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Original Article

Distal skin vasodilation in sleep preparedness, and its impact on thermal status in preterm neonates

Véronique Bach [a, *], Stéphane Delanaud [a], Lucie Barcat [a, b], Emilie Bodin [a, c], Pierre Tourneux [a, b], Jean-Pierre Libert [a]

[a] *PériTox, UMR_I 01, Jules Verne University of Picardy, CURS, Présidence UPJV, Amiens, France*
[b] *Médecine Néonatale et Réanimation Pédiatrique, Pôle Femme Couple Enfant, CHU Amiens, Amiens, France*
[c] *Neurologie Pédiatrique, Pôle Femme Couple Enfant, CHU Amiens, Amiens, France*

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ABSTRACT

Objective: Prior to sleep onset in human adults, distal body temperatures change progressively from wakefulness levels (low skin temperatures and a high core temperature) to sleep levels (high skin temperatures and a low core temperature) due to distal skin vasodilation and greater body cooling. It is not known whether this sleep preparedness exists in preterm neonates, even though sleep has a key role in neonatal health and neurodevelopment. The present study's objectives were to determine whether sleep preparedness (as observed in adults) can be evidenced in preterm neonates, and to assess repercussions on thermal stress.
Methods: During a 12-h night-time polysomnography session, skin temperatures (recorded with an infrared camera), sleep, and wakefulness episodes were measured in 18 nine-day-old preterm neonates.
Results: Fifteen wakefulness episodes were considered. Our results highlighted significant pre-sleep distal skin vasodilation (mainly at the foot: an increase of 0.38 °C in the 20 min preceding sleep onset) for the first time in preterm neonates. This vasodilation occurred even though (1) most factors known to influence pre-sleep vasodilation in adults were not present in these neonates, and (2) the neonates were nursed in a nearly constant thermal environment. The vasodilatation-related increase in body heat loss corresponded to a 0.15°C/h fall in mean body temperature (calculated using partitional calorimetry).
Conclusion: Compensation for this body heat loss and the maintenance of body homeothermia would require a 4% increase in metabolic heat production. In neonates, this type of energy expenditure cannot be maintained for a long period of time.

1. Introduction

It is well known that sleep regulation interacts with body temperature (T) control in both human adults and neonates (for a review, see Ref. [1]). In adults, body temperatures differ in sleep versus wakefulness (W): skin temperatures (Ts) are lower during W than during sleep, whereas the core temperature is higher during W than during sleep (for a review, see Ref. [2]). Interestingly, when this wake–sleep difference is reduced, sleep quality and quantity worsen; conversely, increasing this difference (when it is small) improves sleep [3]. Sleep is preceded by a transition period during which progressive vasodilation occurs. Accordingly, skin Ts values increase. As a result, body heat losses are enhanced, which in turn leads to a decrease in the body core temperature. Sleepiness increases in parallel, and sleep usually occurs during the period during which the core temperature decreases [4]. This process and its consequences have been extensively studied in adults, especially as a way to improve sleep quality in patients with insomnia [5] or narcolepsy [6] or to reduce sleepiness [7].

It is strikingly that very few studies of sleep preparedness have been performed in infants, even though sleep has a key role in neurodevelopment and general health. Sleep is even more important in neonates, and especially preterm neonates cared for in neonatal intensive care units (NICUs). The environmental conditions and frequent nursing care in the NICU tend to compromise

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* Corresponding author. PériTox, UMR_I 01, Jules Verne University of Picardy, CURS, Présidence UPJV, chemin du Thil, F-80036, Amiens, France.
E-mail address: veronique.bach@u-picardie.fr (V. Bach).

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sleep quantity and quality, although development and recovery from illness are of vital importance.

Brown et al. [8], Lodemore et al. [9], and Wailoo et al. [10] analyzed rectal temperature variations during the first hours after (but not before) sleep onset or bed time. Term and/or older neonates had much the same post-onset variations in body temperatures as adults (ie, a rise in distal skin temperatures and a fall in rectal temperatures). Brück et al. [11] reported that the thermoregulatory behavior of vascular tone is mature and functional in full-term neonates. In preterm neonates (weight <1000 g), this control is absent at birth but develops over the following 2–3 days [12]. It has been reported that the thermoregulatory modulation of vascular tone is less well developed in preterm neonates (gestational age at birth: 33 weeks) at day 3 of life (weight: 2300 g) [13] and in small-for-gestational-age neonates than in full-term neonates. Karlsson and Hamel [14] observed that this thermoregulatory modulation first concerns the foot, which is the first body site capable of vasodilation as the full-term neonate ages.

