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Parent–Infant Cosleeping: The Appropriate Context for the Study of Infant Sleep and Implications for Sudden Infant Death Syndrome (SIDS) Research

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Nearly all laboratory research on human infant sleep assumes that solitary sleeping is the normal and desirable environment. Yet solitary sleeping in infancy is a very recent custom limited to Western industrialized societies, and most of the world's peoples still practice parent–infant cosleeping. A hypothesis is presented that cosleeping provides a sensory-rich environment which is the more appropriate environment in which to study normal infant sleep. In addition, two preliminary, in-laboratory, polygraphic investigations of mother–infant cosleeping are reported in normal infants within the peak age range for sudden infant death syndrome (SIDS). Five mother–infant pairs coslept for 1 night in the first study; in the second, three additional pairs slept separately for 2 nights and coslept the third consecutive night. The results suggest that cosleeping is associated with enhanced infant arousals and striking temporal overlap in infant and maternal arousals. Infant sleep also showed subtle alterations with cosleeping, as manifested in increased overlap with corresponding maternal sleep stages and decreased amount of Stage 3–4. These are the first in-laboratory investigations of parent–infant cosleeping. The implications of the hypothesis and preliminary results for research on the normal development of infant sleep and on SIDS are discussed.

KEY WORDS: infant; parent–infant cosleeping; sudden infant death syndrome (SIDS).

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INTRODUCTION

Any research is limited by the validity of its underlying assumptions. Research on human infant sleep has been based almost exclusively on the assumption that solitary sleeping is the appropriate environment in which to appreciate normal infant sleep. The same is true for sleep studies aimed at understanding the sudden infant death syndrome (SIDS). The assumption, however, disregards two important facts: cosleeping with a parent (or caretaker) is the context in which the neurological and physiological systems which control infant sleep evolved; and cosleeping remains today the norm for approximately 90% of the world's population. Only Western industrialized societies conceptualize solitary infant sleep as a normal and desirable sleeping arrangement (Lozoff and Brittenham, 1979; Lozoff *et al.*, 1984; Whiting, 1963; Konner, 1981).

Data supporting the position that parent–infant cosleeping is the more normal or natural environment of infant sleep come from cross-species, evolutionary, and cross-cultural studies that have been reviewed recently (McKenna, 1986; McKenna and Mosko, 1990; McKenna *et al.*, 1990). Our closest living primate relatives, the chimpanzees and gorillas, not only cosleep for the first few years of life, but also remain in almost continual, direct physical contact with a caregiver day and night throughout this period. Unlike human infants, chimpanzee and gorilla infants are able to cling and remain attached to the caregiver's body without assistance (Konner, 1981; Goodall, 1986). But as a result of concomitant brain enlargement and reduction in the size of the pelvic opening caused by bipedalism, humans are born more immature than other primates. For example, chimpanzees experience 55% of brain development after birth, compared with more than 75% for humans. Consequently, human infants are unable to cling or ambulate, causing them to be even more dependent on the caretaker to provide for their basic needs, e.g., protection, nourishment, transportation, and sensory stimulation (Trevathan, 1987; Lancaster and Lancaster, 1983). Paleoanthropologists and archaeologists have concluded that our human ancestors fulfilled these infant needs by carrying the infant, possibly in slings, during the day and by cosleeping at night, as is still done in present day hunting and gathering societies and nonindustrialized cultures (Lozoff and Brittenham, 1979; Whiting, 1963; Bowlby, 1969; DeVore and Konner, 1974; Konner and Worthman, 1980). In fact, solitary sleeping in infancy has been practiced by humans for only about 200 years, and this follows at least 2 to 5 million years of human parent–infant cosleeping (Konner, 1981; McKenna, 1986; Thevenin, 1976). Surveys of contemporary infant sleeping practices around the world reveal that approximately two-thirds of the world's cultures habitually practice mother–infant cosleeping on the same bed or sleeping surface, and the fraction is much higher if the definition of cosleeping is extended to include sleeping in the same room (Burton and Whiting, 1961; Barry and Paxson, 1971). Cosleeping is still the common practice of all Asian cultures today, including urbanized industrial Japan (Caudill and Plath, 1966; Valsiner, 1989). Thus solitary sleeping in infancy is a very recent and mostly Western custom.

Our preliminary research on parent–infant cosleeping assumes that cosleeping is the normal or natural and hence more appropriate environment in which to study infant sleep. Given the human infant's immaturity at birth and the fact that infant sleep evolved in the context of cosleeping, it is only logical to ask what could be the impact on and possible benefit to the infant of cosleeping with a caregiver. Aside from better protection from environmental threats, by necessity cosleeping would also provide a far more rich and varied sensory experience than solitary sleeping. Any bedpartner would inadvertently provide stimulation through vocalizations, radiant heat, changes in body position, respiratory sounds and movements, odors, and perhaps expired gases. When the bedpartner is a caregiver, there would additionally be more intentional stimulation stemming from the role of caregiver, e.g., gestures to check on and comfort the infant.

