ISSN 0803-5326

Vol. 83 / June 1994 /
Supplement 397

ACTA PÆDIATRICA

an International Journal of Paediatrics

The Neurobiology of Infant–Parent Interaction in the Newborn Period

An International Wenner–Gren Center Symposium

Edited by Jan Winberg and Ingemar Kjellmer

Study 192

SCANDINAVIAN UNIVERSITY PRESS

Oslo - Copenhagen - Stockholm

Acta Paediatr Suppl 397: 94–102. 1994

Sleep and arousal, synchrony and independence, among mothers and infants sleeping apart and together (same bed): an experiment in evolutionary medicine

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McKenna JJ, Mosko SS. Sleep and arousal, synchrony and independence, among mothers and infants sleeping apart and together (same bed): an experiment in evolutionary medicine. Acta Pædiatr 1994;(Suppl 397):94–102. Stockholm. ISSN 0803–5326

Although solitary sleeping in infancy is a very recent custom, limited to Western industrialized societies, and most contemporary people practice parent–infant co-sleeping, virtually all laboratory research on sleep in human infants assumes that solitary infant sleep is the normal and desirable environment. We have used evolutionary and developmental data to challenge this view. We suggest that co-sleeping provides a sensory-rich environment which is the more appropriate environment in which to study infant sleep. In addition, two preliminary, in-laboratory, polygraphic investigations of mother–infant co-sleeping are reported in normal infants, within the peak age range for sudden infant death syndrome (SIDS). Five mother–infant pairs co-slept one night in the first study; in the second, three additional pairs slept separately for two nights and co-slept the third consecutive night. The results suggest that co-sleeping is associated with enhanced infant arousals and striking temporal overlap (synchronicity) in infant and maternal arousals, and that, possibly as a result, co-sleeping mothers and infants spend more time in the same sleep stage or awake condition. The implications of the hypothesis and preliminary results for research on the normal development of infant sleep and on SIDS are discussed. □ *Co-sleeping, infant sleep, overlapping arousals, sudden infant death syndrome (SIDS), synchrony*

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Overview of Euroamerican infant sleep patterns from the perspective of human evolution

"... I think it is a sensible rule not to take a child into the parents bed to sleep for any reason (even as a treat when one parent is away on a trip". "... Children can sleep in a room by themselves from the time they are born, if convenient, as long as the parents are near enough to hear them when they cry" (Dr Spock's Baby and Child Care) (1).

Our work challenges fundamental assumptions underlying pediatric sleep research, specifically the idea that infants sleeping separately from parents necessarily constitutes the most beneficial context for infant sleep for all infants and all family circumstances. Given that all human beings practiced parent–infant co-sleeping up until the last 200 years, we point out that the Western societal practice of solitary infant sleep represents a historically novel sleep environment, the consequences of which (either short or long term) have never been considered or explored experimentally (2, 3). Compared with quickly changing Western cultural ideas on where and how infants "should" sleep, we argue that the mechanisms that control infant sleep are unable to change as quickly and, where infants sleep alone, their sleep and breathing mechanisms are functioning in environments for which they were not designed by evolution. Since pediatric sleep researchers have never explored the impact of social sleep on early neonatal and infant development, we do not know if the recent shift by some world cultures to solitary sleep environments is beneficial, benign or deleterious. What is known from anthropological studies is that continuous contact and carrying (including infant–parent co-sleeping) have characterized the human infant's developmental experiences for well over five million years. While this alone does not prove that separate sleep arrangements for infants are necessarily detrimental, it does mean that co-sleeping environments should also be included when questions are raised about infant sleep. Unfortunately, studies of both normal infant sleep and the sudden infant death syndrome have universally failed to acknowledge co-sleeping environments.

At birth the human infant brain is only 25% of its adult brain weight. Indeed, the human infant is the least neurologically mature primate. This suggests

that, in order for infants to survive, and for human (parental) reproductive success to be maximized, natural selection likely favored the co-evolution of highly motivated care givers on the one hand, alongside highly responsive infants on the other (infants designed to respond to and depend on external parental sensory signals and/or regulatory stimulii) (4). From both an evolutionary and developmental perspective, parental contact and proximity during sleep can be seen to represent a developmental bridge for the infant, extending into postnatal environments the role that the mother played prenatally in regulating important aspects of her infant's development (see Fig. 1). Recent laboratory studies showing beneficial physiological effects (stabilizing temperature, heart rates and sleep) of mothers holding their preterm infants using the Kangaroo method of baby care are consistent with this perspective (see (5–8)). That natural selection favored infant responsibility to postnatal parental sensory stimulii (whether the infant is asleep or awake), in much the same way that it favored fetal responsivity to and regulation by the mother's physiological and/or behavioral status prenatally, through fetal–maternal physiological exchanges (see (9)) is a reasonable proposition justifying research into the effects of adult contact on infant sleep physiology.

