Hypoxic and Hypercapnic Events in Young Infants During Bed-sharing

AUTHORS: Sally A. Baddock, PhD,a,b Barbara C. Galland, PhD,a David P.G. Bolton, MRCP, PhD,a Sheila M. Williams, DSc,c and Barry J. Taylor, MBChB, FRACPd

Departments of aWomen’s and Children’s Health, and bPreventive and Social Medicine, University of Otago, Dunedin, New Zealand; and cSchool of Midwifery, Otago Polytechnic, Dunedin, New Zealand

KEY WORDS
co-sleeping, infant, oxygen desaturation, rebreathing, thermoregulation, sudden infant death syndrome, sleep

ABBREVIATIONS
AS—active sleep
BS—bed-sharing
CI—confidence interval
CO2—carbon dioxide
CS—cot-sleeping
HR—heart rate
QS—quiet sleep
Sao2—arterial oxygen saturation
SIDS—sudden infant death syndrome

WHAT’S KNOWN ON THIS SUBJECT: Sudden infant death syndrome remains the major cause of postneonatal death in developed countries. Although infant-parent bed-sharing following antenatal smoking or maternal consumption of alcohol on the bed-sharing night increases the risk of death, the mechanism is not known.

WHAT THIS STUDY ADDS: Bed-sharing infants experienced more oxygen desaturations and episodes of carbon dioxide rebreathing than cot-sleeping infants but showed appropriate behavioral and physiologic responses. A deficit in these responses in vulnerable infants could link to increased risk of sudden infant death syndrome.

abstract

OBJECTIVES: To identify desaturation events (arterial oxygen saturation [Sao2] <90%) and rebreathing events (inspired carbon dioxide (CO2) >3%), in bed-sharing (BS) versus cot-sleeping (CS) infants.

METHODS: Forty healthy, term infants, aged 0 to 6 months who regularly bed-shared with at least 1 parent >5 hours per night and 40 age-matched CS infants were recruited. Overnight parent and infant behavior (via infrared video), Sao2, inspired CO2 around the infant’s face, and body temperature were recorded during sleep at home.

RESULTS: Desaturation events were more common in BS infants (risk ratio = 2.17 [95% confidence interval: 1.75 to 2.69]), associated partly with the warmer microenvironment during BS. More than 70% of desaturations in both groups were preceded by central apnea of 5 to 10 seconds with no accompanying bradycardia, usually in active sleep. Apnea >15 seconds was rare (BS infants: 3 events; CS infants: 6 events), as was desaturation <80% (BS infants: 3 events; CS infants: 4 events). Eighty episodes of rebreathing were identified from 22 BS infants and 1 CS infant, almost all preceded by head covering. During rebreathing, Sao2 was maintained at the baseline of 97.6%.

CONCLUSIONS: BS infants experienced more oxygen desaturations preceded by central apnea, partly related to the warmer microenvironment. Rebreathing occurred mainly during bed-sharing. Infants were at low risk of sudden infant death syndrome and maintained normal oxygenation. The effect of repeated exposure to oxygen desaturation in vulnerable infants is unknown as is the ability of vulnerable infants to respond effectively to rebreathing caused by head covering.

PEDIATRICS Volume 130, Number 2, August 2012

WHAT’S KNOWN ON THIS SUBJECT: Sudden infant death syndrome remains the major cause of postneonatal death in developed countries. Although infant-parent bed-sharing following antenatal smoking or maternal consumption of alcohol on the bed-sharing night increases the risk of death, the mechanism is not known.

WHAT THIS STUDY ADDS: Bed-sharing infants experienced more oxygen desaturations and episodes of carbon dioxide rebreathing than cot-sleeping infants but showed appropriate behavioral and physiologic responses. A deficit in these responses in vulnerable infants could link to increased risk of sudden infant death syndrome.
Bed-sharing (BS) has special significance in many cultures and is widely practiced for perceived benefits in parent/infant bonding, encouraging breastfeeding, and reducing maternal sleep disruption1–3 and infant stress.4 The proximity of mother and infant facilitates increased interactions, waking, and breastfeeding.5–9 Similar benefits also occur during early skin-to-skin contact.10 However, many factors are associated with an increased risk of sudden infant death syndrome (SIDS) during infant–parent BS, including maternal smoking during pregnancy,11 parental consumption of alcohol or sedative drugs,12 excessive parental tiredness,13 sleeping on a couch,12,14 young infant age,15 and excess bedding.13 In this context, it is important to determine behavioral and environmental factors that affect infant physiology and that could increase the risk of SIDS.

