

Cardiovascular Effect of 7.5% Sodium Chloride–Dextran Infusion After Thermal Injury

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Hypothesis: Clinical study can help determine the safety and cardiovascular and systemic effects of an early infusion of 7.5% sodium chloride in 6% dextran-70 (hypertonic saline–dextran-70 [HSD]) given as an adjuvant to a standard resuscitation with lactated Ringer (RL) solution following severe thermal injury.

Design: Prospective clinical study.

Setting: Intensive care unit of tertiary referral burn care center.

Patients: Eighteen patients with thermal injury over more than 35% of the total body surface area (TBSA) (range, 36%–71%) were studied.

Interventions: Eight patients (mean \pm SEM, 48.2% \pm 2% TBSA) received a 4-mL/kg HSD infusion approximately 3.5 hours (range, 1.5–5.0 hours) after thermal injury in addition to routine RL resuscitation. Ten patients (46.0% \pm 6% TBSA) received RL resuscitation alone.

Main Outcome Measures: Pulmonary artery catheters were employed to monitor cardiac function, while hemodynamic, metabolic, and biochemical measurements were taken for 24 hours.

Results: Serum troponin I levels, while detectable in all patients, were significantly lower after HSD compared with

RL alone (mean \pm SEM, 0.45 \pm 0.32 vs 1.35 \pm 0.35 μ g/L at 8 hours, 0.88 \pm 0.55 vs 2.21 \pm 0.35 μ g/L at 12 hours). While cardiac output increased proportionately between 4 and 24 hours in both groups (from 5.79 \pm 0.8 to 9.45 \pm 1.1 L/min [mean \pm SEM] for HSD vs from 5.4 \pm 0.4 to 9.46 \pm 1.22 L/min for RL), filling pressure (central venous pressure and pulmonary capillary wedge pressure) remained low for 12 hours after HSD infusion ($P = .048$). Total fluid requirements at 8 hours (2.76 \pm 0.7 mL/kg per each 1% TBSA burned [mean \pm SEM] for HSD vs 2.67 \pm 0.24 mL/kg per each 1% TBSA burned for RL) and 24 hours (6.11 \pm 4.4 vs 6.76 \pm 0.75 mL/kg per each 1% TBSA burned) were similar. Blood pressure remained unchanged, and serum sodium levels did not exceed 150 \pm 2 mmol/L (mean \pm SD) in either group.

Conclusions: The absence of deleterious hemodynamic or metabolic side effects following HSD infusion in patients with major thermal injury confirms the safety of this resuscitation strategy. Postburn cardiac dysfunction was demonstrated in all burn patients through the use of cardiospecific serum markers and pulmonary artery catheter monitoring. Early administration of HSD after a severe thermal injury may reduce burn-related cardiac dysfunction, but it had no effect on the volume of resuscitation or serum biochemistry values.

Arch Surg. 1999;134:1091-1097

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LARGE-VOLUME infusion of a balanced sodium solution has been a mainstay in the resuscitation of the severely injured patient for decades. Of special concern to physicians involved with resuscitation of the thermally injured are the consequences of administering the enormous fluid volume required for maintenance of mean arterial blood pressure and urine output. This concern stems from the recognition that morbidity associated with volume overload may be quite severe, particularly pulmonary and cardiac decompensation related to edema.^{1,2}

Recent studies have described the use of hypertonic solutions for primary resuscitation and as adjuvant fluid therapy for patients subject to a variety of traumatic insults.³⁻⁹ The studies examining the efficacy of hyperosmolar solutions in restoring intravascular volume after thermal injury or hypovolemic shock frequently showed promising results under very strictly controlled clinical conditions. Despite these findings, the hemodynamic and cardiovascular effects of a hypertonic saline–dextran (HSD) bolus in the resuscitation of large thermal injury remained unclear. Animal studies from our laboratory and others have shown that a small bolus

PATIENTS AND METHODS

This prospective clinical study was conducted at a tertiary burn referral center in patients with thermal injury involving at least 35% TBSA. Inclusion criteria included age between 18 and 66 years, thermal injury greater than or equal to 35% TBSA, and hospital admission within 6 hours of thermal injury. Patients were excluded from consideration for a history of renal disease, bleeding disorder, multisystem trauma, inhalation injury, and pregnancy. Patient participation was in accordance with informed consent guidelines and restrictions approved by the Institutional Review Board of the University of Texas Southwestern Medical Center at Dallas.

