Objective: To evaluate the influence of ambient temperature on infant's sleep and cardiorespiratory parameters during sleep.

Patients and method: 20 healthy infants with a median age of 11.5 weeks (range 7 to 18 weeks) were recorded polygraphically for one night. They were exposed to 3 different ambient temperatures (20°C-25°C-30°C). Ambient and core temperatures were measured throughout the procedure.

Results: Influence of ambient temperature was seen in:

Respiratory parameters: The frequency of central apneas increased significantly with increasing temperatures in REM sleep, but not in NREM sleep.

Heart rate (HR) parameters and HR spectral analysis: Elevation of temperature was characterized by significantly higher basal HR, shorter RR intervals, and lower parasympathetic activity in REM and NREM sleep.

Saturation in oxygen: During total sleep time, rise in temperature induced a decrease in basal oxygen saturation. During REM sleep, a greater frequency of oxygen saturation drops was associated with central apneas.

Core temperature: With increasing ambient temperature, the rise of rectal temperature was mild. Despite this lack of significant increase, similar results were found when sleep and cardiorespiratory parameters were evaluated according to rectal temperatures.

Conclusion: Changes in ambient temperatures associated with mild increases in body temperature significantly modified cardiorespiratory parameters and autonomic controls in healthy infants. The changes associated with increases in temperature were mainly seen during REM sleep.

Key words: Apnea; autonomic nervous system; infant; temperature; sleep; sudden infant death syndrome

INTRODUCTION

THE RELATIONSHIP BETWEEN ENVIRONMENTAL TEMPERATURE AND SLEEP CHARACTERISTICS HAS BEEN STUDIED IN PREMATURE INFANTS, full term newborns, adults and animals. Higher ambient temperatures increase basal heart rate (HR), decrease HR variability and favor the development of central and obstructive sleep apneas, mainly during REM sleep.

Thermoregulatory mechanisms also influence sleep-wake controls, with the longest sleep duration seen at thermoneutrality. Few data are available on the role of ambient temperature on infant's sleep and cardiorespiratory parameters during sleep. Infants are vulnerable to heat stress and excess in environmental temperatures was associated with the development of sudden infant deaths (SIDS).

The present study was designed to evaluate the effects of changes in ambient temperatures on sleep, cardiac, and autonomic characteristics in healthy infants.

METHOD

Patients

Twenty infants with a median age of 11.5 weeks (range 7 to 18 weeks) were studied. There were 11 boys and 9 girls, born at term (range of 37 to 41 weeks gestation), with a median birth weight of 3.250 gr. (range of 2.600 to 3.950 gr.). The infants had been admitted to join a sleep research program on normal maturation of sleep-related behavior. None had a history of sleep problems or apnea. At the time of investigation, no infant showed signs of infection or neurological problems. None was receiving medication. The aim and the methodology of the study were approved by the University Ethical Committee and were explained to the parents, who gave their informed consent.

Monitoring Procedures

Monitoring took place over an eight-hour nocturnal session in a quiet, darkened room. All infants slept on their...
The infants were fed on demand, receiving maternal or cow's milk formula in the usual volumes administered at home. Immediate assistance by an experienced pediatrician was available during the experiment. The infants were wearing their pajamas and were covered with a duvet. The clothing and bedding were evaluated to correspond to 5°C Tog Insulation.20,27 The lower critical temperature corresponding to the thermoneutrality range was defined as 20–22°C.27 The infants' behavior as well as the nursing interventions were charted. The data were collected on computerized polygraph recorders (Morpheus System, Medatec, Brussels, Belgium). The following variables were recorded simultaneously: two scalp electroencephalograms, two electrooculograms, digastic electromyogram, electrocardiogram, thoracic and abdominal respiratory movements, and airflow by thermistors taped under each nostril and on the side of the mouth. An actigram was placed on one arm to measure gross body movements. Oxygen saturation was recorded continuously from a transcutaneous sensor (Ohmeda Biox, Hayward, CA).