It is not possible to extrapolate results from adults to preterm neonates because (1) sleep and circadian rhythms differ markedly and (2) the NICU and the closed incubator constitute a relatively stable environment that does not markedly influence physiological processes.

Finally, it would be interesting to know whether pre-sleep distal vasodilation (if present in preterm neonates) has a physiological impact, notably by influencing the maintenance of body homeothermia. Indeed, neonates lose heat to the environment more easily and rapidly than adults do: this is due to the neonates' higher skin surface area to body mass ratio, low body heat content, small energy substrate stores, and poorly efficient thermoregulatory processes. As a result, a fall in peripheral vasomotor tone can rapidly increase body heat losses, and might thus affect the infant's thermal status.

The objective of the present study was to measure changes in body temperatures before sleep onset and to determine whether sleep preparedness (related to changes in peripheral vasomotor tone in adults) can be observed in preterm neonates. We also sought to assess the repercussions of sleep preparedness on the neonate's thermal stress, since this process involves body heat losses. We assessed the neonate's thermal status from the average mean body temperature (T_b) calculated from partitional calorimetry; this technique provides reliable data on energy expenditure by preterm neonates [15,16]. In fact, the internal (core) body temperature is difficult to measure continuously, and the ideal measurement site is subject to debate because uniform core temperatures are not found anywhere in the body. In the present study, we postulated that T_b is more representative of body heat imbalance (ie, thermal stress) than temperatures measured at the usual body sites (such as rectal, tympanic, or axillary or abdominal skin temperatures. Moreover, T_b changes result from variations in both internal and peripheral temperatures, even though the latter may change in opposite directions [8].

2. Methods

2.1. Neonates

Neonates were recruited in the NICU at Amiens University Medical Center (Amiens, France). The study protocol had been approved by the local investigational review board (CPP Nord Ouest II; reference: RCB/2010-A00337-32). Infants were included after both parents gave their written informed consent. The present study did not modify the neonates' care procedures. Eighteen preterm, single-pregnancy neonates were included (nine females and nine males; mean ± standard error of the mean [SEM] postmenstrual age: 209 ± 2 days; mean birth weight: 1287 ± 75 g, mean head circumference at birth: 27.1 ± 0.4 cm). The main inclusion criteria were a postmenstrual age between 25 and 32 weeks, birth less than 12 h before NICU admission, the absence of congenital malformations or heart disease, and an Apgar score greater than 5 at 10 min. At the time of the experiment (night [N] 9 of life), the neonates (mean body weight: 1299 ± 62 g) were nursed in the supine position in a closed incubator (Satis+, Médipréma, Tauxigny, France) and at thermoneutrality with air servo-control of air temperature (mean value: 32.7 ± 0.3°C; relative humidity: 65%). The room air temperature was 25°C, and the relative humidity of the room air was 50–60%. The neonates were receiving continuous parenteral nutrition and (in some cases) additional continuous enteral nutrition. At night (between 19:00 or 20:00 and 08:00), light levels in the NICU were below 10–20 lux, and noise levels were low. All the neonates were receiving anti-apnea medication (caffeine) at the time of the study. None of the included neonates had infections or neurologic or cardiorespiratory disorders or diseases that could have affected sleep, cardiorespiratory function, or thermoregulation capabilities.

2.1.1. Sleep

During N9 (20:00 to 08:00, when nursing care is minimal), a 12-h polysomnography session (two electroencephalograms, two electrooculograms, a chin electromyogram, and two actimeters [on the right wrist and left ankle]) was performed (using an Alice 4 polysomnograph, Respironics, Nantes, France).

Sleep was scored visually offline (in 30-s windows) as rapid eye movement (REM), non–rapid eye movement (NREM), and transitional sleep [17]. In contrast to studies of evening sleep onset in adults and older children, we considered episodes of W to be taken into account; episodes of W had to occur and end spontaneously, and had to last for more than 10 min. Episodes of W with video-confirmed nursing interventions were excluded from our analysis if the intervention occurred within the 30 min before sleep onset. Finally, episodes of W had to be followed by active sleep, and episodes that were not followed by more than 12 min of sustained sleep were not considered.

