Apnea and periodic breathing in bed-sharing and solitary sleeping infants

CHRISTOPHER A. RICHARD, SARAH S. MOSKO, AND JAMES J. McKENNA²

Sleep Disorders Center, Department of Neurology, University of California, Irvine, Medical Center, Orange 92868; and Department of Anthropology, Pomona College, Claremont, California 91711

Richard, Christopher A., Sarah S. Mosko, and James J. McKenna. Apnea and periodic breathing in bed-sharing and solitary sleeping infants. J. Appl. Physiol. 84(4): 1374-1380, 1998. - Mother-infant bed sharing, compared with the solitary sleeping condition, has recently been associated with several physiological and behavioral effects. Because the physiological effects of bed sharing may also include respiratory changes, we compared the incidence of central and obstructive apneas and periodic breathing in bed-sharing and solitary sleeping infants. Twenty routinely bed-sharing mother-infant pairs and fifteen routinely solitary sleeping pairs slept for 3 nights in a sleep laboratory. After an initial adaptation night, each pair spent 1 night bed sharing and 1 night in solitary sleep in random order. Apnea and periodic breathing were scored from polysomnographic recordings. The frequency of central apnea was significantly increased on the bed-sharing night, compared with the solitary night, regardless of routine sleeping arrangement. There were significantly fewer obstructive apneas on the bed-sharing night than on the solitary night, but only in routinely solitary sleeping infants. In both groups, there was a significantly higher frequency of periodic breathing events on the bedsharing night than on the solitary night. These findings demonstrate that the bed-sharing environment can have a significant impact on respiratory control in the infant. Evidence is also presented to suggest that routine bed sharing may result in subtle neurophysiological and/or developmental differences in infants.

infant central apnea; sudden infant death syndrome; cosleeping

INFANT SLEEP PHYSIOLOGY, like all biological systems, was designed by the process of natural selection in which the success of individuals is determined by their interaction with the environment. Anthropological and ethnographic studies indicate that the sleeping environment within which human (and other primate) infants adapted and evolved was that of parent-infant cosleeping. Lozoff and Brittenham (20) scored ethnographic and behavioral data from 186 tropical, nonindustrialized societies to infer the characteristic pattern of child care. Parent-infant cosleeping was shown to be the sleeping arrangement for 100% of the hunter-gatherer groups and 76% of the other nonindustrialized societies. Tropical hunter-gatherer societies are considered to be ecologically, and therefore adaptively, similar to prehistoric hominids. Based in part on these observations, cosleeping is accepted by anthropologists to represent the sleep environment of prehistoric humans and hominids (5, 16-18). Furthermore, cosleeping is not just interesting from an evolutionary perspective, it remains today the species-typical behavior for most of the world's human population (2, 16). Despite this,

assimilation of this basic human behavior into physiological or behavioral studies of infant sleep has not occurred. Instead, infant sleep research has been modeled exclusively on the recent Western cultural practice of solitary sleeping, i.e., placing infants in a room alone. It is not yet known whether the cultural transition to solitary sleep is associated with positive, negative, or neutral adaptive consequences in the infant.

Parent-infant cosleeping occurs in different forms with varying degrees of parental proximity and contact, including bed sharing (same bed or sleeping surface) and sleeping on separate surfaces in the same room. In addition, the consistency of cosleeping can vary widely, ranging from all night every night to occasional or partial-night cosleeping. Of the various forms of cosleeping, bed sharing is the most different from solitary sleeping because of the greatly enhanced auditory, olfactory, visual, tactile, and thermal stimuli resulting from the close proximity to the parent(s). Recently, several physiological and behavioral consequences of bed sharing were reported. While bed sharing, infants had less stage 3-4 sleep (analog of quiet sleep) and more stage 1-2 sleep than when they slept alone (27). Bed sharing was associated with more frequent arousals in both mother and infant, many of which temporally overlapped (25, 26). Prone infant positioning was minimized during bed sharing, and mother-infant pairs typically slept oriented face to face at very close proximities (29). In addition, breast feeding was significantly increased during bed sharing in both routinely bed sharing (since birth) and routinely solitary sleeping infant groups (21). Considering current ideas on the mechanisms of and risk factors for the sudden infant death syndrome (SIDS), we hypothesized that the effects of bed sharing may provide some degree of protection through increased infant arousability, facilitation of breast feeding, reduction in prone sleeping, and/or increased maternal vigilance (22). This idea has been challenged by Mitchell et al. (24), who reported epidemiological data suggesting that bed sharing may increase SIDS risk, although subsequent analyses greatly reduced the scope of the association (see DISCUS-

Because defective cardiorespiratory control probably plays an important role in events leading to at least some SIDS deaths, understanding how environmental manipulations and caretaking practices affect infant respiration could be critical to developing strategies to reduce SIDS susceptibility. The present study compares the occurrence of apnea and periodic breathing in the bed-sharing and solitary sleeping environments in healthy infants at the peak age for SIDS.