The presence of adults in bed, as well as the use of adult bedding (eg, heavy duvets), expose infants to a different environment compared with sleeping alone in a cot. A component of this is a warmer microenvironment, thus raising peripheral temperature of BS infants.16 The rectal–shin temperature difference, an indicator of thermal comfort,17 is smaller in BS infants, indicating a thermolytic state, whereas in cot-sleeping (CS) infants it is larger, indicating a state of thermogenesis.16 Patterns of thermoregulation, sleep state, and breathing are tightly linked,18 and elevated body temperatures (hyperthermia) can trigger periodic breathing in infants.19

Most studies of infant sleep physiology are of solitary sleeping infants, with few studies describing infants’ responses to the BS environment. Polysomnographic studies of infants at low risk of SIDS report decreased time in quiet sleep (QS)20 and increases in arousals21 periodic breathing,22 inspired carbon dioxide (CO2),23 heart rate (HR),24 peripheral26 and axillary temperature25 and synchronicity of mother–infant sleep cycles26 during BS. The aim of this study was to investigate the usual sleep practices of infants in their own home to identify and compare desaturation events (arterial oxygen saturation [Sao2] <90%), and rebreathing episodes (inspired CO2 >3%) between BS infants and CS infants that could improve understanding of the impact of the different environments of BS and CS on infant physiology.

**METHODS**

**Participants**

The power calculation was based on a study identifying the frequency of high-risk behavior in CS infants.27 From this data, we predicted 50% of BS infants and 20% of CS infants were likely to experience a potentially dangerous event. By using a binomial distribution, it was suggested that 40 to 50 infants were needed to show this difference at the 5% level of significance with 80% power.

Forty infants who regularly slept in the parental bed with 1 or both parents for 5 hours per night were studied, as previously described.2 Briefly, BS families were recruited through local postnatal groups and media advertising. CS infants matched for age and season of gestation (except 2 infants in the ‘long play’ group who were 28 and 32 weeks’ gestation, respectively). Forty infants who regularly slept in the parental bed with 1 or both parents for a minimum of 5 hours per night (BS) and 40 age-matched CS infants who slept in a cot or bassinet for at least 5 hours per night were studied, as previously described.2 Briefly, BS families were recruited through local postnatal groups and media advertising. CS infants matched for age and season of study were recruited from the postnatal ward of a local hospital. Infants were aged 0 to 6 months and were >37 weeks’ gestation (except 2 infants in each group who were 28 and 32 weeks’ gestation, respectively).

The study was approved by the Southern Regional Health Authority Ethics Committee, New Zealand (protocol 97/04/036). Informed consent was obtained from the parents.

**Study Protocol**

Infants were monitored over 2 consecutive nights during their usual sleep practice (bed-share or cot-sleep) in their home. The details of recordings have been described previously.16 Briefly, the first night involved video recording and the second, both video and physiologic recording. The latter data included electrocardiogram, Sao2, and HR (Nellcor N-200, Nellcor, Inc, Hayward, CA), respiratory pattern via chest and abdominal bands (Respitrace model 150; Respitrace Co, NY), airflow via nasal thermistors, and shin and rectal temperature. The level of CO2 in the microenvironment around the infant’s face was measured (NORMOCAP 200 OXY; Datex Instrumentation Corp, Helsinki, Finland) from air sampled through a catheter attached to midway between the inner canthus of the infant’s eye and the lateral edge of the nostrils.

All leads were secured to allow mothers to handle infants freely during the night. All physiologic signals from the recording units were relayed through a portable polygraphic system (BabyLog Link; Christchurch Hospital, New Zealand) and stored on a laptop computer. A small surveillance camera (Panasonic CEC-C38, Osaka, Japan) and infrared light source allowed recordings on a video recorder (Panasonic AG-TL700, Osaka, Japan), set to “long play” (to enable 15 hours of recording on a 3-hour videotape), of the infant’s sleep position, movements, and parent–infant interactions, as previously described.3

Mothers completed a questionnaire providing demographic information.

