Purdue University

Purdue e-Pubs

Weldon School of Biomedical Engineering
Faculty Publications

Weldon School of Biomedical Engineering

1981

Transchest defibrillation under conditions of hypothermia

W A. Tacker

Charles F. Babbs
Purdue University, babbs@purdue.edu

D R. Abendschein

L A. Geddes

Follow this and additional works at: https://docs.lib.purdue.edu/bmepubs

Part of the Biomedical Engineering and Bioengineering Commons

Recommended Citation

Transchest defibrillation under conditions of hypothermia

W. A. TACKER, JR. MD, PhD; C. F. BABBS, MD, MS, PhD; D. R. ABENDSCHEIN, PhD;
L. A. GEDDES, ME, PhD

Biomedical Engineering Center, Purdue University, West Lafayette, Indiana, USA.

ABSTRACT

This study was conducted to determine whether or not hypothermia changes ventricular defibrillation threshold. Ventricular fibrillation was induced by electrical stimulation of the endocardium in pentobarbital anesthetized dogs, both during normothermia and hypothermia produced by circulating 8 °C water through a rubber bladder implanted in the peritoneal cavity. Defibrillation threshold was determined as the shock strength needed to defibrillate the ventricles and differing no more than 10 percent from a shock strength that failed to defibrillate. Defibrillation threshold current was stable for body temperatures ranging from 37 $^{\circ}$C to 22 $^{\circ}$C. Threshold energy increased linearly with decreasing temperature in keeping with the expected temperature-dependent changes in body fluid resistance. Normothermic electrical doses are probably appropriate for defibrillation of hypothermic children.

Key words: defibrillation threshold, drowning, electrical dose, ventricular fibrillation

1

INTRODUCTION

Survival after near-drowning has been reported after prolonged periods of immersion when the victim was young and the event occurred in cold water [1]. Hypothermia itself increases the propensity of the ventricles to fibrillate [2]. It is commonly held that ventricular defibrillation is more difficult under conditions of hypothermia, and it would follow logically that stronger shocks might be required to defibrillate subjects who fibrillate as a result of cold-water immersion. However, use of excessive defibrillation energy increases the risk of producing cardiac damage by the electrical shock [3]. This study was conducted to determine whether or not hypothermia changes ventricular defibrillation threshold.

METHODS

Ten mongrel dogs weighing 5-10 kg were anesthetized with intravenous pentobarbital sodium (30 mg/kg), an agent which itself does not alter the defibrillation threshold [4]. The trachea was intubated and lead III ECG was monitored to document heart rhythm. Fibrillation was produced by a 5-sec burst of 60 Hz electrical stimulation of the right ventricle via a transvenous catheter-electrode. Transchest ventricular defibrillation threshold was determined by repeated trials of fibrillation and defibrillation, using shocks of increasing or decreasing peak current amplitude, each 10% above or below the preceding shock. The defibrillator shocks were delivered via jelled, 10-cm diameter disc electrodes sutured to the shaved right and left hemithoraces. The durations of the damped sine wave pulses were dependent upon subject resistance and ranged from 5-10 msec. The ventricles were never permitted to fibrillate for more than 50 sec and were never refibrillated until pulsatile arterial blood pressure had returned to a stable level.

Defibrillation threshold was measured at normal body temperature (37 °C) and at 3 hypothermic temperatures (32, 27, and 22 °C). Hypothermia was produced by circulating 8 °C water through a rubber bladder implanted in the peritoneal cavity. Peritoneal cooling has the advantage (compared to ice water immersion) of keeping the thorax dry so that defibrillation threshold may be determined using transchest electrodes. Re-warming was accomplished by circulating 40 °C water through the same rubber bladder. A solution of isotonic saline (Impersol) was introduced to the peritoneal cavity to prevent direct contact of the rubber bladder with the bowel and to increase the efficiency of heat exchange. Threshold energy and current values were obtained during both the cooling and re-warming phases of the study. Peak current values were read directly from a storage oscilloscope trace. Delivered energy values were calculated as 1/2 peak current × peak voltage × pulse duration. Threshold values were normalized to allow for direct comparison among dogs, even when the normothermic thresholds varied widely from one subject to another. Normalization was carried out in the following manner. All threshold values for one subject were averaged to obtain a mean threshold. Then each individual threshold was divided by that mean to obtain a normalized threshold. For example, in l dog, mean threshold was 6.8 amperes, and the initial peak current threshold was 6.0 amperes. Hence, that subject would have a normalized threshold at 37 °C of 6.0/6.8 = 0.88.