Our basic hypothesis is that the sensory environment of cosleeping is somehow adaptive. We suggest that cosleeping provides important stimulation which, through evolution, the infant has come to expect and to which it has been prepared to respond physiologically, psychologically, and socially. The only data available on how infants are impacted by cosleeping comes from contemporary hunting and gathering peoples in Africa and urban La Leche League mothers in the United States, both of whom habitually cosleep as well as breast-feed. In contrast to Western industrial society, where frequent nocturnal awakenings after 4 or 5 months of age can be considered abnormal (Anders and Keener, 1985; Hoppenbrouwers *et al.*, 1988; Parmelee *et al.*, 1984) and certainly undesirable, these cosleeping infants arouse frequently to suckle and may not sleep through the night until after the first year or two (Konner and Worthman, 1980; Elias *et al.*, 1986; Super and Harkness, 1982). Despite the appeal to Western parents, the accelerated appearance of consolidated sleep may not be adaptive for the infant, as is implied from recent studies on the role of arousal deficit in the pathogenesis of SIDS (see Discussion).

The idea that cosleeping provides important sensory experience could have significant implications for present-day hypotheses on the mechanisms of SIDS. Most contemporary hypotheses involve defects in arousal, cardiorespiratory control, or maturation (see Schwartz *et al.*, 1988; Guntheroth, 1989). Although no consensus has yet been reached by researchers on the specific mechanisms of SIDS, varied histories and pathological findings in SIDS victims have led to a consensus that these infants represent a heterogeneous group and that the causes of SIDS are multifactorial, whatever the actual terminal mechanism(s) (Willinger, 1989; Ariagno and Glotzbach, 1991; Schwartz and Sagantini, 1988). This has led to two recent research trends: interest in possible environmental factors that might conspire with predisposing constitutional deficits or vulnerabilities to promote the expression of SIDS and appreciation that furthering the understanding of the causation of SIDS will require exploration of the interaction of the suspected mechanisms. Our hypothesis is consonant with both these trends. It focuses on experiential factors that may conspire with the range of SIDS deficits to increase risk. Furthermore, if cosleeping is found to alter the infant's sleep experience significantly, then the question is raised whether these alterations could play a role in the prevention of some subclasses of SIDS. Pertinent to this are some studies which have reported lower rates of SIDS in societies where cosleeping is common practice, although other contributing factors to these statistics also need to be considered (Takeda, 1987; Tasaki *et al.*, 1988; Davies, 1985; Lee *et al.*, 1989).

As a starting point to test the hypothesis that cosleeping provides sensory experience important to infants, we have begun to look for measurable effects of mother–infant cosleeping on conventional measures of infant sleep. Two pilot studies are described below which are intended to illustrate the potential value to the study of infant sleep of assuming that cosleeping is the more normal environment. Both studies were performed in healthy infants 2.5–4.5 months of age because this is within the peak age for SIDS, although these infants were not thought to be at increased risk. Our logic is that if cosleeping is adaptive, then the normal developing nervous system should be responsive to the sensory experience of cosleeping. If so, then findings in normal infants could be relevant to hypotheses on SIDS causation and prevention.

METHODS

Study 1

Five healthy mothers (32–37 years old) with normal pregnancies and deliveries and their full-term infants (three males and two females, 2.5–4.5 months old) shared a single-sized bed during 1 night of polysomnography in the laboratory. The mothers were screened beforehand for history of sleep disorders and drug or alcohol abuse. All were nonsmokers. The infants had normal developmental histories, with no incidences of an apparent life-threatening event and no siblings with an apparent life-threatening event or SIDS. Mothers and infants were Caucasian and none of the pairs routinely coslept at home. Mothers were kept blind to the hypothesis being tested and informed only that the effects of cosleeping on infant sleep were being measured. Although not an inclusion criterion, all of the infants had been weaned prior to the study. Infants were retired at their normal bedtimes and fed as usual beforehand and during the night. Mothers performed all caretaker interventions themselves. They retired later, near their regular bedtimes, and were not instructed on how to position themselves or their infants in bed. Recordings were begun when the mother retired and terminated the following morning after both mother and infant had awakened. EEG (C3/A2), EOG, chin EMG, EKG, and respiratory effort (chest strain gauge) were recorded in both mothers and infants, and all 10 channels were written out simultaneously on a single polygraph.

Recordings were scored for sleep stages using the Rechtschaffen and Kales (Rechtschaffen and Kales, 1968) adult system for the mothers and the system for 3 month olds developed by Guilleminault and Souquet (Guilleminault and Souquet, 1979) for the infants. Both systems assign to each 30-sec recording epoch the predominant stage of sleep or wakefulness, and these are totaled to determine the total sleep time, the amount of each stage, and the sleep efficiency (ratio of total sleep time to total recording time). The systems differ by a higher voltage criterion for slow-wave sleep and the identification of only three sleep stages in infants: Stages 1–2, 3–4, and REM. For comparability to infant sleep stages, Stages 1 and 2 in the adult were combined, as were stages 3 and 4.

The epochal system of stage scoring does not identify short awakenings which span less than 50% of an epoch. Because of our interest in all arousal phenomenon in sleep, we quantified both "epochal awakenings" (EWs), which are identified by the stage scoring systems, and subepochal or transient arousals (TAs), which are not. TAs were scored by a modification of the criterion published by Carskadon *et al.* (1982). They defined a TA as any clearly visible EEG arousal lasting ≥ 2 sec but not associated with any stage change in epochal scoring. We omitted the latter exclusion criterion to allow recognition of all short-lived arousals not scored as EWs, irrespective of whether there is return to the same or a different sleep stage. Although it was not a requirement, by far the majority of TAs in infants and mothers was accompanied by signs of physiological arousal on other channels, i.e., increased heart rate, burst of chin EMG activity, or change in pattern of respiration or EOG.