**Data Analyses**

**Desaturation Events**

A desaturation event was defined as Sao2 <90%28,29 lasting for ≥1 second. The events were reviewed in association with electrocardiogram, respiratory pattern, airflow, and inspired CO2 data, and those attributed to movement artifact were discarded. For each desaturation event, the following measures were taken: the Sao2 nadir and
duration, simultaneous HR (from electrocardiogram), and inspired CO₂. Apneas associated with desaturation events were identified and the duration (from the end of the preceding inspiration until the end of the next inspiration) measured. Apnea was described as central if there was a flattened trace (≥3 seconds) for both chest and abdominal movement as well as nasal airflow, and as obstructive if respiratory effort was evident without nasal airflow. For the 30 seconds preceding the desaturation event, the mean HR, sleep state, and rectal and shin temperatures were measured. QS was indicated by periods of regular breathing accompanied by low HR variation, while active sleep (AS) was indicated by periods of irregular breathing and HR.18

Rebreathing Events
Sections of recording in which CO₂ was >3% were identified (above the level known to stimulate ventilation30). The rebreathe event duration encompassed the rise and fall of that event. The duration of each rebreathe event, and mean and maximum CO₂ levels, were measured. A free of movement artifact 30-second sample free of movement artifact that included the maximum level of CO₂ was identified, and mean values were calculated for SaO₂, HR, respiration rate, and CO₂. A 30-second baseline CO₂ period was identified in the same behavioral state preceding the rebreathe event.

CO₂ Cumulative Exposure
The BabyLog software was used to identify all episodes in which CO₂ averaged over 5 seconds exceeded either 2%, 3%, or 4% for a minimum of 3 seconds. Episodes during awake time or due to movement artifact were deleted, and a cumulative time was calculated for each level of CO₂.

Statistical Analyses
Although BS and CS infants were matched for age and season of study, data for both members of the pair were not always available. The data were therefore analyzed as 2 groups, and a form of regression analysis, adjusted for infant age and season, was used to take the matching into account for all comparisons. Desaturation events were analyzed by using Poisson or logistic regression to estimate the relative risk or odds ratio for the BS group for the variables based on counts or categories, with the CS group used as the reference. All comparisons were adjusted for infant age, season of study, and multiple events per infant but not for breastfeeding because all BS infants and 35 of the 40 CS infants were breastfed. Almost all rebreathe events were observed in the BS infants, and thus a group comparison was not possible. All rebreathe events were analyzed as 1 group. Regression analysis, adjusting for multiple events per infant, was used to investigate changes in HR, breathing rate, and SaO₂ associated with rebreathe events. Separate analyses of infants of smoking mothers was not possible due to the small number of smokers.

RESULTS
BS infants were aged 0 to 6 months, and CS infants were matched for age and season of study. There were no significant differences between groups with respect to mean ± SD for the following characteristics: infant study weight (BS infants: 6580 ± 1301 g; CS infants: 6594 ± 1265 g), gestation (BS infants: 39.8 ± 2.7 weeks; CS infants: 39.5 ± 2.7 weeks), birth weight (BS infants: 3583 ± 735 g; CS infants: 3562 ± 671 g), or maternal age (BS infants: 28 ± 5.6 years; CS infants: 30 ± 5.5 years). The ratio of the number of males compared to females was 0.6 for BS infants, 0.55 for CS infants. All mothers had some tertiary education, most infants were breastfed (BS infants: 100%; CS infants: 88%), and maternal smoking in the second trimester was less common among BS mothers (8% vs 25%) than among CS mothers. The majority of mothers were of European descent, with 10% of BS mothers and 18% of CS mothers identifying as Maori (indigenous New Zealanders). Maternal alcohol consumption was minimal in both groups, ranging from rarely to 3 glasses of wine or beer per week; 17 of 40 BS mothers and 15 of 40 CS mothers reported no alcohol consumption during pregnancy.

