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## Effects of body position on thermal, cardiorespiratory and metabolic activity in low birth weight infants

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### Introduction

Care takers have long noted that low birth weight (LBW) infants seem more comfortable when cared for in the prone position. They also spend more time in quiet sleep (1–3) and often have improvements in ventilation (4) as compared to supine position. These perceptions attracted further interest with the recognition that sudden infant death syndrome (SIDS) was related to prone body positioning during sleep [5–10] and decreased with the introduction of public health measures designed to reduce the incidence of prone sleeping [11]. The very fact that supine position protects against SIDS is counterintuitive, but nonetheless true. Numerous physiological differences related to body position have been reported [1–4,12–23] and reviewed [24], and several hypotheses have been formulated to explain how these differences might render infants more vulnerable to SIDS. One prominent hypothesis relates SIDS to relative increases in body temperature [25–34], thought to be caused by less efficient heat dissipation in the prone position.

Evidence consistent with this hypothesis includes the observation that the victims of SIDS are commonly found in unusually warm environments, often feel warm and/or diaphoretic when discovered, and exhibit higher than expected rectal temperatures at examination or autopsy [32,33]. In addition, SIDS victims have often been wrapped tightly in clothing/bedding and/or a history of a recent febrile illness is often elicited [34].

A detailed study of the interactions among body position, sleep states, heat production, surface temperature profiles and surface temperature gradients, and cardiorespiratory activity in LBW infants may provide important information concerning physiological disturbances that predispose to SIDS, a condition to which LBW infants are especially susceptible as they grow to infancy [6]. The primary objective of this study was to test the hypothesis that despite lower metabolic rate (heat production), prone body position during sleep is associated with systematic

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increases in absolute surface temperatures and reduced surface thermal gradients in growing LBW infants. A secondary objective was to determine whether any observed differences in physiology related to body position would suggest or refine specific testable hypotheses related to thermal balance, cardiorespiratory activity, and cardiorespiratory instability.

## Material and Methods

### Patient population

The patient population was comprised of 32 healthy, growing LBW infants, ranging in birth weight from 805–1590 g and in gestational age from 26–35 wk. All subjects were enrolled in a prospective, double-blind, controlled study of the effects of quality of dietary energy on the rate and composition of weight gain [35]. Stratified randomization was done within three birth-weight ranges, i.e. 750–1000, 1001–1300, and 1301–1600 g and the infants were equally among the diet groups. The study was approved by Institutional Review Board and written consent was obtained from parents of all infants. All infants were being maintained in room air, were free of apnea of prematurity and were receiving no cardiac or respiratory medications. None had sonographic evidence of central nervous system pathology at the time of the studies. The characteristics of study infants are shown in Table 1.

### Experimental Design

The infants received 1 of 5 experimental formulas differing only in the absolute and relative amounts of carbohydrate and fat. Each infant was begun on the experimental formula at the time of initiation of enteral feeds and remained on that formula until discharge from the hospital at a weight of approximately 2200 g. As a part of the feeding study infants were subjected to measurements of energy balance, which included 6h estimates of O<sub>2</sub> consumption and CO<sub>2</sub> production. These studies were performed in the Infant Physiology Laboratory at Children's Hospital of New York beginning when the infant reached full enteral intake of 180 ml/kg.day. Each 6h study was comprised of two sequential three-hour periods of observation separated by a feeding period. Infants were randomly assigned to prone or supine position for the first 3-hr of the study starting after 8:00 AM feed and the position was reversed for the second 3-hr of the study which began after the 11:00 AM feed. They remained in their assigned positions throughout the inter-feeding period, and no further manipulations were performed.

### Experimental Protocol

After they had been on full enteral intakes for a minimum of 3d, infants were brought to the laboratory, located adjacent to the nursery, at approximately 7:30 AM for 6h measurements of thermal profile, cardiorespiratory activity and energy expenditure (indirect calorimetry). Clad only in paper diapers, they were placed on a soft mattress in a Plexiglas open-circuit indirect flow-through calorimeter with a flow in L/min set at twice the body weight in kg. Electrodes for recording vital signs were attached. No external heating source or physical constraints such as swaddling were employed during the study period. The head was not covered. Studies began following the 8:00 AM feeding and continued until the 2:00 PM feeding. The studies were interrupted for the 11:00 AM feeding after which sleeping position was changed. As dictated by the study protocol the volume and composition of the two feeds were identical.