Study 2

In the second study, three healthy mothers (18–36 years old) and their full-term infants (two females and one male, 2.5–4 months old) underwent 3 consecutive nights of polysomnography. Two pairs were Latino and one was Caucasian. One of the Latino mothers was routinely cosleeping with her infant since birth: her decision to cosleep was based on personal choice. All other characteristics of the mothers and infants were as in Study 1, and mothers were again kept blind to the hypothesis being tested. Mothers and infants slept in separate rooms the first 2 nights and they shared a single-sized bed the third night. The first night was viewed as an adaptation night because "first night effects" on laboratory sleep have been measured in adults and infants which resolved by the second night (Bernstein *et al.*, 1973; Sostek and Anders, 1975; Agnew *et al.*, 1966). The second room used for the solitary nights was identical in size, layout and furnishings to that used for the cosleeping night. The rooms were adjacent and the doors between them left open so that mothers were in auditory contacts with their infants and able to perform all caretaker interventions on an ad lib basis. The same physiological variables were monitored as in Study 1 with the additional measurement of airflow (combined oronasal thermocouple).

Mothers in both Study 1 and Study 2 were asked each morning to fill out a short questionnaire which asked whether their own and their infant's sleep were typical of their usual patterns at home. In all cases, mothers responded that the laboratory sleep was typical for both themselves and their infants.

RESULTS

Study 1

Since there was no control (solitary sleeping) condition in this study for comparison, other approaches were developed to ascertain whether cosleeping altered sleep pattern. Using the epochal scoring system, we computed for each mother–infant pair the percentage of their total recording time spent simultaneously in the same stage of sleep or wakefulness. These times of corresponding sleep–wake stages were called simultaneous activity time (SAT). SAT averaged $39 \pm 7\%$ for the five pairs (range, 27–44%). To determine what fraction of SAT could be attributable to chance or to an inherent organization of sleep shared by adults and infants, we also computed SATs obtained by pairing, on the same epoch-by-epoch basis, the recording of each infant with the recording of another, randomly matched

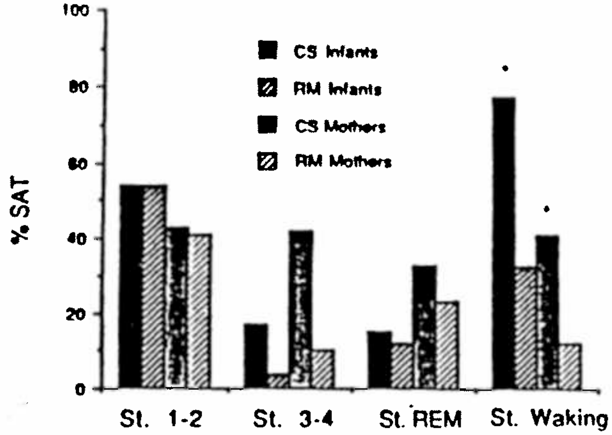

Fig. 1. Mean percentage SAT for individual sleep-wake stages in cosleeping (CS) and randomly matched (RM) mother-infant pairs. $n = 5$ for mothers and infants. For each sleep-wake stage, infant SAT was compared in the CS and RM conditions. Analogous comparisons were made for maternal SATs. (•) Significantly different from RM condition (paired t tests, $p < .01$–$.05$).

mother with whom the infant did not cosleep. The pairings began at the first epoch of each recording, and because total recording times in the randomly matched infants and mothers differed by no more than 13 min, at most this much recording time had to be eliminated from the end portion of any recording to compute SATs. The mean SAT of the five randomly matched pairs was 29 ± 5% (range, 23–34%). Compared to the randomly matched condition, the increase in SAT in the cosleeping condition was significant for the infants (paired t test, $p < .05$). The analogous increase in SAT for the mothers failed to reach significance ($p < .1$).

We also calculated SATs for individual sleep-wake stages in both cosleeping and randomly matched pairs to assess their contribution to the overall SAT (Fig. 1). For infants and mothers, mean % SAT was higher in the cosleeping condition for Stages 3–4, REM, and Waking, although the increase reached statistical significance only for Stage Waking in both infants (paired t test, $p < .05$) and mothers ($p < .01$).

Arousals were also quantified and investigated for temporal overlap in cosleepers. EWs were identified independently in mothers and infants and later categorized for each sleeper according to whether the cosleeper had an overlapping epoch(s) of waking, i.e., an overlapping EW. Infants averaged 20 ± 14 EWs (range, 2–37) and mothers averaged 34 ± 9 (range, 29–49). Of the 102 total EWs recorded in infants, 91 (or 89%) overlapped

