have to consider both effectiveness of depolarization and safety to the heart and adjacent tissues.

While the large muscles of the fore and hind limbs appear to be unsuitable sites for evaluating defibrillators, impedance measurements in dogs have shown that resistance in the limbs (Table III) is similar to that reported for the exposed heart.[10] Furthermore, the use of limbs affords a number of advantages. One can compare different treatment methods in the same animal: e.g., in the present study AC, DC and control sites. A small number of animals may provide decisive information

regarding the relation between lesions and other experimental variables, e.g., number of shocks, or energy and voltage levels. The ease of biopsy and the availability of multiple tissue specimens are additional advantages.

The lesions resulting from AC and DC were in large measure dependent upon the discharge settings selected. Since AC and DC represent two diverse forms of electrical discharge, a comparison could have been made at similar voltage, current or energy level settings. None of these were selected for initial comparison since the objective of the study was to determine the least injurious method for depolarizing the heart. A shock level effective for defibrillation was therefore chosen. The settings employed were 350 volts and 70 WS for the AC and DC instruments respectively. At these settings AC and DC defibrillate about 65 per cent of episodes of ventricular fibrillation in dogs.[5] While these settings are of equivalent physiologic effect, they represent markedly different energy levels. In order to determine the properties of the electric discharge responsible for muscle injury, the two types of discharge were also studied at settings of approximately equal energy but without regard to defibrillating effectiveness. For example, an AC setting of 150 volts was found to approximate the energy delivered by a 70 WS DC discharge, however, 150 volts is well below the AC requirement for causing defibrillation of dogs transthoracically.

Following the application of DC or AC discharges, lesions were morphologically identical but varied in severity and frequency. A single shock of either type provoked no discernible pathologic change. It required 5 shocks of AC and 10 shocks of DC to induce lesions of approximately the same severity. In the first 3 groups of 32 animals the incidence of "positive" biopsy specimens at 7 days was 68 per cent for AC and 37 per cent for DC. The mean grade of AC lesions was greater than that of DC lesions. The most severe lesions (4+) were seen only after AC. Other differences were noted between these two forms of electrical discharge. Increasing the number of shocks resulted in a change in severity of lesions after AC but not after DC. Thus the mean pathologic grade after 10 AC shocks to the hind limbs was 1.6, after 40 shocks it rose to 2.7. For similar numbers of DC shocks the grade remained fixed at about 1.4; the incidence of lesions, however, increased from 38 to 67 per cent. The mass of muscle between electrodes influenced the frequency and severity of lesions after AC but not after DC. For example, 10 AC shocks to the forelimb resulted in a degree of injury similar to that which followed 40 AC shocks to the hind limb. Such a relation between muscle mass and severity of lesion was not observed in DC treated limbs. The energy output of an AC defibrillator increases as

the resistance between the electrodes decreased. Due to its lower resistance, a forelimb receives more energy from a given AC shock, than a hind limb. This may account for the greater severity of AC lesions noted in the forelimb.

The lesions observed were grossly and microscopically different from those following electrical burns.[11] In the present studies skin and subcutaneous tissue were not involved, there was no gross evidence of muscle necrosis, there was uniform absence of inflammatory or vascular change irrespective of when the biopsy was made. The muscle lesions consisted only of disruption of the sarcoplasmic reticulum, clumping of sarcoplasm and necrosis of individual fibers. There was also associated sarcolemmal nuclear proliferation, vacuolization and replacement by fibroadipose tissue. In the case of DC, the lesions were identical whether the biopsy specimen was obtained from beneath the anode or cathode site.

When energies were matched for the two forms of discharge, DC lesions were consistently more severe. The dependence of lesions on the energy of discharge was demonstrated in the case of AC shocks delivered to forelimbs. When the cumulative energy equalled 367 WS no lesions were observed, when it was increased to about 738 WS, 5 of 8 biopsies showed lesions with a mean pathologic grade of 2.0. This doubling of energy, was associated with a 40 per cent rise in voltage and current. The AC defibrillator, though less injurious per unit of energy delivered, loses this advantage in practice because of much greater energy requirements for achieving defibrillation.