2.1.2. Temperatures

An infrared camera (B400, Flir Systems, Issy les Moulineaux, France; emissivity: 0.97, reflected T: 32°C; transmission: 0.89) operated by Therma CAM Researcher 2.9 software (Flir Systems) was attached to the incubator's ceiling (at 40 cm from the neonate) and continuously recorded the neonate's skin temperatures. Skin Ts were measured offline semi-automatically every 5 min at six different body sites: the abdomen (T_{abdo}), the right pectoral region ($T_{pectoral}$), the external aspect of the middle of the left thigh (T_{thigh}), the eye region (T_{eye}), the dorsum of the hand (T_{hand}), and the dorsum of the foot ($T_{foot,}$ left side). Infrared technology enables the accurate measurement skin Ts without disturbing sleep.

In view of the absence of vasomotor control in the abdominal region in neonates, T_{abdo} is strongly correlated with the core body T [18]. T_{abdo} is also easier and safer to record than esophageal or rectal Ts. Lyon et al. [12] consider that the $T_{foot} - T_{abdo}$ difference is a good indicator of skin vasomotor activity. The $T_{hand} - T_{abdo}$ difference was also calculated.

The sleep onset was defined as time = 0 (t_0). Individual body temperatures were considered during the end of each episode of W (up to 20 min before sleep onset = $t_{initial}$) and during the following sleep episode (up to 50 min of sustained sleep and whatever the sleep stage). The mean ± SEM was calculated between −20 and + 50 min, and then plotted against time.

2.2. Partitional calorimetry

As described in detail elsewhere [16], body heat storage (S, kJ hr^{-1} kg^{-1}) was calculated by partitional calorimetry at $t_{initial}$ and at sleep onset (t_0) for each episode of W that fulfilled the above-mentioned criteria. Briefly, heat storage is calculated as the sum of the different body heat exchanges between the neonate's skin surface area and the surroundings. Partitional calorimetry takes account of the incubator air's temperature and humidity, the air velocity, the radiant temperature, the mean skin temperature T_{sk}, and clothing insulation. T_{sk} was assessed as follows: $T_{sk} = 0.15\ T_{foot} + 0.15\ T_{thigh} + 0.19\ T_{hand} + 0.28\ T_{head} + 0.115\ T_{chest} + 0.115\ T_{abdo}$ [19]. Body heat storage can be described by the body heat balance equation:

$$S = M \pm R \pm C \pm K - E_{resp} - C_{resp} - E$$

where M is the metabolic heat production (calculated using Chessex et al.'s equation) [20], conduction (K) is the heat transfer between the skin surface area and any materials with which the body is in contact (mainly the mattress), convection (C) is the heat exchange with the air around the neonate's skin, radiation (R) is the heat transfer between the skin surface and the surrounding surfaces (in the form of nonvisible, electromagnetic energy), evaporation (E) is the transfer via transepidermal water losses and from sweating, and evaporative exchanges (E_{resp}) and convective exchanges (C_{resp}) are heat exchanges through the expired air of the respiratory system. The heat exchanges can be positive (body heat gain) or negative (body heat loss).

Differences in thermal heat storage ΔS were calculated as the difference between the initial value ($S_{initial}$) and the value at sleep onset (S_{t0}). The change (ΔTb, °C/h) in the mean body temperature was calculated using the following equation: $\Delta Tb = \Delta S/3.494$, where 3.494 is the specific heat constant of the body tissues in kJ h^{-1} kg^{-1}.

2.3. Statistical analyses

All statistical analyses were performed with Statview software (version 5.0, Abacus Concepts Inc, Berkeley, CA). Data are provided as the mean ± standard error of the mean (SEM). Wilcoxon tests were used to compare temperature values at the initial time ($t_{initial}$) versus sleep onset (t_0). The threshold for statistical significance was set to $p < 0.05$.

3. Results

Of the 18 neonates studied, only eight (three girls and five boys; mean postmenstrual age: 211 ± 3 days; mean birth weight: 1369 ± 131 g, mean head circumference at birth: 27.3 ± 0.7 cm, mean body mass at N9: 1375 ± 107 g; two were small for gestational age) exhibited episodes of W that fulfilled the above-mentioned criteria. The 8 neonates did not differ significantly from the whole study population in terms of clinical and demographic variables. All the neonates were nursed in a cocoon in a closed incubator, and one wore a bonnet.