METHODS

Thirty-five healthy, breast-feeding, Latino mother-infant poors were recruited from the University of California, Irvine, Birthing Center during their 6- to 12-wk postpartum visits to spend 3 consecutive nights in a clinical sleep laboratory. All met criteria for either routinely bed sharing (RB; n = 20) or routinely solitary sleeping (RS; n = 15) since birth. RB was defined as bed sharing with the mother for at least 4 h per night, 5 days a week; RS was defined as bed sharing not more than one night per week for any part of the night. Two-week shep logs were completed at home just before the sleep recordings to confirm maternal reports of the infants' usual home sleep environment. Mothers were screened to exclude subjects who smoked or used alcohol or drugs, either currently or during pregnancy, or had abnormal pregnancies or deliveries. Pairs were recorded when the infants were between 11 and 15 wk old. Infants were screened to exclude subjects born at <37 wk of gestation, under 2,500-g birth weight, with a history of prolonged apnea or an apparent life-threatening event, with siblings or primary relatives who were victims of SIDS, or with 5-min Appar scores of <8. Both mother and infant were seen by a physician to ensure that they were healthy at the time of the recordings. The mothers were also screened for sleep disorders by a physician trained in sleep medicine. All procedures were approved by the institutional human subjects committee, and all mothers signed informed consents for themselves and their infants.

Each pair spent the first night in their routine (home) -leeping condition, which served as an adaptation night. On the remaining 2 nights, each pair had a bed-sharing night (BN) and a solitary night (SN), randomly ordered, to allow within-subjects comparisons. Both members of each pair were recorded nightly using standard polysomnography consisting of electroencephalogram, electrooculogram, chin electromyogram, respiratory airflow and effort, electrocardiogram (Grass Instruments, Quincy, MA), and infrared video. Mothers and infants went to bed and arose at their normal times. Mothers performed all care-taking interventions themselves and were not instructed on infant sleep positioning, feeding schedules, or the specific aims of the study. Infant sleep was scored manually in 30-s epochs for wakefulness and three sleep stages [1-2, 3-4, and rapid eye movement (REM)] according to the Guilleminault and Souquet (9) system developed for the 3-mo-old infant. Central apnea was scored when there was an absence of airflow accompanied by an interruption in respiratory effort lasting ≥3 s. Obstructive apnea was scored when there was an absence of airflow of ≥3 s despite respiratory effort. In addition, central or obstructive apneas that were associated with an arousal were tabulated. An arousal associated with an apnea could be either an epochal wakening (stage scored as wakefulness) or a ≥3-s transient arousal (25, 34) and could either precede or follow the apnea. Apneas followed by an arousal are of particular clinical interest and were partitioned for further analyses. Periodic breathing was scored when there were at least three central apneas within 20 s with no more than 10 s separating two consecutive apneas. Apneas meeting criteria for periodic breathing were not included in the analysis of central apneas.

Statistical analyses were directed at determining whether the sleep environment affected any feature of apnea or periodic breathing (i.e., frequency, mean duration, or longest occurrence) either acutely (BN vs. SN) or habitually (RB vs. RS). Frequencies were computed as the number of events per hour of total sleep time (overall frequency) or per hour of a given sleep stage. Mean and longest durations also were

analyzed as a function of sleep stage and as overall totals. Central apneas were analyzed with a 2 × 2 repeatedmeasures ANOVA. However, because some infants did not exhibit obstructive apnea or periodic breathing, these variables were nonnormally distributed and showed unequal variances, precluding the use of parametric tests. For those data sets, a Wilcoxon signed-ranks test was used to assess night effects (BN vs. SN) and a Mann-Whitney U-test was used to assess group effects (RB vs. RS). In addition, the two groups were compared in their routine conditions, i.e., RB on the BN (RB-BN) vs. RS on the SN (RS-SN); t-tests were used for the parametric data sets and the Mann-Whitney U-test was used for nonparametric data. These comparisons were included to assess how the data from habitually bed-sharing infants compared with published normative data (i.e., using solitary sleep environment). Central and obstructive appear also were partitioned by duration into 3-s bins (3-5.9, 6-8.9, 9-11.9, 12-14.9, and ≥15 s) to assess whether any differences in the occurrence of apnea were confined to shorter or longer apnea. These binned data were analyzed with nonparametric tests. Significance was assigned when P < 0.05.

RESULTS

Analysis of the sleep logs covering the 14 days preceding the recordings confirmed that RB pairs met criteria for routine bed sharing and that RS pairs met criteria for routine solitary sleeping; RB pairs bed shared on an average of $13.7 \pm 0.5 \, (\text{SD})$ nights, whereas RS pairs bed shared 0.6 ± 0.9 nights. There were no differences in age between the two groups of infants at the time of recording. RB infants consisted of 11 males and 9 females and were an average of 13.0 ± 1.3 wk old at the time of the recording; RS infants consisted of 4 males and 11 females and were 12.9 ± 1.3 wk old.

Central apnea. Analysis of central apnea with ANOVA revealed no significant differences in the mean duration or longest occurrence between nights or groups. For apnea frequency, however, there were significant main effects for the night (within subjects) comparisons where there were increases on the BN in the overall frequency of central apnea due to significant increases in the frequency during stages 1-2 and REM. There were no significant main effects for group (between subjects) or interaction effects. The means ± SE for the frequency data are illustrated in Fig. 1. Comparisons of the two groups in their routine conditions (RB-BN vs. RS-SN) showed no significant differences for any sleep stage, although the differences for stages 1-2 and REM approached significance (P < 0.10). However, when the data were pooled across sleep stages, there was a significantly higher frequency of central apnea in the RB-BN group than in the RS-SN group (P = 0.035).