### Measurement of surface temperatures

Surface temperatures (ST) were recorded from 4-body sites, i.e. forehead, right flank, right forearm and right leg using Incutemp<sup>®</sup> thermistors (Mallinckrodt, St. Louis, MO, USA) [36, 37] Care was taken to insure that the thermistor on the flank was not sandwiched between the infant and the mattress. Environmental temperature, i.e. chamber temperature and room temperature were also recorded using the same Incutemp<sup>®</sup> sensors. Temperatures from all sites

were recorded every 8 seconds. The temperature data was collected using a custom-made, multiplexed, self-calibrating device that logged measurements from each thermistor to a dedicated computer. The computer was linked via a common clock to a sister computer that recorded continuous measurements of the other physiologic variables.

### Measurement of heart rate and respiratory frequency

The electrocardiogram was amplified by a standard heart rate monitor (Hewlett Packard 3680) and the intervals between the RR-waves were processed using a special purpose RR-interval preprocessor. The preprocessor detects R-waves, measures the RR-interval ("1 msec), and passes these times to the computer over a parallel port. All data were analyzed in one minute blocks with each block corresponding to a minute of designated sleep state. Any RR-interval exceeding 667 msec (<90 bpm) or shorter than 300 msec (>200 bpm), representing brief period of motion artifact, was excluded from the analyses. The average number of excluded RR intervals was extremely small, <1% per minute. Mean heart rate (HR) was computed as the inverse of the mean of RR-intervals measured during each block of time. The standard deviation of all accepted RR-intervals (RR-SD, in msec) was also measured each minute as a global measure of heart period variability.

The impedance pneumogram was digitized at a rate of 50 samples/sec, and conditioned using a 13 point moving average/first derivative filter to attenuate periodic low frequency fluctuations and to improve the detections of breaths. A pattern recognition program designed to detect peaks and troughs was then used to mark the beginning and end of each breath in the filtered impedance tracing [38]. The average breath-to-breath (BB) interval was computed each minute and the inverse of this mean was taken as respiratory frequency (f). Brief periods of apnea (breath durations exceeding 3 seconds) and motion artifact (durations less than 0.5 seconds) were excluded from the analysis. Eliminating short apneic periods was necessary because we were interested in the total breath count and variability during regular spontaneous breathing. The standard deviation of breath intervals was taken as a global measure of respiratory variability (BB-SD).

### Measurement of energy expenditure

The open circuit, indirect calorimetry system used to measure oxygen consumption ( $\text{VO}_2$ ) and carbon dioxide production ( $\text{VCO}_2$ ) has been previously described [39]. Flow was set a twice the infant's weight in kg and this typically resulted in the exiting gas samples having approximately a 0.5% difference in fractional concentration from the entering gas stream (room air). Respiratory quotient (RQ) was determined as the ratio of the mean  $\text{VCO}_2$  during the 150 min interfeed interval to the mean  $\text{VO}_2$  during the same interval.

### Coding of behavioral state

Each minute of the study was coded for behavioral state. Coding began 10 min after the 8:00 AM feeding, continued until 11:00 AM feeding, resumed 10 min after the 11:00 AM feeding and terminated just prior to the 2:00 AM feeding. Behavior codes were assigned by direct observation each minute using a scoring system developed and validated in our laboratory [40]. Briefly, active sleep (AS) was coded whenever at least one rapid eye movement was observed during the minute. In addition to small body movements typical of active sleep, movements of whole extremities and the torso were seen in this state. Stretching, yawning, whimpering, sucking and grimacing were also present occasionally. Quiet sleep (QS) was designated when the infant was asleep without rapid eye movements. The use of 1-min epochs without the use of smoothing algorithms leads to more minute to minute variability in state assignment but has the advantage of maintaining a tighter relationship between the assigned state and the simultaneous changes in physiologic variables [41].

## Data Analysis

At the termination of each study, a computer file was constructed which contained minute averages of temperatures from forehead, right flank, right forearm and right leg, HR, RR-SD, f, BB-SD, VO<sub>2</sub>, VCO<sub>2</sub>, and RQ. The temperature data from the forehead and the flank were designated as central temperatures. The forearm and the leg temperatures were referred to as peripheral temperatures. The rationale for designating abdominal flank and forehead temperatures as central temperature was based on transcutaneous thermometry studies which have demonstrated that skin sensors at these sites can reliably monitor central body temperature in preterm human infants [42] and newborn piglets [43]. From the above temperature data, forehead-to-arm, forehead-to-leg, flank-to-arm, flank-to-leg, and forehead-to-environment gradients were computed for each minute. The corresponding behavioral state code was then appended to the minute-by-minute physiologic data. Behavioral codes corresponded temporally to the temperature and cardiorespiratory measurements but, due to unavoidable lags in the open circuit system, preceded the metabolic measurements by several minutes. Hence, minute-to-minute analyses of the metabolic data were not possible, and mean values for each 3h study interval were computed to summarize the metabolic activity of the infant. Data were then sorted for prone and supine positions during QS and AS and compared for each state of sleep, using paired t-tests.

