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Recovery From Postoperative Hypothermia **Predicts Survival in Extensively Burned Patients**

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To clarify the cause of postoperative hypothermia in extensively burned patients, factors affecting postoperative hypothermia were studied in 16 extensively burned adult patients (8 survivors and 8 nonsurvivors) with a burn index greater than 35. Body temperature was monitored continuously in either the urinary bladder or rectum. Hypothermia of less than 35°C occurred in 89% (66 of 74) of the total operations performed in these 16 patients. The rate of temperature rise (RTR) was significantly lower in nonsurvivors $(0.4 \pm 0.2^{\circ}\text{C/h})$ than in survivors $(1.7 \pm 0.9^{\circ}\text{C/h}; p < 0.001)$. Continuous indirect calorimetry performed in seven patients (four survivors and three nonsurvivors) demonstrated that RTR was determined primarily by heat production. The measured energy expenditure reached only 1.7 ± 0.2 times the basal energy expenditure during rewarming in nonsurvivors, whereas it was 2.7 ± 0.9 times the basal energy expenditure in survivors (p <0.01). Surprisingly, in nonsurvivors, the RTR was significantly decreased even during the first 2 weeks. These findings suggest that those who cannot generate heat well in postoperative hypothermia are unable to produce the additional energy required to overcome sepsis.

ypothermia, defined as a core temperature less than or equal to 35°C, is an inevitable complication of any operation with general anesthesia [1,2]. Extensively burned patients, in particular, often experience intraoperative and subsequent postoperative hypothermia. We have known empirically that those patients who have difficulty in rewarming will usually succumb to the burn injury [3]. However, little attention has been paid to this problem. In this study, we investigated the significance of prolonged postoperative hypothermia in extensively burned patients.

PATIENTS AND METHODS

Patients: Sixteen extensively burned adult patients with a burn index exceeding 35 were studied. The burn index was calculated as the second-degree burned body surface area/2 + the third-degree burned body surface area [4]. These burn patients were admitted to the Department of Traumatology of Osaka University Hospital from October 1985 to April 1991. All patients had been in good general health before injury. Eight of the patients died (mean survival: 58 ± 26 days). Informed consent was obtained from each patient prior to being entered in

Operation and postoperative management: The 16 patients underwent a total of 74 débridements and/or skin graft operations. All patients were rewarmed similarly during routine postoperative management in a warm environment (38.0°C) by an air-fluidized bed (Clinisystem UA 101-D, Tokyo, Japan), thermal blankets, and fluid warmers.

Measurement: Body temperature was monitored continuously in the urinary bladder or rectum preoperatively, intraoperatively, and postoperatively. The admission temperature was recorded at the time of admission to our intensive care unit after operation.

In seven patients (four survivors and three nonsurvivors), indirect calorimetry was performed continuously during rewarming using a breath-by-breath system (System RM300, Minato Medical Science, Osaka, Japan) [5]. All of these patients were intubated or had undergone tracheostomy and were mechanically ventilated for at least 12 hours after surgery. The calorimeter was attached directly to the ventilator.

Data analysis: To further assess the physiology of postoperative hypothermia, we defined two parameters. The recovery time was defined as the length of time in hours required for a patient's core temperature to rise to 37.0°C. The rate of temperature rise (RTR) was defined as shown in Figure 1.

The energy expenditure was calculated using a modification of Consolazio's equations as follows [6]: MEE =

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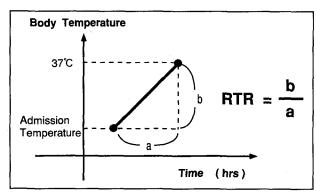


Figure 1. Definition of rate of temperature rise (RTR).

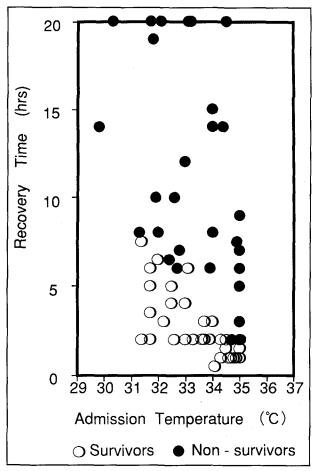


Figure 3. Relationship between the admission temperatures and recovery times. No correlation was observed between those two parameters. However, this population seems clearly to be divided into two groups: survivors and nonsurvivors.

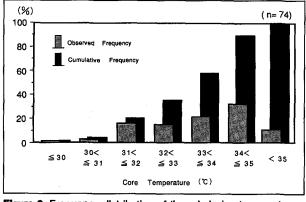


Figure 2. Frequency distribution of the admission temperatures. Postoperative hypothermia with temperature less than or equal to 35.0°C was seen in 89% (66 of 74) of all operations.