RESULTS

Figure l shows normalized threshold energy and current for defibrillation as a function of aortic temperature. Threshold energy increased 2.5% per °C decrement of body temperature, whereas threshold current remained essentially constant. Because threshold current did not vary with body temperature, the change in threshold energy with temperature reflects a change in subject impedance. Figure 2 illustrates that the measured transchest impedance (peak voltage/peak current) for these same 10 animals increased linearly as a function of decreased body temperature. The temperature coefficient was l.8% per °C. Because more energy is dissipated by passage of a fixed current intensity through a higher impedance, the increase in subject impedance accounts for most of the increase in threshold energy.
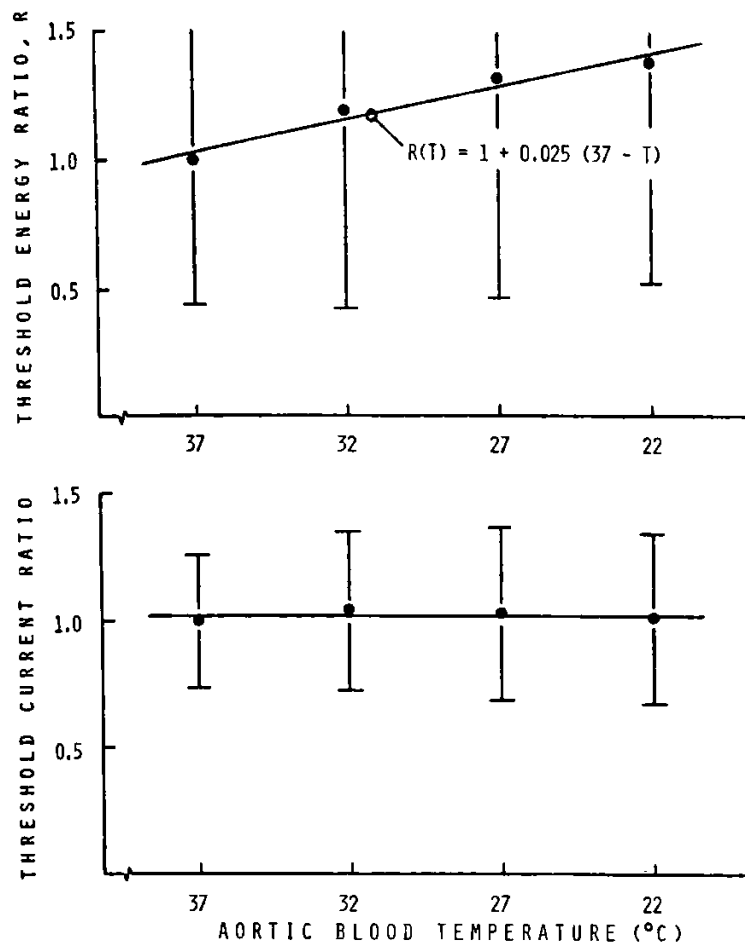

