

Mother-Infant Bedsharing is Associated with an Increase in Infant Heart Rate

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Objectives: We hypothesized that mother-infant bed sharing, compared to solitary sleeping, would be associated with higher infant heart rates. The objective was to compare infant heart rates between the 2 environments and, secondarily, to test for relationships between heart rate and other, previously reported, differences in the same infants.

Design: Heart rate was measured in 15 infants over a bed-sharing night and a solitary-sleeping night. Eight of the 15 infants routinely bed shared with the mother at home; the other 7 routinely slept in a room alone.

Setting: The Sleep Disorders Center, University of California, Irvine Medical Center.

Participants: Fifteen mother-infant pairs who met criteria for routinely bed sharing or sleeping solitarily. All were healthy, and infants were more than 38 weeks gestation at birth and 11 to 15 weeks old at the time of the study.

Interventions: None.

Results: Analysis of variance indicated that, irrespective of routine sleeping condition, heart rate was lower during solitary sleeping than during bed

sharing in all sleep stages. Significant regressions were found with infant temperature. Heart-rate variability was higher during solitary sleeping than during bed sharing (both routine groups) in stages 1 and 2 and rapid eye movement sleep, but only stages 1 and 2 sleep effects were independent of basal heart rate.

Conclusions: Infant heart rate is affected by the mother's presence in the sleep environment. The increase in sympathetic activity in stages 3 and 4 and rapid eye movement sleep might be partly explained by differences in thermoregulation between bed-sharing and solitary-sleeping environments. These results support the notion that sensory differences between bed-sharing and solitary-sleeping environments account for some of the physiologic differences between infant sleep in the 2 sleeping conditions.

Key Words: Co-sleeping, solitary sleeping, infant heart rate variability, infant thermoregulation

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INTRODUCTION

RECENT INTEREST IN THE PHYSIOLOGIC AND BEHAVIORAL CORRELATES OF MOTHER-INFANT BED SHARING IS DRIVEN, IN PART, BY CLAIMS THAT BED SHARING IS A SIGNIFICANT INDEPENDENT RISK FACTOR FOR THE SUDDEN INFANT DEATH SYNDROME (SIDS).¹ Furthermore, some form of co-sleeping or bed sharing represents the condition under which infants adapted throughout human evolutionary history.^{2,3} It seems self-evident that the infant's sensory and social environment during bed sharing is enriched compared to that found during solitary sleeping. It is much less clear whether the physiologic differences between infants in the 2 conditions, presumably related to those environmental differences, are clinically relevant. In terms of infant morbidity and mortality, the various physiologic responses and adaptations to bed sharing that have been previously reported could be construed as positive (increased breast-feeding and arousal frequency, less obstructive apnea, greater maternal vigilance⁴⁻⁷), negative (increased central apnea and periodic breathing and, from a SIDS perspective, higher body temperature in non-rapid eye movement sleep [REM]^{6,8,9}), or neutral (as with small, but statistically significant differences in sleep architecture¹⁰).

Understanding the effects of bed sharing on infant heart rate might be useful in determining how stimuli related to the presence of the mother could affect homeostatic systems during this important developmental period. Increases in basal heart rate can indicate increases in sympathetic relative to parasympathetic activity, and decreases in heart-rate variability can indicate possible cardiovascular control-system insufficiency.^{11,12} In addition, Petersen et al¹³ suggested that developmental changes in infant heart rate are related to developmental changes in the thermoregulatory system. Infant body temperature is significantly

affected by bed sharing versus solitary sleeping, probably with a sleep-stage effect^{8,9}; therefore, it is possible that bed sharing could affect heart rate via its effect on thermoregulation.

Since external sensory input is generally arousing and involves increased sympathetic activity,^{14,15} we hypothesized that infant heart rate would be increased during mother-infant bed sharing. In the absence of prior findings that would predict a specific effect of enhanced sensory input on heart-rate variability, we proposed the null hypothesis, ie, that heart-rate variability would not differ in the 2 environments.

