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NAVAL MEDICAL FIELD RESEARCH LABORATORY

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○ MYOCARDIAL FUNCTION FOLLOWING THERMAL BURN

by

THORNTON W. MERRIAM, JR., LCDR MC USNR

with the technical assistance of

R. Jackson; M. G. Moore, Jr., HMCA USN; H. N. Edwards,
HMCA USN; J. M. Hanner, PFC USMC; J. Abramson,
LCPL USMC; and G. S. Walker, HM3 USN

Bureau of Medicine and Surgery, Navy Department
Task MR005.12-0101.2.2



CAMP LEJEUNE

NORTH CAROLINA

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Physiology Division

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SUMMARY PAGE

THE PROBLEM

To determine whether or not an impairment in the efficiency of the heart muscle occurs following severe thermal burn.

FINDINGS

Utilizing the ventricular function curve as a measure of myocardial contractility, it was found that following 30 per cent body surface area thermal burn in dogs, there is a definite decrease in the efficiency of the heart muscle.

APPLICATION

If the results of this study can be applied to burns in man, they indicate that following severe burns a careful evaluation of the cardiac status of the victim should be carried out. A careful watch for evidence of heart failure should be maintained.

ADMINISTRATIVE INFORMATION

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ABSTRACT

Several investigators have suggested that a defect in myocardial function occurs in the dog following severe thermal burn. A study was therefore undertaken in which myocardial contractility was measured before and after burn. The left ventricular function curve was used as an index of contractility. Ventricular function was measured immediately before and 30 minutes following 30 per cent body surface area burn in 25 dogs, and before and after a sham burn in 13 control dogs. It was found that the burn produced a significantly greater impairment of myocardial contractility than did the sham burn. The mechanism by which the burn produced this alteration in contractility was not apparent from the results of this study.

Myocardial Function Following Thermal Burn

INTRODUCTION

Severe thermal burn produces in the dog an immediate and profound reduction in the cardiac output. This finding was reported originally by Gilmore and Handford (1) and Dobson and Warner (2) and has been confirmed by other investigators (3,4). This decrease in cardiac output is unrelated to changes in right atrial pressure (1), and occurs too rapidly to be explained solely on the basis of changes in plasma volume (1-3). The restoration of plasma volume by the infusion of intravenous solutions does not result in a return of the cardiac output to preburn levels (5). When a cardiac glycoside is administered with the intravenous fluids, however, an increase in cardiac output to preburn values occurs (4). These observations suggest that a defect in myocardial contractility may occur after severe thermal trauma, and that this defect may be reversible.

In the present study, the left ventricular function (VF) curve (6-8) was utilized as an index of myocardial contractility. VF curves were obtained on dogs before and after 30 per cent body surface area thermal burn.

METHODS

Fasting inbred beagles and mongrel dogs of both sexes (mean weight, 9.1 kg; range, 5.0 kg - 16.8 kg) were anesthetized with morphine sulfate (2 mg/kg) intramuscularly, followed in 30 minutes by chloralose (60 mg/kg) and urethane (600 mg/kg) intravenously. The trunks of the animals were shaved. The right and left femoral arteries, the left femoral vein, and the left jugular vein were cannulated. A tracheostomy tube was inserted. The cannula in the right femoral artery was connected to a Sanborn Pressure Transducer (267A)* and arterial pressure recorded on a Sanborn 150 Multichannel Recorder. The jugular cannula was a small bore plastic tube of sufficient length to reach approximately to the level of the right atrium. The left side of the chest was opened by means of a longitudinal incision in the anterior axillary line, transecting four or five ribs. Artificial respiration was carried out with a Respiration Pump Model 607.** The pericardium

* Sanborn Company, Waltham, Massachusetts.

** Harvard Apparatus Company, Dover, Massachusetts.

was opened widely and two small electrodes were attached to the left atrial appendage. These electrodes were connected to a Model S4 Stimulator,* and the heart paced at a constant rate. A rigid metal cannula with an internal diameter of 2.0 mm was inserted into the apex of the left ventricle through the apical dimple, and sutured in place. The cannula was attached directly to a Sanborn Pressure Transducer (267B) and ventricular pressure recorded. The tip of the cannula was used for the zero reference point in recording ventricular pressure.