The number of infant central apneas associated with an arousal, either preceding or after the apnea, was expressed as a percentage of all central apneas for analysis. There were no significant differences in the occurrence of these apnea-arousal events between nights or groups. When only central apneas preceding an arousal were analyzed, there were again no differences between groups or nights.

Partitioning apneas by duration produced different results for the RB and RS groups (see Table 1). In the RS infants, there were significantly more of the short-

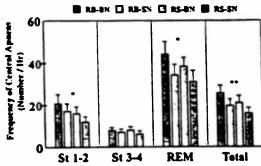


Fig. 1. Mean (\pm SE) frequency of infant central apnea (no./h stage time) in 4 sleeping conditions. Significant main effects for night were found where the frequency was higher on bed-sharing night (BN) [vs. solitary night (SN)] during stages (St) 1–2 and rapid eye movement (REM), and overall (total/h of total sleep time). RB, routine bed sharing; RS, routine solitary sleeping. Significant night effect where BN was greater than SN across groups (df = 33,1): $^{\circ}P < 0.05$; $^{\circ\circ}P < 0.01$.

est central apneas (3-5.9 s) on the BN, compared with the SN, during stage 1-2 (P=0.035). In the RB infants, there were significantly more apneas of 6-8.9 s on the BN during REM and overall (P=0.003 and P=0.017, respectively) and more apneas of 9-11.9 s during stage 1-2 (P=0.009). However, in RB infants, there were

fewer apneas of 9-11.9 s during stage 3-4 on the BN than the SN (P = 0.014). No significant effects were found for central apneas of 12-14.9 s, and apneas of >15 s were not analyzed because of their infrequent occurrence (5 of 20 RB infants, 3 of 15 RS infants).

Obstructive apnea. Nonparametric analyses indicated that obstructive apnea was decreased on the BN compared with the SN, but only in the RS infants (Fig. 2). The frequency was lower for the overall total and for stages 1-2 and REM. In contrast, RB infants showed no significant difference in frequency of obstructive apnea between the BN and the SN. There were no significant differences in apnea frequency or mean duration between the RB infants on their BN and the RS infants on their SN, either overall or for any given sleep stage. Analysis of the percentage of obstructive apnea associated with an arousal showed that only in RS infants were there significantly more obstructive apneas associated with an arousal over the entire SN than on the BN (P = 0.038). Restricting that analysis to those obstructive apneas followed by an arousal was made problematic by the fact that many infants did not exhibit those patterns (i.e., 7/20 RB infants on their BN, 6/20 their SN, 9/15 RS infants on their BN, and 5/15 on their SN), but there was some indication o.

Table 1. Number of central apneas

	Group	3-5.9 s	6-8.9 s	9-11.9 s	12-14.9 6	≥15 s
			Median			
C	RB-BN	30.5	13.5	4.0*	0.0	0.0
Stage 1-2	RB-SN	26.0	11.5	1.5	0.0	0.0
	RS-BN	27.0*	11.0	2.0	0.0	0.0
	RS-SN	19.0	10.0	1.0	0.0	0.0
a. a.	RB-BN	3.5	2.0	1.5	0.0	0.0
Stage 3-4	RB-SN	4.0	3.5	2.5*	0.0	0.0
	RS-BN	3.0	5.0	1.0	0.0	0.0
	RS-BN RS-SN	3.0	3.0	1.0	0.0	0.0
		3.0 64.0	20.5*	1.5	0.0	0.0
REM	RB-BN		12.0	1.0	0.0	0.0
	RB-SN	52.0	17.0	1.0	0.0	0.0
	RS-BN	84.0	13.0	2.0	0.0	0.0
	RS-SN	51.0	27.5°	7.0	1.0	0.0
Total	RB-BN	100.0	29.5	6.5	0.0	0.0
	RB-SN	89.0		5.0	0.0	0.0
	RS-BN	85.0	32 .0	4.0	0.0	0.0
	RS-SN	76.0	25 .0	4.0	0.0	0.0
		1	nterquartile range			
Stage 1-2	RB-BN	15.5-45.3	6.75-25.3	0.0-7.5	0.0-1.0	0.0-0
	RB-SN	14.5-42.3	5.0-22.3	0.0-4.0	0.0-1.0	0.0-0
	RS-BN	10.0-36.5	4.515.0	0.0-5.0	0.00.0	0.0-0
	RS-SN	11.0-30.0	5.5-18.0	0.0-5.0	0.00.5	0.0-0
Stage 3-4	RB-BN	0.8-7.0	1.0-5.5	0.0-2.3	0.0-0.3	0.0-0
	RB-SN	1.0-7.5	2.8-6.5	0.8-4.3	0.0-1.0	0.0-0
	RS-BN	- 1.5-8.0	1.5-7.5	0.0-2.0	0.00.5	0.0-0
	RS-SN	1.5-5.0	0.5-6.0	0.0-3.5	0.0-0.0	0.0-0
REM	RB-BN	53.75-111.3	9.75-26.0	0.75-2.3	0.00.0	0.0-0
	RB-SN	40.25-90.8	9.5-17.5	0.0-4.3	0.0-0.3	0.00
	RS-BN	32.0-78.5	6.0-27.0	0.0-2.0	0.0-0.0	0.0-0
	RS-BN RS-SN	31.5-77.0	5.5-21.5	1.0-2.0	0.00.0	0.0-0
		84.75-148.5	20,2554.0	2.0-13.3	0.0-2.0	0.0-1
Total	RB-BN	55.75-162.0	21.0-47.8	2.5-11.8	0.0-2.3	0.0-2
	RB-SN	64.5-112.5	13.5-47.0	1.0-10.5	0.0-1.0	0.0-1
	rs-bn rs-sn	45.5-112.5 45.5-100.5	15.5-44.5	1.5-11.0	0.0-2.0	0.0-0