METHODS

Thirty-five mother-infant pairs were recruited to spend 3 consecutive nights in the sleep laboratory for polysomnography with video recording. In 15 of those pairs, the electrocardiogram was digitized successfully and constitutes the sample in this report. The 20 other pairs either were not recorded digitally (13 pairs) or the polysomnograms were contaminated by artifact in 1 or more sleep stages (7 pairs). All met selection criteria for inclusion into either routine bed-sharing (RB) or routine solitary-sleeping (RS) groups. All infants were healthy and between 11 and 15 weeks old (a peak age range for SIDS) and had normal gestations. No subject had a family or personal history of SIDS or an apparent life-threatening event. Other details of the sample and selection criteria have been published previously.^{4-6,10} This study was approved by the University of California Irvine Institutional Review Board.

Each mother-infant pair slept the first or "adaptation" night in the sleep laboratory in their routine home condition (bed sharing or solitary sleeping). On the following 2 nights, pairs spent 1 night in their routine condition and 1 night in the other sleeping condition, with the order randomized. Data from the adaptation night were not used in this report. The 3-night protocol accomplished 2 main goals. First, it eliminated the confounding influence of any possible "first-night effect," although subsequent analysis showed no measurable effect in this sample (Richard, unpublished observations). Secondly, this design allowed a 2 × 2 repeated-measures statistical analysis (ANOVA). The within-subject comparison was between the bed-sharing night and the solitary night for both groups (night effect), while the across-subject comparison was between RB and RS sleepers on both nights (group effect).

Disclosure Statement

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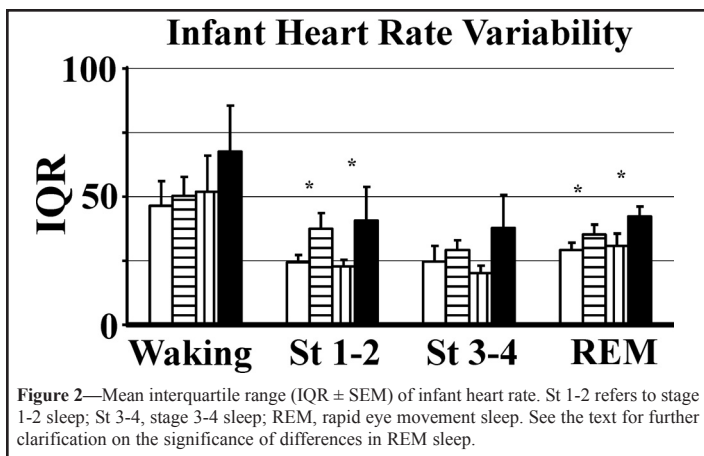
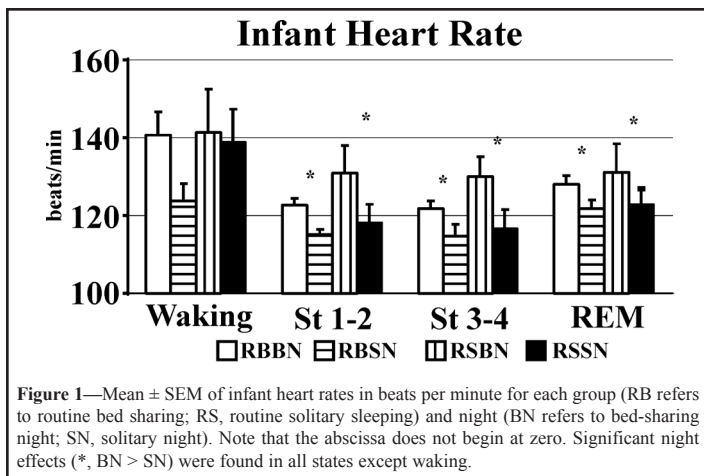
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This study involved standard polysomnographic paper recordings (ie, electroencephalogram, chin electromyogram, eye movement, electrocardiogram, and respiration) and sleep-stage scoring. Temperature recordings also were done but only on the infants. The electrocardiogram signal was digitized at 1 kHz and stored on optical disks. Digitized records were downloaded to a personal computer and subjected to R-wave peak detection for determination of R-R intervals. For each infant on each night, a single continuous block of R-R intervals was chosen from each of the 4 sleep-wake categories: waking, stage 1-2, stage 3-4 and REM sleep.¹⁶ Sample periods were at least 1 minute of artifact-free data from a single, consolidated sleep stage (ie, not including any stage transitions or arousals). Most artifacts were due to movement, crying, or sensor adjustment. For waking, sample periods ranged from 60 to 660 seconds (median, 120 seconds); stage 1-2 samples ranged from 120 to 1200 seconds (median, 600 seconds); the stage 3-4 sample range was 120 to 1620 seconds (median, 600 seconds); and REM samples ranged from 80 to 1020 seconds (median, 450 seconds). All software-detected R waves were confirmed visually and checked for erroneous or multiple marks. The interval data were converted into heart rate on a beat-by-beat basis. Therefore, for each infant, the median heart rate was derived in each of the 4 sleep-wake stages on both nights of the study. Interquartile ranges (IQR) of the R-R intervals indexed heart-rate variability.