**Temperature Measurements**

Core temperature was measured throughout the procedure by means of a rectal temperature probe placed 1 cm inside the infant's rectum, and environmental temperature with an air temperature probe placed within 20 cm of the infant's face. These probes were connected to the polygraph and the temperature signals were displayed continuously and recorded on the sleep recording.

**Changes in Ambient Temperature**

Each infant was assessed at ambient temperatures of 20°C, 25°C, and 30°C. All infants fell asleep at an ambient baseline room temperature of 25°C. The infants were randomly divided into two groups. For 10 infants, the ambient temperature was first raised and then lowered. For the 10 other infants, the temperature was first lowered, then raised. To raise the temperature an electrical convector heater, 3 m from the cot, was used; there was no obvious noise from the fan. The room temperature was lowered to 20°C by opening an upper part of the room window. Care was taken to ensure that the infant was not in a direct draft.

Thermal environment has been changed slowly to avoid the arousal effect of thermal loads.11,12 The infants remained at the desired temperature for 20 minutes for adaptation before physiological variables were recorded during a median of 30 minutes (thermal plateau periods).

A study was interrupted when an infant woke up (opened the eyes and/or cried).

The only variable that changed throughout study periods was the ambient temperature, with each baby acting as his or her own control.28

**Data Analysis**

At each thermal plateau of 20°C, 25°C, or 30°C, 30-second periods of the recordings were analyzed and categorized as either nonrapid eye movement sleep (NREM), rapid eye movement (REM), indeterminate sleep, or wakefulness according to criteria recommended in the literature.29,30 NREM refers to NREM 2 and 3 stages. Gross body movements were measured by actigraphs and confirmed visually. Sleep states changes were defined by the interruption of the state by another state for more than one minute or longer. Median values for oxygen saturation, heart rate, and respiratory rate were calculated on one-minute stable sleep epochs, at least five minutes after any change in body position, movement, sigh, or arousal. Sleep apneas were scored only if they lasted three seconds or more. A central apnea was scored when flat tracings were obtained simultaneously from strain gauges and thermistors. Periodic breathing was defined by the succession of more than two central apneas, separated from each other by less than 20 seconds of breathing. An obstructive apnea was scored when continuous deflections were obtained from strain gauges, while a flat tracing was recorded from thermistors. To avoid artifact scoring due to thermistor displacement, obstructive apneas preceded by body movements, crying, or sighs were rejected. Mixed apneas were defined as central apneas followed directly by obstructive episodes, and were scored together with the obstructive apneas.

The electrocardiogram was recorded in DII, anterolateral position. A drop or a rise in HR referred to changes greater than 10% of basal values. Overall HR variability was defined as the standard deviation of the RR values. A drop in oxygen saturation referred to changes greater than 3% of basal values.

During thermal plateaus, the frequency of NREM, REM, indeterminate sleep, movements, awakenings were calculated by dividing the duration of each sleep-wake stage by the total duration of the thermal plateau, then multiplying by 100, to obtain a percentage. The frequency of apnea was calculated as an index by dividing the absolute number of respiratory events during the thermal plateau by the total sleep time of the plateau. A similar index was also calculated for each specific type of respiratory event and for

---

**Table 1—General characteristics of the infants studied**

<table>
<thead>
<tr>
<th>N° of infants</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M/F)</td>
<td>11/9</td>
</tr>
<tr>
<td>Gestational age (wk)</td>
<td>39 (37-41)</td>
</tr>
<tr>
<td>Postnatal age (wk)</td>
<td>11.5 (7-18)</td>
</tr>
<tr>
<td>Birth weight (g)</td>
<td>3250 (2600-3950)</td>
</tr>
<tr>
<td>Weight at study (g)</td>
<td>5615 (3000-7250)</td>
</tr>
</tbody>
</table>

The figures represent absolute, median and range of values.
each stage of sleep. The same procedures were made for
sleep state changes and for heart rate and oxygen saturation
drops.

