Infant Arousals During Mother-Infant Bed Sharing: Implications for Infant Sleep and Sudden Infant Death Syndrome Research

Sarah Mosko, PhD*; Christopher Richard, PhD*; and James McKenna, PhD‡

ABSTRACT. *Objective.* **Normative values for infant sleep architecture have been established exclusively in the solitary sleeping environment. However, most of the world's cultures practice some form of parent-infant cosleeping. In addition, no previous polysomnographic studies in infants examined the frequency of electroencephalogram (EEG) arousals. This is the first study to assess (a) EEG arousals in infants and their relationship to sleep stages; (b) the impact on arousals of mother-infant bed sharing; and (c) the temporal overlap of infant with maternal arousals during bed sharing.**

Methodology. **Three nights of polysomnography were performed in 35 breastfeeding mother-infant pairs when the infants were 11 to 15 weeks old. An adaptation night was followed by one bed sharing night and one solitary sleeping night. Twenty infants had been bed sharing since birth and 15 were routine solitary sleepers. Both epochal awakenings (EWs), based on 30-second epoch scoring of sleep-wake stages, and more transient arousals (TAs) ≥3 seconds were quantified.**

Results. **Stage 3–4 sleep was associated with a striking paucity of EWs and TAs compared with stages 1–2 or rapid eye movement sleep. Bed sharing facilitated EWs and TAs selectively during stage 3–4 sleep. EWs from stage 3–4 sleep were more frequent on the bed sharing night than on the solitary night in both infant groups. Routinely bed sharing infants also exhibited more frequent TAs in stage 3–4 than the routine solitary sleepers in both conditions. In both groups, the number of infant arousals (EWs + TAs) that overlapped the mother's was doubled during bed sharing, with infant arousals leading most often.**

Conclusions. **Mother-infant bed sharing promotes infant arousals. Together with a previous report that bed sharing reduces stage 3–4 sleep, this suggests that normative values for infant sleep must be interpreted within the context of the sleeping environment in which they were established. Given that arousability is diminished in stage 3–4, we speculate that, under otherwise safe conditions, the observed changes in stage 3–4 sleep and arousals associated with bed sharing might be protective to infants at risk for SIDS because of a hypothesized arousal deficit. The responsivity of the mother to infant arousals during bed sharing might also be protective.** ***Pediatrics*** **1997;100:841–849;** ***bed sharing, cosleeping, solitary sleeping, infant arousals, SIDS, infant sleep.***

From the *Department of Neurology, University of California Irvine Medical Center, Orange, California; and the ‡Department of Anthropology, Pomona College, Claremont, California.

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Reprint requests to (S.M.) Sleep Disorders Center, Bldg 22C, Rt 23, University of California Irvine Medical Center, 101 The City Drive, Orange, CA 92868.

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ABBREVIATIONS. SIDS, sudden infant death syndrome; ALTE, apparent life-threatening event; EEG, electroencephalogram; REM, rapid eye movement; RB, routine bed sharing; RS, routine solitary sleeping; BN, bed sharing night; SN, solitary sleeping night; EW, epochal awakening; TA, transient arousal; ANOVA, analysis of variance; NREM, non–rapid eye movement.

The mechanism(s) of the sudden infant death syndrome (SIDS) remains controversial. Impaired cardiorespiratory controls, hyperthermia, lethal rebreathing of carbon dioxide trapped in bedding, and arousal deficiency are among the postulated mechanisms that are currently the focus of much interest and debate.[1,2] That arousal deficiency could be important stems from the notion that arousal can be a protective response to dangerous conditions or events in sleep. Observations in victims of SIDS support a role of arousal deficiency in SIDS. Infants who subsequently died of SIDS were found to move less in sleep,[3] and parents of SIDS victims reported retrospectively greater difficulty awakening their infants and fewer infant body movements than reported by the parents of healthy infants.[4] Observations in infants at high risk for SIDS also support a role of arousal deficiency. Infants who have suffered an apparent life-threatening event (ALTE) have fewer sleep-related movements[5] and less frequent spontaneous awakenings[6–8] than control infants. Subsequent siblings of SIDS victims have longer periods of uninterrupted sleep,[9,10] fewer body movements in sleep,[11] and fewer movements accompanying obstructive apneas.[12] Furthermore, the arousal response to hypoxic or hypercarbic challenges during sleep may be impaired in both ALTE infants and subsequent siblings of SIDS victims.[13–16]

The majority of SIDS cases occur between 1 and 6 months of age. If arousal deficiency is contributory, seemingly normal developmental changes in infant sleep architecture during this period might act in concert with an arousal deficit to increase an infant's risk. Sleep gradually consolidates over the first 6 months of postnatal life, as shown by lengthening of sustained sleep bouts.[8,17–20] Also, the total duration of quiet sleep (associated with high-voltage slow-wave electroenchephalogram [EEG] or delta) increases and episodes of quiet sleep lengthen progressively.[7,10,18,21,22] The amount of rapid eye movement (REM) sleep and indeterminate sleep, by contrast, declines or remains stable.[10,21,22] Arguably, this selective increase in quiet sleep might undermine infant arousability because arousal threshold has been shown to be high in the EEG delta range in human

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infants and adults as well as in rats.[23–25] That the majority of normal infants under 7 months of age fail to arouse in response to a hypoxic challenge in quiet sleep illustrates that infant arousability is attenuated in quiet sleep.[26] Furthermore, Schechtman et al[27] have provided specific evidence that enhanced quiet sleep (which is associated with EEG delta waves) may contribute to SIDS. They found that siblings of SIDS victims aged 3 to 4 months displayed increased integrated delta amplitude in the early morning hours relative to control infants. Factors that facilitate either sleep consolidation or quiet sleep might represent a particular challenge to infants with impaired arousability.

