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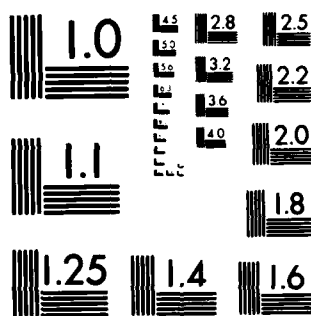
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THE EFFECT OF DIRECT CURRENT TRANSTHORACIC COUNTERSHOCK
ON HUMAN MYOCARDIAL CELLS EVIDENCED BY CREATINE
KINASE AND LACTIC DEHYDROGENASE ISOENZYMES

by

Suzanne Ruth Hansen

A.A., Palomar College, 1974

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A thesis submitted to the
Faculty of the Graduate School of the
University of Colorado in partial fulfillment
of the requirements for the degree of

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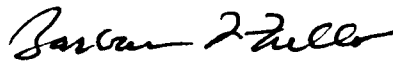
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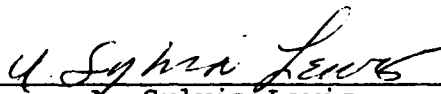
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by



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Date 5/9/56

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The Effect of Direct Current Transthoracic Countershock
on Human Myocardial Cells Evidenced by Changes in
Creatine Kinase and Lactic Dehydrogenase Isoenzymes
Thesis directed by Professor Barbara Fuller

Direct current (DC) transthoracic countershock has been reported to damage myocardial and skeletal muscle cells in humans and animals to varying degrees. The purpose of this study was to evaluate the effects of DC-transthoracic countershock on human myocardial cells by measuring total creatine kinase (CK) and lactic dehydrogenase (LDH) immediately before and at 6, 12, and 24 hours after DC-countershock. Twelve males and 3 females aged 33 to 79 years (mean age 59 years) admitted with a diagnosis of supraventricular dysrhythmias for elective DC-countershock participated in the study. Patients with known or suspected myocardial infarction were not included in the study. Patients were countershocked (delivered energy measured in watt/seconds) using a Life-Pak 6 defibrillator attached to an R-2 unit. Cumulative watt/seconds delivered ranged from 30-1100. Four patients, after 2 countershock attempts, failed to convert to sinus rhythm. These patients received one gram of intravenous procainamide, an antiarrhythmic agent, prior to a third

countershock 4 hours later. This group of patients were analyzed separately as a sub-sample.

The design of this study was prospective, quasi-experimental. There was no randomization due to the specificity of the phenomenon under study. Individual pre-shock enzyme values were used as controls for comparison to mean postshock enzyme values. The presence of cardiac specific isoenzymes was used to infer myocardial cell damage. Data analysis included the Sign Test and the Paired t-test. A correlational matrix was constructed to explore replationships between extraneous variables and the dependent variables.

Results of this study indicate that DC-countershock does not injure myocardial cells when used to correct supraventricular dysrhythmias. Skeletal muscle may be damaged by giving intravenous procainamide and repeating DC-countershock after a period of 4 hours. This damage was evidenced by the presence of skeletal muscle isoenzymes in the serum of 4 patients who received split doses of countershock.

The utility of these findings may be of significance to nursing in prompting further research to explore the possibility that split doses of energy (600 watt/seconds), may lead to skeletal muscle damage.

The form and content of this abstract are approved.

Signed *Julia J. Miller*
Faculty member in charge of thesis

Dedicated to
my mother, Kathleen,
who has always believed
in me, and to
my husband Jim,
who always will.

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I gratefully and sincerely acknowledge the constant encouragement and support of Barbara Fuller, PhD and A. Sylvia Lewis, PhD. Without these dedicated professionals, this project could not have been accomplished.

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CHAPTER I

INTRODUCTION

Electrical countershock has been used to terminate hemodynamically significant cardiac dysrhythmias in humans since the late 1890s. As early as 1775, the use of electric shock was reported by Abildgaard. He described a series of experiments rendering chickens "lifeless" after initial electrical shock with subsequent shocks resuscitating these same animals (Cramp-ton, 1980). Prevost and Batelli (1899) applied electrodes directly to fibrillating dog hearts and used alternating current (AC) and direct current (DC) countershock techniques to terminate fibrillation episodes (Wiggers, 1940). In 1936, Beck first reported successful AC countershock defibrillation to an exposed human heart (Beck, 1936, 1947). By the 1950s, external countershock was preferred over the direct open-chest placement of electrodes and AC shock was no longer employed. Zoll (1956) and his colleagues refined DC-countershock discharge to terminate both atrial and ventricular dysrhythmias (Zoll, Paul, Linenthal, Normal & Gibson, 1956). The first DC-synchronized capacitor

was developed in 1962. Synchronizing the shock to occur after the vulnerable refractory period of the ventricle avoided sending the patient into a more lethal rhythm, specifically, ventricular fibrillation (Lown, Amarasingham, & Neuman, 1962; Lown, Kluger, & Wolff, 1964). The age of synchronized DC-countershock was born, currently a widely used method of terminating hemodynamically significant cardiac dysrhythmias.

Direct current countershock has been reported to precipitate myocardial necrosis in animals studied at necropsy (Dahl, Ewy, Warner, & Thomas, 1974; DiCola, Freedman, Downing, & Zaret, 1976; Ewy, 1983; Lerman, Weiss, Buckley, Becker, & Weisfeldt, 1984). Elevations in serum creatine kinase (CK) enzymes following DC-countershock on humans have been reported (Forrsell, Nordlander, Nyquist, Orinius, & Styrelius, 1975; Hunt & Bailie, 1968; Mandecki, Giec & Kargul, 1970; Reiffel, Gambino, McCarthy, & Leahey, 1978). Further, serum elevations of myocardial specific isoenzymes of creatine kinase and lactic dehydrogenase have been reported following DC-countershock on humans (Ehsani, Ewy, & Sobel, 1976; Pfisterer, Ritz, Scholer, & Vanderschmitt, 1978; Verdejo, Mussenburg, Cardenas, & Cuaron, 1981).

Autopsy studies have failed to reveal myocardial necrosis in human beings following numerous

countershock attempts in unsuccessful resuscitation, but insufficient time may have been the reason. Evolution of histologically detectable changes associated with myocardial necrosis takes more time than is usually available after death and before autopsy actually is accomplished (Ehsani et al., 1976).

The effects of direct current countershock on myocardial tissue are of concern to critical care nurses. Critical care nurses are taking a more active part in the delivery of DC-countershock for the correction of atrial and ventricular dysrhythmias. Does DC-countershock injure myocardial tissue? Does the passage of electrical current through the body harm our clients? In light of these concerns, the purpose of this study was to determine if a relationship existed between the delivery of direct current transthoracic countershock and myocardial injury as evidenced by the presence of cardiac specific creatine kinase and lactic dehydrogenase isoenzymes in human serum. An increased knowledge of the effects of DC-countershock will help the critical care nurse educate clients and assist in planning the pre- and post-shock courses of care.

CHAPTER II

CONCEPTUAL FRAMEWORK

The major concepts and their relationships that provided a rationale for this study included the following: (a) electrophysiologic effects of electric current on myocardial cells, (b) increased plasma levels of intracellular enzymes indicative of cell damage, and (c) factors influencing the amount of electric current that actually reaches myocardial tissue. Each of these concepts will be examined to show their relevance to the research questions put forth in this study.

