The Effect of Occlusive Dressings on the Energy Metabolism of Severely Burned Children

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Metabolic studies were performed on 23 burned children. They were studied sequentially until their burn wounds were healed. A metabolic study lasted 20 minutes, during which continuous measurements were made of O_2 consumption and CO_2 production rates, rectal temperature, average surface temperatures (dressings, skin and wound), body heat content, and rate of body weight loss using a bed scale. These measurements allowed solution of the heat balance equation for each study period. After 24 hours in a constant temperature room kept at 28 C and 40% relative humidity, metabolic studies were initiated when blood was drawn for catecholamine assay, followed by a metabolic analysis, after which dressings were removed and fresh silvadene applied to the wounds. No dressings were applied. Metabolic analyses were repeated after two and four hours of exposure, after which blood for catecholamine analysis was drawn and the study terminated. Without dressings in a thermally neutral environment, burn patients demonstrated an increased rate of heat loss of 27 watts/square meter body surface area (W/M^2), compared with the predicted normal. The major portion of this increment is by evaporation, which increased 300%. The rate of heat production equals heat loss, and is increased 50% above the predicted normal. Occlusive dressings result in a 15 W/M^2 decrease in the rate of heat loss, about evenly divided between evaporative and dry routes, with a corresponding 15 W/M^2 decrease in the rate of heat production. Plasma catecholamine levels of bandaged burn patients are not significantly different from values for healed burn patients, and do not correlate with the rate of heat production. The increased heat production of burn patients is a response to an increased rate of heat loss, not vice versa. The use of occlusive dressings substantially reduces the energy requirements to manageable levels, even in patients with very large burns.

SINCE THE ORIGINAL observation, almost 30 years ago, that patients with large thermal burns were hypermetabolic but not hyperthyroid, investigators have attempted to elucidate the pathophysiology of this phenomenon.[8] Simultaneous direct and indirect calorimetric studies on burned rats showed that they were hypermetabolic, and that the increase in their metabolic rate was secondary to increased evaporative heat loss through the burn wound.[7] Studies of burned humans have produced inconsistent results. However, investigators agree that burned humans are hypermetabolic and that they demonstrate increased evaporative heat loss. In addition, there is agreement that the increases in both heat production and heat loss are proportional to the size of the burn wound. Results from burned humans are ambiguous as to whether increased heat production, under control of the central nervous system, activates a secondary "convenient and corresponding" increase in heat loss, or whether increased heat loss from the burn wound and unburned skin drives a centrally controlled appropriate increase in heat production in order to maintain homeostasis. There is also disagreement as to the primary or secondary role of catecholamines in the cause and maintenance of burn hypermetabolism. Finally, there is disagreement and/or lack of information as to whether burned patients are febrile or hyperthermic and what, if any, is the impact of a chronic increase in body temperature upon the metabolic rate of the burned patient.

Most investigators agree that these questions are fundamentally important in the care of patients with major thermal injuries, for one of the major determinates of successful outcome in the treatment of patients with large burns is the maintenance of an excellent nutritional state. In order to do this, the clinician must meet the unmodified increase in nutritional demands of the burn patient, or else develop treatment methods which lower the nutritional requirements of the patient without compromising principles of good wound care.

The present studies were designed and performed to investigate several of these questions.

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TABLE 1. *Characteristics of the Three Groups of Patients*

	All Burn Patients	≥20% BSA Burn	Healed Burn Patients
Number	23	16	24
Mean age	11 ± 6	11 ± 7	10 ± 6
Median age	9	14	7
Mode age	18	18	14
Mean %* BSA burn	8/29	10/38	11/32

* Full thickness/total area of burn.

Materials and Methods

The study population consisted of 23 burned children and young adults, treated at the Arkansas Children's Hospital Burn Center, with the group characteristics summarized in Table 1. Patients were studied sequentially until their burn wounds were healed. Twenty-four patients with healed burns were studied as a control group. Each experiment upon an individual patient started at 8:00 A.M., when the study subject was placed in a constant temperature room maintained at 28 ± 0.1 C and 40 ± 10% relative humidity, with air movement less than 0.25 m/sec. These environmental conditions meet the requirements for thermal neutrality for nude, uninjured man. Just before entering the study room, the patient's dressings had been changed. Dressings consisted of 12 layers of coarse gauze held in place with Kerlex® rolls. The dressing had an estimated insulative value equal to 0.75 clo. A clo unit allows heat transfer of 5.55 kcal/$M^2 \cdot$ hr or 6.45 W/$M^2 \cdot °C$ temperature gradient across the insulation in question. Thus, a dressing with a clo value of 0.75 would allow heat transfer of 8.6 W/$M^2 \cdot °C$ temperature gradient across the dressing.[12] All burn wounds were treated once or twice daily with dressing change, atraumatic debridement, and the application of fresh Silvadene® or Silvadene® cerium. The study subjects remained in the constant temperature room for 24 hours, while normal nursing care was maintained.

At 8:00 A.M. the second morning, at least eight hours postprandial, metabolic studies were begin. Approximately one half of the study subjects were premedicated one hour before study with 10 mg of diazepam, 10 grains of aspirin and 30 ml of an antacid. Three patients were administered cath mixture by mouth equal to 0.5 ml/kg of body weight. Cath mixture contains 15 mg meperidine, 5 mg chlorpromazine HCl, and Promethazine 5 mg/ml.

Baseline venous blood was obtained for catecholamine determination with patients fasted, quiet, and supine overnight. A metabolic study period was arbitrarily determined to be 20 minutes. Skin and dressing temperatures were determined using a Stoll/Hardy® radiometer, and core temperature with a hospital thermometer or thermocouple. For metabolic measurements, the patient's head and neck were enclosed in a hood, through which outside air was drawn at a rate of 40–45 L/min, then through a hygrometer and flowmeter, after which the air stream was split and 250 ml/min passed through O_2 and CO_2 meters, after which all flow was exited to the room. Details of the indirect calorimetry plumbing and gas analysis have been reported.[7] The patient remained supine on a hospital bed which was mounted on a Potter bed scale, allowing continuous measurement of the rate of body weight loss. The rate of evaporative heat loss was calculated directly from the rate of body weight loss, using .674 W as the heat of vaporization of each gram of water. Dry heat loss was calculated by three different methods: 1) radiational heat loss using the law of Stefan Boltzmann[17]:

$$Q_R = \sigma B_1 B_2 (T_1^4 - T_2^4) \quad (1)$$

where:

Q_R = rate of radiation in Watts
σ = universal radiation constant 5.7×10^{-12} W/cm^2 per $(°K)^4$
B_1, B_2 = blackness of radiating surfaces
T_1, T_2 = temperature of radiating surface in °K

The remainder of the dry heat loss was determined by difference: (heat production − evaporative heat loss − radiating heat loss = conductive and convective heat loss where body heat content remains constant). 2) Dry heat loss was determined using a combined transfer coefficient,[16] 6.83 W/$M^2 \cdot °C \cdot$3) Dry heat loss was determined by difference alone: heat production − evaporative heat loss = dry heat loss, where body heat content remains constant.

The heat loss coefficient for radiation and convection was calculated from the formula[24]:

$$h_O = \frac{M \pm \Delta S - E_V}{T_s - T_a} \quad (2)$$

where

M = heat production
ΔS = change in body heat content
E_V = evaporative heat loss
T_s = average surface temperature of patient
T_a = ambient temperature.

