Myocardial Necrosis
from Direct Current Countershock

Effect of Paddle Electrode Size
and Time Interval Between Discharges

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and Evan D. Thomas

SUMMARY
The effect of varying both paddle electrode size and the time interval between direct current
countershock on myocardial necrosis was studied. Forty-two dogs were divided into seven groups of six dogs
each. All dogs were given ten consecutive, 240 watt-second countershocks (delivered energy into a 50 ohm
load). Three groups were shocked with paddle electrode diameters of 8.0 cm (standard electrodes), two
groups with paddle electrode diameters of 12.8 cm (large electrodes), and two groups with paddle electrode
diameters of 4.3 cm (small electrodes). The time intervals between discharges in the groups shocked with
the standard electrodes were 15 seconds, one minute, and three minutes. The time interval between dis-
charges in the groups shocked with small and large electrodes was 15 seconds and three minutes. Myocardial
necrosis was quantitated by precordial electrocardiographic mapping recorded minutes after, and by gross
and microscopic examination of the hearts four days after direct current countershock.

When the time interval between discharges was shorter, myocardial necrosis was greater. When the time
interval between discharges was constant, more necrosis was produced with smaller-sized paddle electrodes.
It is concluded that large paddle electrodes should be used for delivering direct current countershocks, and
that during elective cardioversion, consecutive discharges should be delivered at time intervals greater than
three minutes.

Additional Indexing Words:
Cardioversion Defibrillation Precordial electrocardiographic mapping

FOLLOWING the development of direct current
(DC) defibrillators, a study comparing myocardial necrosis from alternating current (AC) and direct
current (DC) discharges showed the relative safety of the DC discharges.1 The transient increase in serum
enzyme levels that occasionally occur in man following DC cardioversion have been attributed to chest
wall muscle and not cardiac muscle damage.2 4 A re-
cent study in which isoenzymes of creatinine
phosphokinase (CPK) were measured following elec-
tive cardioversion suggests that, at times, some of the
elevation of CPK is contributed by enzymes from the
myocardium.6

During a study of the determinants of canine transthoracic resistance to DC discharge in this
laboratory, electrocardiographic ST-segment elevation and gross myocardial lesions were frequently
observed after ten consecutive DC countershocks. Similar studies by Patel and Galysh suggested that
gross pathological lesions were more frequent at higher levels of delivered energy.7 Our pilot studies
suggested that the myocardial necrosis was related not only to the amount of delivered energy but also to the
paddle electrode size and the time interval between DC discharges.

Since this observation could be of significant clinical import, the following experiments were
devised to determine the effect of paddle electrode size and time interval between DC discharges on
myocardial necrosis from DC defibrillator or cardioverter discharge.

Methods
Forty-two mongrel dogs weighing between 14.2 and 27.0
kg were divided into seven groups of six dogs each. The

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MYOCARDIAL NECROSIS FROM DC SHOCK

protocol within each group varied only in the time interval between discharges and/or the diameter of the paddle electrode used to deliver the discharge. Each dog was anesthetized with pentobarbital sodium, 25 mg/kg. The thoracic hair was removed with electric clippers. Electrode paste* was generously applied to the paddle electrodes which were then applied to the chest wall. In all experiments, one paddle electrode was placed on the left precordium with its center over the point of maximal cardiac impulse. The second was placed on the opposite lateral chest wall at the same thoracic level. Ten consecutive direct current discharges were delivered to each animal from a Hewlett-Packard 7802C defibrillator at a dial setting of 400 watt-seconds. At this setting this unit delivers 240 watt-seconds across a 50 ohm resistance.

Three groups were shocked with paddle electrodes with diameters of 8.0 cm (standard electrodes), two groups with paddle electrodes with diameters of 12.8 cm (large electrodes), and two groups with paddle electrodes with diameters of 4.3 cm (small electrodes). The time intervals between discharges in the groups shocked with standard electrodes were 15 seconds, one minute, and three minutes. The time intervals between shocks in the groups shocked with the small and large electrodes were 15 seconds and three minutes.

Electrocardiographic and morphologic means were utilized to assess the degree of myocardial injury and/or necrosis. A standard 12-lead electrocardiogram was taken on each dog before and after the discharges were delivered. Electrocardiographic precordial mapping was then done. A grid of 25 points, each two centimeters apart, was painted on the left chest wall with the center of the grid at the point of maximal cardiac impulse (fig. 1). Electrocardiographic recordings were made from each point using the unipolar precordial lead. Any electrocardiographic ST-segment elevation which differed from the baseline recording was measured in millimeters, at a point 0.08 second after the onset of the QRS complex. The ST-segment changes of each of the 25 points on each animal were added. The mean, in millimeters, was recorded for each animal.