Direct current countershock is not an innocuous procedure. DC-countershock can cause physiologic changes in myocardial cells in three major areas: (a) dysrhythmia production (atrioventricular blocks, ventricular fibrillation), (b) hemodynamic compromise (hypotension, tachypnea, tachycardia), and (c) necrosis of myocardial tissue (Tacker & Geddes, 1980). The first two effects are well documented in the literature with human and animal models (Braunwald, 1980; Crampton, 1980; Ewy, 1983; Goldberger, 1982; Turner & Towers,

1965). The third effect, myocardial tissue necrosis, is the focus of this research project. Myocardial tissue necrosis has been reported primarily on animal models sacrificed soon after countershock treatment and on a few human subjects at time of autopsy (Dahl et al., 1974; DiCola et al., 1976; Ewy, 1983, Lerman et al., 1984).

Electric shock causes momentary depolarization of a majority of heart fibers thereby terminating ectopic dysrhythmias (Resnekov, 1975). Following countershock, action potentials progressively decrease in amplitude and duration and are accompanied by an increased failure of membrane repolarization (Jones, J., Lepeshkin, Jones, R., & Rush, 1975). Electrical damage to myocardial cells is seen microscopically as disruptions of mitochondrial cristae with deposition of electron dense mitochondrial particles, swelling of the endoplasmic reticulum and disorganization of the Z-bands in the myofibrils. These changes are explained by a substantially increased intracellular calcium concentration following the shock which also disrupts cellular respiration (Jones, J., Lepeschkin et al., 1975; Jones, J., Paull, Proskauer, Jones, R., Lepeschkin, & Rush, 1975; Karch & Billingham, 1984; Ventriglia & Hamilton, 1983).

Electric current may adversely influence coronary blood flow leading to ischemic changes in the involved tissue. Coronary blood flow is reduced following countershock due to the following: (a) loss of normal autonomic control of coronary arterial tone, (b) acute contracture of myocardial cells from cholinergic stimulation, and (c) reduced inflow of oxygenated blood resulting from electrically enhanced stimulation of coronary alpha receptors. Prolonged interruption of coronary blood flow results in ischemia which, in turn, disrupts cellular metabolism. Sodium pumps are "turned-off," cations and water influx the cell, the cell swells and finally the cell membrane ruptures (Andersen & Reiser, 1983; Crampton, 1980).

When a cell membrane is disrupted, intracellular contents, including enzymes, are liberated into the circulation. Two enzymes of particular importance to the detection of myocardial cell injury are creatine kinase (CK) and lactic dehydrogenase (LDH). Both CK and LDH are normally found in skeletal, myocardial and brain tissue, however, fractionation of these enzymes will identify their specific source. Plasma levels of CK isoenzymes (CKMB) and LDH isoenzymes (LDH1) are most helpful in confirming the diagnosis of myocardial cell injury (Braunwald, 1980; Crampton, 1980; Ewy, 1983; Goldberger, 1982).

Finally, it is the current flow between the electrodes that actually depolarizes the myocardial cells and abolishes chaotic dysrhythmias. This current flow can be influenced by two factors: (a) the amount of energy delivered, or shock strength (Geddes, Tacker, Rosenborough, Moore & Cabler, 1974; Patton, Allen, & Pantridge, 1984; Tacker, Galioto, Giuliani, Geddes, & McNamara, 1974; Tacker, Guinn, Geddes, Bourland, & Krompani, 1978), and (b) transthoracic resistance (Jones, V., Charbonnier, & Long, 1981; Kerber & Hoyt, 1978; Kerber, Kouba, Martins, Kelly, Low, Hoyt, Ferguson, Bailey, Bennett, & Charbonnier, 1984; Tacker & Geddes, 1980).

The amount of energy delivered to the patient is traditionally determined by the attending physician and is based on his/her previous experience and individual patient history. Transthoracic resistance (TTR), however, is influenced by several factors. TTR is increased in people of large body weight/size (Geddes et al., 1974; Kerber & Hoyt, 1978), and people with large chest circumference either due to skeletal deformities or during full inspiration (Ewy, 1983; Jones, V. et al., 1981; Kerber, Grayzel, Hoyt, Marcus, & Kennedy, 1981). The underlying assumption with TTR is that the larger the resistance, the more energy will be required to defibrillate the heart.

TTR can be decreased by the following methods:

(a) using low resistance electroconductive jellies or pads between the skin and electrodes (Tacker & Geddes, 1980; Tacker & Paris, 1983), (b) using large electrodes, i.e., 8-10 centimeters in diameter, and (c) placing electrodes in an anterior/posterior position on the thorax (Kerber, 1984; Kerber, Martins, Kelly, Ferguson, Kouba, Jensen, Newman, Park, Kieso, & Melton, 1984). Delivering successive countershocks 1 to 3 minutes apart can also decrease TTR (Chambers, Miles, & Stratbucker, 1977; Kerber et al., 1981), as can delivering countershock during end-expiration when transthoracic circumference is at a minimum (Crampton, 1980; Ewy, 1983).

With the previous concepts in mind, it seems reasonable to expect that DC-countershock might injure myocardial cells and that elevated plasma levels of intramyocardial enzymes (CK, LDH) could indicate whether such electrically promoted damage had occurred. Further, the same dose of energy could have different effects on tissues depending on the transthoracic resistance encountered. Finally, it follows that the higher the shock strength delivered, the greater the chance for electrically induced cell damage and cell membrane rupture leading to the liberation of enzymes into the blood. These concepts provided the conceptual

framework for this study, the goal of which was to determine if a relationship existed between direct current countershock and myocardial damage.

CHAPTER III

REVIEW OF RELATED LITERATURE

The majority of reviewed research on the relationship between myocardial damage and DC-countershock has been on animals. Direct open chest application of electrodes as well as closed chest application methods have been reported. Research on human subjects record closed transthoracic shock application using various locations of electrodes, varying shock strengths, electrode paddle size, and impedance factors. Animal and human research will be presented separately and chronologically in this literature review.

Animal Studies

Researchers studied 66 mongrel dogs to determine the transthoracic resistance to DC-countershock. Electrodes were applied to the thoracic skin and shocks of 400 watt/seconds were delivered. Thirty-nine dogs received 10 consecutive countershocks at 400 watt/seconds with 8 cm paddles. Fourteen dogs received the same wattage with 4.5 cm paddles. The remaining 13 dogs received 400 watt/seconds with 12.8 cm paddles. The time between the 10 consecutive shocks varied at 15

seconds, 1 minute and 3 minutes. Each group received the same treatments at the 3 different times.

The animals were sacrificed 3-14 days after the DC-countershock. Evidence of myocardial necrosis was evident in most cases; the least amount of damage was found in the dogs who received current through the larger paddles. There was no reported injury to the skin, lung tissue or epicardium in any of the dogs. Pectoral and intercostal muscle necrosis was most severe in the group treated with the 4.5 cm paddles. Conclusions drawn by these researchers were:

(a) increased intervals between shocks decreased the severity of the lesions suggesting that some types of cell derangement can be partially corrected in several minutes, and (b) increased paddle diameter decreased the severity of damage while spreading it more superficially (Dahl, Ewy, Lynch, & Warner, 1975; Warner, Dahl & Ewy, 1975).

Ehsani et al. (1976) studied the effects of DC-countershock on serum creatine kinase isoenzyme activity. In their descriptive study, 11 anesthetized mongrel dogs were subjected to 10 consecutive 240 watt/second countershocks at 15 second intervals using 8 cm electrode paddles. Serum creatine kinase (CK) samples were obtained immediately after countershock. All 11 dogs

demonstrated a prompt rise in total serum CK activity. Isoenzyme determinations of these same samples showed the bulk of CK rise due to the MM fraction indicating skeletal muscle damage, not myocardial (MB) liberation of the enzyme. Six of the 11 dogs with ST segment elevation immediately post-countershock showed some CKMB liberation averaging 52 ± 6 mIU/ml. All dogs were sacrificed 4 days after countershock and those with increased ST segments on electrocardiogram demonstrated gross and microscopic changes indicative of myocardial necrosis. Results were expressed as percentages and averages among the groups of dogs.