Reciprocally, factors that facilitate arousal might be protective against SIDS in vulnerable infants. Furthermore, conditions that enable a care taker to better detect potentially dangerous conditions in the infant might also be protective. We have hypothesized that parent-infant cosleeping (room sharing or bed sharing) might decrease SIDS risk in some infants via effects on either parental or infant sleep.[28–31] This was based in part on the observation that SIDS rates tend to be lower in societies where parent-infant cosleeping is commonplace,[32–36] together with evidence that infant sleep evolved within the context of cosleeping.[37] A laboratory polysomnographic study was designed to compare bed sharing with solitary sleeping in 35 Latino, mother-infant pairs. In the mothers, bed sharing modestly reduced the total duration and episode length of stage 3–4 sleep. Stage 1–2 sleep was reciprocally increased overall, although individual stage 1–2 episodes were also shortened. These stage effects could be explained by an increased arousal frequency found for both stages 3–4 and 1–2.[29] Similarly in the infants, total stage 3–4 duration (analogue of quiet sleep) was reduced and stage 3–4 episodes were shorter during bed sharing. Also, total stage 1–2 sleep was reciprocally increased in infants, but individual episodes of stage 1–2 and REM were both longer.[30] The impact of bed sharing on infant arousals was not reported. None of these effects in mothers or infants habituated when bed sharing was routine. Furthermore, there was no evidence that the reduction in stage 3–4 sleep in infants was explained by rebound of stage 3–4 sleep during solitary sleep before the mothers retired or on other nights when infants slept alone.[30] We suggest that, by limiting the infant'stage 3–4 sleep, bed sharing might enhance the infant's ability to arouse spontaneously in response to a dangerous or life-threatening condition. Furthermore, in the mother, curtailment of stage 3–4 sleep and augmentation of arousals should promote her ability to monitor changes in the infant's status.

In the present study, we describe the impact of bed sharing on infant arousal frequency in these same Latino mother-infant pairs. The temporal overlap of infant arousals with maternal arousals also is assessed.

MATERIALS AND METHODS

Results are presented for 35 mother-infant pairs. Twenty were routinely bed sharing (RB) and 15 were routinely solitary sleeping (RS) since birth. RB was defined as bed sharing with the mother for at least 4 hours per night, 5 nights per week; RS was defined as bed sharing no more than 1 night per week for any part of the night. Two-week sleep logs were completed at home just before the sleep recordings to confirm maternal reports of the infants' usual home sleep environment. For the 33 pairs who completed all 14 nights of the log, the mean number (± SD) of bed sharing nights was 13.7 ± 0.5 for the RB group vs 0.6 ± 0.9 for the RS group.

Subjects were recruited from the Birthing Center at the University of California Irvine Medical Center. The protocol was approved by the University's Human Subjects Review Committee. Informed and signed consent was obtained from all mothers, and they were remunerated for their participation. All mothers were Latina, because bed sharing is an accepted practice in this ethnic group[38] and to control for potential cultural differences in attitude toward and implementation of bed sharing. Other inclusion criteria for mothers were: age <38 years; exclusively or predominantly breastfeeding (no more than two 4-oz bottles of formula per day and none after 3 PM); prenatal care; no present or past history of drug or alcohol abuse; no history of smoking or alcohol or illicit drug use during pregnancy; uncomplicated pregnancies; good health and freedom from sleep disorders; no medications known to affect sleep pattern; and choice of sleeping practice for reasons other than infant temperament. The latter criterion was to eliminate infant temperament as a possible factor in choice of sleeping practice, eg, response to a "fussy" infant. A physician trained in sleep disorders medicine performed the sleep histories. RB mothers were 27.0 ± 5.9 years of age, and RS mothers were aged 24.3 ± 8.5 years, a nonsignificant difference ($P > .05$).

Inclusion criteria for infants were: age 11 to 15 weeks at the time of the sleep studies; good health, with normal growth and development; >37 weeks gestational age and >2500 g at delivery; 5-minute Apgar score ≥8; no history of SIDS in first degree relatives; and no history of prolonged apnea or an ALTE. The RB infants comprised 11 boys and 9 girls, aged 13.0 ± 1.3 weeks when sleep testing was performed; the RS infants comprised 4 boys and 11 girls and were 12.9 ± 1.3 weeks old.

Sleep studies were performed in the University Medical Center Sleep Disorders Center. Mother-infant pairs underwent 3 consecutive nights of polysomnography: an initial adaptation night (matching the routine home sleeping arrangement) followed by a bed sharing night (BN) and a solitary sleeping night (SN) in randomly assigned order. For the SN, infants were placed in a standard crib in a room adjacent to the mothers' with the doors between them open. On the BN, mother-infant pairs shared the same twin size bed used by the mothers for the SN. Infants were maintained on their usual feeding and sleeping schedules, with mothers performing all care taker interventions ad lib. Mothers were blind to all experimental hypotheses and instructed only to prepare their infants for sleep as they would at home. Mothers also retired at their usual times, an average of 66.5 ± 24.7 minutes after their infants (collapsing across groups and conditions). Monitoring was terminated after mother and infant had awakened the next morning at their usual times.