Cardiac output was determined by the continuous sampling dye injection technique using indocyanine green (9), and the Cuvette Densitometer.** Dye was injected into the right atrium via the jugular cannula, and blood withdrawn at a constant rate from the left femoral artery through the cuvette by means of a falling column of mercury. Densitometer output was recorded on the multichannel recorder and cardiac output calculated by the formula described by Hamilton et al. (10).

The data for the VF curves were obtained by the method described by Sarnoff et al. (6, 11). Gain settings for recording left ventricular end diastolic pressure (LVEDP) were adjusted so that a pressure change from 0 to 30-40 cm H₂O produced a full-scale pen deflection. The respirator was turned off while the high speed LVEDP tracings were obtained. Changes in LVEDP were produced by the rapid infusion of heparinized whole blood from a donor dog. Blood was removed slowly via the femoral vein at the conclusion of the VF curve procedure. Withdrawal was continued until LVEDP, cardiac output and mean arterial pressure had returned to pre-infusion levels. Arterial hematocrit was determined at the beginning and end of each VF curve procedure.

An example of the tracings from which the data for VF curves were obtained is given in figure 1. The LVEDP was read at the point on the diastolic pressure curve immediately before the onset of systole. Readings on 10 successive contractions were taken and mean LVEDP obtained. Stroke work in grammeters was calculated by the formula described by Sarnoff and Berglund (6, 7). The VF curve plotted from the data illustrated in figure 1 is shown in figure 2.

* Grass Instruments Company, Quincy, Massachusetts.