From the skin, dressing and core temperatures, the average skin and surface temperatures were calculated using the percentage regional representation proposed by Hardy and DuBois.[15] Average body temperature weighed 80/20 rectal/skin, and a specific heat of .976 W was used to calculate body heat content.[24] The rate of heat production was calculated from the rate of O_2 consumption; the respiratory quotient was calculated

from the volume of CO_2 produced divided by the volume of O_2 consumed per unit time. Formulae for these three calculations are those proposed by Lister.[19] All signals were interfaced with a Digital Equipment Corp.® PDP 11-VO3 computer, for which a specific program was developed to permit the computer to perform, on line, all data alterations, such as summations, amplifications and calculations in real time. This allows a single operator to collect data and be completely current in data processing. Data collected manually, such as radiometer temperatures and barometric pressures, etc., were given to the computer by manual entry through the console. The gas analysis portion of this system was found to be accurate to ±1–2% when burning absolute alcohol in the hood.

The data collected allowed solution of the heat balance equation for each metabolic run:

$$M = E_V + Q_R + Q_C \pm \Delta S \quad (3)$$

where:

M = metabolic rate
E_V = evaporative heat loss
Q_R = radiational heat loss
Q_C = convective heat loss
ΔS = change in body heat content.

Following the first 20-minute metabolic period, during which the patient was undisturbed, fasted, and dressed, the dressings were removed and fresh Silvadene was placed on the burn wounds, and dry sheets placed on the bed. During most experiments, at this point the patient was offered and assisted in eating a regular hospital breakfast. Sequential metabolic analyses were completed at two and four hours after removal of the dressing. With the completion of four hours of wound exposure, venous blood was drawn for catecholamine analysis, and the study terminated with the application of fresh topical ointment and a dressing to the burn wound. The differences between means for data collected from the 16 patients with total burn area greater than 20% of the body surface (10/38 = mean area of full thickness injury over mean total area) were treated for statistical significance using Student's standard t-test and for paired data. The null hypothesis was rejected at the 0.05 level of probability.

Data from all patients were analyzed by computer to determine that linear regression best fit the data. Linear regressions were compared with one another for statistical significance using the method described by Fisher.[11]

Results

Heat Balance of Burned Patients with and without Dressings at an Ambient Temperature of 28 ± 0.1 C and 40% Relative Humidity

The mean values for the heat balance equation for the 16 large burns are summarized in Table 2. The rates of heat production and evaporative heat loss can be measured within 5% error, but the accuracy of measurement of the other components of equation 3 have larger and greatly variable error. Thus when ΔS is small, no matter what the sign, the equation can be solved rather confidently by calculating convective loss by difference. Where ΔS is small, most of the error will be in the distribution of dry heat loss between radiation and convection.

After 24 hours bandaged, resting quietly, comfortable and fasted, the averaged rate of heat production of the 16 burn patients with large burns was 66 W/M², which is 12.4 W/M², or 23%, above the predicted normal for the group[5] ($p < 0.001$) and 8 W/M², or 14%, above the average value for the healed burns ($p < 0.01$). Sixty-eight per cent of the total heat production is transferred to the environment by a 237% increase in evaporative heat loss equal to 45 W/M²,

TABLE 2. *The Mean Values for the Heat Balance Equation for the Burned Patients, Bandaged and Exposed, and the Patients with Healed Burns*

	Number	Hp*	=	E_V	+	Q_r	+	Q_c	+	ΔS
Burned patients bandaged	16	66.0 (±8.1)†	=	45.1 (±8.2)	+	21.6 (±3.0)	+		+	0.2 (±6.3)
Burned patients exposed	16	81.4 (±9.6)	=	51.8 (±11.7)	+	30.1 (±5.6)	+		+	2.0 (±10.4)
Healed burned patients	27	58.0 (±8.4)	=	20.0 (±4.7)	+	24.5 (±4.2)	+	15.8 (±10.4)	−	2.0 (±4.9)
Predicted normal	16	53.6 (±6.7)	=	13.4 (±1.7)	+	32.2 (±4.0)	+	8.0 (±1.0)		0

† Mean values ± standard deviation.

* Heat production and loss expressed as Watts/M², E_v = evaporative heat loss, Q_r = radiation heat loss calculated from the Stefan-Boltzmann equation, Q_c = convective heat loss calculated by difference, ΔS = change in body heat content.

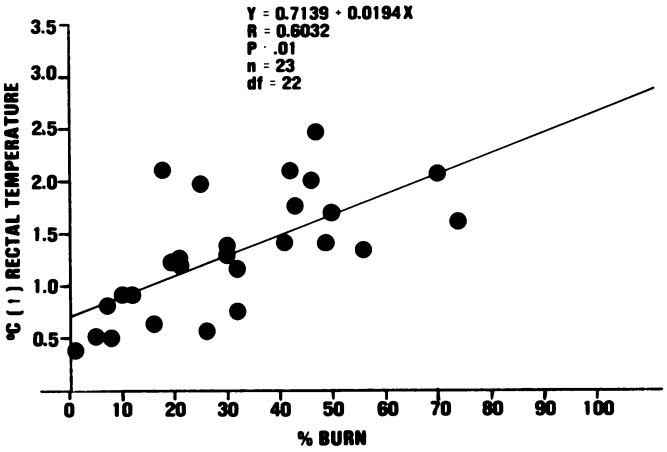

FIG. 1. Correlation between per cent BSA burn and °C increase in rectal temperature for bandaged patients at 28 C.

while only 33% (22 W/M²) of the heat is lost by the dry route (mainly by radiation according to these calculations) compared to normal heat exchange where 70–75% of the heat produced is lost by radiation and convection and 25% by nonregulatory evaporation. After only four hours without dressings, the exposed burned patients demonstrated a further increase in their average rate of heat production to 81.4 W/M², which is 27.8 W/M² or 52% above predicted normal for the group ($p < 0.001$) and 23 W/M² or 40% above the average value for the healed burn group ($p < 0.001$, Table 2). The corresponding increase in the rate of heat loss associated with dressing removal is about equally divided between increases in evaporative and radiational heat loss. Even exposed the heat loss by radiation and convection of burned patients amounts to only 37% of the heat production, while evaporation accounts for 63% of the heat transfer.

These data show that burned patients respond normally to an increase in their rate of heat loss following dressing removal with a corresponding increase in their rates of heat production. The major portion of the increased rate of heat production following thermal trauma is driven by an uncontrolled increase in evaporative heat loss occurring through the burn injury. Further, this is under normal central thermoregulatory control. These data do not support the notion that a *de novo* centrally driven increase in the rate of heat production occurs following thermal injury and that increased nonregulatory evaporative heat loss is just a convenient route to dispose of the increased heat production.

The Role of Increased Body Temperature in the Production of the Hypermetabolism Following Thermal Injury

There is a chronic elevation of body temperature following thermal injury which is directly proportional to the size of the burn injury for bandaged patients (Fig. 1). Whether this elevation is fever or hyperthermia, primary or secondary, is moot and will be discussed later. These questions in no way alter the metabolic consequences of chronic elevation of the body temperature. Both in animals and man, elevation of body temperature is associated with approximately a 13% elevation in the rate of heat production per degree elevation of body temperature.[10,22] This is equivalent to a whole body Q_{10} of 2.3. Most clinical states associated with sustained increase in the rate of heat production, are associated with elevation of the body temperature. Exercise and hyperthyroidism are two examples. Thus, there is reasonable doubt if the rate of heat production can be increased very much without an associated increase in body temperature. Both bandaged and exposed, the 16 burn patients with injury to more than 20% of the BSA demonstrate a 1 C increase in average rectal and body temperatures, and a 1.8 and 1.4 C increase in average skin temperature compared with the healed burn group ($p < 0.01$), while the differences between bandaged and exposed measurements for rectal, skin and average body temperatures were not significant. Only the average surface temperature was significantly different for these patients comparing bandaged and exposed condition (1.8 C, $p < 0.001$). This difference is secondary to the cooler surface temperature of dressings when compared with burned and unburned skin of burn patients, and accounts for the increase in radiational heat loss when bandages are removed. If the rate of heat production of the bandaged patients is adjusted for the effect of increased body temperature using a Q_{10} of 2.3, the remaining 3.9 W/M² or 7% increment in heat production rate is not significantly different from the predicted normal average for the group.