Monitoring in infants and mothers included standard, noninvasive polysomnographic measures (EEGs C3/A2 and O1/A2, left and right electrooculograms, chin electromyelogram, airflow via an oronasal thermocouple [Rochester Electromedical, Tampa, FL] (infants) or thermister [EPM Systems, Midlothian, VA] (mothers), respiratory effort at the chest and abdomen via piezo-crystal belts [EPM Systems, Midlothian, VA], and electrocardiogram) and also infrared audiovideo camera recording. All signals from a given pair were recorded simultaneously each night on a single 22-channel polygraph (Grass 8 plus, Grass Instruments, Quincy, MA). Sleep stages were scored in 30-second epochs using the Rechtschaffen and Kales system[39] in mothers (modified by collapsing across stages 1 and 2 and stages 3 and 4) and the similar Guilleminault and Souquet system[40] in the infants. Two types of arousals were scored. Both stage scoring systems identify epochal awakenings (EWs) that reflect a change in stage scoring to wakefulness (ie, when sleep is followed by an epoch reflecting at least 50% wakefulness). More transient EEG arousals ≥3 seconds (TAs) also were scored. These reflected an abrupt, transient shift in EEG frequency (which could include alpha, beta, or theta frequencies) scored according to established criteria,[41] modified only in that arousals meeting criteria for EWs were scored separately as such. EWs and TAs were summed to obtain total arousals. In addition, arousals of either type in infants were categorized according to

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temporal overlap with arousal in the mother. The three categories were: mother aroused first; infant aroused first; and arousals appeared simultaneous. To prevent experimenter bias in the identification of overlapping arousals, sleep stages and arousals were scored independently in mothers and infants before arousal overlap was determined. All recordings from a given mother-infant pair were scored by the same individual, and interrater reliabilities for the scoring of sleep stages and arousals were >.86 for infant recordings and >.94 for maternal recordings.

Analyses

Arousal Frequencies

EWs and TAs were partitioned by sleep stage and expressed as frequency scores (per hour of a given sleep stage). For both EW and TA frequencies, three main effects and their interactions were assessed first by a 2 × 2 × 3 repeated-measures analysis of variance (ANOVA): they were laboratory condition (BN or SN); routine sleeping arrangement (RB or RS); and sleep stage (1–2, 3–4 or REM). Because the frequencies of both EWs and TAs appeared much lower in stage 3–4 than in the other two sleep stages, the main effect for sleep stage and all interactions involving this term were assessed by two planned comparisons. The first compared stage 3–4 with stages 1–2 and REM; the second compared stage 1–2 with stage REM. Separate 2 × 2 repeated-measures ANOVAs of EW and TA frequencies were performed subsequently for each sleep stage. These reflected our particular focus on infant arousability in stage 3–4 sleep. Furthermore, a significant 3-way interaction effect on TA frequency suggested that a separate analysis be performed for each sleep stage using the factors laboratory condition and routine sleeping arrangement. That is, the significant 3-way interaction indicated that the effects from the two substantive factors, laboratory condition and routine sleeping arrangement, varied across stages of sleep. In order to interpret the substantive factors, separate 2 × 2 analyses were conducted for each sleep stage.

Overlap With Maternal Arousals

For the analysis of overlap of infant with maternal arousals, EWs and TAs were not partitioned by sleep stage, and they were summed to reflect total arousals. Nonparametric tests were used because of non-normal distributions and unequal variances among the groups: the Wilcoxon matched pairs signed ranks test was used for within-group comparisons of the two laboratory conditions (BN vs SN); the Mann-Whitney *U* test was used for comparisons between groups (RB vs RS). For all analyses, significance was assigned to $P < .05$.

TABLE 1. 2 × 2 × 3 ANOVAs for EWs

	df	F	*P*
Night	1	18.29	***<.001***
Group	1	0.09	.763
Sleep stage	1.86*	66.40	***<.001***
3–4 vs 1–2 and REM	1	108.01	***<.001***
1–2 vs REM	1	13.33	***.001***
Night vs group	1	0.05	.828
Night vs stage	1.69*	14.01	***<.001***
3–4 vs 1–2 and REM	1	23.70	***<.001***
1–2 vs REM	1	8.81	***.006***
Group vs stage	1.86*	0.56	.561
3–4 vs 1–2 and REM	1	0.66	.423
1–2 vs REM	1	0.44	.510
Night vs group vs stage	1.69*	1.45	.244
3–4 vs 1–2 and REM	1	0.03	.847
1–2 vs REM	1	2.21	.147

* Corrected for sphericity. Night = laboratory condition; Group = routine sleeping arrangement. Significant *P* values are in bold italics.

TABLE 2. 2 × 2 × 3 ANOVAs for TAs

	df	F	*P*
Night	1	0.16	.690
Group	1	3.88	.057
Sleep stage	1.9*	122.68	***<.001***
3–4 vs 1–2 and REM	1	225.26	***<.001***
1–2 vs REM	1	1.69	.203
Night vs group	1	2.02	.165
Night vs stage	1.67*	0.04	.938
3–4 vs 1–2 and REM	1	0.17	.685
1–2 vs REM	1	<0.01	.964
Group vs stage	1.9*	0.81	.443
3–4 vs 1–2 and REM	1	1.50	.230
1–2 vs REM	1	<0.01	.953
Night vs group vs stage	1.67*	1.71	.195
3–4 vs 1–2 and REM	1	6.39	***.016***
1–2 vs REM	1	0.27	.605

* Corrected for sphericity. Night = laboratory condition; Group = routine sleeping arrangement. Significant *P* values are in bold italics.