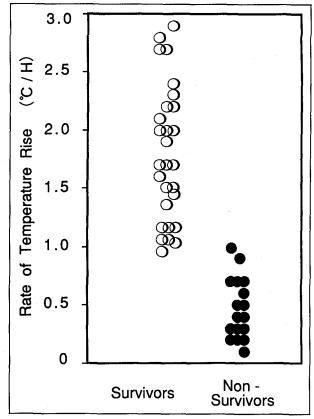


Figure 4. Rate of temperature rise (RTR) in the two groups. RTR values were significantly lower in nonsurvivors than in survivors (p <0.001).

 $[3.796 (\dot{V}O_2) + 1.214 (\dot{V}CO_2)] \times 1440$, where MEE = measured energy expenditure (Cal/d), $\dot{V}O_2$ = oxygen consumption (L/min), and $\dot{V}CO_2$ = carbon dioxide production (L/min).

The results of indirect calorimetry were compared with basal energy expenditure (BEE), which was calculated using the Harris-Benedict formulas as follows [7]:

for males: BEE = 66.47 + 13.75(W) + 5(H) - 6.76(A); and for females: BEE = 655.1 + 9.56(W) + 1.85(H) -4.67(A), where W = weight (kg), H = height (cm), and A = age (years).

Statistical analysis: All values are expressed as the mean \pm SD. The data were analyzed by the Mann-Whitney test. Significance was designated as p < 0.05.

TABLE I
Difference in Patient Characteristics Between
Subgroups*

	Survivors	Nonsurvivors	Signif- icance [†]
No. of patients	8	8	
Age (y)	43.1 ± 17.2	44.5 ± 15.5	NS
Sex			
(Male/female)	5/3	5/3	
Body weight (kg)	61.6 ± 4.3	60.8 ± 5.8	NS
Burn index	54.6 ± 15.9	66.9 ± 13.8	NS
Incidence of inhalation injury	4/8	4/8	
No. of operations	40	34	

NS = not significant.

TABLE II

No Parameter Studied Seemed to Affect the RTR

in Either Group*

	Survivors	Nonsurvivors	Signif- icance [†]
Admission temperature (°C)	33.5 ± 1.2	33.3 ± 1.4	NS
Nonepithelialized area in each operation (%)	39.2 ± 21.6	48.0 ± 19.2	NS
Mean area operated on in each operation (%)	13.8 ± 7.4	14.8 ± 8.6	NS
Length of operation (min)	217 ± 55	199 ± 71	NS
Amount of bleeding during operation (mL)	1,490 ± 1,160	1,690 ± 1,510	NS
Amount of fluid infused and transfused during operation (mL)	3,740 ± 1,340	3,960 ± 2,730	NS

NS = not significant.

[†]Significance was calculated by the Mann-Whitney test.

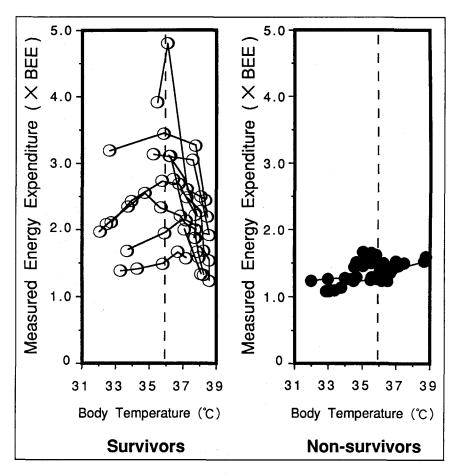


Figure 5. Energy expenditure during rewarming in the two groups. The measured energy expenditure (MEE) increased greatly during rewarming in survivors, whereas it increased gradually with no significant peak in non-survivors (p <0.01). BEE = basal energy expenditure.

RESULTS

Frequency of hypothermia: Figure 2 presents the frequency distribution of the admission temperatures. Indeed, postoperative hypothermia with a temperature less than or equal to 35.0°C occurred in 89% (66 of 74) of the total operations.

Outcome and RTR: The lack of correlation between the admission temperatures and the recovery times is depicted in Figure 3. However, this population seems clearly to be divided into two groups: survivors and nonsurvivors.

Figure 4 shows the RTR values for the two groups.

^{*}Values are expressed as the mean ± SD.

[†]Significance was calculated by the Mann-Whitney test.

^{*}Values are expressed as the mean ± SD.

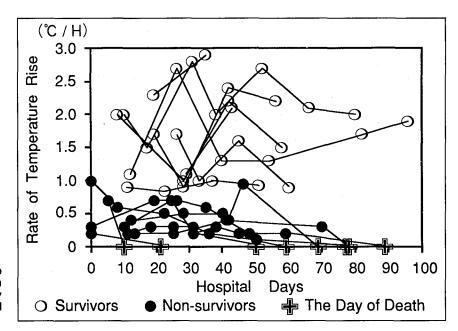


Figure 6. Rate of temperature rise (RTR) during the clinical course. In nonsurvivors, the RTR was already decreased significantly by the second week after the burn injury.

The RTR was significantly lower in nonsurvivors (0.4 \pm 0.2°C/h) than in survivors (1.7 \pm 0.9°C/h; p <0.001).