Researchers examined the cardiac damage produced by DC-countershock over a dose range of 10-90 watt/seconds applied directly to the heart in 26 mongrel dogs (Doherty, McLoughlin, Billingham, Kernoff, Goris, & Harrison, 1979). Electrodes were applied after open chest thoracotomy directly to the pericardial sac. The threshold of injury was noted at 30 watt/seconds with histological damage to the myocytes confined to the epicardial layer. Necrosis of myocardial and skeletal muscle was demonstrated using technetium-99 pyrophosphate scanning. Serum creatine kinase was not found to be elevated in any of the dogs; all dogs were sacrificed 10 minutes after completion of technetium-99

scanning and the hearts removed. Necrosis of tissue was present in all dogs on histologic examination. With low shock levels (<50 watt/seconds) damage occurred primarily in the epicardial layers; dosages greater than 50 watt/seconds demonstrated damage to the endocardial layers as well ($p<0.001$). The use of larger paddles (6 and 7.5 cm) demonstrated primarily diffuse epicardial damage ($p<.01$) with the smaller 2 cm paddles causing concentrated damage to the endocardial tissue. The use of different sized paddles did not appear to affect the total number of damaged cells; the larger paddles only spread the current over a larger area causing more superficial damage. The researchers concluded that short time intervals between shocks can increase cell injury. They hypothesized that this was due to the cumulative effect of the thermal component of the damage.

Tacker, Van Vleet, and Geddes (1979) attempted to quantify the safety margin between defibrillation threshold and damage threshold to the myocardium. These researchers studied electrocardiographic (ECG) changes and serum cardiac (CKMB) isoenzyme levels in 56 male and female mongrel dogs. The dogs were placed into 8 groups depending on the shock strength they received, from 1-20 A/kg of body weight. One group of dogs served as a control group and received no shock

treatment. Shocks were applied using 10 cm paddles to the thorax in non-fibrillating hearts. Serum CK and LDH activity was measured 2 minutes before countershock and every 30 minutes for 2 hours after countershock. Eight of the 56 dogs died immediately after countershock with 12 or more A/kg, all from ventricular dysrhythmias. In the group of dogs that received 9 or more A/kg, the activity of total CK and LDH was increased and was greatest in the groups given the larger shocks. Isoenzymes of CK and LDH were also noted to be increased with increased shock strength.

Koning, Veefkind, and Schneider (1980) studied the effect of countershock on isolated Langendorff-perfused rabbit hearts. The researchers wanted to quantify the critical countershock dose to prevent myocardial damage by either too high energy shock or the necessity of repeated shocks in the case of too low energy shocks. Five bastard rabbit and 5 strain rabbit hearts were isolated and perfused by the Langendorff method. All hearts received the same treatments. Perfusion pressure, PO_2 of the perfusate, temperature, preload and afterload were all controlled in this experiment. The researchers avoided the use of anesthetics to reduce the amount of neuronal influence on the results of the study. Synchronized countershocks between 15-70

watt/seconds were delivered with paddles placed directly on the ventricles. 3.1 cm and 11.0 cm paddles were used to test the influence of paddle size on cardiac damage.

The findings of this study were consistent with previous results specifically in relation to rise in CKMB; significant at 30 watt/seconds ($p < .005$). Increased dysrhythmias, changes in cellular electrical equilibrium and an increased amount of necrotic tissue with greater levels of countershock were reported. Results were expressed as means and the standard error of the mean. It was not clear in the text of this report, which rabbit hearts comprised the control group. In the figures present in the text, values are present for the control group, however, it is unclear what procedures, if any, were performed on this group. The reader questions the purpose of the two groups of rabbits in this study and the vagueness of the composition and treatment of the control group.

In summary, experimental methods utilizing animal models in the laboratory are varied and inconsistent. However, the reported results tend to support the notions that: (a) myocardial damage, evidenced by necrosis on gross and microscopic autopsy examination, is increased with increasing shock strength, (b) paddle size affects the amount and severity of tissue damaged

by the shock, and (c) increasing the time interval between shocks may decrease the thermal component associated with skeletal and myocardial tissue injury.

Human Studies

Human research studies are limited because gross and histologic examinations of the heart tissue following DC-countershock are rarely made except in the event of death. The review of literature does not reveal consistent positive or negative relationships between DC-countershock and enzyme release of CK and LDH. The following studies utilized closed chest countershock techniques and either total serum CK and LDH levels, or isoenzyme determinations of CK and LDH, or both.

Ehsani et al. (1976) measured total and isoenzyme levels of CK in 30 patients receiving DC-countershock. The mean age was 54 years and patients were treated for various tachyarrhythmias without suspected myocardial infarction. Twenty-three patients had atrial fibrillation, 5 had atrial flutter, and 2 had paroxysmal atrial tachycardia. Patients received diazepam or sodium methohexitol as premedication. Anterior/posterior paddles were used with the total cumulative energy delivered between 40-750 watt/seconds. Serum total and isoenzyme determinations were obtained in

all patients and from 25 control patients not receiving countershock, determined by the researchers to be in good general health. Blood samples in the experimental group were obtained immediately after and serially for 24 hours after countershock. Peak total CK activity occurred within the first 4 hours. The results showed that normal control subjects had virtually no total CK or CKMB activity in the serum. Of the 30 patients receiving electrical countershock, 15 had elevated postshock total CK activity (>50 mIU/ml), peaking within 4 hours and gradually declining. However, isoenzyme studies revealed that the increased CK activity was due to isoenzymes in skeletal muscle (CKMM) rather than from myocardial tissue. Only 2 patients had elevated myocardial isoenzyme activity and they both had received more than 425 cumulative watt/seconds. Results were presented in tables representing the averages from the four serial serum determinations. These researchers concluded that DC-countershock generally does not produce a considerable amount of myocardial damage in humans but needs to be utilized with caution until further research has been accomplished. One limitation noted in this study was the lack of pre-shock CK levels from the subjects. Serum was drawn only after countershock so the rise from normal for each individual subject was unknown.

A large study with 94 patients was performed in Poland by Mandecki et al. (1970). All patients were treated with DC-countershock; there was no control group. The age of the patients varied between 22 and 73 years. Valvular heart disease was present in 51 patients, atherosclerotic heart disease in 38, lone fibrillation in 4, and treated thyrotoxicosis in one patient. Seventy cases presented for conversion of atrial fibrillation, 19 with atrial flutter, 4 with supraventricular paroxysmal tachycardia, and 1 case of paroxysmal ventricular tachycardia. Patients were divided into 2 groups, 1 group receiving 1 counter-shock and the other group receiving more than 1 counter-shock. Serum samples for CK were measured immediately before countershock and serially at 1.5 hours and 24 hours after countershock. Cumulative delivered energy ranged from 150-400 watt/seconds. These researchers indicated a significant difference ($p < .05$) between pre- and post-cardioversion total serum CK levels using the "paired experiment method" (a modification of the Students t-test). The investigators proposed the rise due to skeletal muscle isoenzyme but did not utilize isoenzyme tests to validate their claim.