Eighteen patients were enrolled in this study. Prior to initial assessment, all patients received standard-volume resuscitation of RL solution as dictated by the Parkland guidelines (4 mL/kg per 1% TBSA burned, with half the total given over the initial 8 hours). All patients underwent intubation, and mechanical ventilation was continued throughout the study. Eight patients received an infusion of 4 mL/kg of HSD over 30 minutes via central venous catheter. Eligible patients received HSD based on availability from a 24-hour hospital pharmacy. Central hemodynamics were measured in all patients at the earliest possible time with a pulmonary artery catheter.

Fluid resuscitation of all patients was maintained with RL in sufficient volume to maintain a urine output of 0.5 to 1.0 mL/kg per hour. Hemodynamic response to resuscitation was monitored by continuous assessment of heart rate, blood pressure, and urine output. Measurements were made at 4-hour intervals of cardiac output, central venous pressure, pulmonary capillary wedge pressure, and systemic vascular resistance. Hemodynamic, metabolic (serum electrolytes, osmolality), and coagulation (prothrombin time, partial thromboplastin time, platelet count) parameters were regularly measured for 24 hours. Arterial blood was collected for measurement of PO_2 , PCO_2 , and pH as clinically indicated. At the end of 8 and 24 hours, total fluid requirements and urine outputs were calculated. In addition, venous blood samples were collected at regular intervals for the measurement of serum troponin I, creatine kinase, and creatine kinase MB fraction levels to assess cardiac injury.

Statistical significance was determined with an unpaired *t* test, with $P < .05$ representing significant difference compared with patients receiving RL resuscitation only. Analysis of variance was used to determine significance among multiple groups.

(4 mL/kg) of 7.5% sodium chloride in 6% dextran-70 (HSD) given early during resuscitation after several types of trauma improved cardiac function and significantly reduced total fluid requirements.^{3,10-12} Hypertonic solutions may reduce resuscitation fluid volumes by osmotically drawing intracellular water into the depleted extracellular spaces, while coadministered colloid may prolong this effect by retaining this recruited fluid in the plasma.¹³ Hypertonic

Patient Demographic and Injury Characteristics*

	HSD + RL (n = 8)	RL Alone (n = 10)
Age, y	32 ± 2	35 ± 4
Sex, M/F	5/3	7/3
Weight, kg	76 ± 5	79 ± 2
TBSA, %	48 ± 2	46 ± 6
Full-thickness TBSA, %	11 ± 8	13 ± 5

*HSD indicates hypertonic saline-dextran-70; RL, Ringer lactate; and TBSA, total body surface area. Values are mean ± SEM unless otherwise indicated.

solutions have long been known to have positive inotropic effects and may enhance myocardial contractility through the stimulation of sodium-calcium exchange at the myocyte membrane.¹⁴ Other mechanisms may involve autonomic nervous system stimulation or enhancement of pulmonary-cardiac reflexes.¹⁵

We had previously examined the hemodynamic effects of delayed hypertonic saline administration in laboratory experiments designed to mimic conditions commonly found during initial transport, evaluation, and resuscitation of thermally injured patients. This work confirmed that profound cardiac contractile dysfunction exists for several days after cutaneous thermal injury and demonstrated the cardioprotective and volume-sparing effects of hypertonic saline when given immediately after thermal injury as well as when given up to 4 hours after injury.¹² Given these findings and observations, we proposed that a hypertonic saline bolus delivered within hours after thermal injury would benefit a patient population expected to require massive fluid resuscitation and develop burn-related cardiac dysfunction.

In the present study, fluid resuscitation using lactated Ringer (RL) solution was initiated according to the Parkland formula (4 mL/kg per each 1% total body surface area [TBSA] burned) in all patients with severe thermal injury. An HSD bolus was subsequently administered on arrival to all patients who met the inclusion criteria. A primary objective of this clinical study was to evaluate the efficacy of an intravenous HSD bolus in maintaining cardiohemodynamic parameters and reducing resuscitation volume requirements when given as a supplement to standard resuscitation fluid in acute thermal injury.

RESULTS

PATIENT PROFILE

The patient population studied (**Table**) was predominantly men in the fourth decade of life who sustained a severe (>40% TBSA) thermal injury with comparable extent of full-thickness injury. All patients survived through the 24-hour study period.