Two-way, repeated-measures ANOVAs were the primary analysis tool. Separate tests were performed for each sleep-wake state and used to detect either immediate effects (within routine group) or long-term effects (across groups) of sleeping environment. Posthoc tests were not done, as they are inappropriate when the number of comparison groups is 2. Statistical significance was indicated by $P < .05$.

RESULTS

Infant heart rate was affected by bed sharing in a state-specific manner. ANOVAs showed a significant night effect, where heart rate during bed sharing was higher than during solitary sleeping in all sleep stages

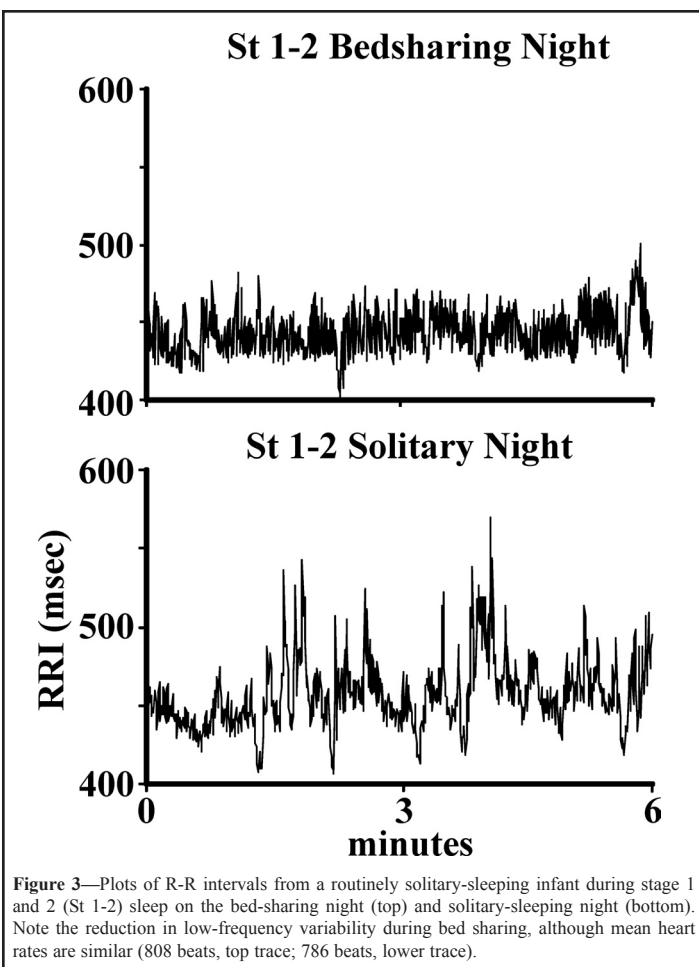


(stage 1-2, $P = .006$; stage 3-4, $P = .002$; REM, $P = .006$) but not in waking ($P > .20$). There were no significant group effects (RB versus RS sleepers) or interactions between group and night in any sleep-wake state. On average, compared to the solitary night, heart rate on the bed-sharing night was 8.8% higher in stage 1-2, 9.2% higher in stage 3-4, and 5.5% higher during REM sleep. Figure 1 shows the mean (\pm SEM) of the median infant heart rates for each sleep-wake stage.

The ANOVA of the infants' IQR showed a significant night effect for stage 1-2 ($P = .017$) and for REM sleep ($P = .024$) but not for stage 3-4 sleep ($P = .12$) or for waking ($P > .20$). In both of the affected sleep stages, the bed-sharing night was associated with a reduction in IQR compared to the solitary night (Figure 2). Although not statistically significant, the pattern in stage 3-4 sleep was similar to that in stage 1-2 and REM sleep. No group (routine sleeping condition) or interaction effects were detected. An example of the reduction in heart-rate variability is illustrated in Figure 3. These tracings of R-R interval are from a single infant who routinely slept alone and are taken from stage 1-2 sleep.

Given that heart-rate variability decreases as heart rate increases,¹⁷ the infant IQR ANOVAs were repeated using the matching heart rates as covariates. Covariant analyses indicated that the reduction in IQR on the bed-sharing night during stage 1-2 sleep was not fully explained by the increase in median heart rate during that sleep stage ($P = .023$, compare to initial $P = .017$). However, the fall in heart-rate variability during REM sleep appears to be due to differences in basal heart rate ($P = .347$; initial $P = .024$).