RESULTS

For all the analyses performed, only that portion of an infant's recording that coincided with the mother's time in bed each night was used to equalize the recording samples in the two recording conditions and to control for time of night effects. Collapsing across the two groups (RB and RS), the resulting infant recording times were 467.1 ± 8.1 (SEM) minutes on the BN versus 461.1 ± 8.6 minutes on the SN, a nonsignificant difference ($P > .05$).

Arousal Frequencies

The results of the 2 × 2 × 3 ANOVAs for frequency of EWs and TAs are presented in Tables 1 and 2, respectively. To help interpret the ANOVA tables, the means (± SEM) under each combination of factors for EWs and TAs are plotted in Fig 1. Although not part of the analyses, total arousals per hour (sum of EW and TA frequencies) also are plotted in the bottom panel of Fig 1. The most striking feature of the plots is the lower frequency of arousals in stage 3–4 compared with either of the other two sleep stages. Figure 1 presents the mean of each combination of the two laboratory conditions (BN and SN) and two routine sleeping arrangements (RB and RS) for each stage of sleep. Averaging these four means for each sleep stage, infants averaged 2.9/hour TAs in stage 3–4 compared with 18.0/hour in stage 1–2 and 19.4/hour in stage REM. For EWs, the infants averaged 1.9/hour in stage 3–4 compared with 5.1/hour in stage 1–2 and 6.5/hour in stage REM. As shown in Tables 1 and 2, for both EWs and TAs, this difference in the means was reflected in a highly significant main effect for sleep stage ($P < .001$ for each outcome measure). Furthermore, planned comparisons indicated that, for both EWs and TAs, the mean frequency in stage 3–4 was significantly less than the combined mean obtained from the other two stages ($P < .001$). A second planned comparison for each outcome measure indicated that stage REM exhibited a significantly higher frequency of EWs than did stage 1–2 ($P = .001$), whereas TA frequency was not significantly different in these two stages.

For EW frequency, a significant main effect was found also for laboratory condition (night effect, $P < .001$) with a highly significant interaction with sleep stage ($P < .001$) (Table 1). Collapsing across the two other factors (routine sleeping arrangement and

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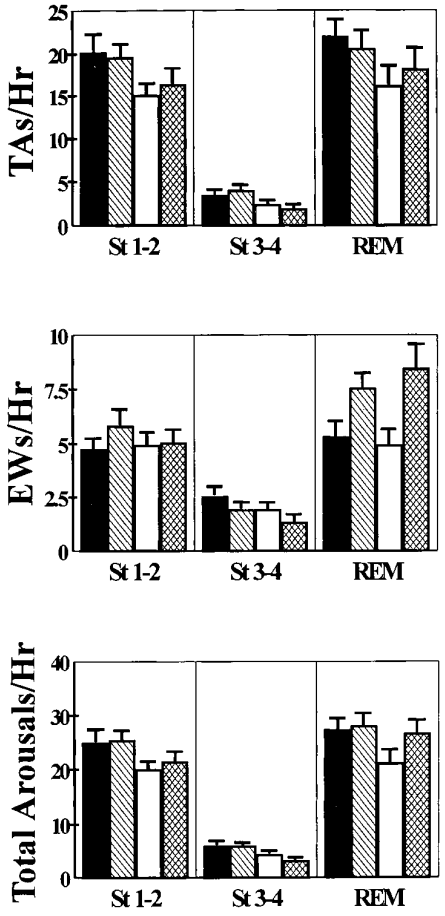

Fig 1. Mean (± SEM) frequencies of TAs (top), EWs (middle), and total arousals (bottom) are plotted separately for each stage of sleep as a function of infant group (RS or RS) and laboratory condition (BN or SN). Results of statistical comparisons are given in Tables 1 through 3.

sleep stage), infants averaged overall 4.1/hour EWs on the BN compared with 5.0/hour on the SN. The two planned comparisons for the interaction of laboratory condition with sleep stage revealed significant interactions effects for both (Table 1). The plotted means in Fig 1 show the nature of the effect of sleep stage on the effect of the laboratory condition. EWs appeared more frequent on the SN than on the BN for stages 1–2 and REM, but for stage 3–4 the opposite was true—EWs appeared more frequent on the BN. The plot also indicates that the result of the planned comparison of stage 1–2 versus REM is explained by a larger effect of laboratory condition for stage REM. The relationships within each stage of sleep were further clarified by the results from the 2 × 2 ANOVAs presented in Table 3.

For TA frequency, other than the main effect for sleep stage, the only other significant outcome of the 2 × 2 × 3 analysis was the 3-way interaction between laboratory condition, routine sleeping arrangement and sleep stage for the term stage 3–4 versus 1–2 and REM (P = .016) (Table 2). This indicated that the relationship between laboratory condition and routine sleeping arrangement was contingent on sleep stage. Also, the main effect of routine sleeping arrangement (group) approached a significant level (P = .057), and this reflected a trend toward more frequent TAs in the RB group that is evident in Fig 1.