RESUSCITATION VOLUME AND URINE OUTPUT

The total volume of resuscitation fluid received by each treatment group was tabulated and plotted (**Figure 1**, left). By 8 hours after injury, the volume of resuscitation as rec-

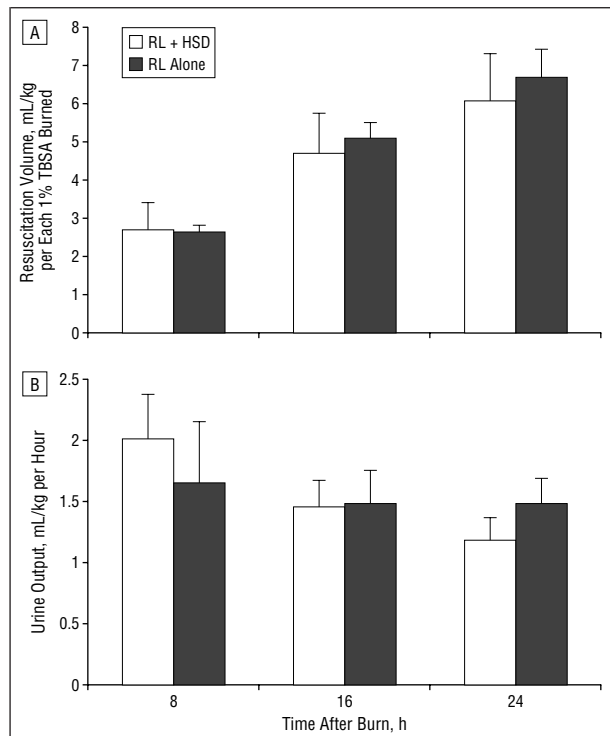


Figure 1. Resuscitation volume requirements (A) and urine outputs (B) during the 24-hour study period for patients receiving Ringer lactate (RL) plus 7.5% sodium chloride-dextran-70 bolus (HSD) and RL alone. The resuscitation volumes required by all burn patients were greatly in excess of volumes predicted by the Parkland formula. TBSA indicates total body surface area. Data are expressed as mean \pm SEM. There were no significant differences between the groups.

ommended by the Parkland burn formula was exceeded regardless of resuscitation scheme and was not different between the 2 groups. Similarly, at 24 hours after burn, the volume of crystalloid required by patients in both groups significantly exceeded the Parkland formula recommendation (4 mL/kg per each 1% TBSA burned), but again there was no difference between groups.

Urine output measurements (Figure 1, right) were consistently above 0.5 to 1.0 mL/kg per minute during the first 8 hours of resuscitation for all burn patients. Urine flow rates diminished in both groups over the 24-hour study period, but there were no significant differences between the groups at any time.

METABOLISM

All patients, regardless of resuscitation scheme, maintained serum electrolyte profiles (sodium, potassium, chloride, glucose, osmolality) within the normal range (serum sodium, 150 ± 2 mEq/L [mmol/L], and plasma osmolality, 302 ± 12 mOsm/L [mmol/L]), for all patients throughout resuscitation [mean \pm SD]. Acidosis, hypoxia, hypercapnea, and hypocapnea were not observed during the resuscitation of any patient. All patients developed a mild thrombocytopenia, but platelet counts below $100 \times 10^9/L$ were not observed. All patients exhibited a moderate rise (1.2-1.5 times normal) in both prothrombin and partial thromboplastin times during resuscitation, with no differences between groups or clinical evidence of hemorrhage.