**HR Spectral Analysis**

Digitized ECG signals were sampled at 300 HZ. An
autoregressive power spectral analysis of the spontaneous
beat-to-beat variabilities in RR intervals was per-
formed. Autoregressive power spectrum was calculated
for three epochs of 256 heartbeats in each thermal plateau.
Premature ventricular contractions or artifact RR intervals
due to gross body movements or arousals were eliminated
by visual analysis of the HR data before HR spectral anal-
ysis was performed. Power spectral analysis of HR fre-
quency fluctuations has shown the existence of two major
spectral components: a low-frequency component (LF)
defined by center frequency of 0.1 Hz (0.04 -0.15 Hz) relat-
ed to ortho and parasympathetic activities and a high-fre-
cuency component (HF) defined by center frequency of 0.4
Hz (> 0.15-2 Hz) reflecting parasympathetic tone. Respiratory frequency during the selected period was mea-
sured manually after being printed on paper. For each 256
RR interval period, the major component in the LF band of
the HR spectrum was related to the major component in the
HF band corresponding to the mean respiratory frequency
as determined by analysis of breath to breath intervals. The
ratio of LF/HF powers for each episode was calculated as
an index of sympathovagal interaction. Spectral compo-
ments were represented as RR intervals (in ms), power (in
msec2), bandwidth (in Hzeq), and normalized power
values, obtained by dividing the power of the frequency
band by the total power (in %), after subtraction of the
direct current component. Optimal autoregressive model
order was determined by minimizing the value of the final
predictor error. Stationary was confirmed by pole dia-
gram analysis. To determine if any observed differences
in spectral analysis results were related to the differences
seen in heart rate, each spectral power was normalized for
heart rate (spectral power was divided by the square of the
mean heart rate value of the analyzed epoch).

**Statistical Analysis**

For each infant, the sleep characteristics were compared
for each thermal plateau. Statistical analysis was performed
with the use of an analysis of variance for repeated mea-
sures for ambient temperature (PA) and for core tempera-
ture (PR). Due to the study design, sleep stades character-
istics were compared between 20°C and 30°C, the results
were analyzed with the use of t-student for matched-pairs.
Statistical significance was defined with a level of <.05.

These statistical analyses were done for all the infants
together and for the two groups of infants separately: for
those for whom the ambient temperature was first raised
and then lowered, and for those for whom the ambient tem-
perature varied in the opposite way.

**RESULTS**

The major characteristics of the 20 infants included in
the analysis are shown in Table 1.

| Table 2—HR characteristics in different temperatures in REM and NREM sleep |
|-----------------------------------------------|---------|---------|--------|--------|--------|
| **REM**                                      | 20°C    | 25°C    | 30°C   | PA     | PR     |
| Basal heart rate (beat/min)                   | 120.9   | 124.28  | 129.7  | 0.043  | 0.05   |
| Mean RR intervals (msec)                      | 506.05  | 486.16  | 468.33 | 0.028  | 0.05   |
| Minimal heart rate (beat/min)                 | 107.32  | 112.37  | 121    | 0.001  | 0.005  |
| RR Variability (msec)                         | 35.39   | 23.03   | 18.5   | 0.040  | 0.005  |
| Frequency of decelerations after central apneas | 0.11    | 0.14    | 0.20   | 0.008  | 0.002  |

| **NREM**                                     |         |         |        |        |        |
| Basal heart rate (beat/min)                   | 116.6   | 122.15  | 127.87 | 0.001  | <0.001 |
| Mean RR intervals (msec)                      | 522.87  | 486.16  | 468.33 | 0.003  | 0.004  |
| Minimal heart rate (beat/min)                 | 107.33  | 115.88  | 121.33 | 0.021  | <0.004 |

The figures represent mean values with standard deviation. Analysis of variance for repeated measures was used. PA: analysis of variance with ambient temperature, PR: analysis of variance with central temperature.
Ambient and Rectal Temperatures

The mean time for the room temperature to rise to 30°C was 42.7 +/- 21 min and to decrease to 20°C was 45.2 +/- 23 min. There was no significant difference in the changes of temperature duration between the two groups of temperatures.