Forssell et al. (1975) studied the effects of DC-countershock on 12 patients without evidence of acute

myocardial infarction, following conversion of supra-ventricular tachyarrhythmias. The patients in the study ranged from 46-70 years. All patients except 1 were treated with digoxin which was discontinued 3 days prior to elective DC-countershock. Oral anti-coagulant therapy was initiated on all patients 3 weeks prior to conversion. Quinidine, an antiarrhythmic drug, was begun 24 hours before conversion. Intravenous diazepam was given as premedication to the procedure. Anterior/posterior paddle placement was utilized, the size of the paddles was not reported. Cumulative shock strength varied between 100-650 watt/seconds. Serum CK levels were drawn 30 minutes prior to countershock and serially immediately after, at 1 hour and 2-4 hours after countershock for a total of 18-51 hours. The total CK rise showed no correlation with either the discharged energy or the number of DC-countershocks. The exact statistical tests utilized in this study were not reported. The only table showed CK levels over time in hours. These investigators mentioned the usefulness of isoenzyme determinations of CK, however, they did not do isoenzyme studies on elevated total serum levels of CK.

Reiffel et al. (1978) reported the effect of DC-cardioversion on CK and LDH isoenzymes in 18 patients

without known cardiac ischemia who underwent elective countershock for conversion of atrial dysrhythmias. Fourteen patients had right or left ventricular enlargement, 11 patients had right or left ventricular failure, 3 patients had arteriosclerotic cardiovascular disease. Serum CK, LDH total, and isoenzyme samples were drawn immediately before, immediately after and serially at 6, 12 and 24 hour intervals following DC-countershock. Intravenous diazepam was used in all patients as premedication. Anterior/posterior paddle placement was used without mention of paddle size. Cumulative delivered energy ranged from 10-600 cumulative watt/seconds. Ten patients received less than 100 cumulative watt/seconds without significant changes in the serum enzymes. Seven patients received cumulative doses greater than 100 watt/seconds of energy and there was no substantial change in total or isoenzyme levels of CK or LDH in these patients. In one patient receiving 600 watt/seconds cumulative dose, total CK was elevated, but the isoenzymes were not elevated. These researchers concluded that occasional and modest CK elevations may follow DC-countershock if high levels of countershock energy are required. They suggested that increases in myocardial enzyme levels associated with acute dysrhythmias probably indicate

cardiac ischemia rather than myocardial damage secondary to DC-countershock.

In an abstract from Pfisterer et al. (1978), the diagnostic value of the myocardium specific isoenzyme CK was presented. In all 12 patients there were elevations of total CK as well as elevated isoenzyme fractions of CK (CKMB) following countershock. Due to the space limitations of the abstract and the non-availability of an English translation of this German report, the exact methods of the experiment were unavailable to this investigator, therefore a critical review of the project was not possible.

Conclusions From Literature Review

In summary, several limitations in the above-mentioned studies on humans exist and the results are more tenuous than those experiments carried out on animal subjects. Isoenzyme determinations of CK and LDH were frequently alluded to as being highly sensitive and specific for myocardial damage. However, only a few researchers actually utilized isoenzymes as a dependent variable in their studies. Only one investigator addressed the effects of cumulative total energy on the isoenzyme levels in humans which may contribute to the thermal component of tissue damage

(Reiffel et al., 1978). Paddle size and location varied between studies as well as the conductive medium used as an interface between the paddles and the skin.

Recent or concurrent myocardial infarction as an intervening variable was considered, but not consistently, and the relationship of ischemia to enzyme rise was rarely addressed. Patients were counter-shocked for both atrial and ventricular dysrhythmias within the same study, although it has been demonstrated that ventricular dysrhythmias lead to ischemia much more rapidly than atrial dysrhythmias. Pre-existing ischemia could have had an effect on the results of the study. Body weight, body surface area, gender, and age were not consistently reported.

The use of anesthetics was not consistently reported. Diazepam was most frequently used as pre-medication but only one study mentioned the effects of anesthetics on neuronal influence of the heart (Konig et al., 1980).

Previous studies, while alluding to DC-counter-shock induced myocardial damage, did not consistently address those variables that may have confounded the results of their study. Thus, the present study attempted to consider body surface area, transthoracic

resistance, paddle placement, and cumulative watt/seconds and the effect these variables may have had on postshock CK and LDH enzyme levels. Patients with known or suspected myocardial infarction were not included in order to decrease the effect pre-existing ischemia may have had on the results of the study.

CHAPTER IV

RESEARCH QUESTIONS AND DEFINITION OF TERMS

Research Questions

Two research questions were addressed in this study. They were:

1. Is there a significant difference between preshock and mean postshock serum creatine kinase enzyme levels?
2. Is there a significant difference between preshock and mean postshock lactic dehydrogenase enzyme levels?

Definition of Terms

Creatine kinase--intracellular enzyme found in brain, skeletal muscle, and myocardial cells. Acts as a catalyst in the transfer of a phosphate group from phosphocreatine to adenosine diphosphate, producing creatine and adenosine triphosphate. Total serum CK represents the total amount of CK that circulates in the bloodstream at any given moment. Normal range for male/female is 45-235 u/l. CKMM

represents CK specifically from skeletal muscle. Normal is <50% activity. CKMB represents CK specifically from myocardial tissue. Normal is <6% activity.

Countershock--delivery of electrical current via a Life-Pak 6 defibrillator capacitor to terminate dysrhythmias.

Hemodynamically significant dysrhythmias--disturbances in rhythm resulting in any of the following: hypotension (systolic <100 mmHg), tachypnea (>28/minute), a decrease in sensorium or decrease in orientation to person, place or time.

Lactic dehydrogenase--intracellular enzyme widespread in tissues. Catalyzes the intraconversion of lactate and pyruvate. Total serum LDH represents the total amount of LDH circulating in the blood at any given moment. Normal range for male/female is 109-193 u/l. LDH1 represents the isoenzyme found in myocardial tissue. Normal range is 18-33% activity.

Myocardial damage--inferred by isoenzyme levels of CKMB and/or LDH1 in the serum above normal levels (see definitions of creatine kinase and lactic dehydrogenase for normal values).

Supraventricular dysrhythmias--alterations in normal cardiac impulse formation leading to rhythms of atrial fibrillation, atrial flutter, and supraventricular tachycardias.

Termination of dysrhythmias--occurs when countershock disrupts a chaotic ectopic rhythm allowing the sinus node to resume conduction.

Transthoracic resistance--the opposition offered by a body to the passage through it of an electric current. There are three determinants of transthoracic resistance:

Body surface area--calculated from height and weight using DuBois Body Surface Chart (Palmer, 1982, p. 141).

Number of shocks received--transthoracic resistance decreases with subsequent shocks.

Transthoracic circumference--measurement of chest circumference at the fifth intercostal space just below the nipple line not to include breast tissue.

CHAPTER V

METHODOLOGY

Study Design

The design of this study was prospective, quasi-experimental. The independent variable was application of direct current transthoracic countershock; the dependent variable was myocardial injury evidenced by the presence of myocardial isoenzymes of CK and/or LDH in the serum following countershock. In this study, control was achieved by using individual preshock enzyme values of CK and LDH as comparison to individual mean postshock enzyme values of CK and LDH. Study subjects were not randomly selected due to the specificity of the phenomenon under study. All subjects received the same pre- and post-treatment measurements except for a subgroup of four patients who, after two unsuccessful conversion attempts, received intravenous procainamide, an antiarrhythmic agent. Also, these four patients received a third shock 4 hours after the infusion of procainamide was completed. The application of countershock varied only in cumulative watt/seconds received, time between

countershocks and the number of countershocks required to terminate the dysrhythmias.