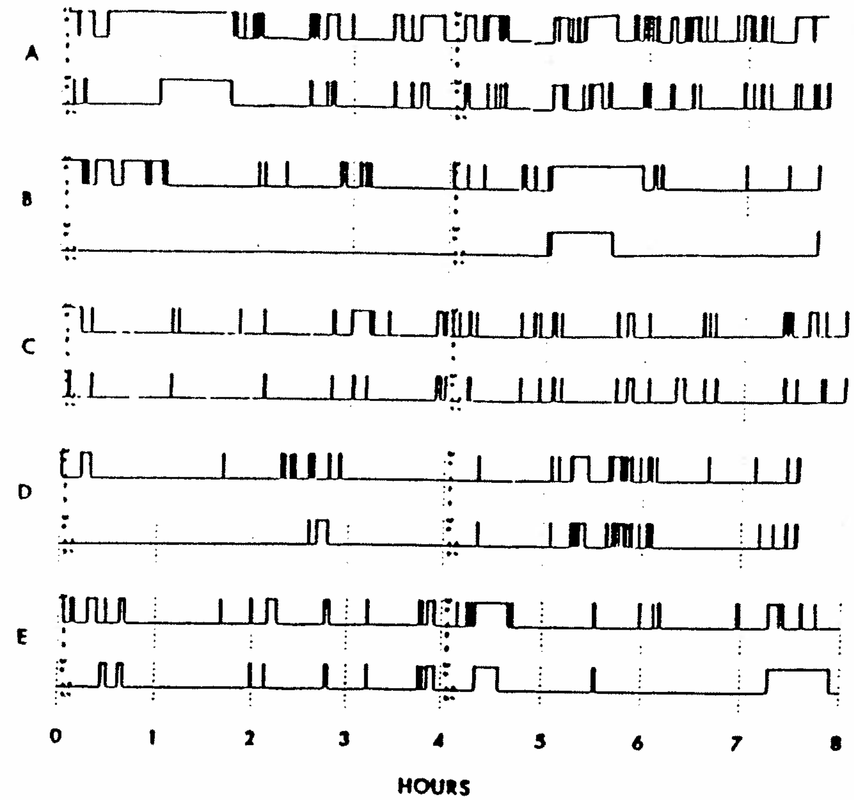

Fig. 2. All-night sleep-wake histograms for five (A–E) cosleeping pairs. In each pair, the top histogram is the mother and the bottom one is her infant. Each histogram has two positions: the top position designates Stage Waking; the bottom designates all stages of sleep since Stages 1–2, 3–4 and REM are collapsed together in the bottom position.

a maternal EW, and for 73% of those the awakenings took place in the same epoch. Of the 169 combined EWs in mothers, 78 (or 46%) overlapped an infant EW, and in 85% of these the awakenings occurred in the same epoch. Eliminating the one pair where the infant had only two EWs all night (both of which overlapped a maternal EW), 84–100% of individual infant's EWs overlapped an EW in the mother, and 50–58% of individual mother's EWs overlapped an infant EW. The high degree of overlap in EWs is also evident in Fig. 2, which shows the all night sleep-wake stage histograms for all five mother-infant pairs, collapsing across sleep stages.

To estimate the degree to which factors other than cosleeping produced these high rates of overlapping EWs, the five pairings described above of records from randomly matched mothers and infants were quantified in the same epoch-by-epoch way for overlapping EWs. Based on this, only 33% of all infant EWs overlapped a maternal EW, and just 15% of all maternal EWs overlapped an infant EW. Comparing the mean percentages of overlapping EWs in the cosleeping vs. randomly matched conditions, the increase with cosleeping was significant for both the infants (paired t test, $p < .01$) and the mothers ($p < .05$).

TAs also were identified independently in each mother and her infant and later categorized according to whether the cosleeper had a concurrent (±5-sec) TA or an overlapping epoch of waking. Cosleeping infants and mothers, respectively, averaged 78 ± 25 (range, 52–115) and 58 ± 12 (range, 28–76) TAs. Of 388 total infant TAs, 114 (or 29%) overlapped a maternal TA and an additional 142 (37%) overlapped a maternal EW. Similarly, 114 (39%) of 290 total maternal TAs overlapped an infant TA, and another 29 (10%) overlapped an infant EW. This means that 66% of all infant TAs overlapped some type of maternal arousal, and 49% of maternal TAs overlapped some type of infant arousal. For individual subjects this value ranged from 47 to 87% in infants and 18 to 63% in mothers. Because the onsets of the maternal and infant arousals appeared nearly simultaneous in many cases, it was not possible to determine reliably which cosleeper aroused first.

Study 2

Since only three subjects were tested, results were not subjected to statistical analysis.

Sleep pattern was similar on solitary sleeping Nights 1 and 2 for both infants and mothers, suggesting that a prominent first night effect was not present (Table I). Even though the usual meaning of the sleep variables is somewhat altered for the infants because they retired 1–3 hr before the start of the recording, there were no trends to suggest that infant sleep was more disturbed the first night. For mothers, REM latency may have been mildly prolonged on Night 1.

Compared to the values obtained on solitary nights, infant sleep pattern variables while cosleeping were similar, with three exceptions (Table I). The number of stage shifts per hour of sleep increased by an average of 29% compared to Nights 1 and 2, Stage 1–2 increased by 28%, and Stage 3–4 decreased by 47%. The increase in stage shifts reflects trends in only two of the infants since Infant 3 showed no change on the cosleeping night. Total sleep time was decreased by an average 39 min, but since sleep efficiency was not reduced this likely reflects the 40 min average shorter