## Results

### Temperature

In prone position and during AS, the infants had higher forehead (36.1 vs. 35.9°C,  $p < 0.018$ ), flank (36.3 vs. 36.1°C,  $p < 0.0007$ ), forearm (35.0 vs. 34.4°C,  $p < 0.0001$ ) and leg (34.1 vs. 33.6°C,  $p < 0.0001$ ) surface temperatures [Table 2] and narrower central-to-peripheral gradients, i.e. forehead-to-forearm (1.1 vs. 1.5°C,  $p < 0.0001$ ), forehead-to-leg (2.1 vs. 2.4°C,  $p < 0.0001$ ), flank-to-forearm (1.3 vs. 1.7°C,  $p < 0.0001$ ) and flank-to-leg (2.2 vs. 2.5°C,  $p < 0.0001$ ) [Table 3]. Despite similar environmental temperatures for prone and supine segments (27.1±1.07°C), the forehead-to-environment gradient in the prone position was significantly higher (8.9 vs. 8.7°C,  $p < 0.05$ ). Similar positional changes were observed during QS.

### Cardiorespiratory measurement

In the prone sleeping position during AS, infants had higher HR (160.7±7.4 vs. 158.6±6.8 bpm,  $p < 0.003$ ), higher f (53.0±4.9 vs. 52.1±3.9 bpm,  $p < 0.04$ ), lower RR-SD (15.4±4.8 vs. 17.5±5.3 msec,  $p < 0.0001$ ) and lower BB-SD (360.9±35.0 vs. 375.3±35 msec,  $p < 0.001$ ) when compared to supine position. Similar positional differences in cardiorespiratory activity were observed during QS, as shown in Table 4.

### Metabolic gas exchange

In the prone position infants demonstrated lower VO<sub>2</sub> (8.9±0.7 vs. 9.6±0.9 ml/kg.min,  $p < 0.00001$ ) and VCO<sub>2</sub> (8.1±0.7 vs. 8.5±0.8 ml/kg.min,  $p < 0.0006$ ). Respiratory quotient was significantly higher in the prone position (0.91±0.08 vs. 0.88±0.07,  $p < 0.01$ ) [Table 5].

## Discussion

Infants sleeping in the prone position exhibited lower metabolic rates than those sleeping in the supine position. Despite this reduction in heat production, central and peripheral surface temperatures were higher in the prone position and the gradients between the central and peripheral sites were narrower, implying increased peripheral perfusion. In addition, prone sleeping was associated with higher heart rates, higher respiratory rates and higher RQ's.

These observations are consistent with the following unifying hypothesis. Sleeping in the prone position, even without excessive wrapping, induces changes in surface temperature profile. Despite reduced heat production (reduced  $\text{VO}_2$  and  $\text{VCO}_2$ ), all surface temperatures increase. This suggests a transfer of heat from the core to the periphery, i.e. cutaneous vasodilatation which results in narrowed central-to-peripheral temperature gradients and most probably an increased cardiac output (increased heart rate). This redistributed blood flow suggests an override of thermal regulation of blood flow, marked by vasodilatation and increased heart rate unaccompanied by changes in overall oxygen delivery (lower  $\text{VO}_2$ ). In other words in the prone position, circulatory control is weighted more in favor of thermal stability and less dependent on metabolic rate. In effect, this modest increase in body temperature induce a peripheral arteriovenous shunt, which in the presence of a reduced metabolic rate would result in an increase in mixed venous oxygen content as oxygen extraction in the periphery would not keep pace with the increased blood flow.

Similarly, the infants displayed signs of thermal override of respiratory control. Relative hyperventilation in the prone position is indicated by the increased respiratory frequency and RQ. It is important to restate that each infant was fed identical feeds before each epoch in prone and supine position. Barring any change in substrate utilization, RQ should not differ on identical intakes and thus an increase in RQ indicates relatively more  $\text{CO}_2$  is eliminated than appropriate for the oxygen consumed. These findings are highly suggestive of increased alveolar ventilation, a not uncommon consequence of warming and  $\text{CO}_2$  stores might be expected to decrease.