Electrode placement was consistent for all patients in the study. The type of premedication varied depending on the preference of the attending anesthetist.

Description of the Major Variables

Independent Variable

The independent variable in this study was the delivery of direct current transthoracic countershock. Dosages of electric current delivered to the patient were recorded in watt/seconds. Individual doses were determined by the physician and dialed into the Life-Pak 6 defibrillator unit prior to discharging the shock. Cumulative doses received per patient ranged from 50-1100 watt/seconds depending on the refractoriness of the cardiac rhythm. Corotin TM electrode pads were used as an interface medium between the defibrillator capacitor and the chest. One pad was placed at the apex of the heart, the other pad was placed between the scapula and centered over the spine. The electrodes were attached to an R2 TM unit per manufacturers' recommendations. The actual discharge of electricity from the capacitor was through

the R2 TM unit and then through the Cortin TM electrode pads.

Dependent Variable

The dependent variable in this study was injury to myocardial tissue inferred by the presence of cardiac specific isoenzymes of creatine kinase and lactic dehydrogenase in the blood. Blood samples were first tested for total CK and LDH enzyme content and reported in units per liter (u/l) of protein. The protocol for the hospital laboratory was that total serum enzymes of CK and LDH would be fractionated only if the values were greater than normal. Therefore, isoenzyme fractionation was performed on CK values greater than 235 u/l and LDH values greater than 193 u/l. The underlying assumption on the part of the laboratory personnel was that fractionation techniques on normal levels of CK and LDH would not yield any information as to the area of release.

Extraneous Variables

Extraneous variables examined in this study included the following: (a) transthoracic resistance, (b) procainamide infusion, and (c) unknown (silent) myocardial infarction. Transthoracic resistance can be calculated from chest circumference, body surface

area, and chest wall thickness (Jones, V. et al., 1981; Kerber & Hoyt, 1978). Chest circumference was measured on all subjects and body surface area was calculated from height and weight using a DuBois Body Surface Chart (Palmer, 1982). Chest wall thickness was not measurable within the scope of this study, but was recognized as another component of transthoracic resistance.

The number of shocks received, cumulative watt/seconds, body surface area, and transthoracic circumference all influence transthoracic resistance. A correlation matrix was done to determine if there was any relationship among these determinants of transthoracic resistance and the dependent variable, myocardial injury.

Intravenous procainamide, an anti-arrhythmic drug, was used on four patients to prevent reversion to supraventricular dysrhythmias following countershock. The patients receiving this drug, after two countershock attempts, received a third shock 4 hours after the infusion was complete. These subjects were included in the initial data analysis, but then were examined as a subgroup of the sample.

It is well known that myocardial infarction leads to myocardial cell necrosis and liberation of isoenzymes

into the blood (Braunwald, 1980; Goldberger, 1982). In addition, myocardial infarction can precipitate dysrhythmias (Tacker & Geddes, 1980). For these reasons, patients with overt signs or symptoms of acute myocardial infarction were not included in the study. However, "silent" myocardial infarctions do occur without overt signs or symptoms, the presence of which was considered to be an uncontrolled extraneous variable.

The Sample

The sample population consisted of all patients admitted to an 80-bed military hospital for DC-countershock for conversion of supraventricular dysrhythmias during the period of 30 September 1984 through 28 February 1986. A convenience, non-probability sample was used due to the inherent nature of the phenomenon under study.

Specific criteria for inclusion into the study were (a) presence of supraventricular dysrhythmia refractory to pharmacologic therapy, (b) absence of recent myocardial infarction, (c) first time countershock, (d) physician decision to cardiovert, and (e) agreement by the patient to participate in the study and permit a chart review. No patient losses

were anticipated. The attending physician informed each patient on the potential risks and benefits of countershock after which a standard Air Force consent form (SF 522) was signed (see Appendix). Each patient participated only once in the study although some did require repeat countershock for recurrent dysrhythmias. Twelve males and three females age 33-79 (mean age 59 years, SD $\pm$ 10 years) met the criteria and agreed to participate in the study.

Of the 15 patients included in the study, 8 had atrial fibrillation, 6 had atrial flutter and 1 had supraventricular tachycardia. Seven patients required 3 consecutive countershocks to abolish the dysrhythmia, 3 patients required 2 shocks each, and 5 patients required 1 shock each. All shocks were administered within 1 to 2 minutes of each other except in the 4 patients who received 2 consecutive countershocks followed by a third countershock 4 hours after infusion of procainamide. Data on demographic and treatment variables are summarized in Table 1.

Data Collection

Body measurements of height, weight, and trans-thoracic circumference were obtained on all patients at the time of inclusion into the study. Prior to the

Table 1

Demographic and Treatment Variables of Clients

Client Number	Age	Sex	Type of Dysrhythmia	Number of Shocks Received	Cumulative Watt/Seconds/Received	IV Procainamide Received
1	55	male	atrial flutter	2	300	no
2	79	female	atrial fibrillation	2	400	no
3	65	male	atrial flutter	3	350	no
4	61	male	atrial flutter	1	100	no
5	64	male	supraventricular tachycardia	1	100	no
6	33	male	atrial fibrillation	2	600	no
7	71	male	atrial flutter	1	100	no
8	61	female	atrial fibrillation	3	350	no
9	46	female	atrial flutter	1	100	no
10	52	male	atrial flutter	1	30	no
11	62	male	atrial fibrillation	3	350	no
12	62	male	atrial fibrillation	3	1000	yes
13	54	male	atrial fibrillation	3	1000	yes
14	65	male	atrial fibrillation	4	1100	yes
15	51	male	atrial fibrillation	3	800	yes

data collection period, a pilot study was done to test interrater reliability among the staff of the Coronary Care Unit.

The pilot study was conducted as follows: the researcher measured height, weight, and transthoracic circumference on a volunteer staff member. Weight was recorded to the nearest pound and height to the nearest inch using a "Selecto" weighted scale. Transthoracic circumference was measured at the fourth intercostal space, perpendicular to the spine (not including breast tissue), using a plastic seamstress tape, and recorded to the nearest centimeter. Individually, eight staff members performed these same measurements on the same volunteer. Obtained results were compared to the researcher's results. There was 100% agreement on these measurements among all eight staff members and the researcher. Thus, an interrater reliability of 1.0 was established. The researcher was not present for actual patient measurements and assumed that patient measurements of height, weight, and transthoracic circumference were obtained in the same manner as in the pilot study.

Samples of venous blood for CK and LDH were drawn immediately prior to, and 6, 12, and 24 hours after the delivery of DC-countershock. Samples were

immediately spun down by the laboratory personnel and refrigerated until assays were done. Total enzyme counts of CK and LDH were done on all samples; iso-enzyme fractionation was done on elevated samples to determine the area of enzyme release. Three patients were discharged prior to the 24 hour postshock blood sampling. Their early discharge resulted in three missing data points.

Pregelled, self-adhesive, Corotin TM electrode pads were placed on the thorax, anteriorally, at the apex of the heart, and posteriorally, between the scapula. The electrode wires were then attached to an R-2 TM unit and directly to a Life-Pak 6 TM defibrillator.

Anesthetics utilized in this study were diazepam, sodium pentothal, and brevital or a combination of diazepam/sodium pentothal, diazepam/brevital or atropine/sodium pentothal. Anesthetics were introduced intravenously via a peripheral line immediately prior to countershock.

Clients received successive countershocks 1 to 2 minutes apart except for a subgroup of four patients. These four patients reverted to their preshock rhythms immediately following the first and second countershocks. Prior to a third shock, these patients

received intravenous procainamide, an antiarrhythmic drug, in an attempt to prevent further postshock reversion episodes.