Fifteen of the 49 observed episodes of W were considered: on average, an episode lasted 23.2 ± 4.1 min (up to 63 min). An episode was always preceded by a long episode of sustained sleep (duration: 45.3 ± 9.4 min) and followed by a long episode of sustained sleep (duration: 73.2 ± 15.1 min). The earliest episode of W began at 20:10 pm, and the latest began at 07:25 (mean start time: 00:31).

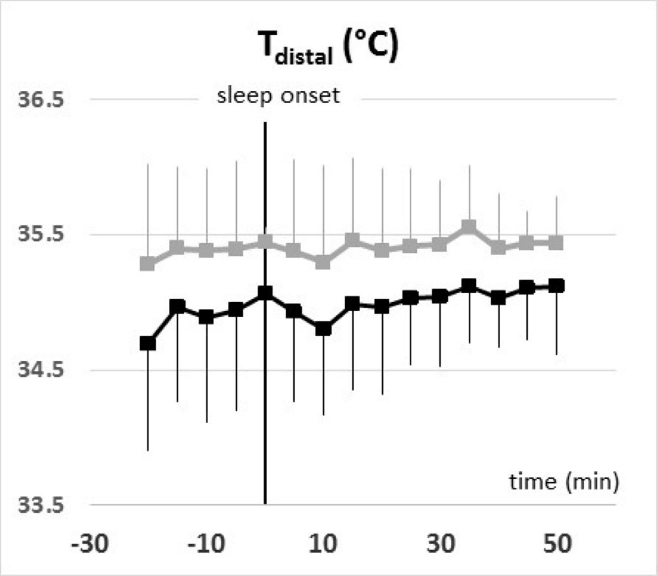

Fig. 1. Distal (T_{foot}, T_{hand}) temperatures (°C) (mean ± standard error of the mean) measured in neonates as a function of the time before and after sleep onset (vertical bar). Black squares correspond to foot temperature data, and gray squares correspond to hand temperature data.

Distal skin temperatures values (Fig. 1) increased markedly (by 0.38 °C) for T_{foot} ($Z = 2.66$, $p = 0.008$) and to a lesser extent (by 0.15 °C) for T_{hand} ($Z = 2.19$, $p = 0.028$). The decrease in skin T_{abdo} was not statistically significant (0.15 °C: $Z = 0.66$, not significant) (Fig. 2). As a result, the distal-to-proximal/abdominal temperature difference (see Fig. 3) increased significantly (by 0.52 °C) for T_{foot} ($Z = 3.02$, $p = 0.003$) and (by 0.30 °C) for T_{hand} ($Z = 2.60$, $p = 0.009$). After sleep onset, the distal temperatures stopped increasing. The distal temperatures decreased somewhat during the first 10 min of sleep (but nonsignificantly; T_{foot} = 0.1 °C/10 min, for example). After first 10 min of sleep, the decrease in distal temperatures continued but was very small.

3.1. Partitional calorimetry

Our calculations showed that the decrease in body heat storage ΔS from $t_{initial}$ to sleep onset was 0.51 ± 0.17 kJ h^{-1}.kg^{-1} (a change

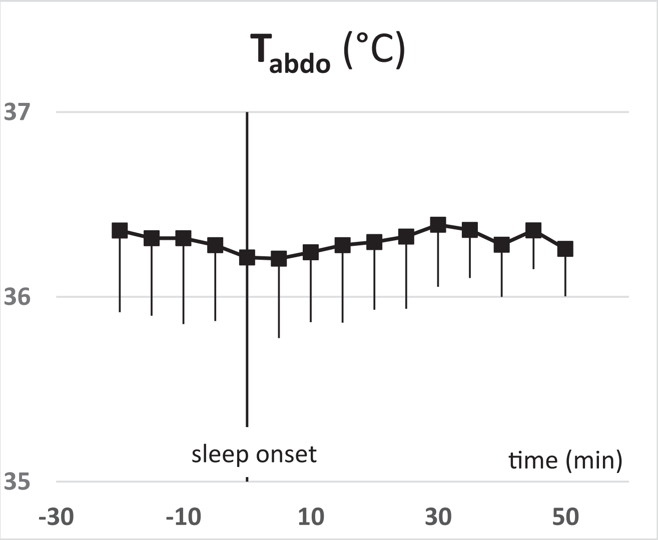

Fig. 2. Abdominal temperature (T_{abdo}) (°C) (mean ± standard error of the mean) measured in neonates as a function of the time before and after sleep onset (vertical bar).