Desaturation Events
There were more desaturation events in BS infants. 255 events were identified from 39 BS infants and 123 events from 40 CS infants. There was no difference on logistic regression between BS and CS infants with respect to the characteristics of events. Most events (85% in each group of infants) comprised a drop in SaO₂ to between 90% and 85%. A drop to <80% was rare (BS infants: 3 events; CS infants: 4 events). Duration of events ranged from 2 to 16 seconds, with 70% lasting <5 seconds. A central apnea of 5 to 10 seconds preceded 70% of desaturation events. Few apneas of >15 seconds were recorded (3 BS infants; 6 CS infants). Events mainly occurred in AS in both groups (AS: BS 61% and CS 87%; QS: BS 39% and CS 13%) with CS infants less likely than BS infants to begin an event in QS (odds ratio: 0.2 [95% confidence interval [CI]: 0.05–0.79]). CIs are large due to the effect of multiple observations per infant.

Table 1 shows that the estimated mean number of desaturation events (per infant per night) were 6.8 for BS infants and 3.1 for CS infants, giving a relative risk of 2.17 (adjusted for sleep time). The ratio was practically the same (2.15) in the 51 infants for whom temperature data were available (the lesser numbers due to parents’ reluctance to have infant rectal temperature measured). Adjusting for the mean overnight rectal–shin temperature difference reduced the risk ratio from 2.15 to 1.54.
indicating that a large part of the difference in desaturation events per night was associated with the warmer temperature of the BS infants (smaller rectal–shin temperature difference). Furthermore, we calculated that for a 1 degree decrease in rectal–shin temperature difference (ie, warmer microenvironment), desaturation events increased by 60% (95% CI: 31–96).

The desaturation events were not associated with elevated inhaled CO₂ or with significant changes in HR. The level of CO₂ 30 seconds before desaturation events was <0.5% for >70% of events in each group (Table 2). A small change in HR of ±30% from baseline occurred during the majority of desaturation events (HR increased in 175 of 378 events and decreased in 188 of 378 events), whereas an increase of >30% from baseline occurred in 4 events (BS infants: 3 events; CS infants: 1 event), a decrease to <70% of baseline occurred for 1 BS event. HR did not drop to <90 beats/min during any desaturation.

### Rebreathing Events

Eighty rebreathe events were identified from 22 BS infants and 1 CS infant (Tables 3 and 4). Although the median duration of an event was 4 minutes, the maximum lasted 59 minutes. The mean level of CO₂ varied from 1.7% to 4.7%. The median total duration of rebreathing per infant per night was 19 minutes, but 1 infant (who spent most of the night with head covered by over-bedding) reached 154 minutes. Mean respiration rate increased from 32 to 38 breaths per minute (P <.001), and mean SaO₂ was maintained at the baseline level of 97.6%, while heart rate increased slightly (126 to 129 beats/min).

The majority of events (70%) were associated with head covering. Of the 5 BS infants who spent some time sleeping prone, events occurred only in 1 while face-down into a pillow and another with face into the mother’s chest. Events also occurred in 1 side-sleeping infant with face into a tri-pillow, and in 1 infant while the mother breathed directly over the infant’s face. Rebreathing events were recorded in 1 CS infant. This infant was swaddled in a muslin wrap and repeatedly pushed the wrap over the face (Fig 1). Two CS infants slept prone for the entire night, but no rebreathing events were identified.

### DISCUSSION

This study found that infants who bed-share experienced more episodes of mild oxygen desaturation associated with the warmer microenvironment and responded more frequently to rebreathe stimuli compared with infants who slept in a cot. More severe desaturation events (<80%) and longer apneic events (>15 seconds) were rare but occurred in both groups of infants. Infants in this study were healthy and, based on demographic characteristics, at low risk for SIDS, and they responded appropriately to the potential stressors. Epidemiologic evidence identifies an increased risk associated with BS for vulnerable infants, and we hypothesize that an infant who does not adjust to the microenvironment created during BS could be at increased risk.

### Rebreathing

BS infants were exposed to more rebreathe events than CS infants, commonly associated with head covering by bedding or mothers’ clothing (during supine or side positioning), and less frequently by the infant’s face into a pillow (during prone or side positioning).