Table I. Common Sleep Pattern Variables

	Night 1	Night 2	Night 3
Infants (n = 3)			
Total recording time (min)	465	434	410
Sleep latency (min)	<1	<1	3.5
REM latency (min)	<1	15	3.4
Waking after sleep onset (min)	54	34	46
Total sleep time (min)	407	395	362
Sleep efficiency	.89	.91	.89
% stage			
1–2	35	37	46[a]
3–4	38	30	18[a]
REM	28	30	32
REM cycle length (min)	56	66	53
No. stage shifts	89	89	104
Stage shifts/hour sleep	14	14	18
Mothers (n = 3)			
Total recording time	465	434	410
Sleep latency (min)	2	<1	6
REM latency (min)	94	64	92
Waking after sleep onset (min)	48	57	29
Total sleep time (min)	413	372	374
Sleep efficiency	.89	.85	.92
% Stage			
1–2	58	53	44[a]
3–4	24	29	35[a]
REM	19	19	20
No. stage shifts	60	56	75
Stage shifts/hour sleep	9	9	12

[a]n = 2 for % Stages 1–2 and 3–4 on Night 3 only because of a respiration artifact in EEG for one pair which obscured Stage 3–4.

recording time the third night. Mothers, too, showed more stage shifts per hour of sleep (33% increase). However, rather than a reduction in slow-wave sleep, % Stage 3–4 was, if anything, increased. That cosleeping produced little objective sleep disturbance is consistent with the morning-after reports from all three mothers that their laboratory sleep, including the cosleeping night, was representative of their usual sleep at home.

On solitary nights, SAT for individual mother–infant pairs ranged from 23 to 38%, with a group mean of 26%. On the cosleeping night the group average increased to 45%, with individual pairs ranging from 26 to 64%. Two of the pairs showed sizable increases in SAT, of 21 and 33%, while cosleeping, whereas Mother–Infant Pair 3 showed a negligible 3% increase.

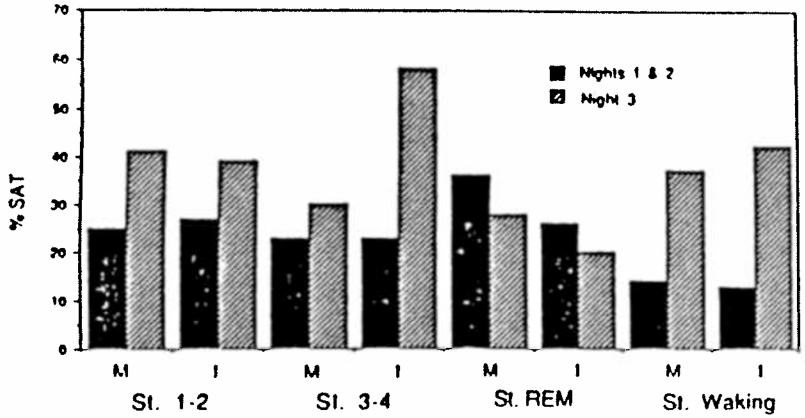

Fig. 3. Percentage SAT for individual sleep–wake stages for three mother–infant pairs, contrasting the average of solitary Nights 1 and 2 with cosleeping Night 3. M, mothers; I, infants.

As shown in Fig. 3, which graphs mean %SAT for individual sleep–wake stages on Nights 1 and 2 and the mean of Night 3, the pattern was the same for mothers and infants: %SAT increased for Stages 1–2 and 3–4 as well as Waking and decreased for Stage REM.

Arousals were expressed as a frequency score (per hour of sleep) in this study to allow for within-pair differences in nightly total sleep time. Also, arousal overlap was more simply defined for EWs and TAs as strict temporal overlap. There was considerable variation between infants in arousal frequency while solitary sleeping. First combining EWs and TAs, infants averaged 14.0/hr arousals across Nights 1 and 2 (range, 7.5–19.7/hr). These were comprised of 9.9/hr TAs (range, 5.9–14.6/hr) and 4.1/hr EWs (range, 1.6–5.6/hr). Mothers showed far less individual variation. Their combined EWs and TAs averaged 8.3/hr, with a range of 8.1–8.6/hr, and these were comprised of 5.4/hr TAs (range, 4.9–5.9/hr) and 2.9/hr EWs (range, 2.8–3.0/hr).

The pattern of arousals during cosleeping also varied between infants. The infant that routinely coslept at home showed the largest increase in arousal frequency (EWs plus TAs) of 65% compared to the mean of the solitary nights: EW frequency increased 113% and TA frequency increased 54%. The second infant showed an overall increase in arousal frequency of just 7%. This reflected, however, a 61% increase in EW frequency with a decrease in TA frequency of 26%, suggesting that arousals were prolonged rather than much more frequent. In Infant 3, there was a negligible (1%) increase in arousal frequency while cosleeping, and the changes in

EW and TA frequencies were also negligible. Since this infant had the highest arousal frequency every night, the possibility is raised of a ceiling effect.

Mothers, too, showed variable changes in arousal frequency while cosleeping. The mother who showed the greatest increase in arousal frequency (113%) also had more frequent EWs (132% increase) and TAs (98% increase). Another mother showed 62% more frequent arousals with 68% more frequent TAs but only 7% more frequent EWs. In the third mother, overall arousals increased minimally, by 5%, with a 20% increase in TA frequency and a 13% decrease in EW frequency. There was no apparent correspondence between the mothers and their infants in terms of change in arousal frequency with cosleeping, e.g., the mother of the infant who showed the greatest increase in arousals exhibited the smallest increase among the mothers.

Figure 4 graphs, for infants and mothers, the percentage of total arousals (EWs plus TAs) each night which overlapped an arousal of either type in the other dyad member. There was a striking increase on the cosleeping night for every infant and every mother. In contrast, the 2 solitary nights differed relatively little from one another, for infants or mothers. From Table II, which separates TAs and EWs, it can be seen that both types of arousals showed a large increase when cosleeping in overlap with arousals in the other dyad member.