The basis for these electrical lesions is not clear. Several possible factors deserve consideration: impaired blood flow, thermal injury, effects of intense contraction, and cell membrane damage. It is unlikely that either intense vascular spasm or vascular occlusion accounted for the lesions. There was no evidence of vascular injury and thrombosis, tissue infarction, or polymorphonuclear cellular infiltration. A thermal effect would have resulted in cutaneous burns, since the skin presents the highest resistance to the passage of electric current. Skin and subcutaneous tissues, however, were completely free of pathologic change. The focal nature of the lesion, which occasionally involved only a single fiber, argues against a generalized thermal effect. Nikitin[12] who studied muscle lesions in white mice following alternating current shock concluded that the intense contraction ruptured sarcolemmal sheaths and caused myofibrillar degeneration. Our finding that AC lesions were more severe than those resulting from DC is consistent with such a mechanical effect, since the tetanic contraction induced by AC develops several times the force of the single twitch provoked by DC. Recent observations in

our laboratory indicate that different degrees of muscle damage result from DC pulses of identical energy but of different waveform. These different waveform discharges appear to produce the same degree of contraction. It may be that suprathreshold electrical stimuli permanently alter the cell membrane of skeletal muscle fibers resulting in changed electrolyte composition of the cell and disruption in its organization. Indeed we have noted a significant release of potassium from muscle subjected to certain forms of electrical shock.

In addition to muscle damage, administration of electrical discharge to the limbs resulted in a substantial incidence of cardiac arrhythmias. This was especially striking with AC where half the shocks through the left forelimb resulted in ventricular fibrillation. This is twice the incidence observed after transthoracic shock at a 350 volt setting. It has been shown that the incidence of ventricular fibrillation with AC shocks in the defibrillating range varies inversely with the voltage of the shock.[5] It appears that when the shock is delivered to the left forelimb, the fraction of the energy reaching the heart is appropriate to provoke ventricular fibrillation.

At the present time AC is still widely employed for defibrillating the human heart. In cardiac operations, especially those involving hypothermia, repeated shocks are frequently necessary to defibrillate the ventricles. Such shocks may compromise the already diseased heart and contribute to operative morbidity and mortality. Main, Aberdeen and Gerbode[13] and Yarborough, Ussery and Whitley[14] have shown significant deterioration in ventricular function after repeated AC shocks delivered directly to the heart. Such changes did not occur following DC discharge. Tedeschi and White[15] have described epicardial and myocardial burns following application of AC shock directly to the heart. The work of Lown, Neuman, Amarasingham and Berkovits[5] showed significant impairment of cardiac function and death following repeated transthoracic AC shocks. The high incidence of ventricular fibrillation following shocks to the forelimbs, indicates the existence of a significant hazard to the operator holding the electrode paddles as well as to other attendant personnel. These findings, together with the high incidence and severity of muscle lesions following defibrillating level AC shocks, suggest that the clinical use of AC discharge be abandoned.

Summary

Pathologic changes were compared following AC and DC discharge through the fore- and hind limbs of 43 dogs. Skin and subcutaneous tissues were not injured. Skeletal muscles showed unique lesions characterized by focal sarcolemmal proliferation, loss of striation, vacuolization,

and ultimately frank necrosis of individual fibers. The lesions were morphologically indistinguishable regardless of the type or polarity of the discharge. At comparable defibrillating energy levels, AC resulted in a higher incidence and greater severity of pathologic change. Lesions first occurred following 5 AC or 10 DC shocks.

When AC and DC were employed at similar energies, the latter resulted in more severe lesions. Discharge energy appeared to be the major factor responsible for pathologic change.

When AC was applied across the forelimbs, there was a high incidence of ventricular fibrillation; this was not observed following DC shock. These findings suggest that the clinical use of alternating current defibrillators should be discontinued.