Our on-going laboratory studies of infant–parent co-sleeping are still in a preliminary phase, but they provide one source of data needed to evaluate our hypothesis that sensory exchanges occurring between infant–parent co-sleeping partners may change the infant's sleep experiences in significant ways. In addition, we speculate that co-sleeping may be potentially helpful to some infants in resisting SIDS, the leading cause of non-accidental deaths in infants in the first year of life in the United States and other Western countries (see (2–4)).

This report summarizes early results from our studies on the effect of same-bed co-sleeping on sleep and arousal patterns of mother–infant pairs. Particular attention is paid to infant arousals since arousal deficiency is thought to play a role in some cases of SIDS (3, 10). We should like to acknowledge that the content of this paper is largely extracted from several more detailed papers which have appeared elsewhere (see (2, 3, 10)).

Methods

We describe two pilot studies of co-sleeping mothers and infants. The first consisted of polysomnographic recordings of five healthy mother–infant pairs (full-term infants aged 2–5 months) who co-slept one night in the sleep laboratory. In the second study, three additional mother–infant pairs were studied for three consecutive nights: they slept apart in adjacent rooms on the first two nights and side-by-side in the same

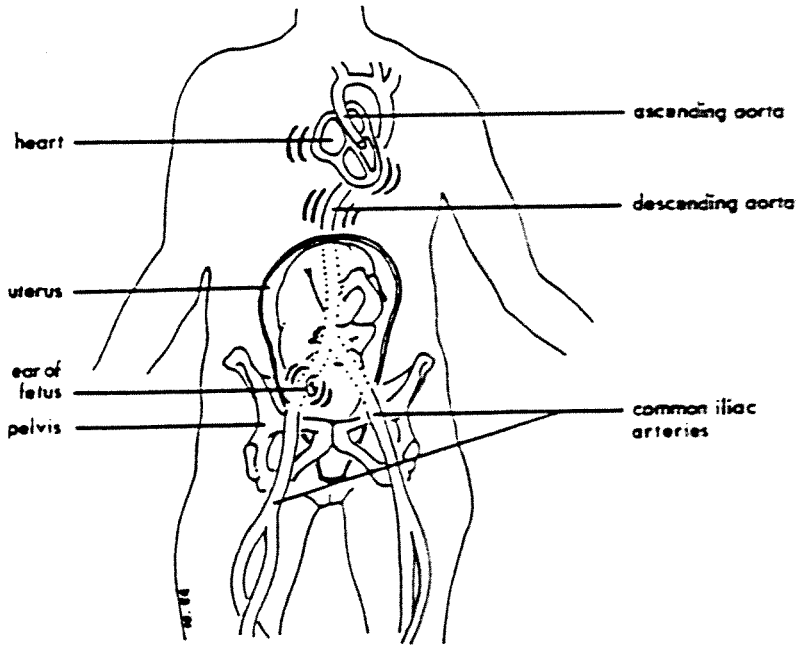

Fig. 1. In addition to hearing pulsating tidal rhythms of blood swooshing by its ears every 0.01 s after ventricular contraction, before birth the fetus is pre-sensitized to other maternal sensory cues and signals involving movement, touch, temperature, nutrient and gas exchanges. We suggest that these sensory experiences of the fetus prepare it for postnatal physiological regulation involving similar sensory modalities. The parent–infant co-sleeping environment is one of several different contexts which helps protect the vulnerable infant as it makes an adaptive transition from complete biological dependency to increasing social and biological autonomy (from (4)).

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bed on the third night. The first night was viewed as an adaptation night as "first night effects" on laboratory sleep have been measured in adults and infants which resolved by the second night (11). Mothers in both studies had normal pregnancies and deliveries, and a medical examination of infants one week prior to the sleep studies showed they all had normal developmental histories.