The significant 3-way interaction effect for the TAs suggested that separate analyses be conducted for each sleep stage to determine the effects of laboratory condition and routine sleeping arrangement. The results of these 2 × 2 ANOVAs are presented in Table 3. For stage 1–2, there were no significant night, group or interaction effects on frequency of either EWs or TAs. For stage 3–4, in contrast, TAs were significantly more frequent in the RB group than the RS group, regardless of laboratory condition (P = .022). Across the two laboratory conditions, TAs were on average 1.6/hour (or 75.6%) more frequent in the RB group compared with the RS group. Furthermore, in stage 3–4 EWs were significantly more frequent on the BN than on the SN, irrespective of routine sleeping arrangement (P = .014). Combining the two groups, in stage 3–4 infants averaged 0.6/hour (or 37.5%) more EWs on the BN compared with the SN. The reverse was true for stage REM in which EWs were on average more frequent by 2.8/hour (or 54.9%) on the SN compared with the BN (P < .001). These opposite effects of laboratory condition on EW frequency in stages 3–4 and REM explain the signif-

TABLE 3. 2 × 2 ANOVAs for EWs and TAs

		BN	SN	Group *P* Value	Night *P* Value	Interaction *P* Value
Stage 1–2						
EWs (/h)	RB	4.7 ± 0.5	5.8 ± 0.7			
	RS	4.9 ± 0.6	5.0 ± 0.6	.766	.153	.256
TAs (/h)	RB	20.1 ± 2.0	19.5 ± 1.6			
	RS	15.0 ± 1.2	16.3 ± 1.8	.067	.743	.368
Stage 3–4						
EWs (/h)	RB	2.5 ± 0.4	1.9 ± 0.3			
	RS	1.9 ± 0.3	1.3 ± 0.3	.127	***.014***	.993
TAs (/h)	RB	3.3 ± 0.6	3.9 ± 0.6			
	RS	2.3 ± 0.5	1.8 ± 0.4	***.022***	.996	.230
Stage REM						
EWs (/h)	RB	5.3 ± 0.7	7.5 ± 0.7			
	RS	4.9 ± 0.7	8.4 ± 1.1	.778	***<.001***	.297
TAs (/h)	RB	21.9 ± 1.8	20.5 ± 2.1			
	RS	16.1 ± 2.3	18.1 ± 2.5	.168	.796	.102

Entries reflect means (±SEM). ANOVA results are given in the three columns on the right. Group = routine sleeping arrangement; Night = laboratory condition. Significant *P* values are in bold italics.

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icant interaction effect between laboratory condition and sleep stage (night vs stage) in the 2 × 2 × 3 analysis (Table 1). For TAs in REM sleep, the frequency did not vary significantly as a function of laboratory condition or infant group.

Overlap With Maternal Arousals

EWs and TAs were not differentiated in the analysis of temporal overlap of infant with maternal arousals. The number of overlapping arousals is graphed in Fig 2 for each group separately on both the BN and SN, and the results of nonparametric test comparisons within and between groups are given in Table 4. For both RB and RS infants, the number of overlapping arousals was roughly doubled on the BN compared with the SN, a highly significant difference for both groups. Combining RB and RS infants, the fraction of infant arousals that overlapped one or more maternal arousals averaged 46.4% on the BN compared with 23.9% on the SN.

For RB infants, further within-group comparisons of the BN and SN revealed highly significant increases on the BN in all three categories of arousals: those where the mother aroused first, where the infant aroused first, and where they appeared simultaneous. However, by far the largest magnitude increase was in the number where the infant aroused first (Fig 3). On average, RB infants exhibited 25.3 more such arousals on the BN. In contrast, the number where the mother aroused first or the arousals appeared simultaneous was on average greater on the BN by 6.3 and 6.0, respectively. Compared with RB infants, the RS infants exhibited the same general pattern of arousal overlap in the two laboratory conditions with two exceptions: the magnitude of the difference between the BN and SN was less for all three categories of overlapping arousals, and the number where the mother aroused first was not significantly higher on the BN. Regardless of which partner aroused first, it is noteworthy that the largest magnitude differences in overlapping arousals were seen consistently in the comparisons of the two groups in their routine sleeping conditions (RB on BN vs RS on SN; Fig 2).

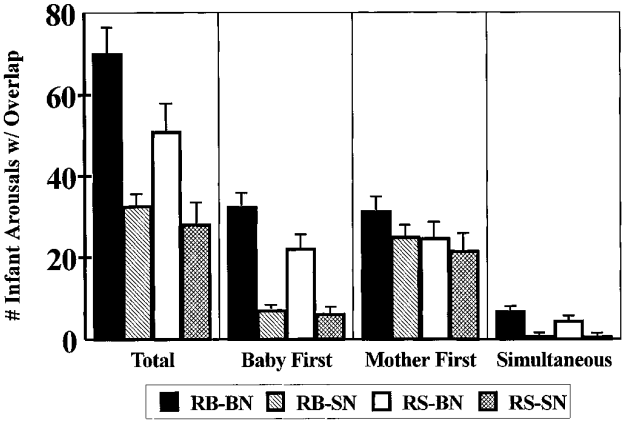

Fig 2. Mean (± SEM) number of infant arousals overlapping maternal arousals is graphed by infant group (RB or RS) and by laboratory condition (BN or SN). Results of statistical comparisons are in Table 4.

TABLE 4. Overlapping Arousals

	RB BN vs SN	RS BN vs SN	RB/BN vs RS/SN
No. overlapping arousals	***<.001***	***.004***	***<.001***
No. mother first	***.018***	.173	***.025***
No. infant first	***<.001***	***.001***	***<.001***
No. simultaneous	***<.001***	***.002***	***<.001***

P values for within-group (Wilcoxon matched pairs signed-ranks tests) and between-group (Mann Whitney *U* tests) comparisons of BN and SN. Significant findings are in bold italics.