These calculations for the metabolic effect of fever show that 34% of the increase in heat production while exposed and 68% while bandaged can be accounted for by the increase in body temperature. This effect of fever appears additive to increased evaporative and dry heat losses, and interaction cannot be excluded; that is, fever surely increases both evaporative and dry heat loss of the burned patient.

Plasma Catecholamine Levels of Bandaged, Exposed and Healed Burn Patients

Plasma catecholamine estimates were performed by the Upjohn Company using a radioenzymatic assay. The mean values for plasma adrenaline (Ad) and noradrenaline (Nad) of bandaged burned patients while quiet and supine, were not significantly different from mean values for the same patients after healing of burn wounds had occurred.

TABLE 3. *Comparison of Plasma Catecholamine Levels of Bandaged and Exposed Burned Patients with Their Paired Values after Healing was Complete*

	Bandaged Burn Patients	Healed Burn Patients	Exposed Burn Patients
Plasma adrenaline	99 ± 73* — p—NS	67 ± 39* — p < .05	251 ± 214*
Plasma noradrenaline	598 ± 273* — p—NS	438 ± 237* — p < .01	1171 ± 635*

N = 8.
* Mean values ± standard deviation in pg/ml. Statistical comparisons were performed using the t test for paired samples.

On the contrary, plasma Ad and Nad concentrations of these same patients after four hours of wound exposure at 28 C were significantly increased compared with values of these patients after healing was complete (Ad 275% increased $p < 0.05$, Nad increased 167% $p < 0.01$, Table 3). Further, both plasma Ad and Nad concentrations for the burned patients after exposure were significantly increased from values obtained just four hours prior in the same patients with intact dressings in the same environment (Ad increased 115% $p < 0.02$, Nad increased 72% $p < 0.01$, Table 4). No normal values for the age range of the study patients are available. The normal adult range of Ad and Nad values provided by Upjohn are shown in Table 4.

These data show that plasma concentrations of catecholamines for burned patients respond quickly to the overall stress imposed on the patient, strongly suggesting a secondary nonspecific response to stress rather than a primary afferent or efferent limb in the cause or effector arc of the hypermetabolism following thermal trauma.

Catecholamines, particularly Ad, given parenterally produce increases in the rate of heat production. Ad may produce up to a 30% increase in the rate of heat production, while Nad does not produce more than a 10% increase.[13] Rats with no adrenal medulla are hypermetabolic following thermal injury,[6] and complete alpha and beta block in burned man only lowers the hypermetabolic state by about 30%.[25] These studies show that serum Ad and Nad reflect the total stress placed upon the patient, are quite labile, and are characteristic of a nonspecific secondary response to thermal trauma.

Heat Loss Coefficients for Burn Patients Bandaged, Exposed and after Complete Healing has Occurred

The heat loss coefficient (h_0) is calculated using equation 2. This equation uses the actual calculated quantity for dry heat loss and the effective temperatures, average surface and ambient, used to accomplish the heat transfer. The h_0 for the same patients, bandaged and exposed, are 5.4 ± 2.7 vs. 5.3 ± 3.3 $W/M^2 \cdot °C$ and not significantly different from one another.

These values for h_0 are, however, both significantly lower than for healed burn patients (8.4 $W/M^{-2} \cdot °C^{-1}$, $p < 0.001$ for both comparisons). This significantly lower h_0 for burned patients accurately reflects the fact that they lose a significantly smaller per cent of their total heat production by radiation and conduction than do healed burn patients or normal subjects. Calculation of conduction coefficients by the equation:

$$\text{Core} - \text{Skin } (W/M^2 \cdot °C) = \frac{M}{T_c - T_s} \qquad (4)$$

where:

T_c = core temperature in °C
T_s = average skin temperature
M = metabolic rate

creates the illusion that a greatly increased amount of heat actually reaches the surface of the patient by tissue conduction, while such is not the case. At least one half of this amount of heat reaches the surface by direct blood flow, and as heat transfer increases in warm environments this figure may reach 85%, of the trans-

TABLE 4. *Comparison of All Plasma Catecholamine Concentrations for 16 Burn Patients While Bandaged and after 4 Hours without Dressings in a Thermally Neutral Environment*

	Bandaged Burn Patients	Exposed Burn Patients	Normal Adult Range
Plasma adrenaline	93 ± 57* — p < .02	200 ± 163*	0–55†
Plasma noradrenaline	632 ± 307* — p < .01	1090 ± 525*	65–320†

N = 16.
* Mean values ± standard deviation in pg/ml.
† Adult normal range in pg/ml. Mean burn wound size = 10/38% BSA (full/total area of burn).

TABLE 5. *Correlated Parameters*

X versus Y	Regression	r	Significance
% Burn – Heat Prod. Bandaged	Y = 61.0 + .1207 X	.2762	NS
% Burn – Heat Prod. Exposed	Y = 64.2 + .4500 X	.7405	$p < .001$
% Burn – % ↑ Heat Prod. Bandaged	Y = 7.881 + .4565 X	.4425	$p < .05$
% Burn – % ↑ Heat Prod. Exposed	Y = 12.889 + 1.1242 X	.7826	$p < .001$
% Burn – Heat Prod. Corrected for Fever Bandaged	Y = 57.531 + .0155 X	.0353	NS
% Burn – Heat Prod. Corrected for Fever Exposed	Y = 59.7970 + .3210 X	.5736	$p < .01$
% Burn – E_v Heat Loss Bandaged	Y = 29.8294 + 0.3426 X	.5223	$p < .01$
% Burn – E_v Heat Loss Exposed	Y = 27.0988 + 0.6590 X	.6842	$p < .001$
% Burn – °C ↑ Rectal T. Bandaged	Y = 0.7139 + 0.0194 X	.6032	$p < .01$
% Burn – °C ↑ Rectal T. Exposed	Y = 0.9766 + 0.0129 X	.3430	NS
% Burn – °C ↑ 80/20 B_T Bandaged	Y = 0.3427 + 0.0192 X	.5695	$p < .01$
% Burn – °C ↑ 80/20 B_T Exposed	Y = 0.5817 + 0.0159 X	.4034	NS
E_v Heat Loss – Heat Prod. Bandaged	Y = 54.9161 + .2413 X	.3619	NS
E_v Heat Loss – Heat Prod. Exposed	Y = 58.4837 + .4187 X	.6037	$p < .001$
Plasma Adrenaline – Heat Prod. Bandaged	Y = 63.2703 + .0120 X	.0842	NS
Plasma Adrenaline – Heat Prod. Exposed	Y = 74.7838 + .0205 X	.3588	NS
Plasma Noradrenaline – Heat Prod. Bandaged	Y = 63.1729 + .0024 X	.0884	NS
Plasma Noradrenaline – Heat Prod. Exposed	Y = 68.3731 + .0113 X	.5771	$p < .01$
↑ Rectal Temp. – % ↑ Heat Prod. Bandaged	Y = 0.6865 + 15.9806 X	.4956	$p < .02$
↑ Rectal Temp. – % ↑ Heat Prod. Exposed	Y = 19.2183 + 19.5750 X	.5130	$p < .01$

ferred heat, further distorting the meaning of conduction coefficients calculated using equation 4.[16] The major portion of the heat transferred from the burn wound to the environment occurs secondary to evaporative heat loss; the heat of vaporization, for the most part, is derived from warm blood delivered directly to the skin and/or burn wound. Burn patients are internally warm and externally cold at an environmental temperature of 28 C, thermal neutrality, for normal man. Anyone observing exposed burn patients under these conditions will confirm that such patients are subjectively cold.