** Guilford Instrument Laboratories, Inc., Oberlin, Ohio.

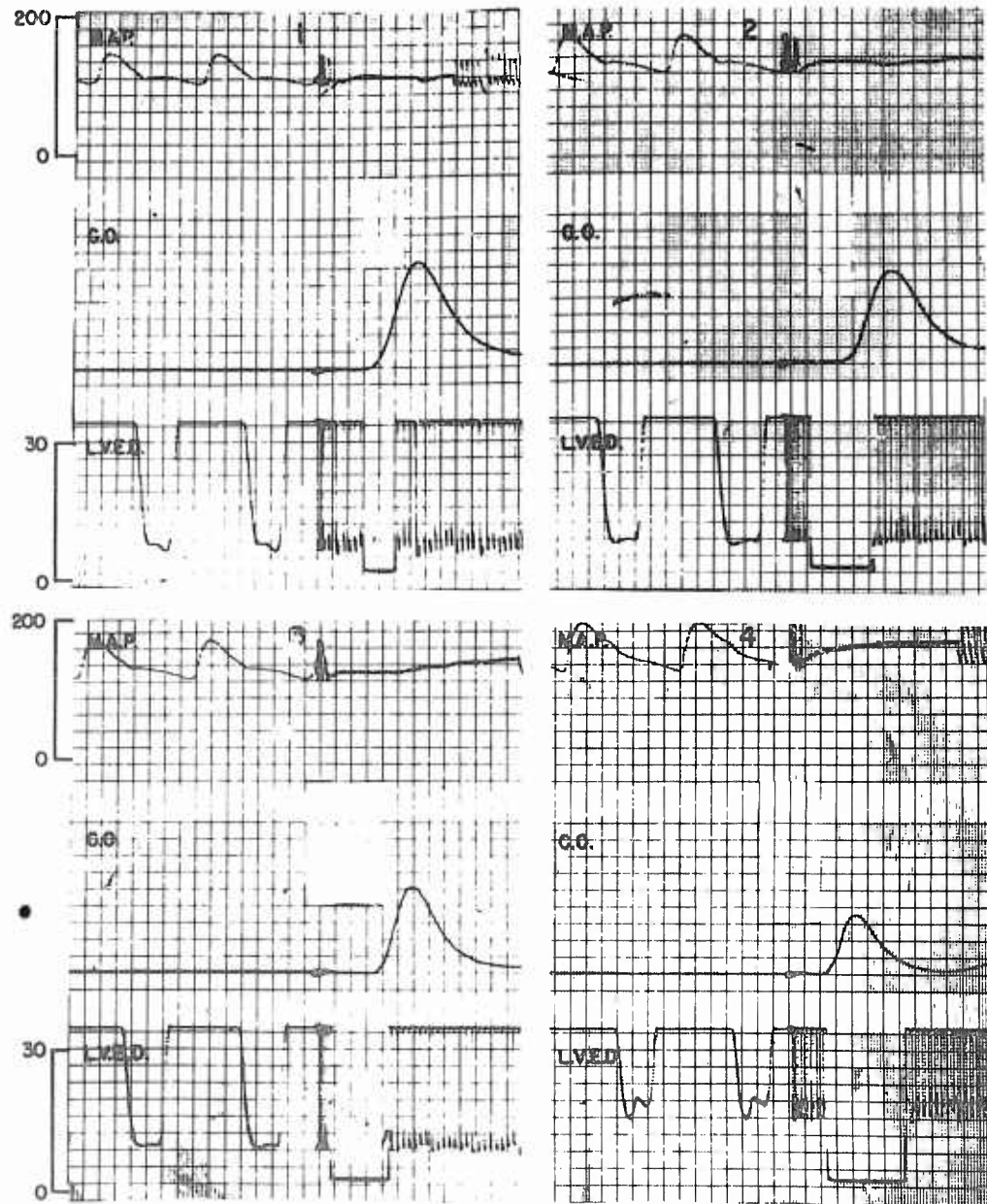


Figure 1. Experiment 10. Dog weight 8.6 kg. MAP = mean arterial pressure (mm Hg). CO = cardiac output: indocyanine green dilution curve. LVEDP = left ventricular end diastolic pressure (cm H₂O). Paper speed: 100 mm per second for LVEDP readings 5 mm per second for the dye dilution curve. Tracings taken at increasing LVEDP values are numbered 1-4, and show increasing arterial pressure and cardiac output as the LVEDP rises. LVEDP readings: 6.0, 7.0, 8.0, and 17.0 cm H₂O, with stroke work values of 8.2, 11.9, 8.0, and 33.4 grammeters respectively.