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LEGENDS FOR FIGURES

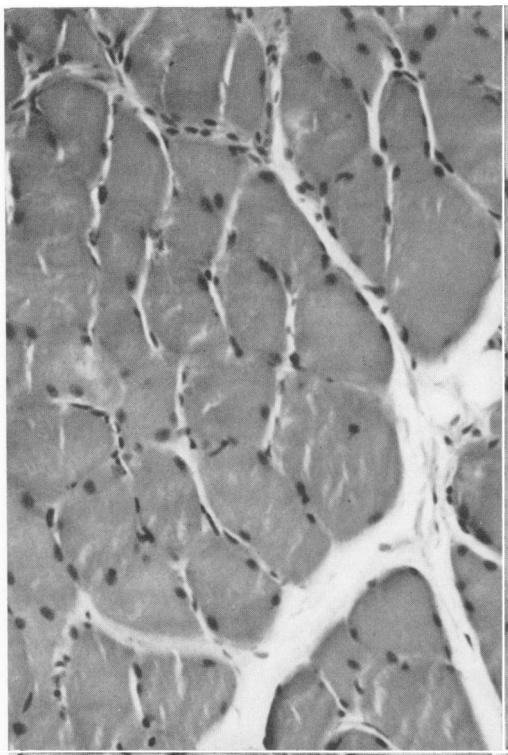
Except where indicated, photomicrographs were prepared from sections stained with hematoxylin and eosin.

FIG. 1. Cross section of normal striated muscle. The average muscle fiber measures 40 to 50 μ in diameter and is composed of myofibrils embedded in a sarcoplasmic matrix and surrounded by the sarcolemmal membrane. The sarcolemmal nuclei lie just beneath the sarcolemmal membrane. × 200.

FIG. 2. Cross section of muscle 7 days after electroshock. There is focal fiber involvement with mononuclear cell infiltration and proliferation of sarcolemmal cells. Other muscle fibers show proliferation of sarcolemmal cells characterized by variation in size and nuclear staining. × 200.

FIG. 3. Longitudinal section of muscle 7 days after electroshock. There are large masses of proliferating sarcolemmal cells which in some areas exceed the width of the myofibril. There is also sarcoplasmic clumping, loss of cross striations and infiltration by mononuclear cells. × 200.

FIG. 4. Cross section of muscle 7 days after countershock. There is marked vacuolization and clumping of the sarcoplasm. In addition there is sarcolemmal nuclear proliferation and mononuclear cell infiltration. × 500.



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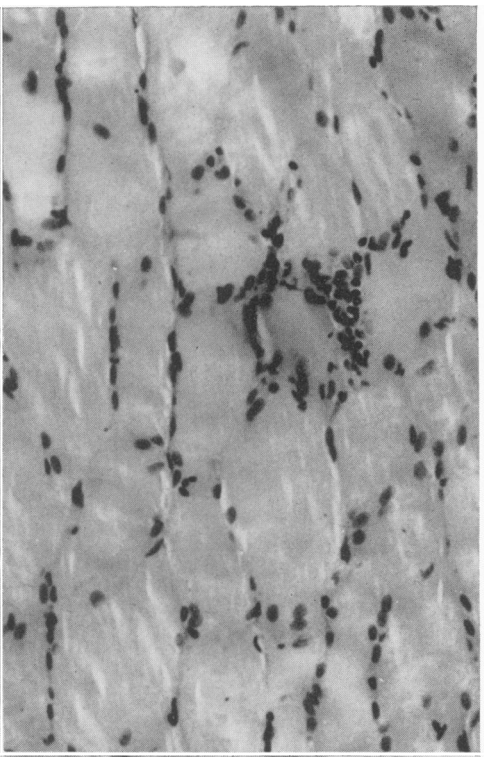