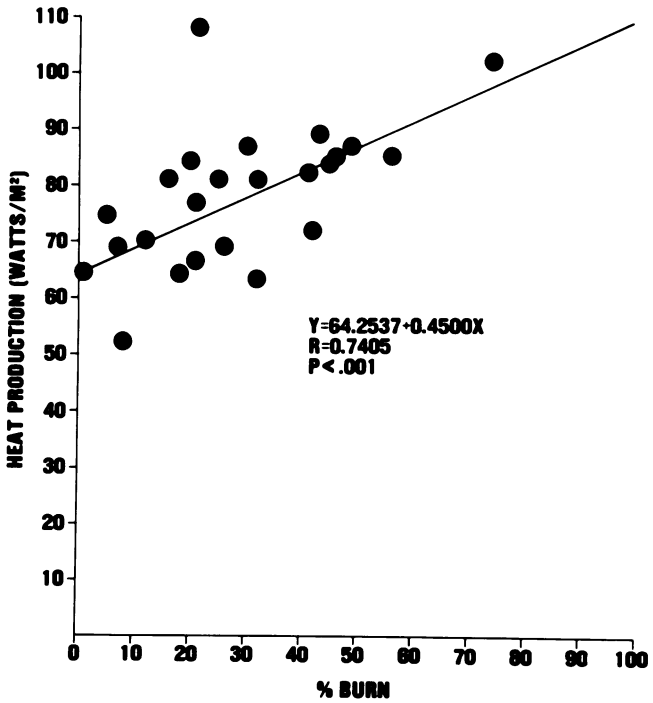

FIG. 2. Correlation between per cent BSA burn and the rate of heat production of patients exposed for 4 hours at 28 C.

Linear Regression Analysis of Some of the Parameters Studied

Table 5 summarizes the data for correlated parameters. Per cent burn surface area (BSA) of burn injury, the most important independent variable, demonstrates positive correlation with heat production exposed and percent increase in rate of heat production both bandaged and exposed (Figs. 2–4). Evaporative heat loss is positively correlated with wound size (bandaged and exposed) and the rate of heat production for exposed patients (Figs. 5–7). The degree centigrade increase in rectal and 80/20 average B_T correlates well with the burn wound size while bandaged, but not exposed (Figs. 1, 8–10). Plasma Ad levels do not correlate with heat production; and plasma Nad levels correlate with heat production only while exposed (Figs. 11–14, and Table 5).

Comparison of the regressions for per cent BSA burn and metabolic rate of our patients exposed with the Brooke data for exposed burn patients shows the slopes are not different. However, there is a significant difference between the regression for our bandaged patients and that for Brooke's exposed patients (Fig. 15).

Correlations do not prove cause and effect relationships, but these studies support the concept of the burn wound as primary in the cause of increased heat loss and hypermetabolism of burned patients, particularly when treated exposed. The absence of correlation between plasma adrenaline levels (the major calorigenic catecholamine) and rate of heat production bandaged or

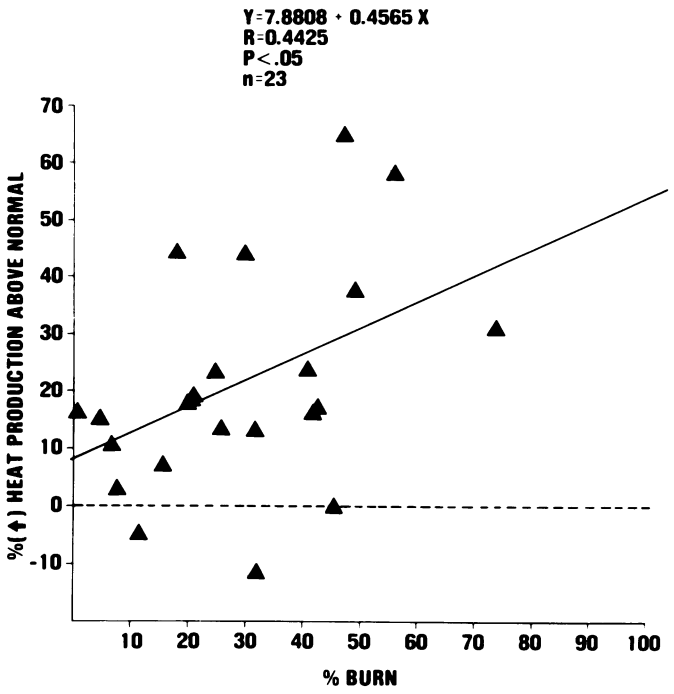

FIG. 3. Correlation between per cent BSA burn and per cent increase in the rate of heat production of bandaged patients at 28 C.

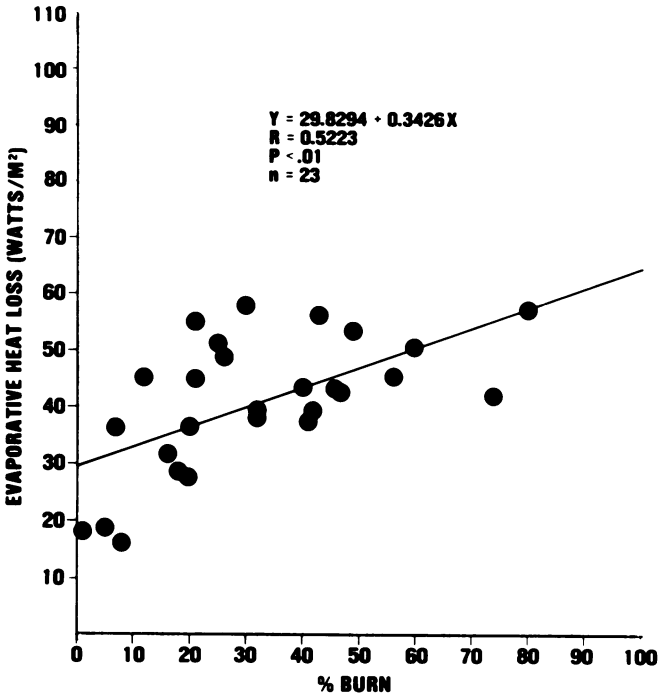

FIG. 5. Correlation between per cent BSA burn and evaporative heat loss of bandaged patients at 28 C.

exposed, and the correlation with noradrenaline levels only exposed does not support the concept that catecholamines play a primary role in the genesis of the hypermetabolism following thermal trauma.