DISCUSSION

These results demonstrate that there is a striking paucity of both EWs and TAs from stage 3–4 sleep, compared with either stage 1–2 or REM, in healthy infants within the peak age range for SIDS. Together with previous evidence that arousal threshold is relatively high in the EEG delta range,[23–25] this supports the premise that infant arousability is comparatively diminished in stage 3–4. This also further legitimizes our primary interest in any changes in stage 3–4 associated with bed sharing, as might pertain to the hypothesized role of arousal deficiency in susceptibility to SIDS.

In our mother-infant pairs, bed sharing promoted both more infant EWs and TAs selectively in stage 3–4. However, bed sharing's effects on EWs and TAs in this stage were somewhat different. EWs were more frequent on the BN, irrespective of whether infants routinely bedshared at home or not. TAs, in contrast, were more frequent in RB infants than RS infants, and this was observed in both the bed sharing and solitary sleeping conditions. This suggests that the bed sharing environment had a facilitory effect on infant EWs in stage 3–4 related to the mother's immediate presence. By contrast, bed sharing's facilitation of TAs depended on bed sharing being habitual but did not require the mother's presence for expression. Certainly a novelty effect (exposure to a new environment) does not explain either of these selective state 3–4 effects—the impact of the bed sharing night on EW frequency was seen in both infant groups (RB and RS), and the impact of routine bed sharing on TA frequency was seen in both laboratory conditions (BN and SN). This suggests involvement of other factors inherent to bed sharing, such as increased sensory stimulation involving potentially every sensory modality.

Although EWs were more frequent in stage 3–4 on the BN, the net effect across the entire night was that EW frequency was overall slightly lower on the BN compared with the SN by an average of 0.9/hour. This was because EWs were less frequent in stage REM on the BN. These opposite effects of bed sharing on EW frequency for stages 3–4 and REM, together with the absence of any significant night or group effects on either EW or TA frequency in stage 1–2 or on TA frequency in stage REM, emphasize further the selectively of bed sharing's facilitation of arousals in stage 3–4 sleep.

Meaningful comparisons of our findings with previous studies of infant arousal patterns are limited by important differences in aims, technology, and crite-

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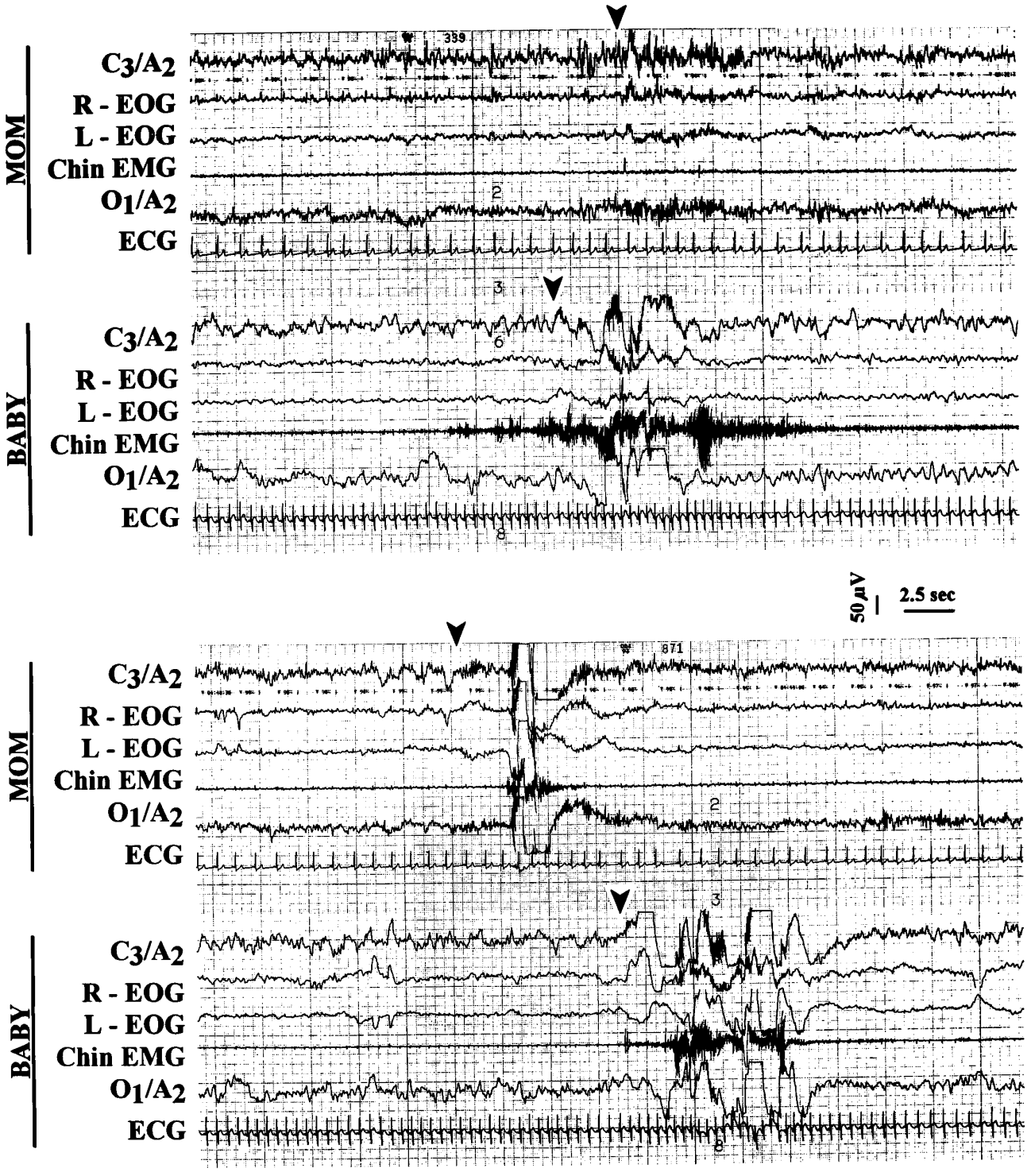