RB, routine bed sharing; RS, routine solitary sleeping; BN, bed-sharing night; SN, solitary night; REM, rapid-eye-movement sle Significant differences are indicated with bold type and asterisk (P<0.05).

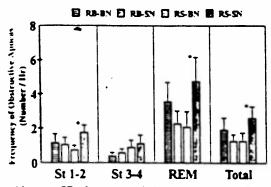


Fig. 2. Mean (\pm SE) frequency of obstructive apneas in infants. Nonparametric analyses showed a significantly higher frequency on BN (vs. SN) during St 1-2 and REM, and overall (total) but only for RS infants. Abbreviations as in Fig. 1. *Significant effect for RS infants only where BN was less than SN, P < 0.05.

more obstructive apneas followed by an arousal in RB intents than RS infants on the BN.

Partitioning obstructive apneas by duration revealed that there were significantly fewer of the short obstructive events on the BN compared with the SN in RS infants (see Table 2). They exhibited fewer obstructive apneas of 3-5.9 s on the BN overall (P = 0.012) and

Table 2. Number of obstructive apneas

	Group	3-5.9 s	6-8.9 s	9-11.9 s	12-14.9 s	≥15 :
			Median			
Stage 1-2	RB-BN	-1.0	1.0	0.0	0.0	0.0
	RB-SN	1.0	0.0	0.0	0.0	0.0
	RS-BN	1.0	0.0	0.0	0.0	0.0
	RS-SN	2.0*	1.0*	0.0	0.0	0.0
Stage 3-4	RB-BN	0.0	0.0	0.0	0.0	0.0
	RB-SN	0.0	0.0	0.0	0.0	0.0
	RS-BN	0.0	0.0	0.0	0.0	0.0
	RS-SN	0.0	0.0	0.0	0.0	0.0
REM	RB-BN	3.0	1.0	0.0	0.0	0.0
	RB-SN	2.0	1.0	0.0	0.0	0.0
	RS-BN	2.0	0.0	0.0	0.0	0.0
	RS-SN	6.0*	0.0	0.0	0.0	0.0
Total	RB-BN	4.5	2.5	0.0	0.0	0.0
	RB-SN	4.5	1.5	0.0	0.0	0.0
	RS-BN	3.0	1.0	0.0	0.0	0.0
	RS-SN	11.0*	2.0*	0.0	0.0	0.0
		Interq	uartile ra	nge		
Stage 1-2	RB-BN	0.0-1.0	0.0-1.0	0.0-0.3	0.0-0.0	0.0-0.0
	RB-SN	0.0-2.0	0.0 - 1.3	0.0-0.0	0.0-0.0	0.00.0
	RS-BN	0.0 - 2.0	0.0-1.0	0.0-0.0	0.00.0	0.0-0.0
	RS-SN	0.5-4.0	0.0 - 3.5	0.0-0.0	0.00.0	0.00.0
Stage 3-4	RB-BN	0.0-0.0	0.0-0.0	0.0-0.0	0.00.0	0.0-0.0
	RB-SN	0.0-1.0	0.0-0.3	0.0-0.0	0.00.0	0.0-0.0
	RS-BN	0.0-0.0	0.0-0.0	0.0-0.0	0.00.0	0.0-0.0
	RS-SN	0.0 - 1.5	0.0-1.5	0.00.0	0.0-0.0	0.0-0.0
REM	RB-BN	0.0-7.5	0.0-0.3	0.0-0.0	0.0-0.0	0.0-0.0
	RB-SN	0.0-4.3	0.0 - 1.3	0.0-0.0	0.0-0.0	0.0-0.0
	RS-BN	0.5-4.0	0.0-1.5	0.00.0	0.00.0	0.0-0.0
	RS-SN	0.0-11.0	0.0-5.0	0.0-0.0	0.0-0.0	0.00.0
Total	RB-BN	0.75-9.3	0.0-4.5	0.0-1.0	0.00.0	0.00.0
	RB-SN	2.25-9.3	0.0-3.3	0.00.0	0.0-0.0	0.0-0.0

Abbreviations as in Table 1. Significant differences are indicated with bold type and asterisk (P < 0.05).