Body Weight Loss For All Burn Patients

None of the patients in this study received intravenous hyperalimentation; however, based on their individual metabolic studies, caloric intake was pushed to

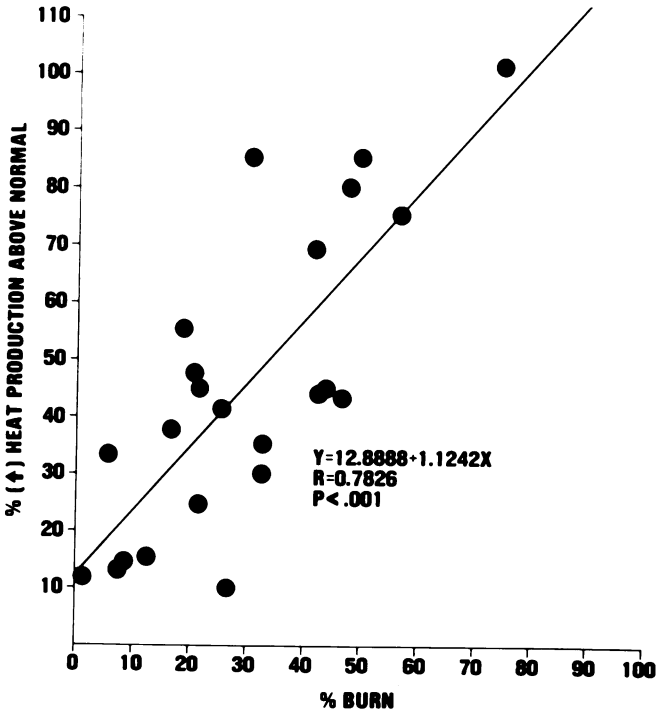

FIG. 4. Correlation between per cent BSA burn and per cent increase in the rate of heat production of patients exposed for 4 hours at 28 C.

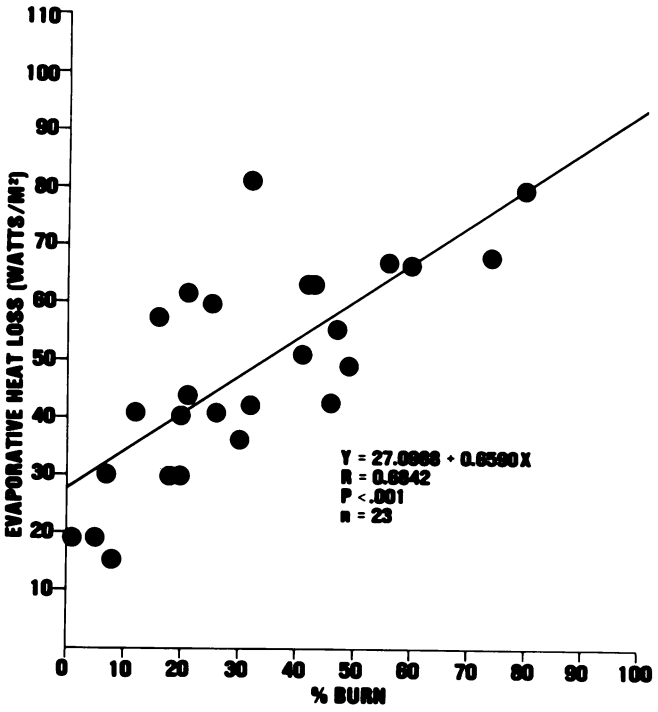

FIG. 6. Correlation between per cent BSA burn and evaporative heat loss of exposed patients at 28 C.

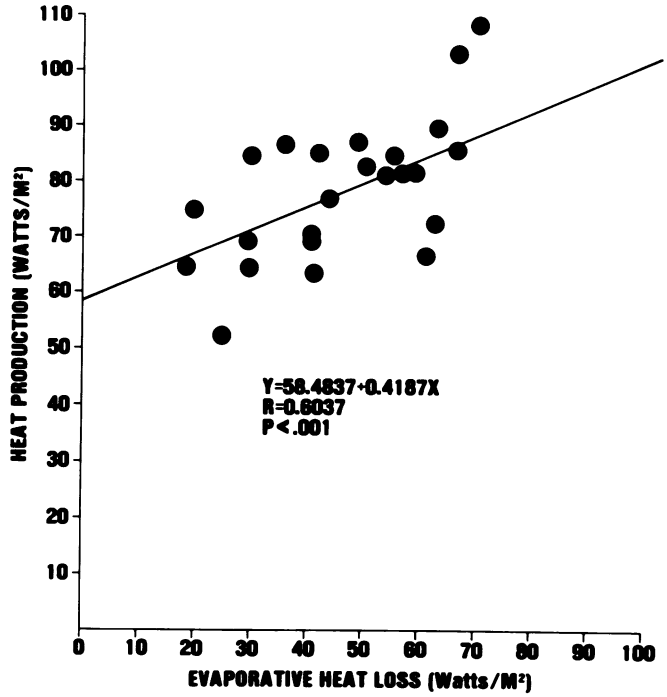

FIG. 7. Correlation between rates of evaporative heat loss and heat production of exposed patients at 28 C.

the indicated level of need or as near as possible allowing for the patient's tolerance. Long-term gastric intubation was very useful in meeting the needs of the patients with the larger burns. Figure 16 shows the body weight loss curves for burn patients grouped by wound size intervals of 10% of the BSA. By the tenth postburn week, only the group of patients with burns larger than 50% of their BSA had lost more than 10%

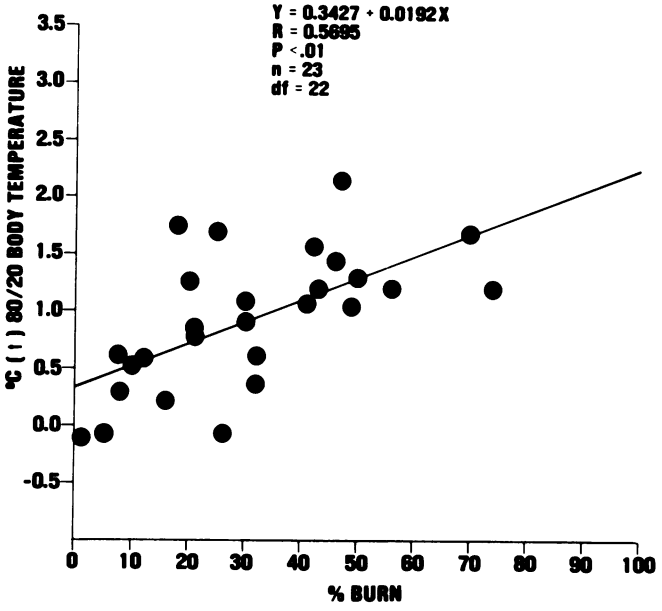

FIG. 8. Correlation between per cent BSA burn and °C increase in average body temperature of bandaged patients at 28 C.

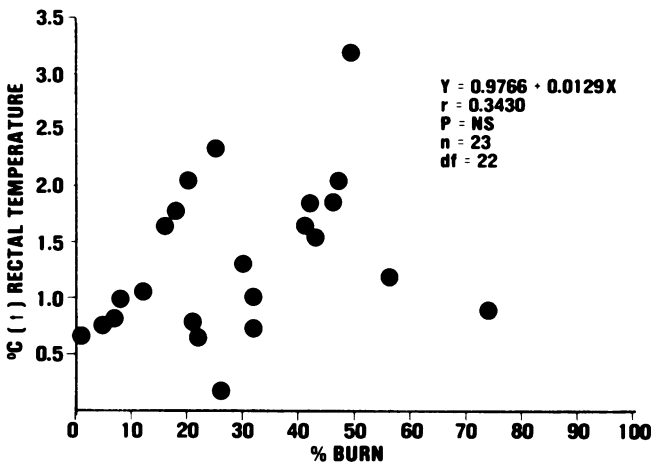

FIG. 9. Per cent BSA burn does not correlate with °C increase in rectal temperature of exposed patients at 28 C.

of their original mean body weight; all other groups had either stabilized or begun to gain weight by this time. These data show that the combination of minimizing the nutritional needs of burn patients by using occlusive dressings and a warm environment along with vigorous oral nutritional support, including tube feedings, allows virtual elimination of body weight loss in all but the patients with very large burns.