Fig 3. Example polysmonographic tracings of overlapping transient arousals in one RB mother-infant pair. Arrows indicate arousal onsets. Top: TA in infant preceding TA in mother, both in stage 1–2 sleep. Bottom: TA in mother preceding TA in infant, both in stage REM sleep. The respiratory channels have been removed from the tracings.

ria used to identify arousals/awakenings. As early as 1957, Moore and Ecko[42] quantified "night waking" in infants, defined by the infant signaling the mother. It was not until two decades later, using in-home video recording, that it was demonstrated that infants typically exhibit additional behavioral awakenings during the night without waking their parents.[43] Since then, numerous polysomnographic studies that included EEG sleep-wake staging have been performed in normal infants within the first postnatal year, as well as in SIDS victims and in infants at high-risk for SIDS. Some of these studies indexed arousals by body movements and reported more frequent movements in REM sleep than non–rapid eye movement (NREM) sleep.[5,8,44] Furthermore, Hoppenbrouwers et al[10] measured state transition probabilities based on sleep-wake staging and reported that infants are more likely to transition to the

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waking stage from active sleep (analogue of stage REM) than from quiet sleep (analogue of stage 3–4). The findings of these polysomnographic studies are in general agreement with the sleep stage differences in frequencies of EWs and TAs reported here. However, EEG arousals based on the standardized criteria more recently developed by the American Sleep Disorders Association[41] that we used have not been applied previously in any infant studies. In addition, we are aware of no studies designed to contrast arousals/awakenings in bed sharing and solitary sleeping infants using any technology or criteria for arousals/awakenings. In fact, the only preexisting data available that shed any light on the impact of bed sharing on infant awakenings were based on simple parental interviews. Singh et al[45] found that awakenings requiring the parent to resettle the infant were reported more commonly among their sample of east Indian infants, all of whom bedshared with a parent or grand parent, compared with the solitary sleeping infants represented in previous studies of night-waking.

The American Sleep Disorders Association criteria for identifying transient arousals[41] have been applied recently to healthy older children and adults by Acebo et al.[46] In their study, however, arousals were not broken down by sleep stage. The TAs (≤15 seconds) they reported were comparable to the TAs we measured. Collapsing across sleep stages in our subjects allowed recalculation of TAs as overall frequency per hour of total sleep. When the two study groups and two laboratory conditions also were collapsed, we found that the infants in our study averaged 14.7 TAs per hour of sleep. Contrasted with the means ranging from 3.2/hour to 5.3/hour reported in older children and adults by Acebo et al,[46] this indicates that short-lived EEG arousals are generally far more common in infancy.

There are two important implications of the augmentation of arousals with bed sharing demonstrated in our subjects. The first concerns the relationship of normative values for infant sleep patterns to the sleep environment in which norms are established. Without exception, polysomnographic norms for infant sleep have been obtained in the solitary sleeping environment. This experimental design bias no doubt reflects the western cultural practice of solitary infant sleeping. Notwithstanding, most of the world's cultures still practice some form of parent-infant room sharing, including sharing the same bed or sleeping surface.[47] Even within the United States, bed sharing is not an uncommon practice, contrary to popular perception. For example, for infants and toddlers, frequent all-night or part-night bed sharing was reported in 19% of whites, 59% of blacks, and 26% of Hispanics in families sampled from New York City and Cleveland.[48,49] In a previous report of sleep architecture in the same 35 infants described herein, we revealed that the amount of stage 3–4 sleep and the duration of stage 3–4 episodes were significantly reduced on the bed sharing night compared with the solitary night, regardless of the infants' routine sleeping arrangement.[30] Together with the immediate effects of bed sharing on EW frequency and the long-term effects on TA frequency discussed presently, these findings demonstrate that normative values for infant sleep established in solitary sleeping infants are not necessarily representative of infants in social sleeping environments. Separate norms should be established.

The second implication concerns the etiology of and risk factors for SIDS. The peak age for SIDS corresponds to a developmental stage when infant sleep in the solitary environment is undergoing consolidation, the amount of quiet sleep is increasing, and sustained bouts of quiet sleep are lengthening.[7,8,10,17–22] Many SIDS researchers believe that arousal deficiency plays an important role in the etiology of SIDS (see beginning of article). If this is true, then manipulations or conditions that facilitate arousability might be protective against SIDS. This might be especially true for quiet sleep, given the ongoing consolidation process, the comparatively low rate of spontaneous arousals, and the relatively high arousal threshold in this stage. The curtailment of stage 3–4 sleep and the facilitation of TAs and EWs in this stage resulting from bed sharing might minimize the occurrence of long periods of consolidated sleep from which infants with deficient arousal mechanisms might have difficulty arousing in response to any potentially life-threatening condition. Furthermore, we have speculated that, during the critical period when infants are vulnerable to SIDS, bed sharing might insure a basal level of "practice" required for the integration or coordination of the neural mechanisms that underlie the arousal response.[28,31] The present finding that TA frequency was higher even on the solitary night in routinely bed sharing infants than in infants who routinely slept alone supports the notion that practice has a sustained impact on arousability.