The above standardized countershock protocol had been established by the Chief of Cardiology prior to the inception of this study. Transthoracic circumference was the only additional measurement included in the existing protocol for the purposes of this study.

Instrumentation

A 1983 model Life-Pak 6 TM defibrillator was used on all subjects in the study. The unit was calibrated monthly by biomedical specialists to ensure dialed energy levels were consistent through a 50 ohm resistance.

A-Gent TM clinical chemistry reagents were used in determining the presence of CK and LDH in obtained serum samples. Reliability of the A-Gent TM CK-NAC test was listed by the manufacturer as a linear regression of $Y=1.22 X=2.8$ with a correlation coefficient of 0.99. The reliability of A-Gent TM LDH test was listed as a linear regression of $Y=0.84 X=1.05$ with a correlation coefficient of 0.987. The hospital laboratory measuring the specimens reported a 98.8% reliability rate in testing procedures for both tests.

Data Analysis

Review of the data collected revealed that 4 of the 15 subjects had received countershock in split doses, 2 shocks in rapid succession and 1 shock 4 hours after an intravenous infusion of procainamide. The data from this group of 4 subjects were analyzed separately, after a demographic profile was obtained for the total sample. Group 1 consisted of 11 patients not receiving IV procainamide; Group 2 consisted of 4 patients who had received IV procainamide.

Three statistical methods were used in data analysis, the Sign Test, the paired samples t-test, and Pearson-product moment correlation. Analysis was performed on the IBM PCXT computer utilizing the Statistical Package for the Social Sciences (SPSS). In statistical analysis, a p value $< .05$ was considered significant.

The Sign Test is an analog of the paired t-test and is a useful non-parametric test to rank a pair of scores in relation to one another on the same subject (Goldstone, 1983). In order to reduce individual variation, the mean of the three postshock enzyme values was calculated on each individual patient and compared to the preshock value of each enzyme. The Sign Test

was used to answer both research questions: (a) "Is there a significant difference between preshock and mean postshock serum creatine kinase enzyme levels?" and (b) "Is there a significant difference between preshock and mean postshock lactic dehydrogenase enzyme levels?" The Sign Test explores whether a statistically significant difference exists between two paired samples. The Sign Test considers the sign only, not the magnitude of difference between paired samples (Bahn, 1972). A plus (+) sign represented postshock values higher than preshock values and a minus (-) sign represented postshock values less than preshock values. Zero difference (0) values were not considered in data analysis. Probability values were calculated using the Bernouli formula (a binomial formula), which assumes a 50% probability that the mean postshock value would be greater than the preshock value (Bahn, 1972). The only underlying assumption of this test is that the variable under consideration has a continuous distribution (Siegel, 1957).

The paired samples t-test is a useful parametric inferential statistical test when there are two dependent variable measures from the same subject. This test was used to answer both research questions

regarding the difference between pre-shock values and mean postshock values of the individual enzymes. The paired samples t-test considers the sign and the magnitude of a relationship between samples (Bahn, 1972). This test allows one to take into consideration the individual variation in dependent samples and requires that data be continuous and at the interval level (Polit & Hungler, 1983). One-tailed probability was used because a directional hypothesis was assumed.

A correlation matrix was constructed to explore relationships among extraneous variables and the dependent measures under study. Pearson's product moment correlation coefficients were computed on the following variables: height, weight, body surface area, thoracic circumference, number of times shocked, anesthetic agent, and rhythm. These extraneous variables were correlated with indicators of the dependent variable, specifically, mean postshock CK levels and mean postshock LDH levels. A significant correlation (for this study, $p < .05$), does not necessarily indicate causation, only that two variables tend to be associated (Duncan, Knapp, & Miller, 1983; Kviz & Knafl, 1980).

CHAPTER VI

RESULTS

The objective of this study was to determine if there was a relationship between the delivery of DC-countershock and myocardial injury as inferred by the presence of cardiac specific isoenzymes in the blood following countershock. The Sign Test and the paired samples t-test were used to answer the two research questions posed by this study: (a) "Is there a significant difference between preshock and mean postshock serum creatine kinase enzyme levels?" and (b) "Is there a significant difference between preshock and mean postshock lactic dehydrogenase enzyme levels?"

The Sign Test

Preshock and mean postshock values for CK were compared separately from the preshock and mean postshock values for LDH. Data from Groups 1 and 2 were analyzed separately.

Group 1 (No Intravenous Procainamide)

Group 1 (n=11) had six patients who had postshock increases over preshock CK values, four patients who

had decreases from preshock CK values and one patient who showed no change in CK value. Five patients had postshock increases over preshock LDH values, and six patients had decreases from preshock LDH values. It is noteworthy that fractionation of supernormal CK samples in one patient showed increased CK isoenzyme levels of CKMM, indicating skeletal muscle damage. Results are displayed in Tables 2 and 3.

Group 2 (Intravenous
Procainamide)

In Group 2 (n=4), the postshock plasma CK levels of all four patients were elevated when compared to preshock levels. Three of the four patients had elevated plasma levels of LDH following countershock, and one patient had a decrease in plasma LDH following countershock. There was no significant difference in this group between preshock and mean postshock values for CK ($p=.06$) or LDH ($p=.25$). Although all four patients had increased postshock CK compared to preshock CK, the sample size was too small to show statistical significance. Tables 4 and 5 display these results.

Paired Samples t-test

The paired samples t-test was used to explore the research questions as stated above. Preshock values of

Table 2

Sign Test, Group 1: Comparision of Preshock Creatine Kinase Values to Mean Postshock Creatine Kinase Values (no procainamide) (n = 11)

Client Number	Preshock CK u/l	Mean Postshock CK u/l	Sign
1	58	137	(+)
2	52	55	(+)
3	102	343	(+)
4	23	16	(-)
5	26	26	(0)
6	119	836*	(+)
7	278	193	(-)
8	6	85	(+)
9	220	143	(-)
10	180	162	(-)
11	24	101	(+)

p = .21 NS

*isoenzyme from skeletal muscle (CKMM)

Table 3

Sign Test, Group 1: Comparision of Preshock Lactic Dehydrogenase Values to Mean Postshock Lactic Dehydrogenase Values (no proclainamide) (n=11)

Client Number	Preshock LDH u/l	Mean Postshock LD u/l	Sign
1	245	181	(-)
2	169	151	(-)
3	114	96	(-)
4	159	167	(-)
5	201	244	(+)
6	302	221	(-)
7	220	224	(+)
8	90	135	(+)
9	471	325	(-)
10	160	207	(+)
11	123	264	(+)

p = .23 NS

Table 4

Sign Test, Group 2: Comparision of Preshock Creatine Kinase
Values to Mean Postshock Creatine Kinase Values (IV
procainamide) (n=4)

Client Number	Preshock CK u/l	Mean Postshock CK u/l	Sign
12	73	470*	(+)
13	340	1537*	(+)
14	133	9724*	(+)
15	111	6264*	(+)

p = .06 NS

*isoenzymes from skeletal muscle (CKMM)

Table 5

Sign Test, Group 2: Comparison of Preshock Lactic Dehydrogenase Values to Mean Postshock Lactic Dehydrogenase Values (IV procainamide) (n=4)

Client Number	Preshock LDH u/l	Mean Postshock LDH u/l	Sign
12	235	225	(-)
13	234	265	(+)
14	220	448	(+)
15	158	240	(+)

p = .25 NS

LDH were compared to mean postshock values of LDH separately for Group 1 and Group 2. One-tailed probability was computed with the degrees of freedom at $n=1$ (Bahn, 1972). Actual numerical values for preshock and postshock enzyme values may be found in Tables 2, 3, 4, and 5 and will not be repeated here.