Discussion

These data show that severity of the hypermetabolic state following thermal trauma can be greatly decreased by the use of dressings and confirm the work of Arturson et al. which showed that the nonfever portion of the hypermetabolism following thermal injury can be controlled by an effective external heat source such as

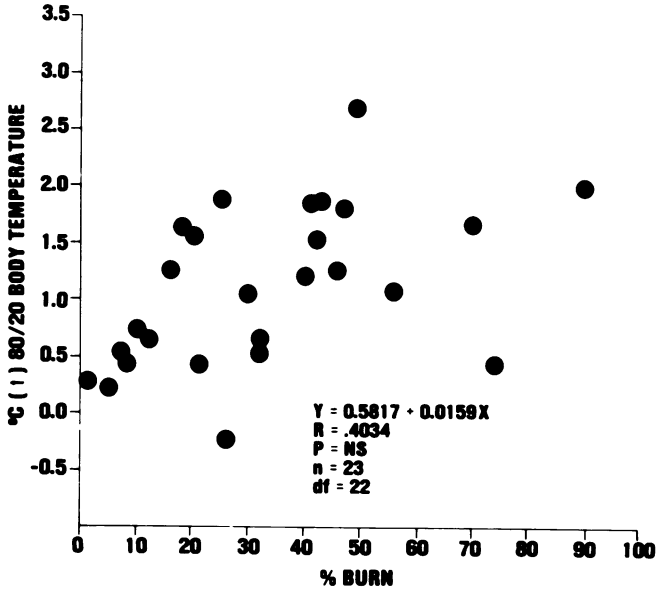

FIG. 10. Per cent BSA burn does not correlate with the °C increase in average body temperature of patients exposed at 28 C.

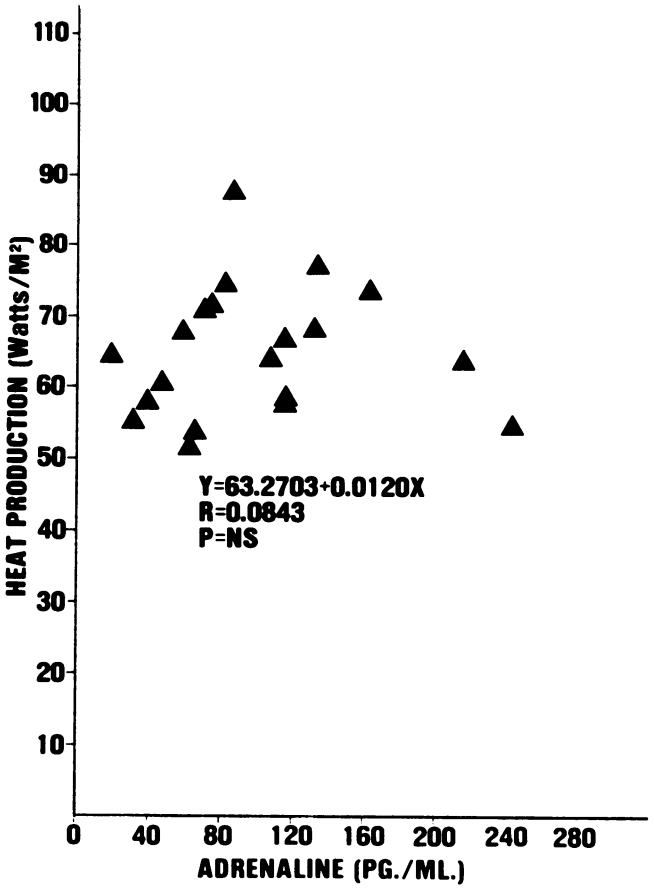

FIG. 11. Plasma adrenaline concentrations do not correlate with the rate of production of bandaged patients at 28 C.

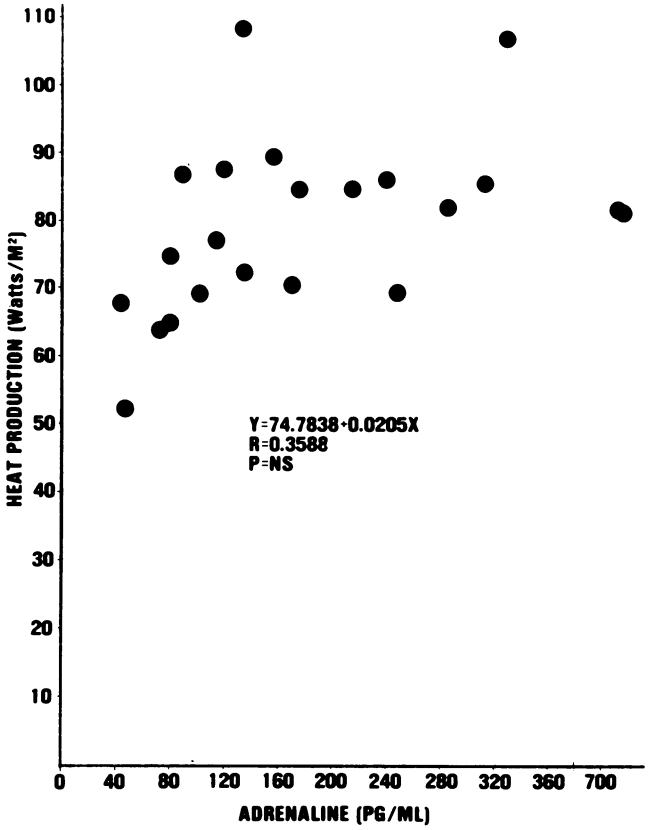

FIG. 12. Plasma adrenaline concentrations are not correlated with the rate of heat production of patients exposed at 28 C.

warm, dry air or infrared heat lamps.[1-4,18] The present data are also qualitatively similar to the results obtained by Neely et al., where in acute metabolic studies, water impervious material was used to cover patients' burn wounds demonstrating a decrease in evaporative heat loss and rate of heat production.[20] Neither Neely's or Arturson's patients demonstrated a return to normal heat production. Arturson corrected heat production for temperature, whereas Neely did not. Both studies, as well as the present study, demonstrate a residual increase in heat production, if corrections are not made for elevation of body temperature.

The present results differ remarkably from data published by the Brooke group.[25] The Brooke burn patients were treated open with topical mafenide in open burn wards maintained well below thermal neutrality for normal man but comfortable for ward personnel in light clothing with coat. Patients were tubbed daily. Metabolic studies were acute, with two ambient temperatures used in a single day in some cases. The subset of patients treated in this manner showed little decrease in rate of heat production when the ambient temperature was increased to 32 C for a few hours. The Brooke group concluded that increased evaporative heat loss of burn patients was only a convenient route to dispose of the extra heat production which results from a centrally driven hypermetabolic state mediated by catecholamines. Their data for heat production rates of burn

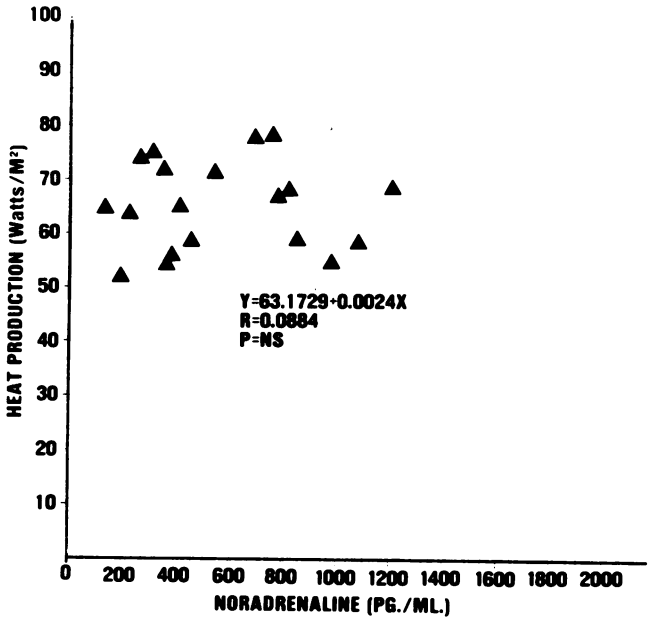

FIG. 13. Plasma noradrenaline concentrations do not correlate with the rate of heat production of bandaged patients at 28 C.