FIG. l. Changes in defibrillation threshold with body temperature. Threshold energy and current ratios were computed by dividing all threshold values for a given animal by the mean threshold value for that animal. The absolute values corresponding to 1.0 on the vertical axes were 1.7 J/kg and 1.4 amp/kg. The temperature scale decreases from left to right, indicating greater depths of hypothermia. Vertical bars indicate ± 1 SD.
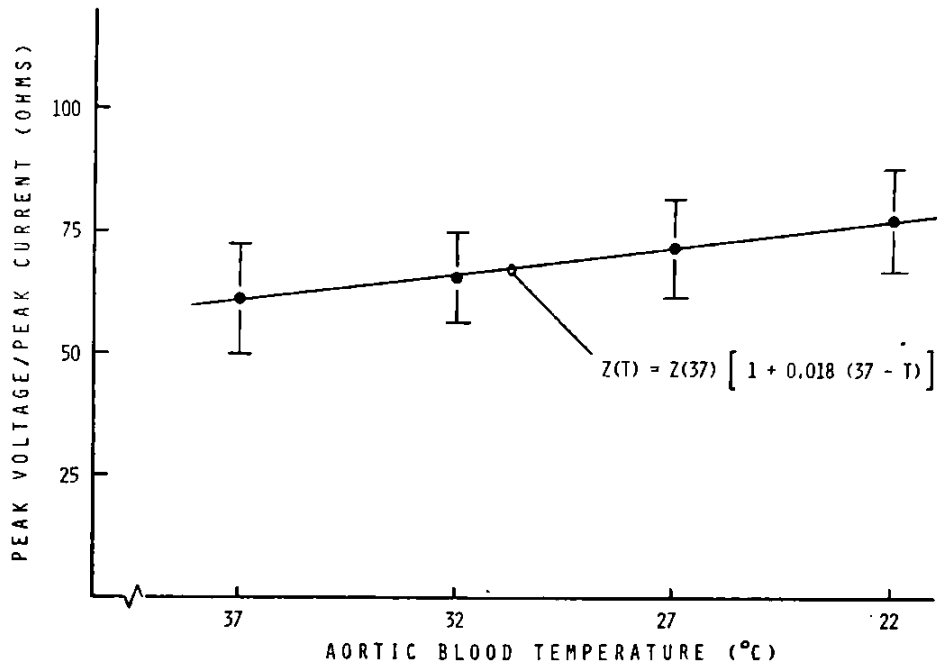
FIG. 2. Changes in transchest impedance to defibrillating current with body temperature in 10 dogs. The temperature scale decreases from left to right, indicating greater depths of hypothermia. Vertical bars indicate ± 1 SD.

DISCUSSION

Although this study does not mimic near-drowning conditions in every respect (i.e., presence of prolonged hypoxia or presence of the diving reflex), it does show that hypothermia alone does not increase the defibrillation threshold when current is used as a measure of shock intensity. Energy threshold increases slightly, but the increase is so small as to have no practical effect on clinical defibrillation dose. These data indicate that during resuscitation efforts on near-drowning victims with concurrent hypothermia, the practical electrical dose for defibrillation is probably unchanged from the dose in the normothermic patient. It should also be emphasized that this study does not address the issue of prompt resumption of cardiac function after defibrillation of the hypothermic subject. The proper use of drugs, pacing, and so forth await investigation.

Patients of small size are most likely to become hypothermic during cold water immersion, and also are most likely to be shocked with excessively strong shocks. Gutgesell et al. [5] recommend a dose of 1 J/lb (2.2 J/kg) for normothermic pediatric patients. This normothermic dose is probably appropriate for hypothermic children.

4

REFERENCES

1. Siebke H, Breivik H, Rad T, et al: Survival after 40 minutes submersion without cerebral sequelae. Lancet 1975; 1:1275

2. Emiroghi F: Fibrillation in normothermic and hypothermic rabbit heart and effect of adrenaline. Arch Int Physiol Biochem 1965; 73:55

3. Babbs CF, Tacker W A, VanVleet JD, et al: Therapeutic indices for transchest defibrillation: Effective, damaging and lethal electrical doses. Am Heart J 1980; 99:734

4. Babbs CF: Effect of pentobarbital anesthesia on ventricular defibrillation threshold in dogs. Am Heart J: 1978; 331

5. Gutgesell HP, Tacker W A, Geddes LA, et al: Energy dose for ventricular defibrillation of children. Pediatrics 1977; 58:898