There was a mild increase in the mean rectal temperature with increasing ambient temperature but the difference did not reach statistical significance (mean of 36.64 ± 0.68°C at 20°C, 36.66 ± 0.56°C at 25°C, 36.87 ± 0.46°C at 30°C (NS)).

The thermal plateau at 25°C was at 11.43 PM (+/- 45 min), the second and third thermal plateaus occurred successively at 1:16 AM (+/- 116 min) and 2:27 AM (+/- 112 min).

Sleep Organization

Due to the study design, sleep states characteristics were compared between 20°C and 30°C.

Following sleep recordings, no significant differences were found for the following variables: total recording time (mean duration of 28 +/- 5.65 min at 20°C and mean duration of 27.3 ± 5.05 min at 30°C), sleep efficiency (mean of 97.5 ± 8.2% at 20°C and mean of 96.5 ± 11.5% at 30°C), frequency of NREM sleep (mean of 36.9 ± 26.9% at 20°C and 28.1 ± 21.1% at 30°C), frequency of REM sleep (mean of 47.4 ± 27.4% at 20°C and 58.5 ± 25.5% at 30°C), frequency of indeterminate sleep (mean of 0.43 ± 0.32% at 20°C and 0.34 ± 0.26% at 30°C), sleep stage changes per hour (mean of 1.25 ± 0.9 at 20°C and mean of 1.1 ± 0.55 at 30°C), movements (mean of 12.7 ± 10% at 20°C and 9.5 ± 9.5% at 30°C), or arousals (mean of 2.5 ± 8.2% at 20°C and 3.5 ± 11.5% at 30°C). Compared to period at 20°C, during the plateau at 30°C, REM sleep frequency tended to increase but the difference did not reach statistical significance. During the two thermal plateaus, all subjects had REM sleep; only two did not have NREM sleep at 30°C, and three at 20°C (NS). The amounts of indeterminate sleep were too low to allow cardiorespiratory analysis; these analyses have been restricted to REM and NREM sleep.

Respiratory Events

The frequency of central apneas increased significantly with increasing temperature during REM sleep (mean 0.10 +/- 0.08 at 20°C, 0.12 +/- 0.10 at 25°C, 0.21 +/- 0.14 at 30°C (PA = 0.009, PR = 0.001). This finding was not found during NREM sleep. With elevation of temperature, no significant differences were found for the following variables in REM sleep: breathing rate (mean 33.4 +/- 9.58 breath per min at 20°C, mean 35 +/- 6.93 breath per min at 25°C, mean 35.4 +/- 6.76 breath per min at 30°C), duration of central apnea (mean of 4.76 ± 1.17 sec. at 20°C, mean of 4.82 ± 1.49 sec. at 25°C, 6.19 +/- 1.81 sec. at 30°C), duration of obstructive event (mean of 5.41 ± 0.82 sec. at 20°C, mean of 6.03 ± 2.22 sec. at 25°C, 6.58 +/- 1.89 sec. at 30°C), frequency of periodic breathing (mean of 0 ± 0% at 20°C, 0.03 ± 0.12% at 25°C, 0.09 +/- 0.24% at 30°C), frequency of obstructive events (0.04 +/- 0.06 at 20°C, 0.05 +/- 0.09 at 25°C, 0.09 +/- 0.13 at 30°C). No significant differences were seen for these parameters with increasing temperature in NREM sleep.

Heart Rate and Heart Rate Variability

As shown in Table 2, elevation of temperature was characterized by significantly higher basal HR, shorter RR intervals, higher minimum HR values during REM, and NREM sleep. With elevation of temperature, smaller HR variability was seen in REM sleep. The frequency of HR decelerations associated with central apneas was higher with increasing temperature in REM sleep. No significant differences were seen in the frequency of decelerations associated with obstructive apneas in REM and NREM sleep.