Four days after the direct current discharges were delivered, the dogs were anesthetized and then sacrificed. The hearts were examined grossly and microscopically. The area of gross discoloration of each heart was estimated in square centimeters. Blocks were taken in buffer 10% formalin and in 4% gluteraldehyde for light and electron microscopic (E.M.) study, respectively. The hearts were then placed in 10% formalin. After fixation for several weeks, the hearts were sectioned parallel to the atrioventricular sulcus. The depth of the gross lesions was noted. Tissue blocks for light microscopy were processed in paraffin and those for E.M. in epon. Microscopic sections from the area of damage were graded as negligible, grade I, II, or III. The macroscopical grading was done by the pathologist (E.W.) on the basis of a combination of the severity and the depth of the lesions. The smaller and milder lesions tended to be quite superficial, i.e., subepicardial. The larger and more severe lesions tended to be full thickness. Grade I lesions were patchy in that many apparently intact muscle fibers were interspersed with the necrotic area and/or the lesion was very superficial. Grade III lesions contained large areas where essentially all fibers were degenerating and the depth of the lesions was full thickness. Grade II lesions were intermediate between Grades I and III, intermediate in both the extent of destruction of cell fibers and in depth. Samples of myocardium which appeared normal grossly showed no microscopic lesions.

To quantitate the amount of necrosis in each heart, the gross area of discoloration in square centimeters was multiplied by the microscopic severity to obtain a myocardial damage index. For example, if the animal’s heart showed a gross lesion with an area of two square centimeters, and the microscopic severity was judged to be Grade II, a myocardial necrosis index of four was given. The pathologist evaluated the morphologic lesions without knowledge of the protocol used on the animal until after the myocardial necrosis score was obtained.

Results

The results are tabulated in tables 1, 2, and 3. There was more electrocardiographic ST-segment elevation

\[\text{Table 1} \]

| Relation of Time Interval Between DC Discharges and Paddle Electrode Size to the Magnitude of Electrocardiographic ST-Segment Elevation |
|---|---|---|
| Small electrodes | Standard electrodes | Large electrodes |
| Time interval: 15 sec | 8.6 ± 7.6 | 2.3 ± 2.8 | 0.1 ± 0.3 |
| Time interval: 1 min | 1.2 ± 3.1 |
| Time interval: 3 min | 0.2 ± 0.4 | 0 ± 0 |

ST segment elevation in millimeters per precordial lead per dog, expressed as mean ± one SD.

\[\text{Table 2} \]

| Relation of Time Interval Between DC Discharges and Paddle Electrode Size to Myocardial Necrosis |
|---|---|---|
| Small electrodes | Standard electrodes | Large electrodes |
| Time interval: 15 sec | 60.2 ± 45.7 | 4.4 ± 1.8 | 3.0 ± 4.5 |
| Time interval: 1 min | 15.0 ± 29 |
| Time interval: 3 min | 4.0 ± 5.4 | 1.1 ± 1.8 | 0.5 ± 0.9 |

Mean ± one SD.

*Redux Paste, Hewlett-Packard part number 651-1008.

**Figure 1**

Left) Grid showing 25 positions for precordial electrocardiographic mapping. Right) Example of grid marked on shaved canine chest.

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when the same amount of direct current electrical energy was discharged transthoracically with the small electrodes and at shorter time intervals (Table 1, 15 seconds versus three minute intervals, \( P < 0.025 \)). An example of precordial electrocardiographic mapping is shown in figure 2.

As shown in table 2, there was more myocardial necrosis when equal amounts of energy were discharged transthoracically at 15 seconds rather than three minutes (\( P < 0.25 \)). When the time interval between discharges was held constant, there was more myocardial necrosis with smaller diameter electrodes (small versus standard and small versus large electrodes, \( P < .025 \)). Table 3 shows the relation between pathologic myocardial necrosis and the mean ST elevation (per precordial lead) for each of the 42 dogs used in the study. The over-all correlation coefficient was high (\( r = 0.89 \)).