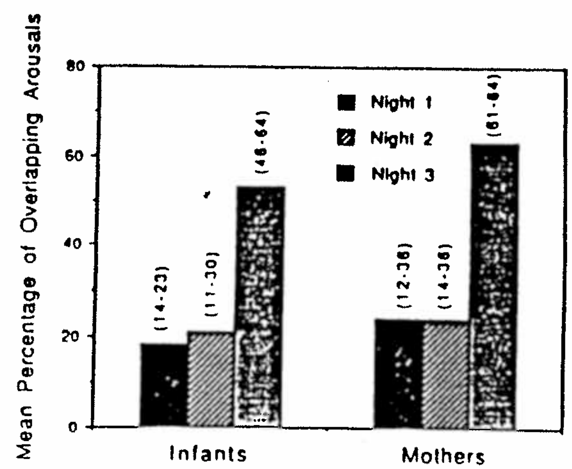

Fig. 4. Mean percentage of overlapping arousals (EWs and TAs combined), contrasting Nights 1 and 2 with cosleeping Night 3. Ranges for individual subjects are given in parentheses.

Table II. Percentage of TAs or EWs Which Overlap Arousal in Other Sleeper

	Night 1	Night 2	Night 3
Infants			
TAs	13	19	36
EWs	23	24	78
Mothers			
TAs	12	16	53
EWs	29	26	84

Table III. Infant Breathing Pauses (per Hour of Sleep)

	Night 1	Night 2	Night 3
Infant 1	33.5	24.0	21.0
Infant 2	11.0	15.4	10.8
Infant 3	—[a]	10.3	9.8

[a]Data unavailable because of a technical problem.

Infant breathing pauses or central apneas ≥2 sec were counted. Infants varied appreciably in apnea frequency, which ranged from 10.3 to 33.5/hr on solitary nights (Table III). The infant who routinely coslept at home (Infant 1) had the highest apnea frequency. All three infants showed a similar sleep stage effect, however, with the fewest apneas in Stage 3–4 (6–12%), compared to Stage 1–2 (20–53%) and Stage REM (37–70%). Also, apneas were more prolonged in Stage 3–4 for all infants on both solitary and cosleeping nights: collapsing across infants and across all 3 nights, apneas in Stage 3–4 averaged 5.6 sec, compared to 4.6 sec in Stage 1–2 and 3.6 sec in Stage REM. On the average, 39% of breathing pauses followed a large breath(s), often as the infant returned to Stage 1–2 or REM following an arousal (Fig. 5). Only 12% of breathing pauses ended in EEG arousal. As shown in Table III, cosleeping was not associated with a remarkable change in apnea frequency, and no appreciable change in apnea duration was detected.

DISCUSSION

These preliminary studies are the first in-laboratory investigations of infant sleep in the context of parent–infant cosleeping. Since the number of subjects studied was not large, the results are discussed as trends, and

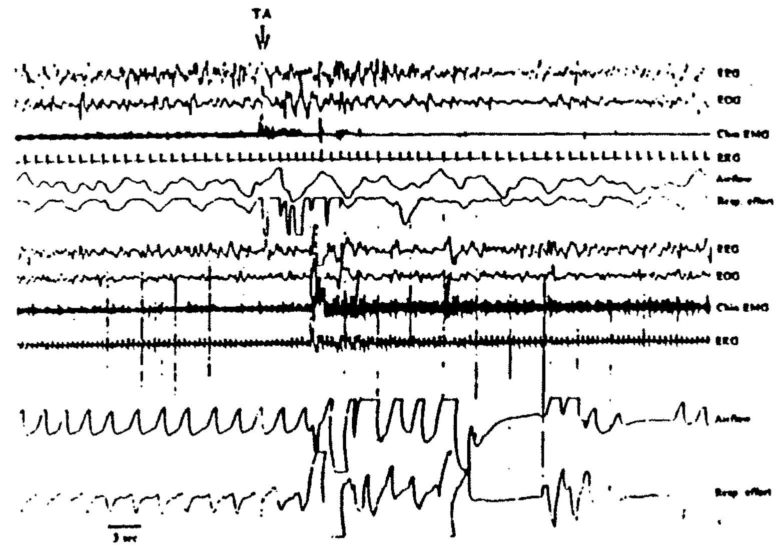

Fig. 5. Polygraphic recording showing a maternal TA followed and overlapped by an infant TA while cosleeping. Note the two brief breathing pauses which occur as the infant returns to sleep.

no firm conclusions are drawn about the specific effects of cosleeping; rather, this discussion is intended to illustrate the kinds of important issues that can be raised by acknowledging the cosleeping environment.