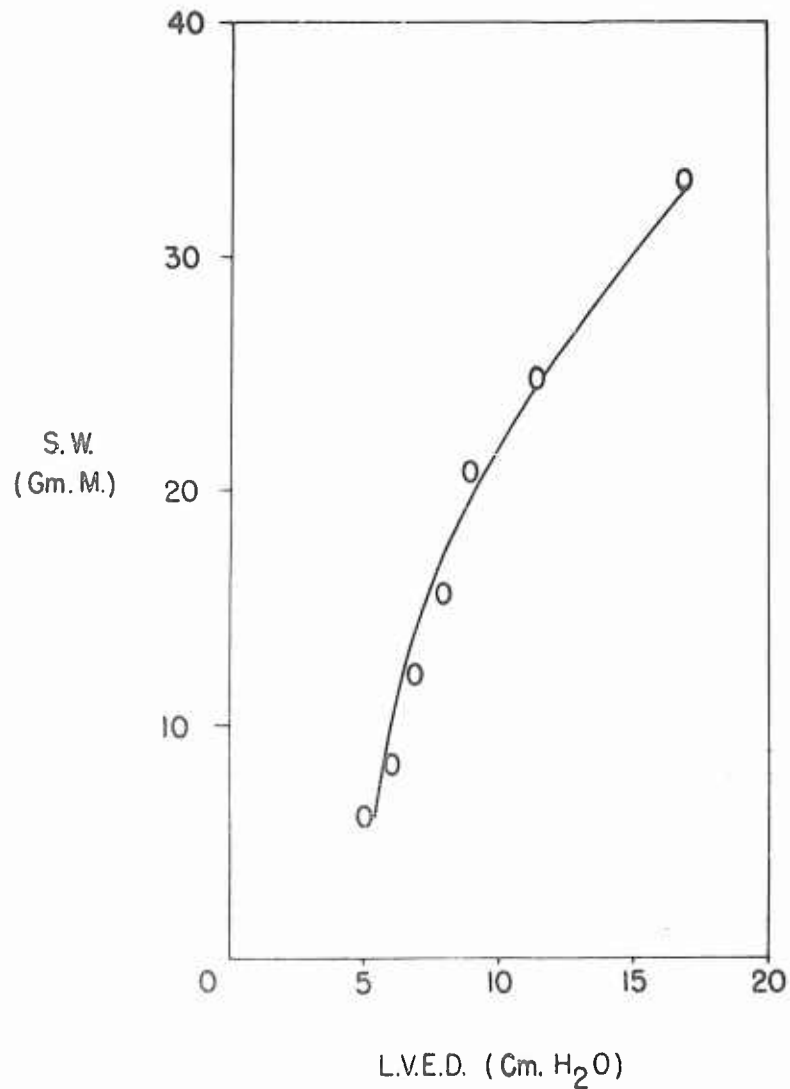


Figure 2. Ventricular function curve plotted from the data in figure 1. SW = stroke work in grammeters. LVEDP = left ventricular end diastolic pressure in cm H₂O. Three points on this curve at LVEDP values of 5.0, 9.0, and 11.5 were not shown in figure 1.

Data for plotting VF curves were obtained from 38 dogs. The animals were then blackened with powdered lampblack and 25 were subjected to a 20 cal/cm² (third-degree) burn of approximately 30 per cent of the body surface area. The remaining 13 control animals received a sham burn. They were manipulated in a manner exactly similar to burning, but were not actually burned. The burn source and technique of burning were described in a previous report from this laboratory (1). The VF curve procedure was carried out again 30-60 minutes after the burn (or after simulated burn). The animals were sacrificed after the postburn measurements had been completed.

In each procedure, the preburn and postburn (or post sham burn) VF curves were plotted, and the direction of the shift in the position of the postburn curve in relation to the preburn curve was noted: to the right (impaired contractility), to the left (improved contractility), or no change. The number of dogs showing shifts in each direction were tabulated in the burned and unburned groups, and the results compared by chi square analysis for multinomial samples (12).

RESULTS

In the control group, the sham burn produced a shift in the VF curve to the right in 6 dogs, to the left in 5 dogs, and no change in 2 dogs. This distribution, expressed as a per cent of the total number, is indicated in figure 3 (unshaded bars).