Figure 3 illustrates the typical appearance of the gross morphologic lesions observed four days post-direct current discharge. As shown in the illustrations, most specimens had lesions that were on opposite sides of the heart like entrance and exit wounds. In some, the lesions were more confluent on the anterior surface of the heart. Examples of the microscopic lesions are shown in figures 4 and 5. The microscopic lesions were characterized by necrosis of myocardial fibers which were replaced by proliferating large mononuclear cells. Although the animals were all sacrificed four days after the DC discharges, there was considerable variation in the extent of the replacement of necrotic myocardial fibers. Some necrotic fibers retained their morphologic characteristics, with cross striation visible. At times, the striations were exaggerated in prominence by dark staining material. The dark staining material was positive for calcium by the von Kossa stain and was electron dense in unstained electron microscopy studies. On electron microscopical examination, this material appeared to be deposited in the mitochondria. Other necrotic fibers were extensively fragmented, with loss of staining intensity, and still others were replaced by large mononuclear cells, many containing mitotic figures. Considerable variability in the amount of destruction was found, not only from animal to animal, but from field to field in the same specimen.

![Figure 3](image-url)

**Figure 3**

Examples of gross myocardial appearance four days after ten direct current defibrillator discharges. **Top** Necrosis produced with small (4.5 cm diameter) electrodes with discharges at 15 second intervals. Note the large whitish discolored area on each side of the heart. Their size is similar to the paddle electrode size used, even though the electrodes were applied to the intact chest skin. **Bottom** Necrosis produced by electrodes of 8.0 cm diameter. Note white areas on the lateral aspects of the heart. One lesion is lateral to the left anterior descending coronary artery and the other larger one is on the infero-lateral surface of the right ventricle. Their area is much smaller than that of the electrode used.

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Discussion

The advantages of treating certain cardiac arrhythmias with electrical cardioversion are well known and widely accepted. Possible adverse effects on the myocardium have been suggested by the appearance of arrhythmias, electrocardiographic ST-segment elevation, serum enzyme elevations, development of pulmonary edema, and acute left ventricular decompensation following elective cardioversion.\textsuperscript{6-16} Arrhythmias such as the transient appearance of a premature ventricular contraction are recognized as commonly occurring after this treatment, but most severe arrhythmias that develop have been attributed to concomitant administration of toxic or near toxic doses of quinidine and/or digitalis, or the advanced stage of the underlying cardiac disease.\textsuperscript{17} There is reason to doubt these conclusions.\textsuperscript{6}

Russian authors have reported "marked changes in the myocardium" in five of 220 patients who had acute left ventricular insufficiency following elective cardioversion.\textsuperscript{19} Transient electrocardiographic ST-segment elevation has been reported in the English

Table 3

<table>
<thead>
<tr>
<th>Time Interval of Countershock</th>
<th>Small (4.3 cm) ST elevation</th>
<th>Myocardial necrosis*</th>
<th>Standard (8.0 cm) ST elevation</th>
<th>Myocardial necrosis</th>
<th>Large (12.8 cm) ST elevation</th>
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*Myocardial damage index determined by multiplying the area of discoloration on gross examination in square centimeters by a grade assigned for the severity of the lesion examined microscopically.

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literature following elective cardioversion.8-11 Both
good correlation found between the mean ST-
segment elevation and the amount of myocardial
necrosis (table 3, r = 0.89) and the finding that all
animals with ST-segment elevation had myocardial
necrosis suggest that any electrocardiographic ST-
segment elevation seen following transthoracic DC
dischARGE is indicative of myocardial necrosis. Sixteen
animals had myocardial necrosis without ST-segment
elevation, indicating that ST-segment elevation is a
specific but relatively insensitive indicator of myocar-
dial necrosis. Serial electrocardiograms were not
done on these animals, but a standard 12-lead electrocar-
diogram done prior to sacrifice on the fourth day
showed no ST-segment elevation.

Although elevated serum enzymes have been
reported following elective cardioversion,2-4,11 most
investigators have concluded that serum enzyme
elevation resulted from chest wall muscle and not car-
diac muscle damage.2-6 A recent study of the effect of
cardiOversion on the serum isoenzymes of CPK
showed that the MB or myocardial fraction of CPK
was elevated in a small percent of patients.6

We have produced myocardial necrosis in a dog in
our laboratory with one DC discharge delivered with
"pediatric-sized" paddle electrodes.20 This experience
and the present study suggest that larger paddle elec-
 trodes should be used for cardioversion and defibrilla-
tion. Preliminary studies from this laboratory have
shown that 12.8 cm diameter electrodes are as effective
as 8.0 cm diameter electrodes in defibrillating the
dog.21