Group 1 (No Intravenous
Procainamide)

In Group 1 ($n=11$), there was no statistically significant difference between preshock and mean postshock values of CK (t -value = 1.35, $df = 10$) or LDH (t -value = .15, $df = 10$).

Group 2 (Intravenous
Procainamide)

In Group 2 ($n=4$), there was no statistically significant difference between preshock and mean postshock values of CK (t -value = 2.00, $df = 10$) or LDH (t -value = 1.59, $df = 10$).

Pearson's Product Moment Correlation
Between Extraneous Variables
and Dependent Variables

Pearson's product moment correlation coefficients were computed between extraneous variables and measures of the dependent variable. Extraneous variables included height, weight, body surface area, thoracic

circumference, number of times shocked, and cumulative watt/seconds. Measures of the dependent variable included mean postshock CK and mean postshock LDH. Groups 1 and 2 were analyzed separately.

Group 1 (No Intravenous
Procainamide)

For Group 1 (n=11), statistically significant correlations were only seen between cumulative watt/seconds and mean postshock CK level ($r = .6352$, $p = .036$). All correlations between extraneous and dependent variables are displayed in Table 6.

Group 2 (Intravenous
Procainamide)

Group 2 (n=4), showed no statistically significant correlations between the extraneous variables and the dependent variables (Table 7).

Mean postshock LDH was negatively correlated with all extraneous variables in Group 1 and with weight and transthoracic circumference in Group 2. The reasons for these negative correlations are unknown.

Table 6

Correlation Coefficients for Extraneous Variables and
Dependent Variables, Group 1 (n=11)

Extraneous Variables	Dependent Variables	
	Mean post-shock CK	Mean post-shock LDH
Height	$r^1_2 = .3891$ $p^2 = .237$	$r = -.1823$ $p = .592$
Weight	$r = .2561$ $p = .447$	$r = -.2335$ $p = .490$
Transthoracic Circumference	$r = .0124$ $p = .971$	$r = -.1189$ $p = .728$
Cumulative Watt/Seconds	$r = .6352$ $p = .036^3$	$r = -.3231$ $p = .332$
Number of Times Shocked	$r = .1822$ $p = .592$	$r = -.4778$ $p = .137$
Body Surface Area	$r = .3964$ $p = .227$	$r = -.2584$ $p = .443$

¹ r = correlation coefficient

² p = probability value

³ = statistically significant

Table 7

Correlation Coefficients for Extraneous Variables and
Dependent Variables, Group 2 (n=4)

Extraneous Variables	Dependent Variables	
	Mean post- shock CK	Mean post- shock LDH
Height	¹ $r_2 = .8540$ $p^2 = .146$	$r = .4586$ $p = .541$
Weight	$r = -.6958$ $p = .304$	$r = -.9120$ $p = .088$
Transthoracic Circumference	$r = -.7640$ $p = .236$	$r = -.8661$ $p = .134$
Cumulative Watt/Seconds	$r = .1045$ $p = .896$	$r = .6709$ $p = .329$
Number of Times Shocked	unable to compute	unable to compute
Body Surface Area	$r = -.2705$ $p = .729$	$r = -.7808$ $p = .219$

¹ r = correlation coefficient

² p = probability value

CHAPTER VII

CONCLUSIONS

Discussion

The purpose of this study was to determine if transthoracic DC-countershock could lead to myocardial cell damage resulting in the liberation of myocardial isoenzymes of CK and LDH into the blood. The results of this study indicated no such damage. There were no statistically significant differences between preshock values and postshock values of either enzyme. In the five patients whose total CK enzymes were above normal limits, only skeletal muscle isoenzymes of CK (CKMM) were found. Based on these findings, a conclusion can be drawn that DC-countershock does not lead to myocardial tissue damage when delivered under the conditions set forth in this study. This conclusion supports previous studies wherein isoenzyme determinations were included (Reiffel et al., 1978).

Previous work demonstrated that transthoracic resistance is influenced by body surface area, thoracic circumference and the number of shocks received. Further, transthoracic resistance plays a key role in

determining the amount of energy needed to defibrillate the heart (Jones, V. et al., 1981; Kerber & Hoyt, 1978; Kerber et al., 1984; Tacker & Geddes, 1980). A major assumption from previous studies is that more energy (cumulative watt/seconds) is needed to defibrillate a person with high transthoracic resistance. However, this assumption is not supported by this study. Neither body surface area ($r=.5270$, $p=.096$) nor thoracic circumference ($r=.4100$, $p=.210$) correlated significantly with cumulative watt/seconds needed to convert the dysrhythmia.

The amount of energy delivered (cumulative watt/seconds), showed a significant positive correlation with the mean postshock CK level for patients in Group 1 ($r=.6252$, $p=.036$). Only one patient in this group had supernormally elevated postshock CK enzyme, which was found to be from skeletal muscle (CKMM). This finding supports previous research studies (Forssell et al., 1975; Hunt & Bailie, 1968; Mandecki et al., 1970; Reiffel et al., 1978).

In four patients (Group 2), a time lapse of 4 hours occurred between the second and third countershocks. All four patients had received intravenous procainamide and received cumulative doses of energy of 800 watt/seconds or more. All four patients had supernormally

elevated CK enzymes postshock. Fractionation of these revealed that the elevation was due to skeletal muscle (CKMM) not myocardial tissue. CKMM was demonstrated in the serum of one patient receiving a cumulative energy dose of 600 watt/seconds. Previous reports have noted that increased dosages of electrical current may injure skeletal muscle and are consistent with the results of this study (Ehsani et al., 1976; Reiffel et al., 1978).

It is unclear at this time whether the skeletal muscle injury was related to the cumulative energy delivered, intravenous procainamide, or the prolonged time between consecutive countershocks. Previous research suggests that cumulative energy and the long time between any shocks cause the skeletal muscle injury after DC-countershock (Crampton, 1980; Ewy, 1983).

Nursing Implications

In ever expanding roles, registered nurses are called upon to deliver DC-countershock to patients in an attempt to abolish chaotic dysrhythmias. The results of this study should assist the nurse in educating the patient regarding the potential risks and benefits associated with DC-countershock. The unanticipated finding of skeletal muscle damage encountered after

high cumulative energy doses (greater than 600 watt/seconds) may serve as a reminder to the practitioner when ordering the energy dose for termination of dysrhythmias.

Four patients (Group 2) had evidence of CKMM in their serum following successive countershocks over a period of 4 hours. It is unclear at this time whether the liberation of CKMM enzymes was due to one or more of the following: (a) intravenous administration of procainamide after two unsuccessful countershocks prior to a third shock, (b) the time interval between successive countershocks, or (c) the cumulative watt/second received. Awareness of these possible contributors to skeletal muscle injury may assist the critical care nurse in educating the patient and the physician on the possibility of skeletal muscle injury following successive countershocks over a period of hours, especially when an intravenous antiarrhythmic drug is administered.

In this study, there was a significant positive correlation between cumulative watt/seconds received and mean postshock CK ($r=.6352$, $p=.036$). In the presence of refractory dysrhythmias, perhaps low energy doses could be used initially and gradually increased until the desired effect is achieved. If the cumulative

watt/seconds received could be minimized, perhaps the liberation of creatine kinase postshock could be minimized or even avoided.