Identical analyses of the mothers' heart-rate data revealed a significant night effect for stage 1-2 sleep, where the bed-sharing night was associated with a slower heart rate than the solitary night ($P = .032$). In addition, there was a significant group effect during stage 3-4 sleep, with lower heart rates in the RB than in the RS sleeping mothers ($P = .042$). The mothers' heart-rate data (mean of the medians \pm SEM) are shown in Table 1. Heart-rate variability in the mothers was not significantly dif-



ferent between nights or between routine sleeping conditions (data not shown).

Secondary Analysis

This population sample has been studied extensively for differences in other dependent variables (eg, sleep architecture, apnea, arousal, and body temperature) in relation to bed sharing or solitary sleeping.^{4-8,10} Regression analysis was used to derive possible associations between those previously reported affected variables and infant median heart rates for the current subgroup of 15 infants. The regression analysis was sleep-stage specific and grouped by the routine sleeping condition, in-laboratory sleeping condition, or both (Table 2).

Several variables showed either no relationship or weak and inconsistent relationships to median heart rate. These included sleep-stage total time and mean duration, central and obstructive apnea frequency, and several measures of breast-feeding. However, 1 variable—mean axillary temperature—showed significant and consistent correlations with heart rate in all of the sleep-wake stages where heart rate was affected by sleep environment (Table 2). In addition, mean temperature and heart rate had the strongest correlation coefficients across all sleep stages.

During stage 1-2 sleep, significant regressions between axillary temperature and heart rate were found for all infants, ie, both RB and RS groups, but only when the 2 nights were combined (RB: $r^2 = 0.205$, $F = 4.85$, $P = .045$; RS: $r^2 = 0.380$, $F = 7.73$, $P = .019$). In this sleep stage, the RS sleeping infants had the strongest coefficient, compared to the RB group (.380 versus .205), and the steepest regression slope (55.4 versus 27.0).

During stage 3-4 sleep, there was a strong relationship between body temperature and heart rate in the RS sleeping infants (both nights combined) and a weaker but significant relationship for the solitary-sleeping nights (both groups combined). This likely reflects the strong regression for the RS sleeping infants on their solitary night ($r^2 = .838$, $F = 26.82$, $P = .006$) that contributed to both of the larger groupings.