Sleep pattern (electroencephalogram (EEG, i.e. C3/A2), electrooculogram (EOG) and chin electromyogram (EMG)), respiration (chest wall movement and sometimes oro–nasal airflow) and EKG were monitored non-invasively each night will all mother and infant channels recorded simultaneously on a single polygraph.

In the second study one mother had been co-sleeping nightly with her infant since birth whereas all of the other seven mothers studied had not. Mothers positioned their infants for sleeping as usual each night with no instructions. Electrodes were attached first to the infant since they retired before mothers at home. The recordings were begun when the mothers retired close to their normal bedtimes and the recordings were terminated the next morning when both mother and infant had awakened spontaneously near their usual time. Mothers slept next door to their infants on solitary sleeping nights with the bedroom doors left open so that they remained in auditory contact. Mothers responded to infant crying, etc, on an ad lib basis each night and performed all care-giver interventions themselves.

Polygraphic recordings were scored for sleep stages in 30-s epochs according to accepted criteria. The Rechtschaffen & Kales (12) system for young adults was utilized for the mothers and the scoring system for the three-month-old infant developed by Guilleminault & Souquet (13) was used for the infants. Identification of sleep–wake stages in both scoring systems depends on three simultaneous parameters: EEG, EOG and chin EMG. Five sleep stages are identified in adults: stage REM plus four stages of non-REM (NREM) sleep, designated stages 1, 2, 3 and 4. In the three-month-old infant, only three stages are defined: stage REM, stage 1–2 and stage 3–4. A major difference between the infant and adult systems (besides the lack of differentiation between stages 1 and 2 and also 3 and 4 in the infant) is the higher voltage criterion for delta waves ($>150\,\mu V$) in the infant. In the process of data reduction, stages 1 and 2 in the adult were combined to obtain a combination stage 1–2 and likewise stages 3 and 4 were combined for comparability to infant sleep stages. Table 1 defines the terms and abbreviations used in the polygraphic studies.

This epochal system of sleep stage scoring assigns to each 30-s epoch either wakefulness (W) or one stage of sleep based on the predominant (greater than 50%) sleep or wakefulness pattern occupying the epoch.

Table 1. Definitions of terms used in our polygraphic sleep studies.

Epoch	Scoring period of 30 s
Sleep stages	1–2 (light sleep), 3–4 (deeper sleep), REM (active, rapid eye movement), NREM (quiet sleep)
W	Wakefulness
EW	Epochal waking (individual awake >15 s)
TA	Transient arousal (EEG arousal >2 but <15 s)
SAT	Simultaneous activity time. Time mother–infant pair simultaneously spent in same sleep–wake stages
SE	Sleep efficiency (percentage of time in bed person is asleep while being recorded)
K complexes	High-voltage EEG spike with a spindle tail, found to occur in stage 2 of sleep; not related to dream activity; thought to be elicited by auditory stimulation

Although awakenings of 15 s or longer that meet these criteria (i.e. epochal awakenings (EWs)) are automatically identified by the epochal system, shorter duration subepochal arousals occupying less than 50% of an epoch are not. Because of our interest in all arousal phenomena in sleep, we quantified these subepochal or transient arousals (TAs), modifying the criteria for TAs published by Carskadon *et al.* (14). They defined a TA as any clearly visible EEG arousal lasting >2 s but not associated with any change in the sleep stage epochal scoring system. We have omitted the latter exclusion criterion in the scoring of TAs to recognize all short-lived arousals irrespective of whether there is return to the same or different sleep stage.

TAs among infants were scored among infants when there was typically either an abrupt increase in the predominant EEG frequency or a sudden burst of distinctively higher voltage slow waves or K complexes. In the mothers, TAs were evidenced by an increase in EEG frequency accompanied by bursts of K complexes. Although it was not a requirement, by far the majority of all TAs in infants and mothers were accompanied by signs of arousal on other channels, i.e. increased heart rate, burst of chin EMG activity, change in respiration or change in EOG pattern (e.g. to slow rolling eye movements or blinking).

In both studies, after the conclusion of morning sleep, all mothers completed a questionairre. They were asked if their own and their infant's sleep were typical of their usual patterns at home. In all cases the mothers responded affirmatively.