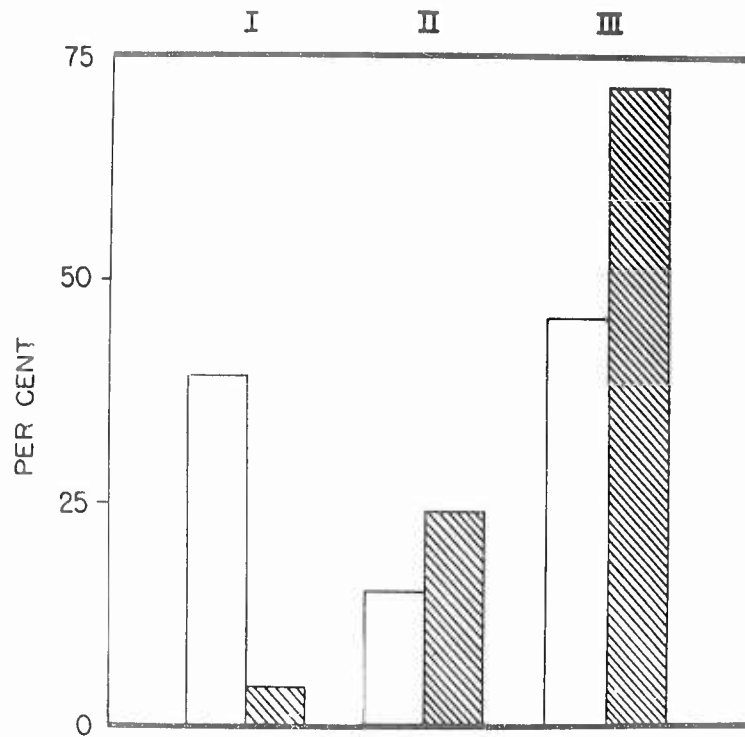


Figure 3. Bar graph showing the distribution of the changes in position of the ventricular function curve after thermal burn (shaded bars) and after simulated burn (unshaded bars). I = shift to the left; II = no change; III = shift to the right. For comparison, the number of dogs showing a shift in each of the directions is expressed as a per cent of the total number of dogs in that group. There were 25 dogs in the burned group and 13 in the control group.

Among the 25 burned dogs, 18 showed a shift of the VF curve to the right following burn, six remained unchanged, and in only one was there a shift of the postburn curve to the left. This distribution is shown in figure 3 (shaded bars). Chi square analysis comparing the relative frequency of occurrence of each possible postburn VF curve position in the burned and control groups revealed a significant difference between groups ($X^2 = 7.613$, $P = <.05$).

In 11 of the 18 burned dogs in which a shift to the right was noted, the shift was to a marked degree, as indicated by a change in the slope of the curve as well as a change in the position. In only one of the six controls in which a shift to the right was noted did such a marked change occur.

Figures 4-7 are examples of VF curves obtained on individual dogs before and after burn. They illustrate the range in configuration of the normal preburn curves, as well as the variation in response to burn.

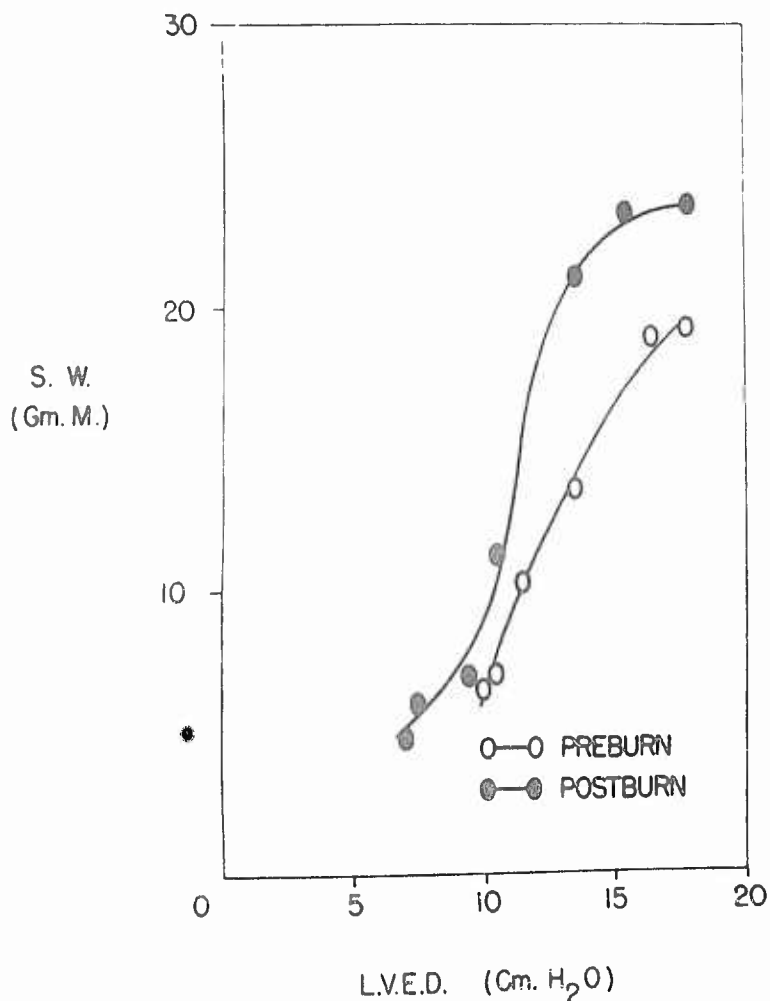


Figure 4. Experiment 31. Dog weight 8.6 kg. Symbols same as in figure 2. Ventricular function curves before and after burn in which the position of the postburn curve was to the left of the preburn curve.