---

**TABLE 1** Number of Desaturation Events per Night in BS Infants and CS Infants, Including the Effect of Overnight Temperature

<table>
<thead>
<tr>
<th>Event</th>
<th>No. of Infants</th>
<th>Estimated Group Mean (Range)</th>
<th>Risk Ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BS</td>
<td>79</td>
<td>6.8 (0–44)</td>
<td>3.1 (0–16)</td>
<td>2.17 (1.75–2.68)</td>
</tr>
<tr>
<td>CS</td>
<td>51</td>
<td>8.5 (0–44)</td>
<td>3.8 (0–16)</td>
<td>2.15 (1.69–2.73)</td>
</tr>
</tbody>
</table>

| BS Category – Adjusted for rectal–shin temperature difference | 51 | 7.7 (0–44) | 3.6 (0–16) | 1.54 (1.17–2.02) | .002 |

Adjusted for age, season, and multiple events per infant.

**TABLE 2** No. (%) of Desaturation Events Associated With Varying Levels of CO₂ at the Infant’s Face

<table>
<thead>
<tr>
<th>CO₂ (%)</th>
<th>BS (n = 30)</th>
<th>CS (n = 24)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.4</td>
<td>200 (78)</td>
<td>84 (71)</td>
<td>—</td>
</tr>
<tr>
<td>0.5–0.9</td>
<td>39 (15)</td>
<td>25 (21)</td>
<td>—</td>
</tr>
<tr>
<td>1.0–1.4</td>
<td>9 (4)</td>
<td>8 (7)</td>
<td>.79</td>
</tr>
<tr>
<td>1.5–1.9</td>
<td>2 (1)</td>
<td>1 (1)</td>
<td>—</td>
</tr>
<tr>
<td>2–2.9</td>
<td>5 (2)</td>
<td>0</td>
<td>—</td>
</tr>
<tr>
<td>&gt;3</td>
<td>0</td>
<td>0</td>
<td>—</td>
</tr>
</tbody>
</table>

| Total no. of events | 255 | 118 |

Multiple logistic regression adjusted for season, age, and multiple events per infant was used for comparison.

CO₂ data missing for 5 CS desaturation events. No desaturation events identified in the remaining 9 BS infants and 16 CS infants.

**TABLE 3** Characteristics of Rebreathe Events

<table>
<thead>
<tr>
<th>Event duration, min</th>
<th>BS (n = 30)</th>
<th>CS (n = 24)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum event CO₂, %</td>
<td>4</td>
<td>4</td>
<td>3–5.7</td>
</tr>
<tr>
<td>Mean event CO₂, %</td>
<td>2.5</td>
<td>1.7–4.7</td>
<td>—</td>
</tr>
</tbody>
</table>

Total event duration/infant, min 19 3–154

n = 25 infants, n = 80 observations.
Ball has also reported more head covering. The basis for the rebreathe events may be the head covering per se or the mother breathing over the infant. In response, infants may either increase ventilation effectively or remove themselves from the rebreathe situation or prompt action by the mother. Mothers also spontaneously remove bedding during head covering events, indicating the importance of a responsive mother during BS. Rebreathing may be hazardous due to the potential asphyxial microenvironment or the cumulative impact of repeated exposure to a mildly hypoxic environment, although, conversely, low levels of CO2 may be protective because of the stimulatory effect of the CO2 on ventilation. Rebreathe events were identified in only 1 CS infant, who was swaddled in a muslin wrap and who pulled the wrap over her face several times. Furthermore, there were some periods when the head could be covered but with relatively little accumulation of CO2, most likely explained by the existence of air channels between the bedding and the infant’s face.

Infants often remained head covered for extended periods of time (up to 59 minutes) and maintained oxygen saturation.

We and others have shown that increased head covering in BS infants compared with CS infants is due to increased mobility of the bedding. We demonstrated here that infants maintained their SaO2, in part due to an increased rate of breathing, as also shown by Ball. The ventilatory response to asphyxia is mainly an increase in tidal volume, but we could not measure this because we used an uncalibrated respitrace system. Our data indicate that normal homeostatic respiratory responses to the rebreathe environment protected these infants.

However, infants with hypoxemia resulting from an infection may not respond as well, and whether the degree of chronic hypoxia that some BS infants are exposed to is sufficient to blunt ventilatory responses is unknown. The vulnerability of the non-responsive infant may lie in intrinsic abnormalities of formation, or immaturity of development of certain brain regions that subserve respiratory and cardiovascular control, which may be due to or aggravated by external factors such as maternal smoking during pregnancy or prematurity.