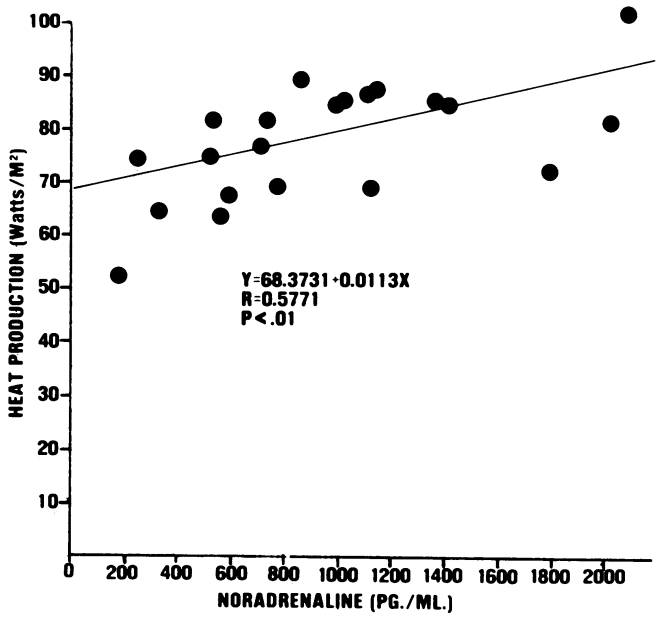

FIG. 14. Plasma noradrenaline concentrations are positively correlated with the rate of heat production of the exposed patients at 28 C.

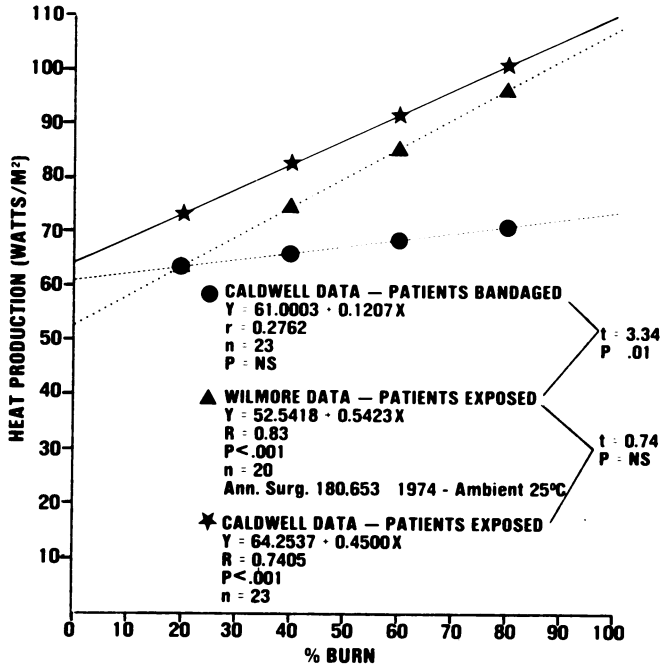

FIG. 15. A comparison of the regressions for per cent BSA burn and rate of heat production for the burn patients in the present study, bandaged and exposed and the regression for the patients studied by Wilmore while exposed. There is a significant difference between the slope of the regression for the present bandaged patients and Wilmore's exposed patients.

patients after alpha and beta block, *showing 64% of the original increase in heat production still present*, do not support the contention that catecholamines are the primary mediators of the hypermetabolic response to thermal injury.

The present data show that the rate of heat production of burn patients follows the rate of heat loss, not vice versa. Plasma catecholamine levels of burn patients change as if a secondary response and are not different from values for healed burn patients when comfortable at rest in occlusive dressings (Table 3). The major portion of the absolute increase in catecholamines is Nad, which has little calorigenic effect in mammals not cold acclimated. Nad is entirely alpha in its action except for the heart and likely plays a role in the

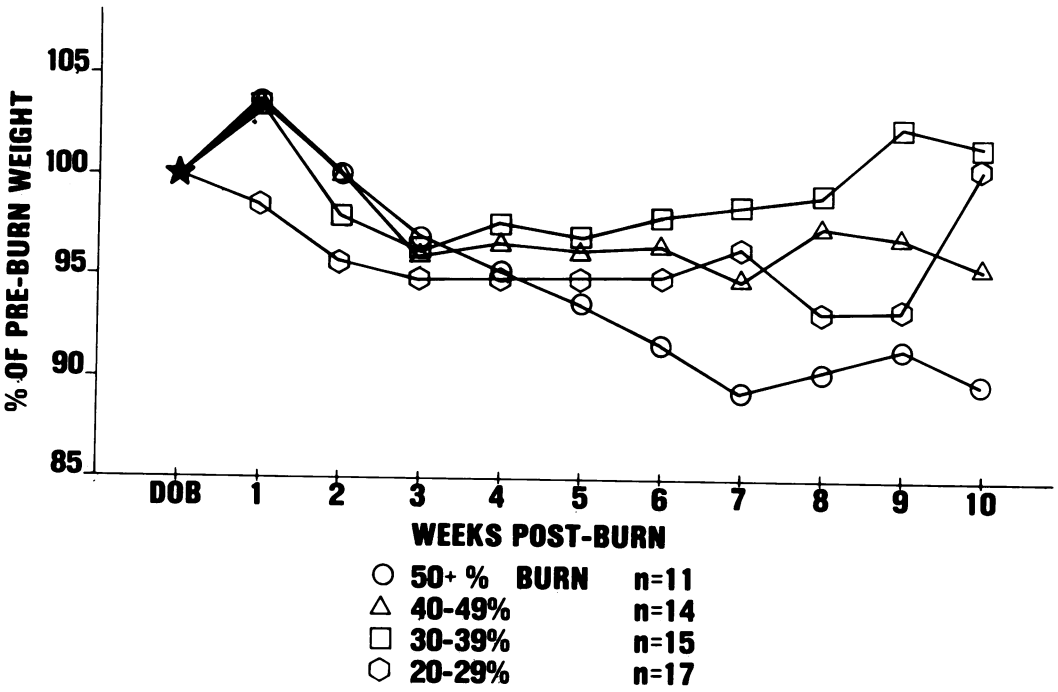

FIG. 16. The change in body weight, as per cent of preburn weight, for a group of patients with occlusive dressings. Burn wound size intervals are 10 per cent.

maintenance of a hyperdynamic circulatory system of patients with major thermal injury.

A major unanswered question concerning the metabolism of burned patients is why noncolonized, uninfected patients demonstrate a chronic elevation of average body temperature roughly proportional to the size of the burn wound (Fig. 1). Is the chronic elevation of the core and average body temperature fever or hyperthermia?[23] It contains some physiologic characteristics of both: In support of fever, are the facts that the elevated body temperature is defended and that the preferred ambient temperature is also elevated for the burn patient. Against the interpretation of fever is the fact that antipyretics have no effect upon the body temperature of burn patients. Like the hyperthermia associated with exercise, which is proportional to the severity of the exercise,[21] the increment in rectal temperature following thermal injury is proportional to the per cent increase in the rate of heat production bandaged or exposed, and proportional to the size of the burn only when bandaged (Figs. 1 and 17).