The most important findings suggested by these studies are that cosleeping infants tend to arouse more frequently and with far greater overlap with maternal arousals, which implies that many arousals are partner-induced. Also, infant sleep stages are altered by cosleeping as manifested in decreased Stage 3–4 and greater simultaneous overlap with maternal sleep–wake stages. It is possible that these effects of cosleeping are related in that more frequent and maternal-induced arousals could interfere with the development or maintenance of infant Stage 3–4 sleep. Also, overlapping arousals could secondarily produce greater overlap with maternal sleep stages since sleep generally lightens following arousals, and conversely a period of fewer arousals in one cosleeper could facilitate the development of deeper sleep in the other cosleeper. Except for the increase in arousal overlap with maternal arousals, these infant trends were not expressed equally in all infants. Since all but one of the mother–infant pairs studied did not routinely cosleep at home, the question is raised whether the cosleeping effects measured could reflect more the novelty of cosleeping than effects that would persist with habitual cosleeping. The fact that the one infant (In Study 2) who routinely coslept with its mother showed all of the above trends prominently and also had the largest increase in arousal frequency with cosleeping suggests that the effects of cosleeping do not habituate. Nevertheless, we propose that individual variation in infant sensitivity and habituation to cosleeping is to be expected and that sensitivity will probably also vary during development as the infant matures and dependence on the parent or caregiver diminishes.

If replicated in future studies, the trends we observed could challenge some of the conclusions about normal development that have emerged from studies of solitary sleeping infants. If cosleeping infants arouse more frequently, then the conclusion that achieving progressively longer periods of sustained sleep at night across the first several months is a normal developmental milestone (Anders and Keener, 1985; Hoppenbrouwers *et al.*, 1988; Parmelee *et al.*, 1984; Harper *et al.*, 1981b) and hence desirable or healthy for all infants may be more an artificial product of the solitary environment than an accurate representation of normal development. Solitary sleeping studies have also consistently measured a large increase in the time spent in Quiet Sleep (analogue of Stage 3–4) across the first several postnatal months (Anders and Keener, 1985; Hoppenbrouwers *et al.*, 1988). Our observations raise the question of whether this, too, could be an artifact of solitary sleeping. Longitudinal studies of infants cosleeping from birth will be needed to address these developmental issues.

An important question which emerges from our studies concerns the possible benefits to infants of arousing more frequently and also simultaneously with the mother and of having less Stage 3–4 or Quiet Sleep. One obvious short-term benefit of more frequent mutual arousals is that the infant has more opportunities to suckle. Human milk is low in fats and proteins and high in carbohydrates, which are metabolized relatively rapidly (Ben-Shaul, 1962), suggesting that infants are adapted to frequent or "continuous" feeding (Lozoff and Brittenham, 1979; Konner, 1981). Also obvious is that the mother's enhanced arousals together with her proximity to the infant increase the likelihood that she can detect when caretaker interventions are needed and take appropriate action. Less obvious potential benefits become evident when our findings are considered in the context of contemporary hypotheses about SIDS.

Prominent researchers have reported evidence that arousal deficit plays an important role in SIDS. Harper *et al.* (1981a) and Hoppenbrouwers *et al.* (1989) found that subsequent siblings of SIDS victims have comparatively longer periods of uninterrupted sleep, suggesting impaired ability to make the transition from sleep to wakefulness. Other researchers have reported less frequent spontaneous awakenings in near-miss SIDS infants (Kahn *et al.*, 1986; Challamel *et al.*, 1981; Coons and Guilleminault, 1985). Furthermore, deficient arousal responses during sleep to hypoxia or hypercarbia in near-miss SIDS infants and subsequent siblings of SIDS victims have been measured (McCulloch *et al.*, 1982; Brady *et al.*, 1985). These findings are important because arousal is thought to be integral to the infant's defense against some crises or life-threatening events in sleep, e.g., prolonged apnea or cardiac arrhythmia. More frequent arousals while cosleeping could percent the occurrence of long periods of uninterrupted sleep from which some infants might have difficulty arousing in response to a physiological crisis. Furthermore, since some arousals while cosleeping are clearly extraneous (partner-induced), one can speculate that cosleeping might even provide important "practice" for the neurological and physiological mechanisms which underlie the arousal response. We know that arousal is a complex event entailing not only characteristic EEG changes, but also changes in a variety of other physiological parameters including cardiac activity, respiratory drive, muscle tone, and reflexes. Daily "practice" of these various systems might strengthen, coordinate, or integrate them in important ways. One can speculate that such practice could be critical to some infants who, without a basal level of practice, would otherwise have difficulty arousing spontaneously to a crisis during the vulnerable phase of development when SIDS occurs. Importantly, the increase in arousal frequency with cosleeping did not reduce the overall amount of nocturnal sleep achieved or sleep efficiency in either the infant or mother. This is important since studies have shown that inducing severe sleep fragmentation decreases the arousal response (Bowes *et al.*, 1980).

The fact that developmental studies in solitary sleeping infants consistently find a large increase in time spent in Quiet Sleep during the months when infants are at highest risk for SIDS could be important to hypotheses about the role of arousal since arousal threshold is thought to be high in the delta portion of Quiet Sleep (Ashton, 1973; Williams *et al.*, 1964; Busby and Pivik, 1983). Our finding that cosleeping may reduce or limit the amount of Stage 3–4 suggests another way that the arousal response may be enhanced and some infants protected by cosleeping: cosleeping might prevent the premature emergence of large amounts of Quiet Sleep seen when infants sleep in isolation. Hoppenbrouwers *et al.* (1988, 1989) have shown in longitudinal studies that the longest duration of Quiet Sleep increases from 1 week to 6 months of age. This apparent development pattern might be altered by cosleeping. Limiting Stage 3–4 or Quiet Sleep might also be important to infants in view of our observation that infants average consistently longer breathing pauses in Stage 3–4, although we observed no sustained (≥15-sec) apnea in any of the eight infants studied. Furthermore, because of the high arousal threshold in Stage 3–4 together with the observation that many short breathing pauses immediately follow an arousal with return to Stage 1–2 or REM (Fig. 5), we also raise the possibility that cosleeping might provide the opportunity to "practice" terminating breathing pauses in stages other than Stage 3–4 where apneas may represent less of a survival threat. This is, of course, highly speculative.