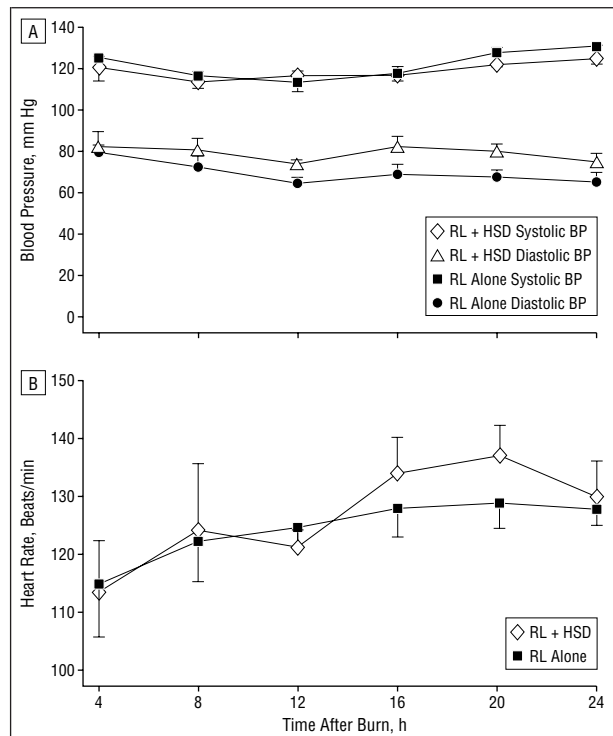


Figure 2. Arterial blood pressure (BP) measurements (A) and postburn heart rates (B) in patients who received Ringer lactate (RL) plus sodium chloride-dextran-70 (HSD) bolus vs RL alone. Data are expressed as mean \pm SEM. There were no significant differences between the groups.

HEMODYNAMICS

Systolic and diastolic blood pressures (Figure 2, left) were adequately maintained by both resuscitation regimens and did not differ between groups. On admission to the hospital, all patients demonstrated significant tachycardia. Over the 24-hour study period, all patients exhibited steadily increasing heart rates, but no differences were noted between the groups at any time (Figure 2, right). Examination of central venous pressures (Figure 3, A) and pulmonary capillary wedge pressure (Figure 3, B) demonstrated that filling pressures increased throughout resuscitation for all patients; however, central venous pressure and pulmonary capillary wedge pressure were lower in the patients treated with HSD throughout the entire study period compared with those treated with RL. This difference was significant at 8 and 12 hours after burn injury. Systemic vascular resistance (Figure 3, C) was slightly elevated early during resuscitation, then steadily declined over time, with no difference between groups.

CARDIAC FUNCTION

The cardiac index remained within normal limits (2.8-4.2 L/min per square meter) for all patients during the first 16 hours of resuscitation (Figure 3, D); at 20 hours after burn, all patients demonstrated a hyperdynamic cardiac response (cardiac index >4.2 L/min per square meter). Plotting stroke work as a function of filling pressure (pulmonary capillary wedge pressure) provided an assessment of relative cardiac contractility at specific times