0.5-7.0

0.0-3.5 0.0-0.0 0.0-0.0 0.0-0.0

1.0-14.5 0.0-13.0 0.0-1.0 0.0-0.0 0.0-0.0

RS-BN

RS-SN

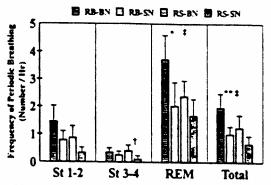


Fig. 3. Mean (\pm SE) frequency of episodes of periodic breathing. There was a significantly higher frequency on BN (vs. SN) during St 1–2 and REM, and overall for RB infants ($^{\circ}P < 0.05$; $^{\circ}P < 0.01$) and a higher frequency on BN during St 3–4 for RS infants ($^{\dagger}P < 0.05$). Additional analyses showed significantly greater frequencies in RB-BN compared with RS-SN groups ($^{\dagger}P < 0.05$). Abbreviations as in Fig. 1.

during stages 1-2 and REM (P = 0.028 and P = 0.017, respectively). There were also significantly fewer apneas of 6-8.9 s overall (P = 0.013) and during stages 1-2 and REM (P = 0.021 and P = 0.030, respectively). Obstructive apneas of >8.9 s were too rare for meaningful analysis; only 15 of 35 infants had obstructive apneas of >8.9 s and only 5 of those had apneas of >11.9 s and 2 of \geq 15 s.

Periodic breathing. Bed sharing was associated with a higher frequency, longer mean duration, longer maximal duration and longer total duration of periodic breathing. In RB infants, there was a significantly higher frequency of periodic breathing on the BN, both overall and for stage REM. However, in RS infants there was a significantly higher frequency on the BN only in stage 3-4 (Fig. 3). The amount of time spent in periodic breathing as a percentage of sleep stage time was significantly greater on the BN for RB infants during REM and stage 1-2, but again in RS infants only during stage 3-4 (Fig. 4). This pattern (where RB infants were affected in REM and occasionally in stage 1-2 and RS infants in stage 3-4) was repeated, in large part, in the other measures of periodic breathing. The

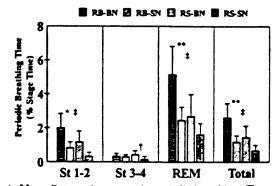


Fig. 4. Mean %stage time spent in periodic breathing. There were significantly higher percentages of St 1-2 and REM, and overall on BN in RB infants and a higher percentage of St 3-4 on BN for RS infants. In addition, there was more time spent in periodic breathing in RB-BN group than in RS-SN group. Symbols for significance as in Fig. 3. See Fig. 1 for abbreviations.

mean duration of periodic breathing on the BN was significantly longer for RB infants overall and in REM. The longest occurrences of periodic breathing on the BN were greater only for RB infants in REM and stage 1–2. Comparison of the two groups in their routine conditions (RB-BN vs. RS-SN) showed that frequency was significantly higher for the RB group overall and during REM. A similar comparison for longest duration showed that the RB-BN group had significantly longer maximal durations in stages 1–2 and REM. The RB infants on their BN also had an overall longer mean duration than the RS infants on their SN, mainly due to an increase during stage 1–2.

DISCUSSION

This study suggests that the bed-sharing environment is associated with more central apneas, fewer obstructive apneas, and more periodic breathing in infants than is the solitary environment. During bed sharing, infants of both groups had a higher frequency of central apnea during stages 1-2 and REM (and overall). In RS infants, this largely reflected an increase in the shortest apnea (3-5.9 s) in stage 1-2; in RB infants, it reflected increases in apnea of 6-8.9 s during REM and of 9-11.9 s during stage 1-2. Therefore the bed-sharing environment seems to increase the occurrence of central apnea and may also have subtle effects on apnea termination. In contrast to central apneas, obstructive apneas were decreased by bed sharing, but only in RS infants who had a lower frequency overall and specifically in stages 1-2 and REM. Similar to central apnea, the change in the number of obstructive apneas was confined to shorter events (i.e., 3-5.9 and 6-8.9 s), although in this case the difference represented a decrease in frequency for those bins.

It is not yet apparent how bed sharing increases central apnea while decreasing obstructive apnea. However, it is noteworthy that the frequencies of obstructive apnea were generally decreased on either group's nonroutine night (RB-SN and RS-BN) compared with their routine night, even though the RB group's differences did not reach significance. This may indicate that changes in obstructive apnea are a response to the novelty of the sleep environment. Consistent with this speculation, there were no significant differences for any obstructive apnea variables between the two groups in their routine conditions.

The amount of periodic breathing was also significantly increased in both infant groups on the BN. RB infants had a higher frequency of periodic breathing and a longer mean duration over the entire night (overall) and specifically during REM, whereas RS infants exhibited more frequent periodic breathing only during stage 3-4. When the amount of periodic breathing was represented as minutes or as a percentage of each stage, there were relative increases on the BN (compared with SN) for RB infants overall (percentage of total sleep time), reflecting increases during stages 1-2 and REM, whereas RS infants had a higher percentage during stage 3-4 only. These significantly

affected variables were generally found to be different between the two groups in their routine conditions as well

To address possible mechanisms responsible for the differences in periodic breathing between groups and between sleeping conditions, it may be helpful to consider how the two groups responded to their nonroutine conditions relative to each other. For the periodic breathing variables, both groups responded to the nonroutine night by approaching the mean values seen in the other group on that night. That is, when RB infants slept solitarily, their means approximated those for the RS infants on their SN, and RS infants on their BN showed average values approaching those for RB infants on their BN. Therefore, unlike the relationships of the obstructive apnea variables, there is no indication of a novelty effect on the periodic breathing variables. Rather, it appears that the bed-sharing condition itself may exert facilitatory effects on periodic breathing that do not habituate. In support of this hypothesis are the findings of significant differences between the two groups in their routine conditions for most of the periodic breathing variables, indicating baseline differences between the two sleep environments.