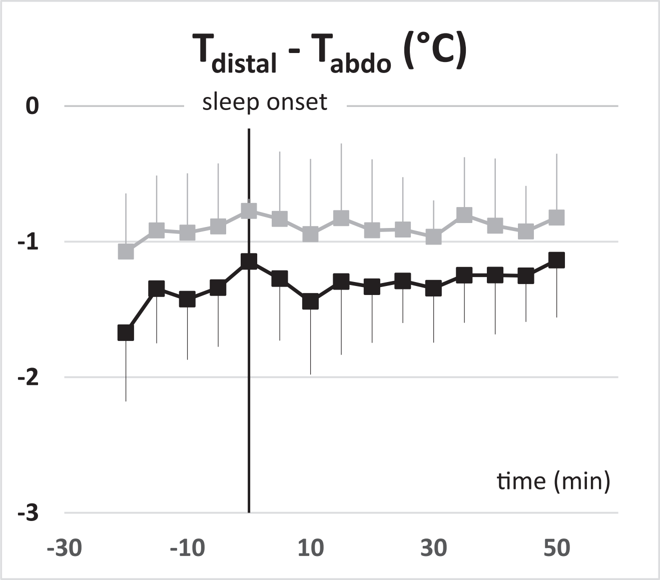

Fig. 3. Distal-to-proximal temperature gradients ($T_{foot} - T_{abdo}$; $T_{hand} - T_{abdo}$) (°C) (mean ± standard error of the mean) measured in neonates as a function of the time before and after sleep onset (vertical bar). Black squares correspond to foot temperature data, and gray squares correspond to hand temperature data.

from -12.76 ± 0.55 to -13.27 ± 0.60 kJ $h^{-1}.kg^{-1}$, $Z = 2.50$, $p = 0.013$), ie a decrease of 0.15 ± 0.05 °C/h in the average mean body temperature ΔT_b. This decrease corresponded to 4.0% ± 1.3% of the metabolic rate measured at $t_{initial}$.

4. Discussion

To the best of our knowledge, the present study is the first (1) to have established how distal skin temperatures vary before sleep onset in preterm neonates, and (2) to have assessed the repercussions of these variations on body heat storage and the core body temperature. Our results argue in favor of a "vegetative preparedness to sleep" in preterm neonates, such as that first described in adults by Magnussen in 1938 (cited in Ref. [21]). This sequence involves several steps, with significant increases in distal skin temperatures due to vasodilation at the hand (0.15 °C/20 min) and especially at the foot (0.38 °C/20 min). This vasodilation triggers heat losses, since these body regions have high convective heat transfer coefficients and are particularly efficient for convective and evaporative heat losses. The ultimate consequence is a decrease in body heat content over the 20 min preceding sleep onset. We previously reported that in neonates, episodes of W are shorter (consistently with the increased sleepiness and sleep propensity observed in adults [22,23]) when skin temperatures (and especially foot) are higher [24].

Kräuchi et al. [25] stated that at least five factors participate in the increased distal-to-proximal gradient leading to sleepiness. Interestingly, the environmental and behavioral parameters that have been demonstrated to have a major role in the pre-sleep increase in vasodilation and sleepiness in adults (cognitive relaxation, body relaxation [26], and reduced light intensity [25]) cannot operate in preterm neonates. In the present study, the neonates were in a nearly constant night-time environment in the NICU and in the closed incubator, notably with regard to environmental parameters (light, noise, and air temperature) and the nursing routine (feeding, etc) during pre-sleep versus sleep periods. Even though lying down has a major role in distal vasodilation [27,28], the absence of postural changes (as in our neonates) does not totally suppress vasodilation in adults [25]. This raises the question of the role of circadian rhythms in sleep onset: in adults, sleepiness increases and sleep onset usually occurs during the fall in core body temperature during the evening. In preterm neonates, it is generally considered that circadian rhythms (and body temperature circadian rhythms in particular) become established at 6 weeks of life [9,29,30]. However, several studies have demonstrated the emergence of low-magnitude circadian rhythms before this time point [31,32]. Furthermore, the amplitude of circadian rhythms is lower in younger infants than in older ones [33]. Similarly, circadian melatonin rhythms are absent for the first few weeks of life [34,35]. Hence, the onset of melatonin secretion (which is also of great importance in the evening vasodilation in adults) [23,36] is probably not involved in sleep preparedness in neonates, especially when they are nursed in a nearly constant light environment, as in the present study.