<table>
<thead>
<tr>
<th>Table 3—Oxygen saturation characteristics in different temperatures in total sleep time and REM sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TOTAL SLEEP TIME</strong></td>
</tr>
<tr>
<td>Basal saturation (%)</td>
</tr>
<tr>
<td>REM SLEEP</td>
</tr>
<tr>
<td>Basal saturation (%)</td>
</tr>
<tr>
<td>Frequency of saturation drops after central apneas</td>
</tr>
<tr>
<td>Minimum saturation (%)</td>
</tr>
<tr>
<td>% saturation drops after central apneas</td>
</tr>
</tbody>
</table>

The figures represent mean values with standard deviation. Analysis of variance for repeated measures was used. P A: analysis of variance with ambient temperature, P R: analysis of variance with central temperature.
Oxygen Saturation

Elevation of temperature was characterized by a decrease in basal saturation in oxygen during total sleep time and a higher frequency of saturation drops associated with central apneas in REM sleep. Minimum saturation values were lower and the percent of saturation drops was higher in REM sleep with increasing ambient temperature (Table 3). These findings did not reach statistical significance with core temperature changes.

No significant differences were seen in the frequency of saturation drops associated with obstructive apneas in REM and NREM sleep.

HR Spectral Analysis

As shown in table 4, with increasing ambient temperature, HF normalized power significantly decreased in REM and NREM sleep. These findings were also found with core temperature changes in NREM sleep but not in REM sleep. With temperature elevation, the HF bandwidth was larger in NREM sleep but not in REM sleep.

There were no significant differences in the total power values, LF and HF powers, LF normalized powers, LF and HF frequency, LF bandwidth, or LF/HF ratios in both REM and NREM sleep. Normalization of the spectral powers by heart rate did not change the findings.

Central Temperature

Despite of lack of significant changes in central temperature, similar significant results were obtained using rectal temperature as a variable instead of ambient temperature, except for HF normalized power, minimum saturation values and percent of saturation drops after central apnea in REM sleep.

During thermal challenges, no infant showed abnormal clinical signs and no intervention was required. All observations were independent of the infant’s sex, type of feeding, time of feeding, temperature schedule (initial increase or decrease in ambient temperature), gestational, or postnatal age.

DISCUSSION

Sleep Stages

In this study, changes in ambient temperature were not associated with significant changes in sleep characteristics. Within our experimental conditions, the lower range of thermal neutrality was estimated to be between 20 to 22°C.27 The highest range of thermal neutrality could not be evaluated, because of lack of data on caloric insulation and metabolic rates.27 Despite these uncertainties, the lack of relation between thermal loads and sleep organization seen in the present study may be related to the absence of significant increase in core temperature and/or the short duration of the thermal loads.

Changes in Cardiac Parameters

Elevation of ambient temperature was associated with higher basal HR, shorter RR intervals, and lower HF normalized powers. The findings were seen during REM and NREM sleep. During REM sleep, elevation of ambient temperature was also related to a smaller HR variability and an increased frequency of HR decelerations following central apneas. Similar changes in HR and HR variability were reported during thermal loads.3,13,10

There were only a few reports on the effects of changes in ambient temperatures on HR spectral analysis in adults,38,39 but none in infants. In our subjects, elevation of

| Table 4—HR spectral analysis in different temperatures in REM and NREM sleep |
|-----------------------------|--------|--------|--------|-----|-----|
| REM                      | 20°C  | 25°C  | 30°C  | P A | P R |
| High-Frequency Component |        |        |        |     |     |
| Power (ms²)              | 135.35| 127.06| 75.19 | NS  | NS  |
| Normalized power (%)     | 13.19 (+/-5.23) | 12.77 (+/-6.51) | 9.65 (+/-3.77) | 0.031 | NS  |
| Band width (Hz eq)       | 0.14 (+/-0.04) | 0.14 (+/-0.04) | 0.13 (+/-0.03) | NS  | NS  |
| Low/High frequency ratio (%) | 6.31 (+/-4.67) | 6.11 (+/-4.6) | 7.7 (+/-4.1) | NS  | NS  |