Although our studies did not measure the impact of cosleeping on the infant's thermal environment, speculation about a possible thermoregulatory benefit of cosleeping is warranted in view of recent evidence that temperature may play a role in triggering SIDS. Several researchers have postulated an important role of environmental and core temperature in SIDS through their powerful impacts on sleep architecture, arousal, and respiration. Deviations from thermoneutrality might lead to SIDS through a variety of postulated mechanisms (Fleming *et al.*, 1990; Glotzbach and Heller, 1989). Cosleeping obviously enhances the caretaker's opportunity to experience personally and hence make appropriate corrections when the infant's sleep environment becomes too hot or cold. As our data additionally suggest, a mother's tendency to awaken in response to infant arousals (some of which might occur as a consequence of deviations from thermoneutrality) would also enhance her ability to make appropriate thermal adjustments. Furthermore, there is the simple fact of her protective maternal behaviors and immediate proximity: our videotape recordings show that during even very short arousals, mothers very often stroke or pat the head, back, or chest of their infant, gestures which would allow her to sense if the infant was too hot or too cold.

Venturing well beyond our data, one can speculate on other possible benefits of cosleeping. The majority of recent discussions of SIDs acknowledges the likelihood that many instances of SIDS involve subtle premorbid deficits in respiratory controls (Schwartz *et al.*, 1988; Guntheroth, 1989). Stewart and Stewart (1991) recently proposed that since, like adults, infants are able to process and respond to environmental stimulation in sleep (Kahn *et al.*, 1986; Ashton, 1973), appropriate external stimuli might compensate for a respiratory control defect. In a study of sleeping infant dogs and pigs, they reported that presentation of repetitive auditory stimuli could influence respiratory rate. Although the significance of such rate changes is not known and similar studies have not been performed in human infants, their results do raise the question of whether breathing sounds (or perhaps respiratory movements) of a cosleeping parent could provide compensatory respiratory stimulation for some infants with defective respiratory controls. In addition, we have begun an investigation of whether an infant's atmospheric environment could be altered in a meaningful way by cosleeping. Our videotapes reveal that the faces of mothers and infants can be separated by as little as 2 or 3 in. when cosleeping. We have investigated

end-tidal CO_2 levels a few inches from an adult's nares and measured atmospheric CO_2 concentrations as high as 3% (unpublished observation). Although when breathed continuously, 2% CO_2 has been shown to increase ventilation in sleeping infants (Haddad *et al.*, 1980), it remains to be tested whether the intermittent exposure which might occur while cosleeping would significantly impact infant respiration.

It is also important to mention a recent hypothesis that cosleeping could be a risk factor for SIDS (Mitchell, 1993). This is based on a New Zealand study which showed that infants whose deaths were ascribed to SIDS were more often found cosleeping with another family member. Although the mechanism of these deaths is not known, it is speculated that overlaying or hyperthermia might be involved. Given that the majority of the world's cultures still practices cosleeping, the laboratory study of cosleeping is equally important to the discovery of any potential detrimental as well as beneficial effects on infants.

In closing this discussion, we state that we do not expect that any one of the above possible benefits of cosleeping will be found to be critical to all infants at risk for SIDS, but as infant vulnerabilities no doubt vary greatly in nature and degree, so may the relative importance of the different types of sensory experiences that are inherent to cosleeping. We also acknowledge that there could be circumstances, such as parental inebriation, where cosleeping could be hazardous. Also, we suspect that cosleeping may affect infant sleep in important ways that will not be apparent by applying the more conventional types of measurements as made in our study and most other studies of infant sleep. For example, any role that olfaction may play in the sleeping infant has not been investigated. Evidence that young infants learn their mother's odor and preferentially orient (by head turning) toward it in sleep and waking (Cernoch and Porter, 1985; Sullivan *et al.*, 1991; Russel, 1976; Schaal *et al.*, 1980; MacFarlane, 1975) could well have implications for infants cosleeping with their mothers. Although not formally investigated, it was our observation that mothers placed their infants so that they were oriented toward themselves and that infants spent much more time with their heads facing toward their mothers. This mutual orientation would maximize both olfactory exchange and exchange of expired gases; it could also serve to minimize the prone sleeping position which has recently been identified as a possible risk factor for SIDS (Guntheroth and Spiers, 1992).

In summary, this discussion is meant to outline some of the issues and directions for future research that emerge when the assumption that solitary sleeping is the most appropriate environment in which to study infant sleep is challenged. The trends we observed in cosleeping mother–

infant pairs will require extensive replication and elaboration of study techniques before any conclusions can be made. However, what is indisputable at the present time is that cosleeping provides a much more enriched, varied sensory environment than solitary sleeping, and human infant sleep evolved within this enriched environment. The implicit assumption made in the abrupt Western cultural shift 200 years ago to infants sleeping in isolation that infants are adequately prepared biologically for this shift has neither been acknowledged nor tested. This omission takes on special importance given the growing consensus among researchers that solving the problem of SIDS will entail improving both the prenatal and the postnatal environments of the infant.

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