Several physiological factors associated with the bed-sharing environment could be hypothesized to contribute to the apneic changes described in this report. Bed-sharing infants have a small but significantly higher axillary temperature than solitary sleeping infants (Richard, unpublished observations). There is some evidence suggesting that increases in body temperature may lead to more apnea and/or periodic breathing (7, 19). Although axillary temperature is not always a good index of body temperature, it is certainly possible that our bed-sharing infants were slightly hyperthermic (compared with solitary sleeping infants), which could account for at least part of the increase in apnea-periodic breathing reported here. Higher ambient temperatures may also be associated with an increase in apnea frequency (1), but this is an unlikely explanation for our findings because the room temperatures were maintained at similar levels across groups and nights and were somewhat cool (20-21°C). In addition, there was less stage 3-4 and more stage 1-2 in both groups on the BN compared with the SN (27). These differences in sleep architecture are not likely to account for our results, since the present data were expressed in frequencies (no. per hour of a sleep stage). In this same sample of subjects, prone infant positioning was minimized during bed sharing in both groups. This behavior (positioning) is not likely to affect apnea frequency or duration, according to at least three independent groups (13, 28, 35).

Finally, the reported increase in infant arousals during bed sharing (25) may be important, since apneas are often associated with arousals. To address this possibility, the number of central and obstructive apneas associated with an arousal was expressed as a percentage of total central or obstructive apneas, respectively. There was no difference in the number of central apneas associated with arousal, either between nights

or between groups, suggesting that the effects of bed sharing on central apnea and arousal are not tightly linked. There was, however, a significant difference in the percentage of obstructive apnea associated with an arousal, but only in RS infants in which there were more of these events on the SN compared with the BN. Because apneas followed by arousal are of clinical interest, those data were analyzed separately (data not shown), although many infants had no obstructive apneas followed by an arousal. As before, there were no significant differences for central apneas, but there was some indication of more obstructive apneas followed by arousal in the RB infants of their BN than in RS infants on their BN. The increase in the occurrence of all apnea-arousal events (either precedence) on the SN, compared with the BN, in RS infants may relate to the finding that the frequency of obstructive apnea on the SN was twice that on the BN in RS infants, whereas their arousal frequency also increased (nonsignificantly) on the SN (25). Therefore the increase in the percentage of apnea and/or arousal events in the RS infants does not necessarily imply that the increase was due to any specific control mechanism, since the probability of a purely temporal association was also increased, and routinely bed-sharing infants did not exhibit the effect. In addition, caution should be used in the interpretation of these data, since they include arousals followed by apneas, which, especially for obstructive apneas, may not necessarily be "true" apneas. That is, some of the obstructive apneas measured in this study that follow an arousal may be an artifact of movements accompanying the arousal. There was no significant correlation between the amount of periodic breathing and the frequency of sleep stage shifts or the frequency of arousals in bed-sharing infants (data not shown).

Comparison of our findings with previously reported norms for infant apnea is problematic for several reasons. First, infant apnea has been defined with widely differing criteria. For example, apnea as short as 2 s were recognized by some investigators, whereas others required at least 6 s. Second, many investigators used wide ranges of infant ages, which almost certainly increased sample variability. Also, the majority of researchers used the quiet sleep and active sleep categories for infant sleep staging, probably because they also apply to the newborn. One factor that must be taken into account with these and any comparisons is the large range of values that has been noted in the literature (see Ref. 11).

There were many variables for which significant differences were found between the two groups in their routine conditions, especially for periodic breathing. These comparisons are interesting because they reveal yet more physiological differences resulting from a particular, commonly chosen child-care practice. In other words, if these results are replicated, they could modify current "baseline" or normative physiological values for infants who bed share. Taking into account the ecological and adaptive history of human sleep physiology and the fact that most of the world's peoples

still practice some form of cosleeping, it can be proposed that solitary sleep represents a novel or modified sleep environment which might induce changes in infant sleep physiology. Although this does not necessarily mean that solitary sleeping is inherently deleterious or maladaptive, it does suggest that the modern cosleeping infant is, from an evolutionary perspective, in a more similar or "expected" environment. This perspective also includes the idea that the infant's physiological control systems almost certainly were designed (by natural selection) to respond to the presence of a cosleeping parent. This perspective is further strengthened by the fact that human infants are the most altricial of all mammals (18), requiring intensive and extended parental caretaking to thrive and develop.