**Desaturation Events**

Desaturation events to <90% were seen more often in BS infants than in CS infants, although the importance of this event as a risk factor for SIDS is unclear. Episodic apnea and periodic breathing occur in normal infants and are not observed more frequently in future SIDS victims, albeit that an increase in obstructive apneas has been reported. We note that almost all of the desaturation events in our study were preceded by central apnea and

### TABLE 4 Baseline and Rebreathing Events: Mean ± SD Values for SaO2, Breathing, and HR

<table>
<thead>
<tr>
<th>Event</th>
<th>Baseline CO2 = 0.5%</th>
<th>Rebreathing Event, CO2 = 3%</th>
<th>Difference</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>SaO2, %</td>
<td>97.6 ± 1.1</td>
<td>97.6 ± 1.2</td>
<td>0.06 ± 0.9</td>
<td>-0.2 to 0.3</td>
<td>.637</td>
</tr>
<tr>
<td>Breaths per minute</td>
<td>32 ± 7</td>
<td>38 ± 8</td>
<td>5.6 ± 6.0</td>
<td>4 to 7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>128 ± 12</td>
<td>129 ± 11</td>
<td>2.9 ± 6.5</td>
<td>1 to 5</td>
<td>.003</td>
</tr>
</tbody>
</table>

n = 23 infants, n = 80 observations. Data are presented as mean ± SD values for 30-second periods during baseline conditions and 30-second periods during rebreathing events.
were accompanied by the observation of a warmer microenvironment, as similarly observed by Richard et al.\textsuperscript{22} Oxygen saturation levels of <80% were not common and occurred in 2% of the infants (3 events in BS infants with 4 events in CS infants). Even in these more severe events, desaturation was not associated with bradycardia. The lowest HR recorded in any event was 90 beats/min. This finding is consistent with other studies of healthy infants, and apnea without bradycardia is considered a normal event.\textsuperscript{47,49,50} Richard and Mosko\textsuperscript{24} have reported a higher HR with less HR variability in BS infants compared with CS infants, partly explained by the higher axillary temperature. It is of interest that animal studies have shown that repeated episodes of hypoxia blunt arousal responses.\textsuperscript{37} We suggest that frequent desaturations in vulnerable BS infants could be a contributory factor in their risk for SIDS.

Thermal Microenvironment

Thick bedding on adult beds raises the in-bed temperature while infant–mother contact reduces the surface area available for heat loss,\textsuperscript{51} leading to increased infant peripheral temperature.\textsuperscript{16,52} Warmer temperatures can trigger periodic breathing in premature infants at near term age\textsuperscript{10} but not in very premature infants at 5 days of age.\textsuperscript{53} Body heat loss (and thus energy expenditure) rather than body temperature is also associated with increased frequency and duration of apneic events in premature infants.\textsuperscript{54} Skin-to-skin contact in premature infants has been shown to have a greater effect on bradycardia and desaturations than hyperthermic conditions induced by elevated incubator temperature.\textsuperscript{55} In that study, skin-to-skin contact did not raise core temperature, but the semi-upright infant position during skin-to-skin contact was suggested to contribute to the unstable respiration. Whether this also occurs in older infants is unknown, but it is probably less of a concern in healthy infants with maturation of cardiorespiratory control.

Sleep Architecture

BS infants have less QS than CS infants, possibly affording protection due to increased arousability in AS.\textsuperscript{20} However, depression of the hypoxic ventilator response occurs in AS,\textsuperscript{56} and desaturation events were more common in our study in AS. Interestingly, BS infants had a larger percentage of desaturation events in QS than did CS infants. Richard et al\textsuperscript{22} reported increased apneas in QS in BS infants associated with elevated axillary temperature in QS only, suggesting a link between desaturations, temperature, and sleep state.

Sleep Position

Prone sleeping infants up to 5 months of age and supine infants at 2.5 months are very limited in their ability to remove blankets from over their head.\textsuperscript{57} In this study, few infants were placed prone to sleep in either group, but the microenvironment could be similar to BS in terms of the warmer microenvironment and potential for rebreathing.\textsuperscript{54,58} Increased arousals\textsuperscript{21} may not be sufficient to protect the infant if the head is also covered by bedding. We have previously reported if the infant’s head is covered, the mother is involved in uncovering of the head ~60% of the time, either initiating action herself or after prompting by the infant.\textsuperscript{56} This suggests a mechanism for the risk of BS with a mother impaired by alcohol or drugs\textsuperscript{52} who may be unable to respond to her infant.