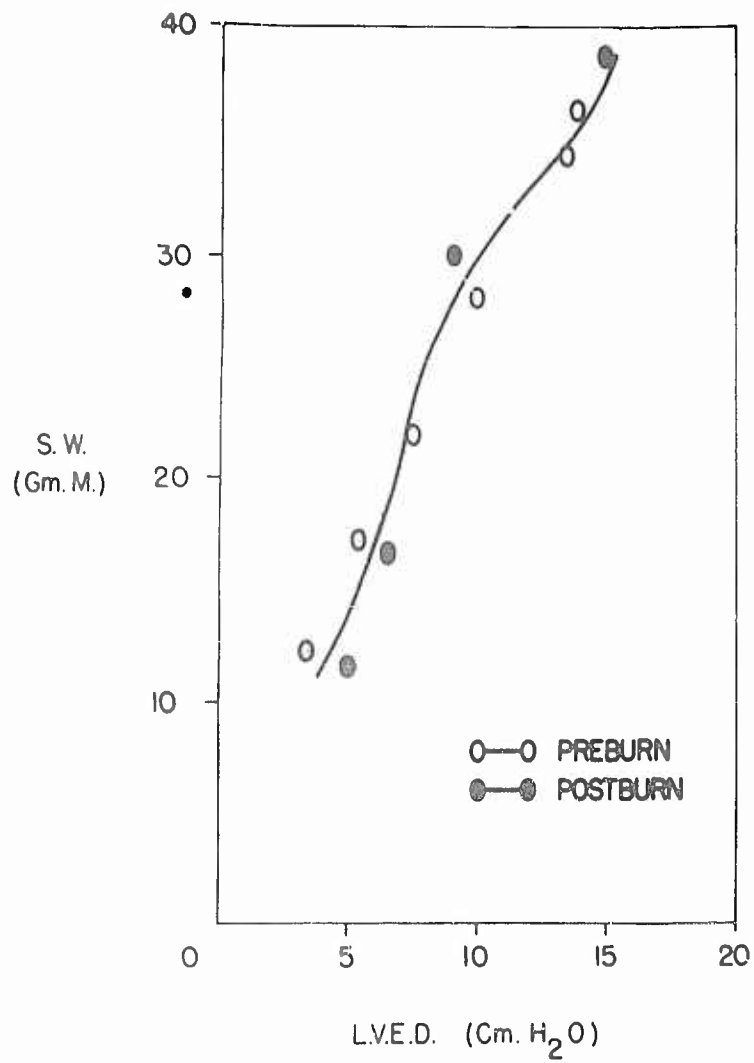


Figure 5. Experiment 35. Dog weight 15.3 kg. Symbols same as figure 2. Ventricular function curve before and after burn in which the preburn and postburn curves were in the same position.