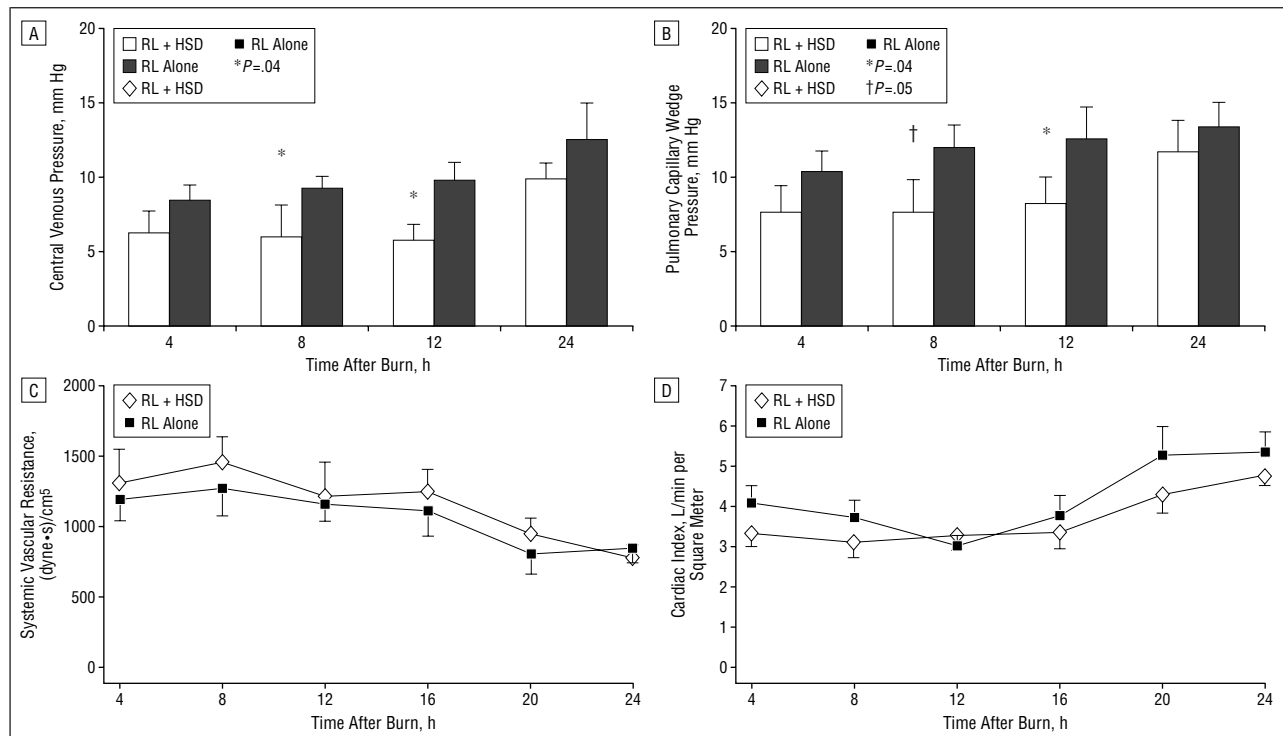


Figure 3. Central venous pressure (A) and pulmonary capillary wedge pressure (B) increased throughout the resuscitation period for all patients. Patients who received Ringer lactate (RL) plus sodium chloride–dextran-70 (HSD) demonstrated significantly lower filling pressures at 8 and 12 hours after burn compared with those who received RL alone. There were no significant differences between the groups in systemic vascular resistance (C) and cardiac index (D). Data are expressed as mean \pm SEM.

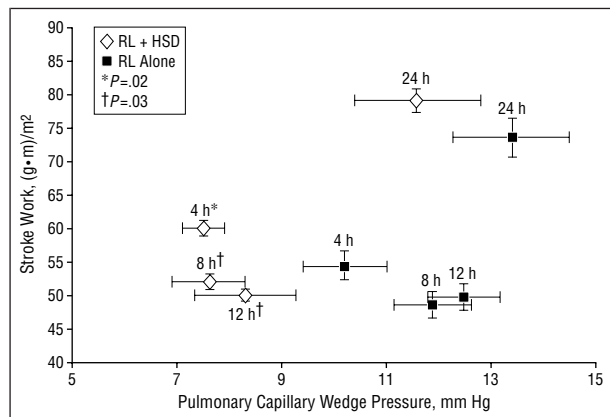


Figure 4. Cardiac contractile function during resuscitation as a function of the amount of stroke work [stroke volume \times (mean arterial pressure – pulmonary capillary wedge pressure) \times 0.0136] generated at a specific filling pressure. Patients who received Ringer lactate (RL) plus sodium chloride–dextran-70 (HSD) were able to generate stroke work similar to that of those who received RL only at lower pulmonary capillary wedge pressures by 4, 8, and 12 hours after burn. Data are expressed as mean \pm SEM.

after burn (**Figure 4**). This analysis demonstrated that patients given an HSD bolus maintained cardiac indices comparable with those of the patients treated with RL, although at significantly lower filling pressures. This discrepancy in cardiac function was apparent at 4, 8, and 12 hours but not 24 hours after burn.

Evaluation of serum creatinine kinase levels (**Figure 5**, A) and the creatine kinase MB fraction (**Figure 5**, B) demonstrated nonspecific postburn elevation of these serum markers for cardiac injury in all patients,

with no clear relationship between groups or time after burn. Serum levels of troponin I, a highly specific and sensitive serum marker of myocardial injury, were significantly elevated after burn in all patients regardless of the resuscitation regimen. Postinjury levels of troponin I were significantly elevated in patients treated with RL at 8 and 12 hours after burn compared with those treated with HSD (**Figure 5**, C).

COMMENT

Thermal injury is associated with significant fluid losses into burned and unburned tissue during the first 24 hours after injury. As a result, adequate resuscitation has been difficult to define, with significant morbidity associated with both underresuscitation and overresuscitation. If fluid resuscitation is not begun promptly and with adequate volume, shock and irreversible organ failure may result. With large-volume resuscitation of thermal injury there is significant extravasation of fluid from the intravascular to the extravascular space, often associated with significant weight gain and tissue edema.^{13,16} Possibly through this mechanism, large-volume resuscitation leads to deterioration of pulmonary and cardiovascular function, contributing to significant morbidity and mortality. This is of major clinical importance in patients with preexisting disease, such as elderly patients with cardiac disease, or in those who have sustained pulmonary trauma, as occurs with inhalation injury.¹⁷

While the common thread in all burn resuscitation regimens is the use of sodium-containing solutions, there is disagreement on the ideal sodium concentration.¹⁸⁻²¹