The implications from this study not only affect nursing practice, they also affect physician practice and could set the stage for an interdisciplinary collaborative approach to research in DC-countershock. DC-countershock is common practice in critical care settings due to its availability and effectiveness in terminating hemodynamically significant dysrhythmias. It should be remembered, however, that delivering electrical current through the body is not an innocuous procedure.

Limitations

The major limitation in this study was the sample size. Data were collected over 17 months and all patients admitted to an 80-bed military hospital were included if they met the criteria for the phenomenon under study. During the 12th month of data collection, a new Chief of Cardiology was assigned to the hospital. This change in practitioners decreased the entry of new subjects into the study as the new Chief of Cardiology was less aggressive in the use of countershock. Patients were being treated pharmacologically for

dysrhythmias and only one patient received countershock in the last 5 months of the study.

Missing values on three patients were also seen as limitations. These three patients were discharged from the hospital prior to the 24 hour postshock blood sampling. Because the sample size was already limited, it was imperative that these patients be included in data analysis. Therefore, postshock values of CK and LDH had to be averaged. This averaging may have effected the outcome of analysis because it brought high postshock levels of enzymes down; significance may have been masked by averaging these postshock enzyme values.

Many extraneous variables were identified in this study. Had the sample size been larger, the effects of these extraneous variables could have been analyzed using multivariate analysis of variance (MANOVA) (Polit & Hungler, 1983). However, for MANOVA to be used, at least five subjects would have been needed for each extraneous variable measured. For example, four different anesthetic agents were used, either alone or in combination, for a total of six possible premedications, a total of 30 patients would have been needed to use MANOVA for this set of variables.

A control group in this study would have been helpful in looking at serum enzymes over time. For example, preshock CK values in this study varied from 6 u/l to 340 u/l. It would have been interesting to see if that wide a variation of enzyme levels existed in patients without underlying heart disease.

Transthoracic resistance was considered to be an intervening variable which could have been decreased by timing the countershock during the end-expiratory phase of the respiratory cycle (Kerber, 1984). Electrode paddles were placed in the anterior/posterior position to reduce resistance and had that been coupled with the delivery of countershock at end-expiration, transthoracic resistance might have been reduced even further.

Suggestions for Further Study

The timing of countershock during end-expiration of the respiratory cycle has been shown to decrease transthoracic resistance and should this study be repeated, care should be given to ensure the shock is delivered during end-expiration. The actual current delivered to the patient was not calculated based on dialed energy and transthoracic resistance. With advancing technology, defibrillating units have been

developed that have the ability to display the actual amount of current delivered and the transthoracic resistance encountered, thus giving the practitioner a more accurate indication of the amount of current actually reaching myocardial tissue.

Three sets of questions arise from the findings of this study regarding the presence of skeletal muscle CK isoenzyme postshock. First, did the presence of procainamide in four patients affect the skeletal muscle cells making them more vulnerable to the effects of electric current? Reports on IV procainamide and countershock are not available in the literature. If procainamide does correlate with skeletal muscle injury, perhaps its use prior to countershock should be reconsidered by practitioners.

Second, does repeated countershock after a period of 4 hours compound injury to skeletal muscle cells due to a thermal component? Further, are there measures that can be taken to decrease the thermal component of injury when successive countershocks are necessary in terminating refractory dysrhythmias?

Finally, a third question arises as to the effects of high cumulative doses of electrical energy on myocardial and skeletal muscle cells. In this study all five patients receiving greater than 600 watt/seconds

cumulative energy showed skeletal muscle damage. Myocardial damage was not seen in the patients in the study, however, the risk of causing damage to myocardial cells is certainly apparent in prolonged resuscitation efforts and deserves further study.

Research studies designed to answer the above questions could clarify the effects DC-countershock has on the human body. The last reported research on human subjects was in 1984 (Kerber et al., 1984). New technology in the areas of high output defibrillators, electrode pads, low resistance conductive mediums and pharmacologic agents to treat dysrhythmias may not only change the way we deliver DC-countershock, but also the effects of DC-countershock on the body.

Summary

Direct current transthoracic countershock has been used successfully for years to terminate hemodynamically significant cardiac dysrhythmias. Hazards associated with countershock have been reported on animal and human subjects, however, these results do not seem to be consistent in the literature. The purpose of this study was to determine if DC-countershock could harm myocardial cells leading to the liberation of cardiac specific isoenzymes into the blood.

The results of this study suggest that DC-countershock is not associated with myocardial damage and that its use in terminating hemodynamically significant dysrhythmias is relatively safe under the conditions outlined by this study. The amount of countershock delivered should be tempered with judgment regarding the body surface area and transthoracic resistance of the patient. Consecutive countershocks should be delivered within minutes of each other. Waiting 4 hours between countershocks coupled with the infusion of procainamide, can lead to a rise in skeletal muscle creatine kinase, suggesting that injury can occur to skeletal muscle tissue following split dose countershock.

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APPENDIX

MEDICAL RECORD	REQUEST FOR ADMINISTRATION OF ANESTHESIA AND FOR PERFORMANCE OF OPERATIONS AND OTHER PROCEDURES
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A. IDENTIFICATION**1. OPERATION OR PROCEDURE****B. STATEMENT OF REQUEST**

1. The nature and purpose of the operation or procedure, possible alternative methods of treatment, the risks involved, and the possibility of complications have been fully explained to me. I acknowledge that no guarantees have been made to me concerning the results of the operation or procedure. I understand the nature of the operation or procedure to be _____
(Description of operation or procedure in layman's language)

which is to be performed by or under the direction of Dr. _____

2. I request the performance of the above-named operation or procedure and of such additional operations or procedures as are found to be necessary or desirable, in the judgment of the professional staff of the below-named medical facility, during the course of the above-named operation or procedure.

3. I request the administration of such anesthesia as may be considered necessary or advisable in the judgment of the professional staff of the below named medical facility.

4. Exceptions to surgery or anesthesia, if any, are: _____

(If none, so state)

5. I request the disposal by authorities of the below named medical facility of any tissues or parts which it may be necessary to remove.

6. I understand that photographs and movies may be taken of this operation, and that they may be viewed by various personnel undergoing training or indoctrination at this or other facilities. I consent to the taking of such pictures and observation of the operation by authorized personnel, subject to the following conditions:

- a. The name of the patient and his/her family is not used to identify said pictures.
- b. Said pictures be used only for purposes of medical/dental study or research.

(Cross out any parts above which are not appropriate)

C. SIGNATURES

(Appropriate items in Parts A and B must be completed before signing)

1. COUNSELING PHYSICIAN/DENTIST: I have counseled this patient as to the nature of the proposed procedure(s), attendant risks involved, and expected results, as described above.

(Signature of Counseling Physician/Dentist)

2. PATIENT: I understand the nature of the proposed procedure(s), attendant risks involved, and expected results, as described above, and hereby request such procedure(s) be performed.

(Signature of Witness, excluding members of operating team)

(Signature of Patient)

(Date and Time)

3. SPONSOR OR GUARDIAN: (When patient is a minor or unable to give consent) I, _____

sponsor/guardian of _____ understand the nature of the proposed procedure(s), attendant risks involved, and expected results, as described above, and hereby request such procedure(s) be performed.

(Signature of Witness, excluding members of operating team)

(Signature of Sponsor/Legal Guardian)

(Date and Time)

PATIENT'S IDENTIFICATION
(For typed or written entries give Name—last, first, middle, grade, date, hospital or medical facility)

REGISTER NO.

WARD NO.

STANDARD FORM 522 (Rev. 10-75)
 General Services Administration &
 Interagency Comm. on Medical Records
 FPMR (41 CFR) 101-11.606-3
 522-106

DATE
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