On the basis of epidemiological studies, some investigators have reported that bed sharing may increase SIDS risk. Mitchell et al. (24) reported, in their New Zealand study, that cosleeping with anyone (bed partner not defined) had a significant multivariate odds ratio for SIDS of 2.02, but, in subsequent analyses, that increase in odds ratio was attributed to their large Maori sample (23) and, further, to Maori mothers who smoke (33). They also reported that bed sharing was not an independent risk factor in their non-Maori subjects and was not a risk factor for all nonsmoking groups (33). The New Zealand results were replicated, in part, in England by Fleming et al. (6) in that they reported a significant odds ratio for bed sharing only in mothers who smoke. From the present study, it is impossible to surmise relative risk of SIDS between the bed sharing and solitary environments based on differences in central or obstructive apnea, since it is not yet clear how apnea relates to SIDS susceptibility. Many studies have addressed the incidence of central and/or obstructive apnea in high-risk infants and SIDS victims and produced conflicting results (8, 10, 12, 32). Although a stronger case has been made for increased periodic breathing in SIDS victims and in high-risk infants (3, 14, 15), these results also are called into question by other reports of no increase in periodic breathing (8, 12, 31). Periodic breathing is thought by some researchers to indicate neurophysiological immaturity in the respiratory control system of infants (4, 30), which could provide a conceptual link between the amount of periodic breathing and SIDS susceptibility. Therefore further studies are needed to determine whether the impact of bed sharing on infant respiration could affect SIDS risk significantly.

In conclusion, this study indicates that motherinfant bed sharing is associated with increases in infant central apnea and periodic breathing in a sleepstage-dependent manner. Most of these effects apparently reflect an immediate impact of the bed-sharing environment, suggesting that some physical feature of that environment (e.g., sensory exchange with the mother) can directly increase the occurrence of apnea. In addition, the expression of those bed sharing-related effects may depend on the routine sleeping condition of the infant, suggesting that routine bed sharing has long-lasting physiological consequences. This may, in turn, indicate that routine bed-sharing affects the development of the infant's respiratory control system. This study also extends the argument that the bed-sharing environment is not equivalent to the solitary sleep environment, since the greatest differences were found between the two groups while sleeping in their routine conditions. We conclude that infant sleeping environment should be considered when interpreting data from infant sleep studies.

The authors express their gratitude to Sean Drummond, Naz Kajani, Dr. James Ashurst, Dr. Lynn Hunt, Dr. Mindy Cetel, and Dr. Peter Fotinakes for excellent assistance.

This study was funded by National Institute of Child Health and Human Development Grant HD-27462.

Address for reprint requests: C. Richard, Sleep Disorders Center, Bldg. 22C, Rt. 23, University of California, Irvine, Medical Center, 101 The City Drive, Orange, CA 92868.

Received 22 April 1997; accepted in final form 3 December 1997.

REFERENCES

- Bach, V., B. Bouferrache, O. Kremp, Y. Maingourd, and J.-P. Libert. Regulation of sleep and body temperature in response to exposure to cool and warm environments in neonates. Pediatrics 93: 789-796, 1994.
- Barry, H., III, and L. M. Paxson. Infancy and early childhood: cross-cultural codes. 2. Ethology 10: 466-508, 1971.
- Brady, J. P., and E. M. McCann. Control of ventilation in subsequent siblings of victims of sudden infant death syndrome. J. Pediatr. 106: 212-217, 1985.
- Cherniack, N. S., and G. S. Longobardo. Cheyne-Stokes breathing An instability in physiologic control. N. Engl. J. Med. 288: 952-957, 1973.
- Devore, I., and M. J. Konner. Infancy in hunter-gatherer life: an ethological perspective. In: Ethology and Psychiatry, edited by N. White. Toronto, Canada: Univ. of Toronto Press, 1974, p. 113-141.
- Fleming, P. J., P. S. Blair, C. Bacon, D. Bensley, I. Smith, E. Taylor, J. Berry, J. Golding, and J. Tripp. Environment of infants during sleep and risk of the sudden infant death syndrome: results of 1993-5 case-control study for confidential inquiry into stillbirths and deaths in infancy. Br. Med. J. 313: 191-195, 1996.
- Gaultier, C. Apnea and sleep state in newborns and infants. Biol. Neonate 65: 231-234, 1994.
- Guilleminault, C., R. Ariagno, R. Korobkin, L. Nagel, R. Baldwin, S. Coons, and M. Owen. Mixed and obstructive sleep apnea and near miss for sudden infant death syndrome.
 Comparison of near miss and normal infants by age. Pediatrics 64: 882-891, 1979.
- Guilleminault, C., and M. Souquet. Sleep states and related pathology. In: Advances in Perinatalogy and Neurology, edited by R. Korobkin and C. Guilleminault. New York: SP Medical and Scientific Books, 1979, vol. 1, p. 225-247.
- Hoppenbrouwers, T., J. E. Hodgman, D. McGinty, R. M. Harper, and M. B. Sterman. Sudden infant death syndrome: sleep apnea and respiration in subsequent siblings. *Pediatrics* 66: 205-214, 1980.
- Hoppenbrouwers, T., J. E. Hodgman, and L. Cabal. Obstructive apnea, associated patterns of movement, heart rate, and oxygenation in infants at low and increased risk for SIDS.. Pediatr. Pulmonol. 15: 1-12, 1993.
- Kahn, A., D. Blum, E. Rebuffat, M. Sottiaux, J. Levitt, A. Bochner, M. Alexander, J. Grosswasser, and M. F. Muller. Polysomnographic studies of infants who subsequently died of sudden infant death syndrome. *Pediatrics* 82: 721-727, 1988.
- Kahn, A., J. Groswasser, M. Sottiaux, E. Rebuffat, P. Franco, and M. Dramaix. Prone or supine body position and sleep characteristics in infants. *Pediatrics* 91: 1112–1115, 1993.
- Kelly, D. H., H. Golub, D. Carley, and D. C. Shannon. Pneumograms in infants who subsequently died of sudden infant death syndrome. J. Pediatr. 109: 249–254, 1986.