Subgroups of patients: The two groups were similar with respect to age, body weight, and burn index (Table I). The presence of an inhalation injury showed no correlation with the RTR.

Other factors that seemed to affect the RTR were also compared between the two groups (Table II). However, no significant difference was found. In other words, these factors did not account for the difference in the RTR values between the two groups.

Energy expenditure: Figure 5 illustrates the energy expended during rewarming. In survivors, the MEE increased remarkably and reached a peak value of 2.71 ± 0.86 times the BEE at about 36.0° C during rewarming. In nonsurvivors, however, the MEE increased slowly with no significant peak and increased to only 1.65 ± 0.18 times the BEE during rewarming (p <0.01).

RTR values during the clinical course: Figure 6 shows the RTR values during the clinical course. In non-survivors, surprisingly, the RTR had already decreased significantly in the first 2 weeks. In nonsurvivors, therefore, the RTR decreased gradually throughout their clinical course.

COMMENTS

In an attempt to prevent intraoperative hypothermia, anesthesiologists have used several techniques: higher ambient air temperature, warming blankets, administration of warmed fluids and blood, and heated humidified gases. Intraoperative hypothermia, however, has been almost inevitable in most operating rooms [1,2,8]. This is simply because anesthesia disables the normal physiologic mechanism by which thermal balance is maintained and makes patients poikilothermic [9,10]. Our results confirmed these observations.

The adverse effects of postoperative hypothermia are

well known [9,10]. There is little known, however, of any relationship between postoperative hypothermia and the patient's prognosis. Slotman et al [11] reported only on mortality correlated with the magnitude of hypothermia. Therefore, to further assess such a correlation, we compared the RTR values in survivors to those in nonsurvivors given that the two groups were similar with regard to age, body weight, burn index, and the type and number of operative procedures. Our results indicate that the RTR was significantly lower in nonsurvivors than in survivors. We could not find, however, any factor that accounted for the difference in the RTR values between the two groups.

The RTR is influenced by three major factors: (1) method of rewarming, (2) heat loss, and (3) heat production. Each of these factors will be discussed below:

- (1) Method of rewarming: All patients were rewarmed identically during rewarming. Moreover, many studies have demonstrated that warming devices are less effective than we expected [2,12-14]. The method of rewarming, therefore, seems to have had little influence on the RTR values in our study.
- (2) Heat loss: During rewarming, heat loss occurs by radiation, conduction, convection, and evaporation. Since all patients were managed in an identical environment during rewarming, patients did not differ with respect to heat losses by radiation, conduction, and convection. In extensively burned patients, however, evaporative heat loss through the nonepithelialized area influences the RTR markedly [15]. From our data, however, no relationship could be seen between the RTR and the amount of nonepithelialized area existing at the time of the operation. Heat loss, therefore, does not seem to explain entirely the difference in the RTR values between the two groups.
- (3) Heat production: Given the previous discussion, we believed that neither the method of rewarming nor heat loss had much effect on RTR values. Consequently,

we focused on heat production. As shown in Figure 5, the difference in heat production between the two groups is enough to account for the difference in the RTR between the two groups. In other words, among the three major factors, only the difference in heat production could explain the difference in the RTR values between the two groups. We believe, therefore, that the RTR is affected primarily by heat production.

Body temperature is maintained within a narrow range (36.0°C to 37.5°C) by a powerful regulating system [9,15]. Flacke [16] noted that the demands of temperature regulation take precedence even if they compete with the requirements of other important homeostatic mechanisms. Therefore, it is important to emphasize that postoperative hypothermia is a potentially life-threatening stress for extensively burned patients.

From the above considerations, we think that the decrease in the RTR is due primarily to the patients' inability to increase their heat production. Those who cannot respond to postoperative hypothermia may not have the energy reserve required to respond to the other stresses of burn injury. Postoperative hypothermia may be a test to estimate the patient's ability to respond to other stress. In other words, patients who cannot respond to the stress of hypothermia may not be able to overcome other stresses (infection, hemorrhage, operation, etc).

Our results (Figure 6) support this speculation; in nonsurvivors, the RTR was already significantly decreased in the first 2 weeks, and all of these nonsurvivors consequently died of sepsis. From these data, we suggest that the RTR during rewarming is useful to predict the patient's ability to respond to additional stress.

We have begun to investigate why nonsurvivors cannot generate heat in hypothermia; we are currently exploring this question by studing how carbohydrates and lipids may be restricted and the role of circulatory failure. According to Fick's equation, oxygen consumption is the product of cardiac output and arteriovenous oxygen content difference. It is still unclear which of the two has greater influence on the reduced oxygen consumption in nonsurvivors.

This study demonstrates that postoperative heat production was impaired in nonsurvivors even during the first 2 weeks when there was no evidence of sepsis. Thus,

we suggest that the RTR after operation could serve as an index of the patient's prognosis. Further study of the two different responses during postoperative hypothermia is warranted.

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