Even though sleep processes develop progressively in neonates [30], it is important to note that vasodilation exists. There are great differences in sleep between adults and neonates. In neonates, sleep is polyphasic, and episodes of wakefulness are shorter and spread out over the 24-h period, whereas human adults display monophasic, long-lasting, daytime wakefulness. Sleep in neonates begins with active sleep, whereas slow-wave sleep (the precursor of which in neonates is quiet sleep) begins sleep in adults and is sometimes considered to have a particular role in sleep-thermoregulatory processes [37]. Finally, neonates have a shorter time scale for homeostatic sleep pressure than older infants, in whom longer periods of daytime wakefulness merge [38].

Since higher distal skin temperatures increases sleep propensity in preterm neonates [24], one can question the possible repercussions of distal vasodilation on core body temperatures and body heat content. In the present study, the changes in body heat storage were calculated using partitional calorimetry; in routine clinical practice, it is difficult to apply direct calorimetry by measuring energy expenditure and oxidative processes via O_2 consumption and CO_2 production. Our results indicate that the peripheral vasodilation induces body cooling, as evidenced by a fall in the body temperature of 0.15 °C/h. This fall is quite large, although it was smaller than those observed in 6- to 12-year-old infants (1.5 °C over 20 min [39]) and adults (1.6 °C over 20 min [21]). We calculated that in order to maintain body homeothermia, this fall would be compensated by a 4% increase in metabolic heat production. Even though a preterm neonate can afford to increase its metabolic heat production to this extent, the fact that the heat content is related to the body mass, and is therefore much lower for a neonate than for an adult, might explain why peripheral skin vasodilation does not continue after sleep onset (Fig. 1), in contrast to what is found in adults [21] and in older infants [39]. In contrast, Brown et al. [8] reported that the distal skin vasodilation after sleep onset is lower in young neonates (less than 8 weeks old) than in older infants. However, judging by the reported air temperature and the absence of an incubator, it appears that Brown et al. studied term neonates. The vasoconstriction observed after sleep onset therefore helps to prevent prolonged heat loss. It probably results from sleep structure differences between neonates and adults; active sleep appears after sleep onset (and is characterized by an increase in sympathetic tone), whereas the autonomic balance is in favor of parasympathetic tone during non−rapid eye movement sleep, the first stage after sleep onset in adults. Lindqvist et al. [40] have observed that in neonates, vasomotricity is controlled more by the sympathetic system than by the parasympathetic system.

Interestingly, we observed the effects of vasodilation in neonates being treated with caffeine, an anti-apneic compound with vasoconstrictive effects. In the future, it would be interesting to compare our present results with those in similar infants not treated with caffeine, to establish whether caffeine's vasoconstrictive effect dampens pre-sleep vasodilation and thus might perturb sleep state initiation. Indeed, a decrease in vasoconstriction might account for the adverse effects of caffeine on sleep [41]; it has been shown that low distal vasodilation and low distal temperatures alter sleep patterns in adults [42,43].

5. Conclusion

For the first time, our study highlighted the occurrence of distal skin vasodilation (especially at the foot) prior to sleep in preterm neonates. This vasodilation is part of the “vegetative preparedness to sleep” observed in adults and older children, and occurs even though (1) most of the factors known to have a major role in pre-sleep vasodilation in adults are not yet present in these neonates, and (2) the neonates are nursed in a nearly constant environment. The resulting fall of 0.15 °C/h in the mean body temperature must be compensated for by a 4% increase in metabolic heat production to maintain body homeothermia; however, this increase cannot be maintained for a long period of time.

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Conflict of interest

The authors have no financial relationship relevant to this article to disclose.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.12.026.

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