During REM sleep, there was a strong relationship between body temperature and heart rate for the RB ($r^2 = 0.442$, $F = 12.90$, $P = .003$) and a weaker relationship in infants on their bed-sharing night ($r^2 = 0.263$, $F = 5.63$, $P = .035$). No other significant regressions were found. Therefore, infant heart rate is positively correlated with temperature during solitary sleeping in stage 3-4 sleep but during bedsharing in REM sleep.

Previous analyses suggested that the higher mean temperatures on the bed-sharing night might relate to a higher frequency of movement arousals during bed sharing.⁸ Therefore, regressions of heart rate were performed with the frequency of transient arousals (electroencephalogram frequency increase of more than 3 seconds, without movement) and movement arousals (transient arousal with movement). The frequency of movement arousals during REM sleep showed strong regres-

sions with heart rate on the solitary night (both routine groups combined; $r^2 = 0.435$, $F = 11.78$, $P = .004$). In addition, each routine group exhibited individually significant regressions between movement-arousal frequency and median heart rate on the solitary night: (RB solitary night: $r^2 = .512$, $F = 8.31$, $P = .028$; RS solitary night: $r^2 = 0.508$, $F = 7.19$, $P = .044$).

DISCUSSION

The main finding is that infant heart rate and variability are affected significantly by bed sharing with the mother. In all sleep states (stages 1-2, 3-4, and REM), heart rates were significantly higher during bed sharing than during solitary sleeping. This was true for both RB and RS sleepers (ie, without a significant group effect), although the effect tended to be greater in RS sleeping infants (see Figure 1). This suggests that the differences in heart rates primarily reflected immediate effects of the different sleeping environments rather than neurodevelopmental adaptations to the infants' routine sleeping condition. Significant heart-rate differences were not found in infants during waking.

The most likely mechanism of the higher heart rates in infants while bed sharing is the relative enrichment of environmental sensory stimuli (physical and social) compared to solitary sleeping. There is evidence that infants actively maximize their exposure to sensory stimuli from the mother during bed sharing, even when they do not routinely bed share. Bed-sharing infants spend most of their time facing the mother and do so at close proximity.^{7,18} While the mother and the size of the sleeping surface largely determine the proximity, infant orientation to the mother seems to be an active infant behavior, since during even supine sleep infants preferentially face toward their mothers.^{7,18} One could speculate that there may be multiple purposes for this infant behavior, such as promoting security, facilitating emotional attachment, and providing stimulation or drive to homeostatic systems, eg, via arousal and increased sympathetic activity.

While the various sensory stimuli that exist in the bed-sharing environment are not well defined, an initial survey of the physiologic and behavioral differences between bed-sharing and solitary-sleeping infants has been reported for a larger sample of subjects participating in this same study.^{4-8,10} This broad range of physiologic data on each infant (and mother) allows associations to be tested between the dependent variables that may modulate heart rate. The results of those analyses could then

Table 1—Heart Rate of 15 Mothers During Bedsharing and Solitary-Sleeping Conditions

Sleep State	Bed-sharing Night	Solitary-sleeping Night
Routine Bed Sharers (n=8)		
AW	68.7 ± 2.9	70.9 ± 2.6
Stage 1-2	63.4 ± 2.9	65.7 ± 2.6
Stage 3-4	64.5 ± 2.7	67.9 ± 2.7
REM	70.0 ± 3.6	74.1 ± 2.8
Overall	66.6 ± 2.9	69.4 ± 2.4
Routine Solitary Sleepers (n=7)		
AW	77.9 ± 3.0	75.6 ± 3.7
Stage 1-2	70.0 ± 2.4	74.5 ± 3.0
Stage 3-4	71.4 ± 2.3	79.7 ± 5.8
REM	73.9 ± 3.3	79.0 ± 2.8
Overall	73.3 ± 2.5	77.2 ± 3.1

AW refers to awake; REM, rapid eye movement.
Data are presented as mean beats per minute ± SEM.