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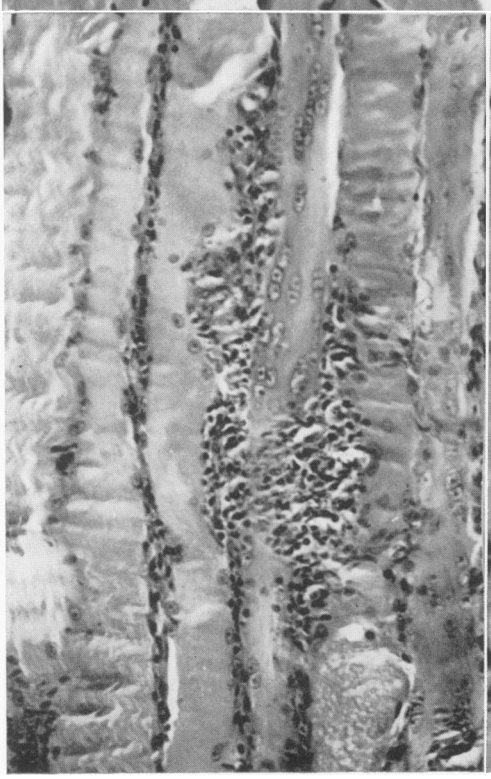

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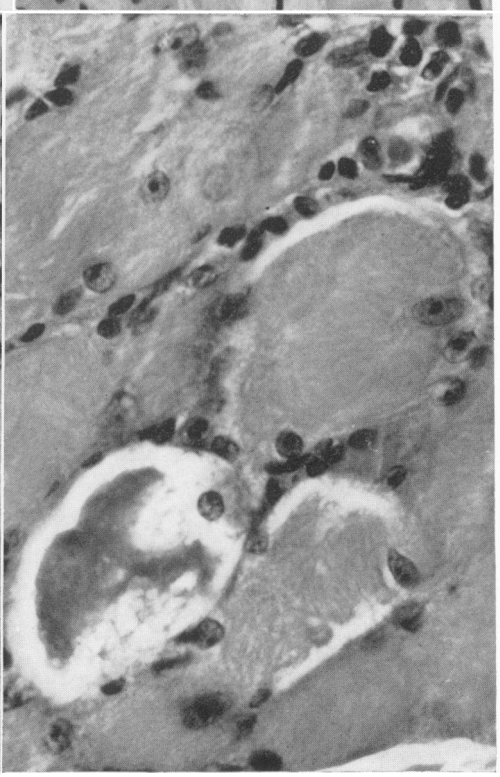

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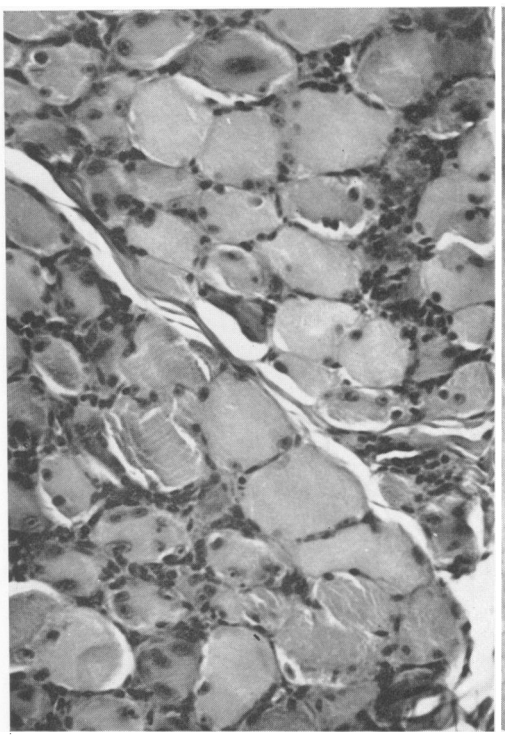
FIG. 5. Longitudinal section of muscle 7 days after electroshock. There are "trains" of proliferating sarcolemmal nuclei and segmental necrosis of the myofibril as indicated by marked clumping of the sarcoplasm. Fragmentation of a myofibril appears in the center of this figure. × 500.

FIG. 6. Cross section of muscle showing a diffuse lesion 7 days after electroshock. There is marked sarcolemmal cellular proliferation, mononuclear cell infiltration, vacuolization and clumping of the sarcoplasm. × 200.

FIG. 7. Cross section of normal control muscle shows that each myofibril is completely encased by a sarcolemmal membrane. The fibers vary in size and number of nuclei. The nuclei are similar in appearance and have approximately the same amount of basophilic staining chromatin material. Gomori's reticulum stain. × 500.