Results

Study 1

Arousals for both mothers and infants were quantified and examined for temporal overlap. For both mothers and infants, EWs were identified independently from the polygraphs and later categorized for each sleeper according to whether or not the co-sleeper had an over-

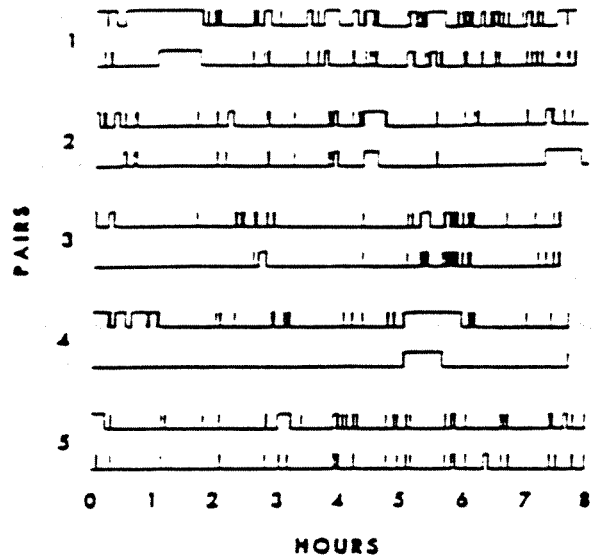

Fig. 2. Sleep–wake transitions of co-sleeping mother–infant pairs over an 8-h period. Each pair (1–5) is a co-sleeping mother and infant. When the line is at the top, the mother or infant is awake, when the line is at the bottom, the mother or infant is asleep. Note how many times the mother and infant awaken for a minute or so at the same time (see also where the spikes line up, or where their awakenings overlap for more extended periods, such as when the infants nurse). This computer generated illustration shows how much synchrony between co-sleeping mothers and infants occurs over 8 h of their sleeping in the same bed (from (10)).

lapping epoch(s) of waking, i.e. an overlapping EW. Infants averaged 20 ± 14 Ews (range 2–37) and mothers 34 ± 9 (range 29–49). Of the total 102 EWs recorded in infants, 91 (89%) overlapped with a maternal EW and for 73% of these the awakenings took place in the same epoch. Of the 169 combined EWs in mothers, 78 (46%) overlapped with 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 during the whole night (both of which overlapped a maternal EW), 84–100% of individual infant EWs overlapped EWs in the mother and 50–58% of the individual mother 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 mother–infant pairs, collapsing across sleep stages.

To estimate the degree to which factors other than co-sleeping produced these high rates of overlapping EWs, the record of each infant was randomly paired with that of a different mother and the records were compared on the same epoch-by-epoch basis to examine overlapping EWs. For the five randomly matched pairs, 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 co-sleeping versus randomly matched conditions, the increase with co-sleeping was significant for both the infants (paired *t*-test, $p < 0.01$) and the mothers ($p < 0.05$).

Smaller TAs were also identified independently in each mother and infant and later categorized according to whether or not the co-sleeper had a concurrent (± 5 s) TA or an overlapping epoch of waking. Co-sleeping infants and mothers averaged 78 ± 25 (range 52–115) and 58 ± 12 (range 28–76) TAs, respectively. Of the 388 total infant TAs, 114 (29%) overlapped a maternal TA and an additional 142 (37%) overlapped a maternal EW. Similarly, 114 (39%) of the 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 all maternal arousals 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 maternal and infant arousals appeared almost simultaneously in many cases, it was not

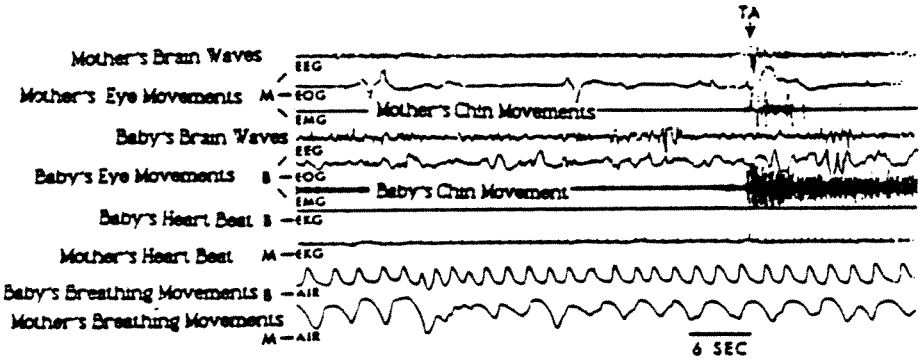

Fig. 3. In this segment of time, the mother has a small arousal (a transient arousal, TA) as indicated by a change in her brain waves, eye movement waves and her chin position. Within 1 s or so of the mother's arousal, the infant opens its mouth and its heart rate increases: the infant also experiences an arousal. We call this a synchronous arousal (see Table 3).