Strengths

We had a large sample size, and monitored 40 BS infants and 40 CS infants, producing ~10 hours of overnight data per study on family behavior, environmental factors, and infant physiology. Although the sample size for temperature analysis was reduced to 51 infants, it remained considerably larger than similar published studies.\textsuperscript{12,8,31} We monitored infants in their home, where families might be more likely to carry out usual nighttime practices. Our design allowed observation of infants in their usual sleep arrangement rather than a crossover design that requires infants to sleep both in their usual arrangement and an assigned arrangement.\textsuperscript{7,8,31} There is evidence to indicate differences in behavior between regular bed-sharers and occasional bed-sharers.\textsuperscript{21} Lastly, we have reported previously that the sensors did not affect infant behavior.\textsuperscript{16}

Limitations

The sample population was largely at low risk of SIDS. The low number of maternal smokers among the bed-sharers, together with the high standard of maternal education and high breastfeeding rate, suggests our sample did not cover the full spectrum of bed-sharers in which social deprivation, low maternal education, smoking, and lack of breastfeeding are other key factors in sudden deaths associated with BS.\textsuperscript{10} The main reasons for BS in our group were related more to parenting style\textsuperscript{5} rather than to cultural or economic influences.

Conclusions

The presence of the mother and other bed-partners, and the physical environment of the adult bed clearly led to a different sleep environment for the BS infant compared with the CS infant, resulting in beneficial and potentially compromising situations. Infant homeostatic responses and frequent maternal interactions seemed to keep these low-risk infants safe. However, we
suggest that it is potentially hazardous for an infant to sleep in the same bed as their parent, if the infant and/or mother are unresponsive. We acknowledge that BS is a practice valued by many; thus, it is important to identify the specific dangers related to this practice. Keeping the infant smoke-free at all times, including during pregnancy, is perhaps the most important message, as well as avoiding consumption of alcohol or sedating drugs on the BS, keeping the infant's face clear; and avoiding the use of thick insulation as overbedding. Studies with high-risk infants are required to advance understanding of the specific mechanism(s) leading to their increased vulnerability.

ACKNOWLEDGMENTS
We thank the families that participated in this study and Charrissa Makowhar-emahihi and Amanda Phillips for research assistance.

REFERENCES
24. Richard CA, Mosko SS. Mother-infant bed-sharing is associated with an increase in infant heart rate. Sleep. 2004;27(3):507–511
33. Chiodini BA, Thach BT. Impaired ventilation in infants sleeping facedown: potential...
57. Skadberg BT, Markestad T. Consequences of getting the head covered during sleep in infancy. Pediatrics. 1997;100(2). Available at: www.pediatrics.org/cgi/content/full/100/2/66
Hypoxic and Hypercapnic Events in Young Infants During Bed-sharing
Sally A. Baddock, Barbara C. Galland, David P.G. Bolton, Sheila M. Williams and Barry J. Taylor

Pediatrics 2012;130;237; originally published online July 16, 2012; DOI: 10.1542/peds.2011-3390

Updated Information & Services
including high resolution figures, can be found at:
/content/130/2/237.full.html

References
This article cites 58 articles, 17 of which can be accessed free at:
/content/130/2/237.full.html#ref-list-1

Subspecialty Collections
This article, along with others on similar topics, appears in the following collection(s):
Fetus/Newborn Infant
/cgi/collection/fetus:newborn_infant_sub
SIDS
/cgi/collection/sids_sub
Respiratory Tract
/cgi/collection/respiratory_tract_sub

Permissions & Licensing
Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
/site/misc/Permissions.xhtml

Reprints
Information about ordering reprints can be found online:
/site/misc/reprints.xhtml

PEDIATRICS is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. PEDIATRICS is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2012 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 0031-4005. Online ISSN: 1098-4275.
Hypoxic and Hypercapnic Events in Young Infants During Bed-sharing
Sally A. Baddock, Barbara C. Galland, David P.G. Bolton, Sheila M. Williams and Barry J. Taylor

*Pediatrics* 2012;130:237; originally published online July 16, 2012;
DOI: 10.1542/peds.2011-3390

The online version of this article, along with updated information and services, is located on the World Wide Web at:
/content/130/2/237.full.html