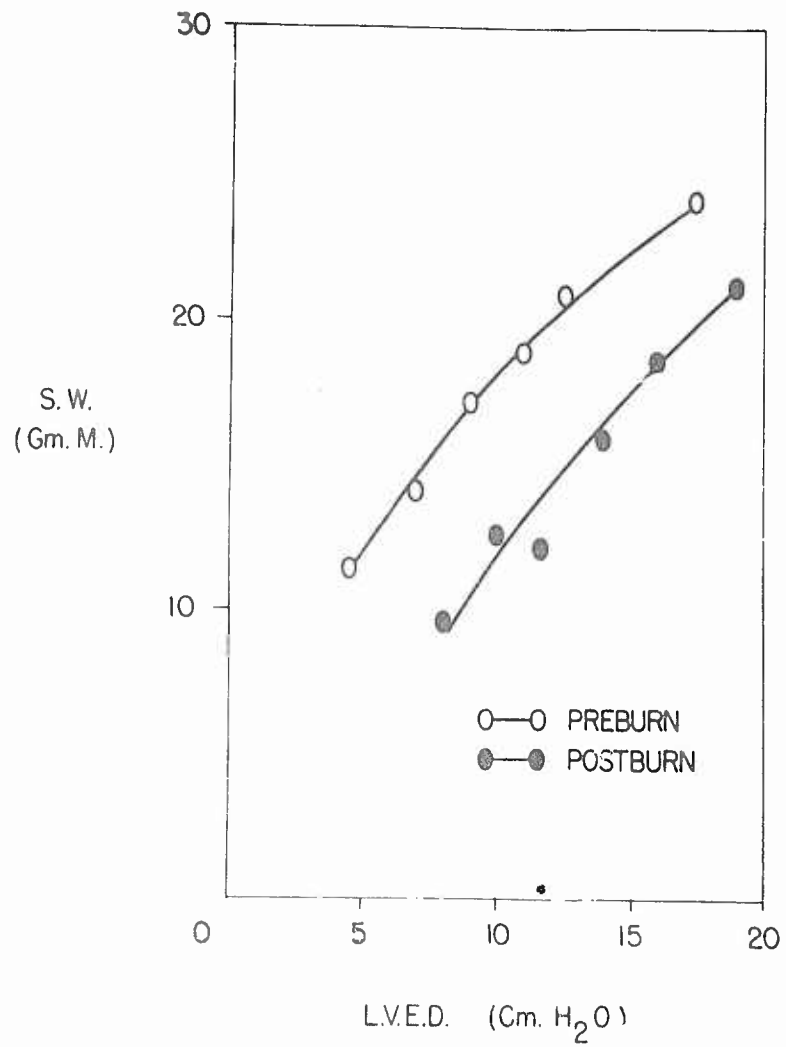


Figure 6. Experiment 30. Dog weight 9.4 kg. Symbols same as figure 2. Ventricular function curves before and after burn in which the position of the postburn curve was to the right of the preburn curve.