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possible to reliably determine which co-sleeper aroused first (see Fig. 3).

Applying the epochal scoring system, we also examined the percentage of total recording time each co-sleeping mother–infant pair spent simultaneously in the same stage of sleep or wakefulness. These times of corresponding sleep–wake stages were called simultaneous activity times (SAT). SAT averaged 39 ± 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 for the five randomly matched mother–infant pairs as described above. The pairings began at the first epoch of each recording and because total recording times of these mothers and infants differed by nor 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 co-sleeping condition was significant for the infants (paired t-test, $p < 0.05$). The analogous increase in SAT for the mothers failed to reach significance ($p < 0.1$).

SATs were also calculated for individual sleep–wake stages in both co-sleeping and randomly matched pairs to assess their contribution to the overall SAT. For infants and mothers, mean percentage SAT was higher in the co-sleeping condition for stages 3–4, REM and waking, although the increase reached statistical significance only for stage waking in both infants (paired t-test, $p < 0.05$) and mothers ($p < 0.01$).

Table 2. 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	0.89	0.91	0.89
% 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 per hour sleep	14	14	18
Mothers ($n = 3$)			
Total recording time (min)	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	0.89	0.85	0.92
% 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 per hour sleep	9	9	12

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

Study 2

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 2). There were no trends to suggest that infant sleep was more disturbed on 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 co-sleeping were similar, with three exceptions (Table 2). The number of stage shifts per hour of sleep (transitions from one state to another) 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 reflected trends in only two of the infants since infant No. 3 showed no change on the co-sleeping night. Total sleep time was decreased by an average of 39 min, but as sleep efficiency (ratio of total sleep to total recording time) was not reduced, this likely reflects the 40-min average shorter recording time on the third night. Mothers too showed more stage shifts per hour of sleep (33% increase). However, rather than a reduction in slow-wave sleep, the percentage of stage 3–4 sleep was, if anything, increased. That co-sleeping produced little objective sleep disturbance is consistent with the morning after reports from all three mothers that their laboratory sleep, including the co-sleeping 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 co-sleeping 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 co-sleeping, whereas mother–infant pair No. 3 showed a negligible 3% increase.

With respect to the percentage SAT for individual sleep–wake stages on night 3 compared to nights 1 and 2, the pattern was the same for mothers and infants: percentage 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/h arousals for nights 1 and 2 (range 7.5–19.7/h). These were comprised of 9.9/h TAs (range 5.9–14.6/h) and 4.1/h EWs (range 1.6–

5.6/h). Mothers showed far less individual variation. Their combined EWs and TAs averaged 8.3/h (range 8.1–8.6/h) and these were comprised of 5.4/h TAs (range 4.9–5.9/h) and 2.9/h EWs (range 2.8–3.0/h). The pattern of arousals during co-sleeping also varied between infants. The infant that routinely co-slept 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%. However, this reflected a 61% increase in EW frequency with a decrease in TA frequency of 26%, suggesting that arousals were prolonged rather than more frequent. In infant No. 3, there was a negligible (1%) increase in arousal frequency while co-sleeping and the changes in EW and TA frequencies were also negligible. Since this infant had the highest arousal frequency every night, the possibility of a ceiling effect is raised.

Mothers too showed variable changes in arousal frequency while co-sleeping. The mother which 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 co-sleeping, e.g. the mother of the infant that showed the greatest increase in arousals exhibited the smallest increase among the mothers.

Table 3 shows 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 co-sleeping night for every infant and every mother. In contrast, the two solitary nights differed relatively little from one another, for infants or mothers. From Table 3, which also separates TAs and EWs, it can be seen that both types of arousals showed a large increase in overlap with arousals in the other dyad members when co-sleeping.

Table 3. Mean percentage of total arousals (EWs plus TAs) overlapping any arousal (EW or TA) in the other on the solitary (nights 1 and 2) and during co-sleeping (night 3).[a]

	Night 1	Night 2	Night 3
Infants	13% TAs 24% 23% EWs	19% TAs 21.3% 24% EWs	36% TAs 52.7% 78% EWs
Mothers	12% TAs 18% 29% EWs	16% TAs 24% 26% EWs	53% TAs 62.7% 84% EWs

[a] For example, and reading the upper left of the table, we see that, on average 24% of the infants' arousals overlapped some type of arousal in the mother; on average 13% of the infants' overlapping arousals were transient arousals, while (on average) 23% of the infants' overlapping arousals were epochal wakenings (EWs). As indicated below, these data are reported for the first night when mothers and infants slept solitarily in adjacent rooms.