FIG. 8. Cross section of muscle with severe lesions 7 days post electroshock. The sarcoplasmic membrane is completely destroyed indicating necrosis of the myofibril. There are proliferating sarcolemmal cells and infiltration by mononuclear cells. Compare with Figure 7. Gomori's reticulum stain. × 500.



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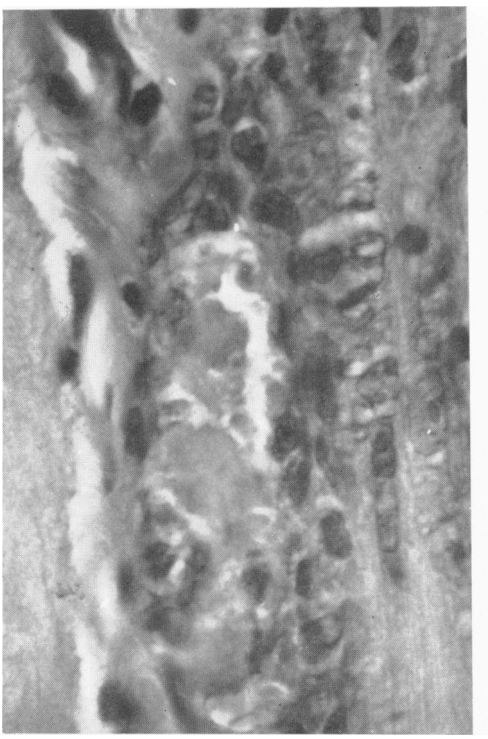

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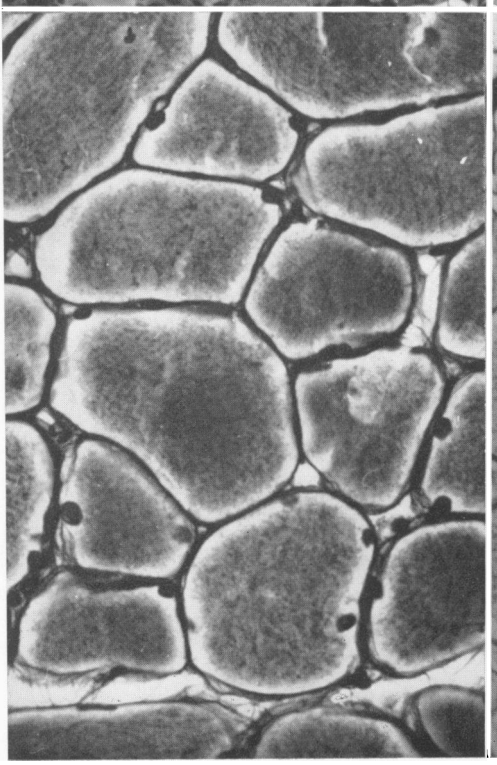

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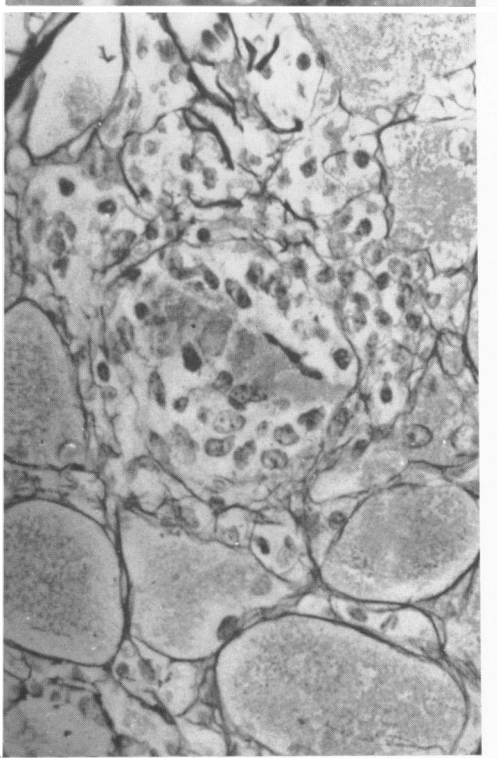

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