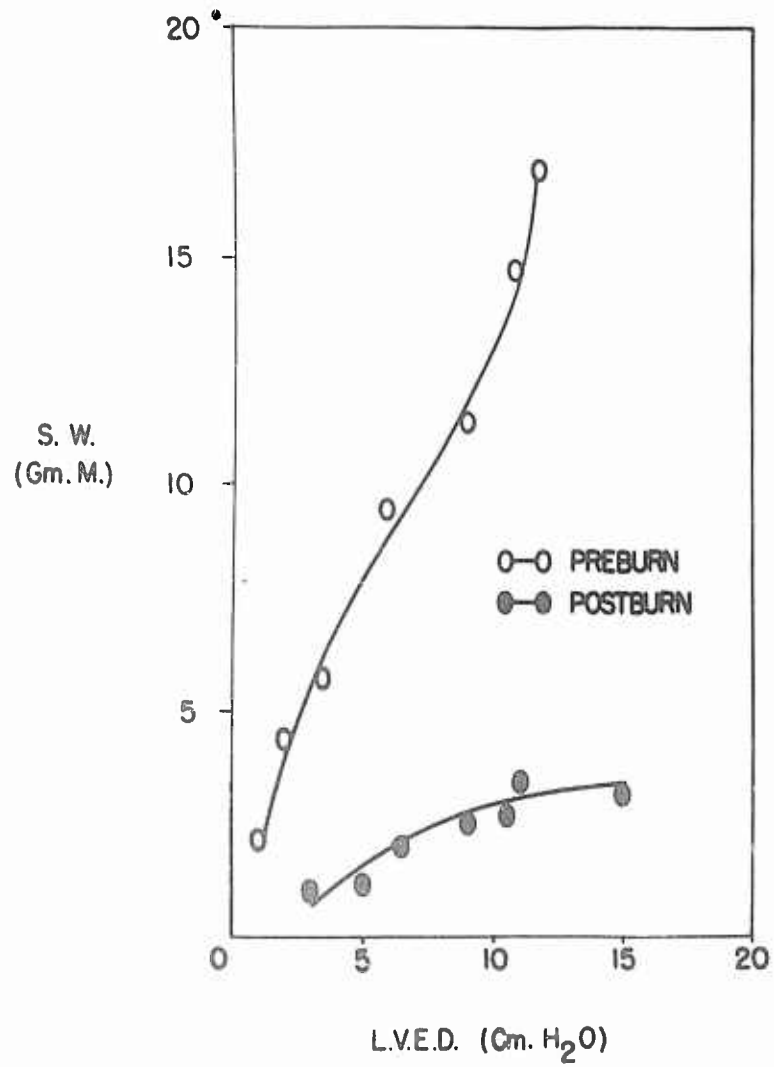


Figure 7. Experiment 22. Dog weight 5.9 kg. Symbols same as figure 2. Ventricular function curve before and after burn in which the position of the postburn curve was to the right of the preburn curve and there was a decrease in the slope of the postburn curve.

Mean arterial pressures below 70 mm Hg were encountered immediately postburn in 9 of the 25 burned dogs, and following manipulation in 3 of the 13 controls. In all cases the hypotension was associated with a shift of the VF curve to the right. This hypotension was not associated with significant changes in calculated peripheral vascular resistance (13) or LVEDP. Hematocrit did not vary by more than 10 per cent during any procedure.

DISCUSSION

The data presented suggest that in the animals studied, an impairment of myocardial contractility occurred following thermal burn. The control dogs subjected to a similar manipulation without burn showed significantly less evidence of impairment.

An impairment of myocardial contractility, as indicated by a shift of the VF curve to the right, can be produced by any one of several mechanisms. Elevated blood pressure produces a shift in the curve to the right by causing carotid sinus stimulation and a reflex decrease in cardiac sympathetic nerve activity (14). An increase in arterial pressure was not encountered after burn in this study but has been noted by other observers (2) immediately following burn, presumably related to peripheral vasoconstriction. However, Gilmore found that pretreatment with dibenamine did not prevent the decrease in cardiac output which followed thermal burn, although it prevented this vasoconstriction (15). It therefore appears unlikely that the contractility change is related to increased carotid sinus pressure. Severe anemia results in a shift of the VF curve to the right (6, 7) but could not have produced the changes noted in this study since hematocrit did not change by more than 10 per cent between the preburn and postburn VF curves. Other investigators have indicated that red cell mass decreases only minimally or may increase during the early postburn period (16). Vagal stimulation does not alter the relationship between LVEDP and stroke work (9). Direct impairment of the myocardium therefore appears to be the most plausible explanation for the contractility change which occurred postburn.

A shift of the VF curve to the left occurred in a greater per cent of control dogs than burned dogs. Stellate stimulation, infusion of sympathomimetic drugs, and decreased carotid sinus pressure produce a shift of the VF curve to the left (6-8, 14). In this study, when hypotension was present, it was associated with a shift of the curve to the right. Increased sympathetic activity is therefore the most likely cause of the shift of the curve to the left. In spite of a probable in-

crease in sympathetic activity, however, only one of 25 burned dogs showed a shift to the left.

The mechanism by which the burn produced these findings is not clear. The elaboration of a toxic substance with properties which are inhibitory to the myocardium has been suggested as a possible explanation (1,2). Another is an inadequate sympathetic-adrenal medullary response to the burn. This phenomenon has been described after thermal burn, although not in the immediate postburn period (17). Myocardial ischemia secondary to hypovolemia and/or splanchnic pooling is a third alternative. Postburn electrolyte imbalance must also be considered, although electrolyte changes which have been reported in dogs postburn are not of great magnitude until several hours after burn (18). The results of the study do not indicate which of these factors, if any, is primarily responsible for the observed defect in contractility.

The possibility that the changes observed in this study were the result of alterations in myocardial distensibility rather than contractility cannot be ruled out. If toxic factors are involved in the VF curve shifts, they could modify distensibility as well as contractility.

SUMMARY

Ventricular function (VF) curves were measured in 25 dogs before and after 30 per cent body surface area thermal burn, and in 13 control dogs before and after a sham burn. It was found that the burn significantly depressed the VF curve, as compared to the effect of sham burn in control dogs. The mechanism by which this decrease in VF was produced was not indicated in this study.

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