Discussion

This research on infant–parent co-sleeping is the first to use an evolutionary perspective to reconceptualize two related areas of pediatric research: normal infant sleep and SIDS. Traditional research paradigms have not proven fully adequate for SIDS (since the basic questions about SIDS and its prevention remain unsolved) and they have been consistently biased as regards normal infant sleep in that only solitary infant sleep has been studied. An evolutionary perspective provides a rich and well-founded beginning point for challenging research assumptions underlying traditional infant sleep and SIDS research which reflect our recent (Western) cultural history, a history which favors individualism and autonomy over social interdependence and familialism (4) and, thereby, a conceptualization of the infant which is (in part) in conflict with its evolutionary history. In fact, by way of this research we have helped to introduce the newly emerging field of evolutionary medicine, which integrates neurobiological, biomedical and biocultural data for purposes of formulation and testing of medical hypotheses.

However, the studies we have described here represent only a small beginning. Obviously, the limited number of subjects studied do not permit any conclusions. This discussion of our findings serves only as a vehicle to illustrate the types of questions that might be raised by acknowledging the co-sleeping environment. Our primary intention is to use these data to call attention to the importance of the characteristics of the sleep environment as they relate to the pattern of normal infant sleep and to potential SIDS co-factors that have previously been ignored or dismissed altogether.

Two of the potentially most important findings suggested by our studies are that co-sleeping infants arouse more frequently and with far greater overlap with maternal arousals, which implies that the arousals are partner-induced. Also, infant sleep stages are altered by co-sleeping, as manifested by decreased stage 3–4 and greater simultaneous overlap with maternal sleep–wake stages. It is possible that these effects of co-sleeping are related in that more frequent and maternal-induced arousals could interfere with the development or maintenance of infant stage 3–4

sleep, a stage of sleep for which arousal threshold is high. Also, overlapping arousals could also produce greater overlap with maternal sleep stages (i.e. greater SAT), since sleep generally lightens following arousals and conversely a period of fewer arousals in one co-sleeper could facilitate the development of deeper sleep in the other co-sleeper. 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 co-sleep at home, the question is raised if the co-sleeping effects measured reflected more the novelty of co-sleeping than effects that would persist with habitual co-sleeping. The fact that the one infant (in study 2) who routinely co-slept with its mother showed all of the above trends prominently and also had the largest increase in arousal frequency during co-sleeping, suggests that the effects of co-sleeping do not habituate. Nevertheless, we propose that individual variation in infant sensitivity and habituation to co-sleeping is to be expected and that sensitivity will also vary during development as the infant matures and dependence on the parent or care giver diminishes.

If these findings are replicated in our continuing studies, they could challenge long-held conclusions about normal development that have emerged from studies of solitary sleeping infants. That is, if co-sleeping infants do arouse more frequently and have less stage 3–4 or quiet sleep, then the conclusion that achieving progressively longer periods of sustained sleep across the first several postnatal months is a normal developmental milestone (15–18) 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.

Related to this is the important question of the possible benefits to infants of arousing more frequently and simultaneously with the mother, and of having less stage 3–4 or quiet sleep. Harper et al. (19) and Hoppenbrouwers et al. (20) found that subsequent siblings of SIDS victims had comparatively longer periods of uninterrupted sleep, suggesting impaired ability to make the transition from sleep to wakefulness. Other researchers have reported similar findings, such as less frequent spontaneous awakenings in near-miss SIDS infants (21–23). Furthermore, deficient arousal responses during sleep to hypoxia or hypercarbia in near-miss SIDS infants and subsequent siblings of SIDS victims have been measured (24, 25). 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 co-sleeping could prevent the occurrence of long periods of uninterrupted sleep from which some infants might have difficulty arousing in response to a physiological crisis.

Since some arousals while co-sleeping are clearly extraneous (partner-induced), one can speculate that co-sleeping 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 practices 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 early development when SIDS occurs. Importantly, the increase in arousal frequency measured with co-sleeping did not reduce the overall amount of noctural 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 (26).