Alterations in the balance between requirements for metabolic gas exchange and cardiorespiratory function might lead to potentially serious consequences. Lambs subjected to controlled heat stress develop irregular breathing and apnea presumably because of hypocapnia secondary to thermoregulatory increases in alveolar ventilation [44]. The lambs and a cohort of newborn human infants studied concurrently were noted to have bursts of enhanced  $\text{CO}_2$  elimination alternating with brief periods of apnea as body temperatures increased. Unfortunately no measurements of arterial  $\text{CO}_2$  activity were made. Similarly, piglets stressed by elevated environmental temperatures develop hyperpnea and hypocapnic alkalosis before ultimately progressing to vascular collapse and shock [45]. Chemosensitivity to  $\text{CO}_2$  and  $\text{O}_2$  in rats is highly temperature sensitive [46]. It should be noted that under most elevated temperature conditions, metabolic rate would be expected to increase as a general increase in metabolic activity occurs. Apparently, the premature infant hyperventilates without increases in metabolic rate secondary to the relative increase in the body temperature due to decreased ability to lose heat in the prone position. These changes may be exaggerated further if the heat losses are reduced by over-bundling or an excessively higher environmental temperature.

Also the developmental changes in the thermoregulatory system may have implications during the most vulnerable period for SIDS. The increase in the ratio of body mass to surface area along with the rising metabolic rate during early infancy results in a much higher net heat loss per unit surface area in a 2–4 month old infant than in a newborn. This, together with the thicker layer of subcutaneous fat and more effective peripheral vasomotor response to cold over the same time period suggests that thermal balance is shifted in favor of heat conservation in a 2–4 month old infant. Thus, unlike a newborn, a 2–4 month old infant is less vulnerable to cold stress but perhaps has greater vulnerability to heat stress [47]. Alternatively, a rise in metabolic rate (from an acute infection, for example) could result in a significant change in thermal balance. There is anecdotal evidence that heat stress may be associated with sudden infant death or with severe hypoventilation [32,48]. In the Avon studies infants with SIDS, particularly those over 70 days of age, were more heavily wrapped and were more likely to have had the heating on all night than control infants matched for age, date, and neighborhood. There was no significant excess of viral infections in the infants with SIDS, but those who had

virus infections were much more heavily wrapped than control infants with similar infections, suggesting that the combination of heavy wrapping and virus infection may be more important than either factor alone [34]. There is, therefore, some physiological evidence that infants of 2–4 months of age may be more vulnerable to heat stress than younger infants, and limited evidence, from clinical studies, that this may occur and be associated with SIDS.

In addition to differences in resting activity in the prone versus supine position, investigators have documented differential physiological responsiveness in the two positions. Infants are more difficult to arouse [22] more susceptible to orthostatic hypotension [20] and, in general, less autonomically responsive in the prone position. When measured, increased environmental or body temperature is usually noted to amplify these differences in reactivity [49].

There are also limitations and assumptions that must be kept in mind while interpreting our findings. Although likely to parallel each other under stable conditions, changes in heat storage cannot be accurately estimated from heat production and surface temperature profile alone. Unfortunately, long periods of indwelling rectal temperature monitoring are not feasible in our small infants. Similarly under stable conditions, respiratory rate and heart rate generally track minute ventilation and cardiac output in LBW infants, however both are indirect estimates, and inferences regarding these variables must be qualified accordingly. The RQ data, drawn from long periods of measurement with identical quantities and qualities of intake and presumably no change in the pattern of substrate utilization, strongly indicate proportionately more CO<sub>2</sub> is eliminated in the prone position than in the supine position, but estimates of the actual changes in CO<sub>2</sub> activity in the blood, total body CO<sub>2</sub> stores and pH were not made. A change in substrate utilization cannot be ruled out. Also, gradients of surface temperature can be expected to parallel gradients in cutaneous blood flow but peripheral blood flow was not directly measured. Taken together these findings, along with many similar findings reported by others, indicate LBW infants are warmer in the prone position and that these differences in thermal profile are associated with changes in cardiorespiratory function that influence well being.

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**Table 1**

Characteristics of the study population.