The most widely used burn resuscitation fluid is RL solution, a balanced, slightly hyponatremic salt solution (sodium, 130 mmol/L); significant systemic edema occurs after large-volume administration. The volume of required resuscitation in the first 24 hours is often calculated by use of a scheme described as the Parkland formula (4 mL of RL per kilogram of body weight per each 1% TBSA burned).²² Although there are several resuscitation formulas in use today, many employ large volumes of isotonic crystalloid solutions and delayed administration of colloid. Regardless of the resuscitation technique, severely burned patients frequently receive fluid volumes that exceed the prescribed guidelines.²³

In an effort to reduce the volume of fluid resuscitation, several animal and clinical studies have evaluated the efficacy of small-volume hypertonic salt resuscitation regimens. Horton et al¹⁰ have done extensive research in a burn shock model (guinea pig: 45% TBSA scald) using a small-volume intravenous bolus of HSD (4 mL/kg given over 30 minutes immediately after burn injury) as a supplement to standard RL resuscitation. Crystalloid resuscitation with HSD supplementation improved cardiac contractile performance, decreased cardiac myocyte injury, and reduced total fluid resuscitation volume 24 hours after burn. In 1973, Monafó et al²⁴ described resuscitation of 81 adult and 25 pediatric burn patients (burns >20% TBSA) with several concentrations of hypertonic salt solutions administered in a continuous fashion. The data indicated that resuscitation volumes required were 20% to 25% less than those calculated by the Parkland formula. These investigations concluded that hypertonic solutions provided a simple, safe, and efficacious resuscitation regimen in acute burns.

The clinical use of HSD given as an early bolus during the resuscitation of severe thermal injury remained unproved. Our choice of HSD given early during resuscitation was based on previous animal and clinical data indicating both safety and efficacy.^{10,24} Using maintenance of urine output as an end point to establish the adequacy of resuscitation and to assess end-organ perfusion, we evaluated the effect of HSD on postburn hemodynamics, biochemical profiles, cardiac function, and required resuscitation volumes. We were able to access and treat patients by an average of 3.5 hours after burn. Delays in initiating treatment were often related to prolonged transport times or an inability to obtain informed consent. Despite the relatively rapid (30 minutes) infusion of a solution of sodium chloride, 2400 mmol/kg per liter, we did not observe hypotension, hyperosmolality, or elevated sodium concentrations, as reported in other studies.²⁵ Patients treated with HSD demonstrated no evidence of renal insufficiency, urticaria, bronchospasm, or anaphylaxis, which have been anecdotally associated with HSD treatment.

From our studies of postburn resuscitation in animal models,^{6,7,10-12} we postulated that the early use of a hypertonic solution would reduce the total volume of resuscitation. Both standard RL and HSD resuscitation schemes adequately maintained urine output and were considered effective. We found significant differences between volume requirements predicted by the Parkland formula and the actual volumes infused in both groups,