We speculate further that there are other means through which bed sharing might be protective against SIDS. The one with perhaps the most face value concerns the proximity of the mother to the infant, which should enable her to more effectively monitor changes in the infant's status. We recently reported, in these same mother-infant pairs, that mothers aroused 30% more often when they bed shared than when they slept alone.[29] Furthermore, the present results demonstrated an approximate doubling on the bed sharing night in the temporal overlap of infant with maternal arousals. Given that the largest increase in overlapping arousals by far reflected instances where the infant aroused first, these findings imply a high level of responsivity on the mother's part to the infant that did not habituate with routine bed sharing. A high degree of maternal attentiveness is also strongly suggested by the close proximity and face-to-face orientation generally maintained by these mothers during bed sharing.[50] Another way that bed sharing might be protective against SIDS could be through facilitation of breastfeeding. Bed sharing significantly increased the frequency and total duration of nighttime breastfeeding in our subjects, whether or not they were routine bed sharers.[51] Several epidemiologic studies have found that breastfeeding reduces the risk for SIDS.[52–55] By

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facilitating breastfeeding, bed sharing might confer some degree of risk reduction. Also, prone sleeping is now widely accepted as a risk factor for SIDS.[56] That prone positioning was minimized in our subjects when they bedshared[50] suggests another avenue through which bed sharing might be protective.

The authors are careful to point out that any potential benefits of bed sharing as pertains to susceptibility to SIDS are theoretical at this time. In fact, in a recent epidemiologic study from New Zealand, bed sharing (defined as bed sharing with anyone) was found to increase significantly the risk for SIDS when practiced in association with maternal smoking.[57] A subsequent epidemiologic study in southern California[58] failed to find increased risk for SIDS associated with similarly defined bed sharing when passive smoking was controlled for in the analysis; and a recent epidemiologic study in the United Kingdom[59] found that bed sharing with a parent(s) was associated with increased risk only in conjunction with parental smoking. It is also noteworthy that the New Zealand study found that room sharing (as opposed to bed sharing) with one or more adults conferred significant protection against SIDS, whereas room sharing with children did not.[60] This suggests that, at least under some circumstances, proximity to the parent(s) during sleep may be protective, as we are proposing. Further epidemiologic studies that additionally control for potentially important factors that could affect how bed sharing impacts infants ultimately will be needed to define in what contexts bed sharing (or room sharing) might be benefical or detrimental to infants with regard to SIDS risk. Such factors include the relationship of the bed partner to the infant, cultural differences in attitudes toward bed sharing with infants,[38,61] individual differences in reasons for bed sharing, differences in parental attitudes about responding to and physical contact with the infant during the day as well as at night,[62] and the type of surface used for bed sharing.

As this is the first study to measure the impact of bed sharing on infant sleep and arousals, appreciation of its limitations is especially important. Some have been discussed in detail previously[29,30] and include the small bed size used for bed sharing and limitations on the extent to which the results would generalize to other subject populations. It is unlikely that a passive effect of bed size alone explains the facilitation of infant arousals with bed sharing, given (a) the close face-to-face proximity maintained by mothers,[50] (b) the observations that mothers typically managed the infant's position relative to her during periods of breastfeeding, often actively enclosing the infant within their arms (unpublished observations), and (c) that mother-infant bed sharing in a single bed (or couch of similar size) is probably not an uncommon occurrence in the population we sampled. Further studies in other populations will be required to determine the extent to which our findings generalize to nonbreastfeeding mothers, to other cultural groups and to bed sharing with the father or other family members.

Another limitation stems from the absence of information on the architecture of daytime sleep in the infants we studied. Conceivably, more bed sharing-induced arousals might undermine infant arousability in the daytime by causing a "rebound" reduction of arousals during daytime solitary sleep. Such a rebound seems unlikely, however, at least for TAs insofar as that TAs were facilitated in RB infants when the mother was absent on the SN.

In conclusion, stage 3–4 sleep in infants was associated with a striking paucity of EWs and TAs, compared with the other sleep stages. Bed sharing increased the frequency of EWs from stage 3–4 sleep, whether or not infants habitually bedshared at home. Infants who routinely bedshared also exhibited more frequent TAs in stage 3–4, whether or not the mother was present on a given night. There was also an approximate doubling during bed sharing in the number of infant arousals that temporally overlapped arousal in the mother. The largest increase in overlapping arousals reflected those where the infant aroused first, suggesting that mothers maintained a high degree of sensitivity to their infants during bed sharing, even when practiced habitually. These differences between bed sharing and solitary sleeping infants indicate that normative values for infant sleep must be interpreted within the context of the sleeping environment in which they were established. We speculate that the selective facilitation of infant arousals in stage 3–4 sleep might be protective to infants at risk for SIDS because of an arousal deficit. Enhanced maternal sensitivity during bed sharing to changes in the infant's status might also be protective.

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Sarah Mosko, Christopher Richard and James McKenna

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