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Gestational age	26–35 weeks (30.3 ± 2.5)*
Birth weight	805–1590 grams (1284 ± 237)*
Postconceptional age at study	33–38 weeks (35.1 ± 1.3)*
Study weight	1667–2293 grams (2103 ± 117)*

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\* Range (mean ± SD)

**Table 2**  
Surface temperatures in prone and supine positions during quiet and active sleep (mean  $\pm$  SD).

Temperature Site	Quiet Sleep		Active Sleep		p
	Prone	Supine	Prone	Supine	
Forehead (°C)	36.1 $\pm$ 0.4	35.9 $\pm$ 0.5	36.1 $\pm$ 0.4	35.9 $\pm$ 0.4	<0.018
Flank (°C)	36.3 $\pm$ 0.5	36.0 $\pm$ 0.5	36.3 $\pm$ 0.4	36.1 $\pm$ 0.4	<0.0007
Forearm (°C)	35.0 $\pm$ 0.9	34.4 $\pm$ 1.2	35.0 $\pm$ 1.0	34.4 $\pm$ 1.0	<0.0001
Leg (°C)	34.2 $\pm$ .9	33.6 $\pm$ 0.9	34.1 $\pm$ 0.9	33.6 $\pm$ 0.8	<0.0001
Environment (°C)	27.1 $\pm$ 1.0	27.2 $\pm$ 0.9	27.1 $\pm$ 1.1	27.2 $\pm$ 1.0	NS

**Table 3**  
Thermal gradients in supine and prone positions during quiet and active sleep (mean  $\pm$  SD).

Thermal Gradients	Quiet Sleep			Active Sleep		
	Prone	Supine	p	Prone	Supine	P
Forehead to Leg ( $^{\circ}$ C)	2.0 $\pm$ 0.7	2.2 $\pm$ 1.0	NS	2.1 $\pm$ 0.8	2.4 $\pm$ 0.7	<0.0001
Forehead to Forearm ( $^{\circ}$ C)	1.0 $\pm$ 0.9	1.4 $\pm$ 1.2	<0.001	1.1 $\pm$ 1.0	1.5 $\pm$ 0.9	<0.0001
Flank to Leg ( $^{\circ}$ C)	2.1 $\pm$ 0.8	2.4 $\pm$ 0.9	<0.0001	2.2 $\pm$ 0.8	2.5 $\pm$ 0.9	<0.0001
Flank to Forearm ( $^{\circ}$ C)	1.2 $\pm$ 0.9	1.6 $\pm$ 1.2	<0.0001	1.3 $\pm$ 1.0	1.7 $\pm$ 1.0	<0.0001
Forehead to Environment	8.9 $\pm$ 1.1	8.6 $\pm$ 1.1	<0.05	8.9 $\pm$ 0.9	8.7 $\pm$ 1.0	<0.05

**Table 4**  
Heart rate (HR), respiratory frequency (f) and the variabilities in heart rate (RR-SD) and respiration (BB-SD) in the prone and supine position during quiet and active sleep (mean  $\pm$  SD).

	Quiet Sleep			Active Sleep		
	Prone	Supine	p	Prone	Supine	p
<b>HR (bpm)</b>	155.2 $\pm$ 9.0	152.1 $\pm$ 9.8	<0.007	160.7 $\pm$ 7.4	158.6 $\pm$ 6.8	<0.003
<b>RR-SD (msec)</b>	9.3 $\pm$ 3.5	12.3 $\pm$ 4.9	<0.00005	15.4 $\pm$ 4.8	17.5 $\pm$ 5.3	<0.0001
<b>f (bpm)</b>	53.6 $\pm$ 8.8	51.7 $\pm$ 7.9	<0.01	53.0 $\pm$ 4.9	52.1 $\pm$ 3.9	<0.04
<b>BB-SD (msec)</b>	220.9 $\pm$ 53.7	260.7 $\pm$ 66.7	<0.005	360.9 $\pm$ 35.0	375.3 $\pm$ 35.1	<0.001

**Table 5**

Oxygen consumption ( $VO_2$ ), carbon dioxide production ( $VCO_2$ ) and respiratory quotient (RQ) in supine and prone position (mean  $\pm$  SD).

	Prone	Supine	p
$VO_2$ (ml/kg.min)	8.9 $\pm$ 0.7	9.6 $\pm$ 0.9	< 0.00001
$VCO_2$ (ml/kg.min)	8.1 $\pm$ 0.7	8.5 $\pm$ 0.8	< 0.0006
RQ	0.91 $\pm$ 0.08	0.88 $\pm$ 0.07	< 0.01