Table 2—Adjusted r^2 values from regression analysis with median R-R intervals for each sleep-wake state.

Sleep Stage	Infant Category	Mean Axillary Temperature	Movement Arousal Frequency
1-2	RB	.205	
	RS	.380	
	BN		
	SN		
	Other†		RSSN
3-4	RB		
	RS	.584*	
	BN		
	SN	.266	
	Other†		RSSN*
REM	RB	.442*	
	RS		
	BN	.263	
	SN		.435*
	Other†		RBSN, RSSN

Only significant r^2 values are given ($P < .05$)

* P values < 0.01 .

All regression values used in the analysis, except breast-feeding, are sleep-stage specific. Frequencies are no./h of that sleep stage.

†Indicates significant regression coefficients for an individual subgroup, eg, routine bed sharers (RB) on their bed-sharing night (BN).

REM refers to rapid eye movement; RS routine solitary sleepers; SN, solitary night.

suggest possible mechanisms for the increased heart rate during bed sharing.

As mentioned in the Results, regressions with median heart rate were run on a number of variables, all found previously to be significantly affected by bed sharing or solitary sleeping. These variables were measures of breast-feeding (frequency, mean duration, and total minutes); apnea (central apnea frequency, obstructive apnea frequency, and periodic breathing [percentage of stage time]); sleep architecture (sleep-wake stage time); arousal (waking frequency and frequency of transient arousals with and without movement); and mean axillary temperature. Mean axillary temperature was the only variable that showed consistently significant regression coefficients within a sleep stage and did so in each sleep stage.

Axillary temperatures in these infants were higher during bed sharing in non-REM sleep stages.⁸ It has been previously reported that body temperature varies with heart rate in infants.^{19,20} In addition, Petersen et al¹³ recently reported a potential developmental relationship between thermoregulation and basal heart rate in infants. We chose axillary temperature to index body temperature, since rectal probes are invasive and can require frequent reinsertion through the course of a night. Long-term continuous axillary recording (as in the present study) avoids most of the discrepancies that plague single-point measures of axillary temperature or other indexes of core body temperature.²¹⁻²³

In stage 1-2 sleep, the results suggest a relationship between mean axillary temperature and heart rate in all infants, since both the RB and RS infants as a group (both nights combined) exhibited significantly positive regressions. The RS group had a somewhat stronger relationship than did the RB infants.

Significant stage 3-4 relationships between temperature and heart rate were limited to solitary sleeping, both routinely and immediate. Therefore, these infants had significantly lower temperatures and lower heart rates than bed-sharing infants and a stronger coupling between the 2 measures. This suggests that during stage 3-4 (slow-wave sleep), body temperature is an important source of influence on heart rate only in the solitary sleepers, whereas nonthermal factors play a larger role in the bed-sharing environment.

In contrast, REM-related effects were found only in the bed-sharing infants (routinely or immediate), suggesting that the increase in heart rate may be due to increased body temperature during that sleep state. However, these bed-sharing infants did not experience a significant increase in axillary temperature during REM sleep.⁸ Therefore, while there is a stronger coupling between temperature and heart rate during bed sharing, factors not related to body temperature are likely involved in the increase in bed-sharing infant heart rate in REM sleep. However, the possibility exists that the stronger coupling between body temperature and heart rate may itself result in higher heart rates in bed-sharing infants.

The interpretation that differences in heart rate between solitary-sleeping and bed-sharing infants are related to differences in thermoregulation does not exclude the possibility that other variables affected by sleeping condition also modulate heart rate. Heart rate in both groups of infants on the solitary night had significant coefficients with the frequency of transient movement arousals during REM sleep, indicating that during solitary sleeping in REM sleep there is a relationship between the frequency of movement arousals and median heart rate that doesn't seem to exist during bed sharing. The specific relationship between sleep state, heart rate, body temperature, and movement arousal, if any, is much too complex (especially in REM sleep) to be addressed with the current analyses and begs further attention.