Whatever the cause of the increased body temperature, its effects follow the laws of physics. Although an increase in the rate of heat production is not necessary to have increased body temperature, an increase in body temperature is invariably associated with an increase in the rate of heat production.[10,14] Is it possible to double the rate of heat production in man without increasing the body temperature? Probably not. Thus, the cause of increased body temperature becomes an important consideration; is it primary or secondary? Is it good or bad? Is an increase in body temperature necessary for the patient to meet the energy requirements imposed by the burn injury?

Burn patients react as if their hypothalamic thermoregulatory set point had been shifted upward, and the shift is proportional to the size of the injury. Burn patients are clearly warm internally, but cold externally at normal ambient conditions of thermal neutrality (28 C and 40% relative humidity).

The metabolic consequences of increased body temperature, whether primary or secondary to increased heat production, seem to be additive to the effects of increased heat loss from the burn wound. Interaction seems most likely—that is, increased body temperature produces a further increase in evaporative and dry heat losses of burned patients.

The use of either occlusive dressings or a constant external energy source adjusted by the patient can drastically reduce, but not eliminate, the hypermetabolism of burned patients resulting in manageable nutritional demands for the host, with less weight loss and a lower mortality rate. Chronic exposure treatment of patients with extensive burn wounds at environmental temperatures of thermal neutrality or below produces a maximal hypermetabolic response per unit burn size, resulting in nutritional requirements impossible to meet for many such patients. Pain, fever and anxiety, etc. induce additional energy requirements for burn patients and complicate the study of any patient not quietly at rest, but rather in pain, or emotionally disturbed.

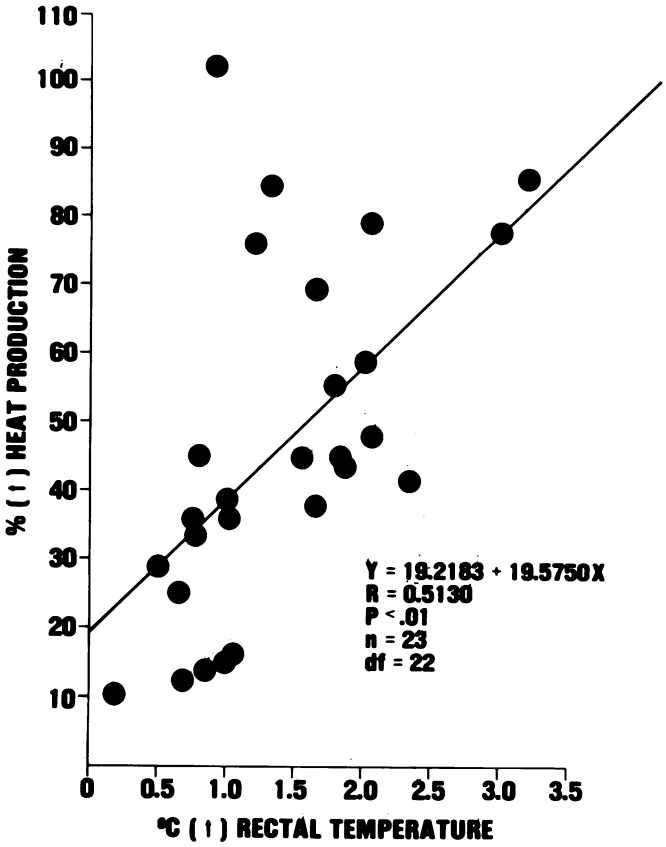

FIG. 17. Correlation between °C increase in rectal temperature and per cent increase in rate of heat production for exposed patients at 28 C.

The eventual colonization of the burn wounds of most patients with thermal injuries covering more than 50% of the BSA further complicates the study of energy metabolism of such patients, since the metabolic effects of bacterial exo and endotoxin must be considered.

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Discussion

DR. WILLIAM A. NEELY (Jackson, Mississippi): Dr. Caldwell has shown without doubt that there is hypermetabolism in burn patients. These slides from our previous study demonstrate again what Dr. Caldwell has shown.

(slide) Please note that this burn patient was hypermetabolic, almost two times normal; this is the normal range. And this is covered, and this is uncovered. Please note that there is tremendous hypermetabolism in the patient uncovered, and all this returns to normal when the patient is grafted.

(slide) The temperature at the time of the study is shown here, in bar graphs. You see that all these differences are statistically significant by the standard deviation.

(slide) This slide shows exactly the same thing, (slide) as does this slide. You will notice that in some of these studies the temperature of the patient was normal.

You will note that the burn patients are particularly hypermetabolic; that this is not abolished when the patient is covered, thus preventing evaporative water loss. I'm sure Dr. Caldwell will say our occlusive dressing would not prevent heat loss, but I will say that I was comfortable in the plastic material that we have put over these patients. And this hypermetabolism does not return to normal until the patient's skin is grafted.

One of these patients, as I mentioned earlier, did not have any temperature elevation in his entire course. This is, of course, rare, but still demonstrates that the patient is still hypermetabolic, even with a normal temperature. There is no doubt that this hypermetabolism is related to both injury and water loss, the latter preventable.

For eons, animals have been covering injury without the benefit of metabolic interference. The question at hand is whether hypermetabolism is favorable, or is an unfavorable reaction to injury, and should we, perhaps, interfere metabolically?

DR. WILLIAM MONAFO (St. Louis, Missouri): It has been known for more than 25 years that extensively burned patients are hypermetabolic—appreciably more so than patients with nonthermal trauma. Since the degree of hypermetabolism is extreme, the question arises whether there may be something unique about heat-injured tissue, as opposed, for example, to tissue injury due to kinetic energy.

About 20 years ago, it was shown that thermally injured skin leaks water vapor at an abnormally high rate—up to tenfold or so more than normal. Moreover, it is known that the water vapor barrier in the skin is superficial—constituted principally by lipids in the superficial cornified layers of the epidermis, so that the cutaneous injury need not be deep in order for the barrier to be destroyed. Since the heat of vaporization of water at body temperature is nearly 0.6 calories per gram of water, it seemed likely that this additional caloric requirement was likely the principal explanation for the hypermetabolic state of the burn patient.

Subsequently, however, data from burned man apparently conflicted with this hypothesis, in that obviating evaporative water loss, for example, by covering the wounds with impermeable plastic, did not necessarily lower oxygen consumption. The situation is made more complex because core temperatures tend to be significantly elevated in burned man, a phenomenon which by itself elevates metabolic rate.

One alternative explanation that has arisen for the principal cause of burn hypermetabolism is that the hypermetabolism is primarily driven by an increase in catecholamine secretion. These are the issues addressed in the present study.

The data we have heard add weight to the concept that cutaneous evaporative water loss is indeed the most important driving force in burn hypermetabolism, since the increase in insensible water loss that attended removal of the dressings resulted in increased energy expenditure and since heat production (corrected for core temperature) was not significantly elevated above normal in these children while they were bandaged. Moreover, only when the dressings had been removed were plasma catecholamine levels significantly elevated, a finding which of course makes the mediating role of those hormones somewhat questionable.

But the design of the study was such that all patients were examined in the same sequence—that is, first with their dressings in place, and then after they had been removed. One wonders if the findings would have differed had this sequence been reversed. I therefore ask Dr. Caldwell whether he has any data collected in the reverse sequence—either from these patients or possibly from others. If he does, and if the results are similar to those in the experiments he has just reported, the data would be even more persuasive. I would also like him to say whether the increase in evaporative water loss that was observed after the dressings had been removed was statistically significant.