NREM

| High-Frequency Component |
|-----------------------------|--------|--------|--------|-----|-----|
| Power (ms²)              | 250   | 137.22| 147.41| NS  | NS  |
| Normalized power (%)     | 33.03 (+/-20.18) | 31.46 (+/-12.9) | 20.79 (+/-11.33) | 0.018 | 0.050 |
| Band width (Hz eq)       | 0.06 (+/-0.03) | 0.07 (+/-0.03) | 0.1 (+/-0.04) | 0.035 | 0.046 |
| Low/High frequency ratio (%) | 2.34 (+/-1.8) | 2.32 (+/-2.11) | 3.36 (+/-2.88) | NS  | NS  |

The figures represent mean values with standard deviation. Analysis of variance for repeated measures was used. P A: analysis of variance with ambient temperature.
ambient temperature was associated with a significant decrease in HF normalized power. The finding could possibly reflect a reduction in cardiac vagal control. These observations are in agreement with reports on adults subjects exposed to passive heating, in whom the changes in autonomic controls were attributed to a combination of vagal withdrawal, small elevation of orthosympathetic activity, and metabolic mechanisms.

Breathing Parameters

Higher ambient temperatures were associated with an increased frequency of central sleep apneas. The findings were most marked in REM sleep. Similar findings were reported in premature infants, as well as in animal studies. It was suggested that hyperthermia could lead to a transient decrease in respiratory chemoreceptor sensitivity. Animals exposed to thermal challenges developed apneas in response to respiratory irritants and exhibited an enhancement of the laryngeal closure reflex. The latest effect was particularly marked in the youngest animals and was attributed to an age- and temperature-dependent change in axonal conduction and synaptic transmission velocities. The modest thermal load in our infants did not modify breathing rate, frequency of obstructive apneas, or percentage of periodic breathing. An increase in periodic breathing has been reported in premature infants and in young animals exposed to higher ambient temperatures. Our observation could be associated with a decrease in periodic breathing propensity with advancing age.

The significant increase in apnea frequency was observed in REM sleep only. During REM sleep, both sensory and motor functions are impaired. This combination of decreased sensory and motor function probably contributed to the marked impairment of ventilatory responses during REM sleep and may be important in the initiation and continuation of apneas.

Changes in Saturation of Oxygen

Elevation of ambient temperature was characterized by a decrease in basal oxygen saturation and a greater frequency of oxygen drops associated with central apneas. The last effect was seen mainly during REM sleep. The lower oxygen saturation values seen with higher temperatures could be related to an increase in basal metabolism. The influence of temperature was significantly greater in REM sleep than in NREM sleep. During REM sleep, infants are more at risk for O2 saturation drops because of relatively low O2 tensions, high incidence of apnea, high metabolic O2 consumption and fall in O2 stores.

Clinical Implications

Hyperthermia due to overdressing or high environmental temperature is considered to be a contributing factor to SIDS. In the present study, exposing healthy sleeping infants to an elevation of ambient temperature led to changes similar to those reported as risk factors for SIDS, such as decrease in respiratory stability and changes in HR autonomic controls. It remains to be evaluated whether these findings contribute to the occurrence of sudden death during sleep in an infant exposed to overheating during sleep.

CONCLUSION

Elevation of ambient and corresponding mild core temperatures led to significant changes in cardiac and respiratory control systems. These changes could contribute to our understanding of the mechanisms implicating higher ambient temperatures in the mechanism of the death of some infants.

ACKNOWLEDGMENTS

The study was supported by the Fondation Scientifique Universitaire Erasme.

REFERENCES