Other possible mechanisms for the effect of sleep environment on heart rate include a generally lower basal sympathetic tone during solitary sleep (as opposed to environmental stimuli-based reflexive changes during bed sharing) that results from sources not addressed in this study, such as differences in mean arterial pressure. During solitary sleeping, infants were occasionally placed prone by mothers in our study, potentially causing a decrease in infant heart-rate variability²⁴; it was during

bed sharing, however, that infants exhibited the fall in heart-rate variability, and infants were never placed prone while bed sharing.⁷ It is an open question whether solitary sleeping is associated with stress responses in the infant, either hormonal or autonomic, that might alter basal blood pressure or heart rate or that alter cardiovascular reflexes to sensory stimuli. Nevertheless, the original premise that the differences between bed-sharing and solitary-sleeping infants result, at least in part, from greater sensory input in the bed-sharing environment remains useful, although the processing of those signals into the modulation of heart rate is complex and sleep-stage specific.

The fall in heart-rate variability in infants while bed sharing was unexpected, since the increase in transient arousals found during bed sharing⁵ would be expected to transiently, but repetitively, increase heart rate. Even more problematic is that only in stage 1-2 sleep is the difference in variability not explained by the effect of different basal heart rates. Therefore, the lower variability during bed sharing may be the result of changes in autonomic control related to sleep environment that are specific to stage 1-2 sleep. Further study is required to delineate the frequencies in heart rate that may be preferentially affected by the bed-sharing or solitary-sleeping condition.

The clinical significance of the differences in heart rate and its variability between the 2 sleeping environments is not clear; clinical outcomes were not a part of the experimental design. However, it does appear that bed-sharing infants, compared to solitary-sleeping infants, have a relative increase in sympathetic activity. Considering that these results resemble certain autonomic differences in some SIDS infants, one might speculate that higher heart rate during bed sharing increases SIDS risk. However, there is strong epidemiologic evidence that bed sharing is not an independent risk factor for SIDS.^{25,26} Yet, it is interesting that bed sharing increases the SIDS risk related to maternal smoking^{25,26} and that the expression of that increased risk may be related to deficiencies in arousal^{27,28} that are accompanied by abrupt increases in sympathetic activity.²⁹ It would be premature at this point to conclude that any positive or negative clinical outcomes are associated with the heart-rate changes reported here.

An interesting contrast was found between mother and infant heart-rate responses to the bed-sharing environment; in non-REM sleep, bed-sharing infants had a higher heart rate and mothers a lower heart rate. A general explanation is that, while bed sharing, a mother and an infant are not equivalent environmental stimuli to each other. Furthermore, they are not equivalent responders to sensory input, given the dramatic differences in developmental state, cognition, and psychology. Nevertheless, the bed-sharing mothers' lack of cardiac activation to the bed-sharing environment is interesting. Factors other than environmental stimuli may play a role in the slower heart rates of bed-sharing mothers or, just as validly, a role in the higher heart rates in solitary-sleeping mothers. Hormonal factors related to bed sharing may slow heart rate, such as effects stemming from the greatly increased level of breast-feeding during bed sharing.⁴ On the other hand, solitary-sleeping mothers might have greater anxieties related to child-care responsibility because of the separation from their infants.

In summary, the bed-sharing sleeping condition is associated with an increase in infant heart rate, compared to the solitary condition, in all 3 infant sleep states. In addition, heart-rate variability is lower during bed sharing in stage 1-2 and REM sleep. Attempts to link median heart rates to a specific physiologic or behavioral feature of the bed-sharing environment suggested that a possible modulation of heart rate by body temperature might exist for stages 3-4 and REM sleep. The source of the difference in heart-rate variabilities remains unknown, but during stage 1-2 sleep, the effect is most likely the result of a change in autonomic balance that is independent of the effect on heart rate.

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