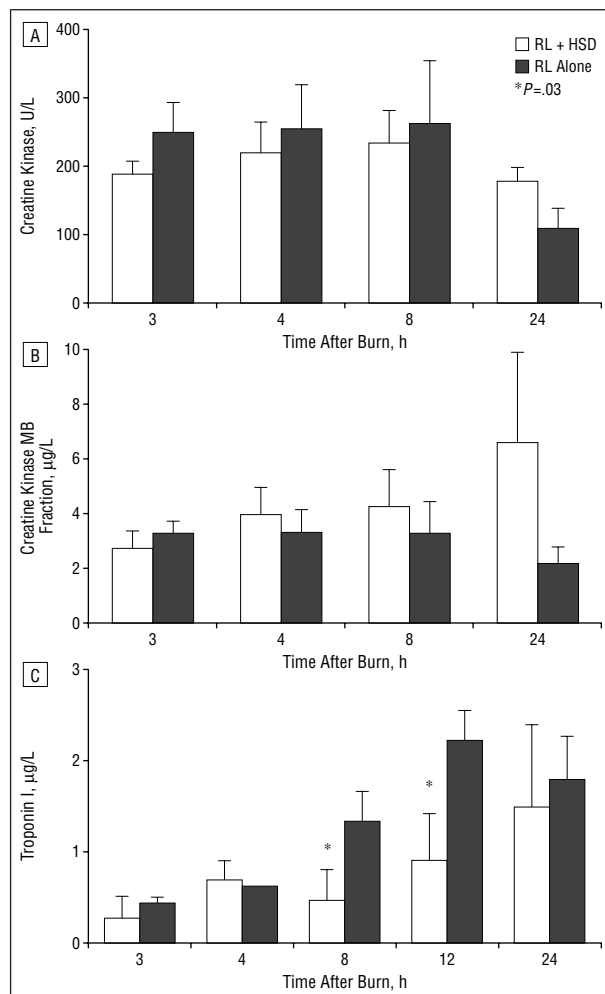


Figure 5. Serum markers of cardiac function. The creatinine kinase (A) and creatinine kinase MB fraction (B) levels demonstrated a nonspecific elevation throughout resuscitation, with no difference between groups. C, Troponin I was detectable in the serum of all burn patients who demonstrated mild postburn cardiac injury. At 8 and 12 hours after burn, patients who received Ringer lactate (RL) plus sodium chloride–dextran-70 (HSD) had significantly less detectable troponin I than those who received RL only. Data are expressed as mean \pm SEM.

a discrepancy that was especially profound during later stages of resuscitation. The exclusion of concurrent trauma, preexisting disease, age extremes, and inhalation injury eliminated these as explanations for the excessive volumes required. Both groups consisted of young, previously healthy individuals of comparable age and weight with large partial-thickness burns of similar size and full-thickness involvement. Although patients given HSD consistently required slightly less volume for resuscitation than those treated with RL, this difference was never significant.

The data clearly demonstrate that a 4-mL/kg HSD bolus failed to reduce resuscitation volumes as observed in the experimental setting. The aggressive resuscitation required by all patients (far in excess of volumes predicted by the Parkland formula) probably precluded any meaningful comparison of volume of resuscitation between groups. The beneficial effects of early HSD bolus that were so apparent in the laboratory were most likely offset in the clinical setting by the dilutional

effects of massive fluid administration. The large resuscitation volumes administered to both treatment groups were reflected in urinary flow rates greater than 1 mL/kg per hour at all stages of resuscitation. In contrast with many studies of HSD resuscitations, our clinical resuscitations were not controlled for experimental purposes or designed to meet particular experimental conditions. By protocol design, all postburn resuscitations proceeded at the discretion of the physician in attendance, not the principal investigators. The strict regulation of hourly urine outputs during a burn resuscitation is often both difficult and possibly dangerous outside the confines of a controlled laboratory setting.

TYPICALLY, DURING the resuscitation of any critically ill patient, extra fluid is administered as a bolus in response to transient changes in blood pressure or heart rate. It is apparent from our data that patients with severe thermal injury consistently received resuscitation fluid in excess of predicted volumes despite a lack of complicating factors. Given these unexpectedly high volumes, our study lacks sufficient statistical power to demonstrate any significant difference in resuscitation volumes between these groups. While this may represent a limitation of our study, we believe aggressive fluid administration during trauma and burn resuscitations occurs with greater frequency than is often reported in the literature.²³ Our findings, therefore, may represent a more accurate picture of the effects of HSD in common clinical practice as compared with rigidly controlled experimental settings.

Interestingly, patients treated with HSD were able to maintain an elevated cardiac index at lower filling pressures than the RL group, a difference that was statistically significant at 4, 8, and 12 hours after burn. These data suggest that maintenance of cardiac function at lower preload volumes was related to HSD-mediated preservation of postburn cardiac contractility. This cardioprotective effect was immediate, as demonstrated through later analysis of invasive monitoring measurements at 4 hours after burn (immediately following HSD treatment). The effect of HSD on cardiac function was also transient; by 24 hours after burn both treatment groups had similar cardiac indices and high filling pressures. These findings are consistent with previous reports describing the positive inotropic effects of sodium and osmolality on cardiac contractile function.²⁶ Wildenthal et al¹⁴ evaluated the effects of increased serum osmolality on left ventricular function and showed that acute hyperosmolality (400 mmol/kg) significantly increased the maximum rate of left ventricular pressure rise while the left ventricular end-diastolic pressure remained constant or decreased. Extensive animal research has been carried out in both burn and nonburn shock animal models evaluating the cardiac response to intravenous bolus hypertonic fluid resuscitation.³⁻⁶ These studies showed that a bolus of 7.5% sodium chloride (2400 mmol/L) transiently improved cardiovascular function after hemorrhagic shock.⁷ In a guinea pig burn shock model, an initial bolus of 7.5% sodium chloride followed by ad-