Transthoracic impedance to direct current dis-
charge is greatest with small paddle electrodes and
progressively decreases with increasing electrode size,
even when only one of the two electrodes is increased in
size.22 In the present study, the mean impedance
was 30 ± 1 ohm with the 12.8 cm electrodes and
66 ± 1 ohm for the 4.5 cm electrodes. If damage is
less when the transthoracic impedance is less, one
should select not only the appropriate paddle elec-
trode size, but also the chest wall interface that results
in lowest impedance. Studies have shown that im-
pedance during direct current countershock is less
when electrode paste is used and increases progress-
vously with saline soaked gauze pads, electrode
creams, and with bare electrodes against the chest
wall.23

There were animals in each group (table 3) in which
there was no necrosis. This large variation in suscep-
tibility suggests that myocardial necrosis to DC
defibrillator discharge is related to other factors
besides delivered energy, paddle electrode size, and
time interval between discharge.

It is probable that myocardial necrosis can occur in

man from DC electroshock delivered during elective
cardiOversion. Myocardial necrosis also probably oc-
curs in patients who undergo multiple defibrillations.
The clinical implications of this study are that myocar-
dial necrosis can be minimized by utilizing large pad-
dle electrodes and/or increasing the time interval
between discharges.

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References

1. LOWN B, NEUMAN J, AMARASINGHAM R, BERKOVITS BV: Com-
parison of alternating current with direct current elec-
troshock across the closed chest. Am J Cardiol 10: 223, 1962
2. SLODOS SJ, FALCOV RE, KATZ MJ, WEST M, ZINMAN MA: Serum
enzyme changes following external direct current shock ther-
apy for cardiac arrhythmias. Am J Cardiol 17: 792,
1966
3. WARBASSE JR, WESLEY JE, CONNOLLY V, GALLUZZI NJ: Lactic
dehydrogenase isoenzymes after electroshock treatment of
cardiac arrhythmias. Am J Cardiol 21: 496, 1968
4. TURNER JRB, TOWERS JRH: Complications of cardioversion.
Lancet 2: 612, 1965
5. CORBITT JD, SYBERS J, LEVIN JM: Muscle changes of the
anterior chest wall secondary to electrical countershock. Am
J Clin Pathol 51: 107, 1969
6. EHSANI AA, EWSY GA, SOBEL BE: CPK isoenzyme elevations
after electrical countershock. (abstr) Circulation 45 (suppl
IV): IV-129, 1973
7. PATEL AS, GALYSH FT: Experimental studies to design safe
external pediatric paddles for a DC defibrillator. IEEE Trans
Biomedical Engineering 19: 228, 1972
8. MORRIS JJ JR, PETER RH, MCFITOSH HD: Electrical conversion
of atrial fibrillation. Immediate and long term results and
9. MORRIS JJ JR, EXTUM M, NORTH WG, KONG Y, MCFITOSH H:
The changes in cardiac output with reversion of atrial fibril-
lation to sinus rhythm. Circulation 31: 670, 1965
10. OBAM S, DAVIES JPH: Further experience of electrical con-
version of atrial fibrillation to sinus rhythm: analysis of
100 patients. Lancet 1: 1294, 1964
11. ABIRG H, CUMHLED I: Direct current countershock complica-
12. HONEY M, NICHOLLS TT, TOWERS MK: Pulmonary oedema
following direct current defibrillation. Lancet 1: 765, 1965
13. PALMSINGMO JA: Pulmonary oedema after defibrillation. Lancet
2: 439, 1965
14. RUSEKOV L, MCDONALD L: Pulmonary oedema following
treatment of arrhythmias by direct-current therapy. Lancet
1: 506, 1965
15. RABINO MD, LIOFF W, DREJFUS LS: Complications and
limitations of direct current countershock. JAMA 190: 417,
1964
16. REALE A: Acute effects of countershock conversion of atrial
fibrillation upon right and left heart hemodynamics. Circula-
tion 32: 214, 1965
17. LOWN B, KLEIGER R, WOLFF G: The technique of cardioversion.
Am Heart J 67: 282, 1964
18. LOWN B: Electrical reversion of cardiac arrhythmias. Br Heart J
29: 469, 1967
19. MAKARYCHEV VA, TSHEKHEM BM, GUBVICH NL:
Circulation, Volume 50, November 1974
Characteristics of the electrical impulse applied to stop cardiac arrhythmias. (translated from Byulleten 'Eksperimental'noi Biologuii i Meditsiny 62: 32, 1966)


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