- Kelly, D. H., A. M. Walker, L. Cahen, and D. C. Shannon. Periodic breathing in siblings of sudden infant death syndrome victims. *Pediatrics* 66: 515-520, 1980.
- Konner, M. J. Evolution of human behavior development. In: Handbook of Cross-Cultural Human Development, edited by R. Monroe and B. Whiting. New York: Garland, 1981, p. 3-51.
- Konner, M., and C. Worthman. Nursing frequency, gonadal function, and birth spacing among Kung hunter-gatherers. Science 207: 788-791, 1980.
- Lancaster, J. B., and C. S. Lancaster. Parental investment: the hominid adaptation. In: How Humans Adapt: A Biocultural Odyssey. edited by D. J. Ortner. Washington, DC: Smithsonian Inst., 1983, p. 33-56.
- Lindgren, C., L. Jing, B. Graham, J. Grogaard, and H. Sundell. Respiratory syncytial virus infection reinforces reflex apnea in young lambs. *Pediatr. Res.* 31: 381-385, 1992.
- Lozoff, B., and G. Brittenham. Infant care: cache or carry. J. Pediatr. 95: 478-483, 1979.
- McKenna, J. J., S. S. Mosko, and C. A. Richard. Bedsharing promotes breast feeding. Pediatrics 100: 214-219, 1997.
- McKenna, J. J., E. B. Thoman, T. F. Anders, A. Sadeh, V. L. Schechtman, and S. F. Glotzbach. Infant-parent co-sleeping in an evolutionary perspective: implications for understanding infant sleep development and the sudden infant death syndrome. Sleep 16: 263-282, 1993.
- Mitchell, E. A., A. W. Stewart, R. Scragg, R. P. K. Ford, B. J. Taylor, D. M. O. Becroft, J. M. D. Thompson, I. B. Hassall, D. M. J. Barry, E. M. Allen, and A. P. Roberts. Ethnic differences in mortality from sudden infant death syndrome in New Zealand. Br. Med. J. 306: 13-16, 1993.
- Mitchell, E. A., B. J. Taylor, R. P. K. Ford, A. W. Stewart, D. M. O. Becroft, J. M. D. Thompson, R. Scragg, I. B. Hassall, D. M. J. Barry, E. M. Allen, and A. P. Roberts. Four modifiable and other major risk factors for cot death: the New Zealand study. J. Paediatr. Child Health 28, Suppl. 1: S3-S8, 1992.
- Mosko, S., C. Richard, and J. McKenna. Infant sleep and arousals during bedsharing (Abstract). Pediatr. Pulmonol. 20: 340A, 1996.
- Mosko, S., C. Richard, and J. McKenna. Maternal sleep and arousals during bedsharing with infants. Sleep 20: 142-150, 1997.
- Mosko, S., C. Richard, J. McKenna, and S. Drummond. Infant sleep architecture during bedsharing and possible implications for SIDS. Sleep 19: 677-684, 1996.
- Orr, W. C., M. L. Stahl, J. Duke, M. A. McCaffree, P. Toubas, C. Mattice, and H. F. Krous. Effect of sleep state and position on the incidence of obstructive and central apnea in infants. Pediatrics 75: 832-835, 1985.
- Richard, C. A., S. Mosko, J. McKenna, and S. Drummond. Sleep position, orientation and proximity in bedsharing infants and mothers. Sleep 19: 685-690, 1996.
- Rigatto, H., and J. P. Brady. Periodic breathing and apnea in preterm infants. I. Evidence for hypoventilation possibly due to central respiratory depression. *Pediatrics* 50: 202-218, 1972.
- Schafer, T., D. Schafer, and M. E. Schlafke. Breathing, transcutaneous blood gases, and CO₂ response in SIDS siblings and control infants during sleep. J. Appl. Physiol. 74: 88-102, 1993.
- Schechtman, V. L., R. M. Harper, A. J. Wilson, and D. P. Southall. Sleep apnea in infants who succumb to the sudden infant death syndrome. *Pediatrics* 87: 841-846, 1991.
- Scragg, R., A. W. Stewart, E. A. Mitchell, R. P. K. Ford, and J. M. D. Thompson. Public health policy on bed sharing and smoking in the sudden infant death syndrome. NZ Med. J. 108: 218-222, 1995.
- Sleep Disorders Atlas Task Force of ASDA. EEG arousals: acoring rules and examples. Sleep 15: 174–184, 1992.
- Waters, K. A., A. Gonzalez, C. Jean, A. Morielli, and R. T. Brouillette. Face-straight-down and face-near-straight-down positions in healthy, prone-sleeping infants. J. Pediatr. 128: -616-625, 1996.