ministration of 1 mL of RL per kilogram per each 1% of TBSA burned given as a constant infusion also provided significant cardiac protection.^{10,11} More recently, Horton et al¹² addressed the question as to whether delaying HSD administration up to 8 hours after burn injury would provide cardioprotection after burn trauma. This study confirmed that HSD given within 6 hours of burn trauma ablated postburn cardiac contractile defects. Horton et al further concluded that HSD-mediated cardioprotection was not related solely to a shift of fluid from intracellular to extracellular compartments, but also involved HSD-associated alterations in sodium-calcium exchange and a functional enhancement of the calcium-regulated cardiac contractile apparatus. Although the serum sodium and osmolality levels following an HSD bolus were transiently increased in experimental models, this was not detectable in the serum of any burn patient. If a transient rise in the serum sodium level does trigger a calcium-driven enhancement of cardiac function, then this mechanism of action may account for HSD preservation of postburn cardiac function in the face of normal serum sodium and osmolality levels.

Severe thermal injury has also been associated with the formation of oxygen-derived free radicals, which are thought to play a role in the development of local and remote tissue injury. The cardiodepressive effects of free radicals are well recognized, and free radical scavengers have been shown to reduce cardiac injury and improve function. Several studies have also demonstrated that HSD administration significantly reduces postburn oxidant-induced lipid peroxidation from systemic and mesenteric circulation, possibly as a result of free radical scavenging and anti-inflammatory and vasodilatory properties.^{5,13,15} Whether HSD preservation of postburn cardiac function is related to a reduction in systemic or cardiac free radical concentrations will require further investigation.

Troponin I, a regulatory protein found in the muscle contractile apparatus, has been shown to be a very sensitive and specific serum marker of cardiac injury in several clinical settings.²⁷ It is a highly specific indicator of focal cardiac ischemia as well as global myocardial stress after a severe systemic stress.²⁸ Recent animal studies have shown that measurement of troponin I provided accurate assessment of the degree of cardiac dysfunction after thermal injury.^{29,30} While serum creatine kinase and creatine kinase MB fraction levels are often elevated after traumatic and burn injury, they are primarily related to mechanical or burn-mediated damage of underlying skeletal muscle and are not related specifically to cardiac injury. The cardiospecificity of troponin I provides unequivocal evidence of myocardial injury with the appearance of this biochemical marker in the systemic circulation. While none of our patients had demonstrable cardiac failure, infarction, arrhythmia, or focal ischemia during the resuscitation period, all had severe and worsening tachycardia under conditions of rapid volume expansion. Circulating troponin I levels were elevated by 4 hours after burn and remained elevated for the entire 24-hour resuscitation period in all burn patients. At 8 and 12 hours after burn, troponin I levels were significantly

lower in the HSD resuscitation group compared with the RL group. These data suggest that severe cutaneous thermal injury in previously healthy individuals induced a mild degree of detectable cardiac injury. Also, the data show that HSD administered early during crystalloid resuscitation diminished the burn-associated rise in troponin-I levels. Diminished postburn serum troponin I levels correlated temporally with the preservation of postburn cardiac contractile function following HSD administration. However, both HSD-associated improvements in cardiac contractility and diminished troponin I levels seemed to be transient. This may reflect the effect of a single bolus HSD infusion and may be related to rapid dilution by an overwhelming RL infusion. A more sustainable effect on postburn cardiac function may be possible with continuous administration of HSD. In the future, the subtle derangements of cardiac physiology demonstrable in burn patients with invasive hemodynamic monitoring, confirmed by detection of troponin I, may be more clearly defined by sensitive noninvasive monitoring modalities, such as bedside echocardiography.

CONCLUSIONS

We compared a standard postburn resuscitation scheme using RL with a regimen in which RL was supplemented with an early bolus infusion of HSD. Both resuscitation plans were adequate and efficacious in maintaining postburn perfusion. The absence of deleterious hemodynamic or metabolic side effects following HSD infusion in patients with major thermal injury confirms the safety of this resuscitation strategy. Postburn cardiac dysfunction was demonstrated in all patients through the use of cardiospecific serum markers and pulmonary artery catheter monitoring. Early administration of HSD after a severe thermal injury seems to have reduced burn-related cardiac contractile dysfunction through an unknown mechanism. Given these findings, we are encouraged to further study the use of HSD as an adjuvant to crystalloid resuscitation in the patient populations at highest risk for postburn cardiac dysfunction, such as elderly patients with cardiac disease, those with multiple injuries, and those with direct pulmonary injury, as occurs with smoke inhalation.

This study was presented in part at the annual meeting of the American Burn Association, Nashville, Tenn, March 16, 1996.

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