Developmental studies of solitary sleeping infants consistently find a large increase in time spent in quiet sleep during the months when infants are at highest risk of SIDS. This could be important to the hypothesis about the role of arousal in SIDS since arousal threshold is thought to be high in the delta portion of quiet sleep (27–29). Our finding that co-sleeping may reduce or limit the amount of stage 3–4 sleep suggests that the arousal response might be enhanced and some infants protected by co-sleeping: co-sleeping might prevent the premature emergence of large amounts of quiet sleep seen when infants sleep in isolation. Hoppenbrouwers et al. (16, 20) have shown in longitudinal studies that the longest duration of quiet sleep increases from one week to six months of age. This apparent developmental pattern might be altered by regular co-sleeping.

Venturing well beyond our data, we can also speculate on other possible benefits of co-sleeping. The majority of recent discussions of SIDS acknowledge the likelihood that many instances of SIDS involve subtle premorbid deficits in respiratory control (30, 31). Following McKenna (4), Stewart & Stewart (32) recently proposed that since infants are able to respond to environmental stimulation in sleep (21, 27), appropriate external stimuli might compensate for a respiratory control defect. In a study of infant dogs and pigs, they reported that presentation of repetitive auditory stimuli could influence respiratory rate. Although the significance of such rate changes are not known and similar studies have not been performed in human infants, their results do raise the question of whether or not breathing sounds (or perhaps respiratory movements) of a co-sleeping parent could provide compensatory respiratory stimulation for some infants with defective respiratory controls. In addition, we have begun an investigation of whether or not an

infant's atmospheric environment could be altered in a meaningful way by co-sleeping. Our videotapes reveal that the faces of mothers and infants can be separated by as little as 2 or 3 in when co-sleeping. We have investigated end-tidal carbon dioxide levels a few inches from an adult's nares and measured atmospheric carbon dioxide concentrations as high as 2.2%. Although when breathed continuously 2% carbon dioxide has been shown to increase ventilation in sleeping infants (33), it remains to be tested whether or not the intermittent exposure which might occur while co-sleeping would have a significant impact on infant respiration.

We also suspect that co-sleeping may affect infant sleep in important ways that will not be apparent by applying the more conventional types of measurements made in ours 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 (head turning) towards it in sleep and waking (34–38) could well have implications for infants sleeping with their mothers. Although not yet investigated formally, it was our observation that co-sleeping mothers placed their infants so they were oriented towards themselves and that infants spent the most time with their heads facing towards their mothers. For two infants videotaped by infra-red cameras, well over 90% of their sleep time was spent facing 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 (39).

Lastly, we acknowledge a recent epidemiological study from New Zealand in which bed sharing was found to be associated with SIDS in the Maori population (4). From this, the authors have concluded that co-sleeping is a risk factor for SIDS and that co-sleeping has "outlived its historical usefulness". Although we acknowledge that undoubtedly there may be specific circumstances under which co-sleeping could be dangerous (such as when an adult is inebriated or desensitized by drugs or when dangerous or unclean bedding materials are used), we believe that the categorical conclusion that bed sharing is a risk factor for SIDS is unfounded for several reasons. First, studies in other cultures (such as industrialized Japan (41–44) or Asian immigrants to western countries (45, 46)) have generally found lower SIDS rates in peoples who practice co-sleeping. Second, within the same New Zealand study, the association between co-sleeping and SIDS did not hold up in the white population or in South Pacific islanders who also commonly bed share. Furthermore, the fact that the association did not hold even in Maori men, or in non-smoking Maori women, suggests that a very specific set of circumstances led to the association of co-sleeping with SIDS. It is also possible that for white control populations there is significant under reporting of the amount of same-bed co-sleeping which occurs. This probably reflects the unwillingness of parents to readily admit to co-sleeping in a culture which largely condemns it, or which defines it extremely narrowly (1, 47).

Certainly, replication of the New Zealand study is needed, together with a thorough investigation of the interaction between bed sharing and smoking. Further epidemiological studies of co-sleeping and SIDS rates (including low-risk populations) also are needed since it has been through epidemiological studies that essentially all of the suspected risk factors for SIDS have been identified. The unique contribution that future research such as our own can make is to elucidate a range of both physiological and behavioral mechanisms presently unknown to researchers that will help to more accurately interpret epidemiological data, irrespective of whether or not those data indicate a reduced or higher risk of SIDS while co-sleeping.

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