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# Infant–Parent Co-Sleeping in an Evolutionary Perspective: Implications for Understanding Infant Sleep Development and the Sudden Infant Death Syndrome[1]

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**Summary:** Evidence suggests that infant–parent co-sleeping represents the species-wide pattern of sleep in which human infant physiology evolved. The hypothesis evaluated in this manuscript is that the co-sleeping environment may foster development of optimal sleep patterning in infants and confer other benefits, including reducing the risk of the sudden infant death syndrome (SIDS). These postulations by McKenna are considered from different perspectives by the coauthors. Using evolutionary, cross-species, crosscultural, physiological and behavioral data, our objective was to present a conceptual framework for assessing the developmental consequences of solitary sleeping and infant–parent co-sleeping. **Key Words:** Co-sleeping—SIDS—Sudden Infant Death Syndrome—Evolution—Pediatric sleep problems.

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## I. INTRODUCTION

Virtually all studies of infant sleep, including investigations involving presumed pathologies in the organization of sleep and arousal, have been carried out on infants sleeping in isolation from the parents or caregivers. Therefore, the extent of our knowledge of

the ontogeny of sleep in a variety of environments (hospital, sleep laboratory or home), has been derived exclusively from solitary sleeping infants.

The purpose of this manuscript is to examine from an evolutionary perspective the validity of certain assumptions underlying contemporary infant sleep research, particularly the idea that solitary infant sleep environments are "normal", and, hence, the most appropriate context for the study of the development of human infant sleep. The first section of the paper consists of an overview of the hypotheses of McKenna (1,2) that (a) parent–infant co-sleeping confers benefits to infants that are not provided by the solitary sleeping environment and (b) the co-sleeping environment may confer protection from potential hazards associated with infancy, particularly the sudden infant death syndrome (SIDS). The results from the electrophysiological recordings of co-sleeping mother–infant pairs are discussed briefly in relation to these hypotheses. The frequency and nature of sleep disturbances in infants and children are then related to the dynamic social and psychological environment by Sadeh and Anders.

Evelyn Thoman raises critical questions about McKenna's concept of a "natural ecology". The cardiorespiratory and thermoregulatory research of Schechtman and Glotzbach, respectively, provides physiological and clinical perspectives on SIDS and the sleep environment. Finally, specific suggestions are made as to what directions co-sleeping studies might take.

## II. AN EVOLUTIONARY ANALYSIS OF INFANT SLEEP

**James J. McKenna**

### A. Infant physiology and sleep environment

Infant sleep physiology evolved under conditions of continuous parental contact (3). In fact, for the vast majority of nonwestern cultures, diverse forms of parent–child co-sleeping remain the predominant sleeping arrangement for infants and young children throughout the first few years of life (4).

Cross-cultural studies of infancy, human evolutionary data and recent psychobiological studies of primate development suggest that the sensory-rich social sleep environment with which human infants evolved may confer certain physiological or psychosocial advantages on infants that solitary sleep environments do not provide. An evolutionary perspective suggests that the ecology of infant sleep cannot fully be understood, or clinical models of "normative" (species-wide) patterns constructed, without examining sleep as it unfolds in a co-sleeping (social) environment—the "environment of evolutionary adaptedness" (5).

I do not dismiss as unimportant present sociocultural values or lifestyles that promote solitary infant sleep, both in the home and as a standard research condition for studying infants in the laboratory. But sociocultural values underlying infant care practices and sleep research change much faster than does the biology of the infant, whose developing nervous system reflects millions of years of successful adaptations to infant–parent co-sleeping. The recent western pattern of solitary infant sleep may have adverse physiological or neurodevelopmental consequences for infants heretofore not considered.

I do not suggest that solitary infant sleep causes SIDS or that SIDS deaths can be eliminated simply by co-sleeping. Nor do I propose that co-sleeping is optimal for all parents and infants, or that traditional, solitary infant sleep studies should be abandoned. But clinicians should consider that our recent cultural history has determined the nature of infant sleep studies. To ignore what both developmental and evolutionary studies of primates and other mammals have yielded over the last 20 years concerning the important physiological regulatory effects caregivers assert on their infants (6–8) is to overlook insights that may be critical to the ability of the SIDS research paradigm to accommodate potential explanations of a disorder for which multiple etiologies and co-factors are strongly suspected.

Co-sleeping from the standpoint of the infant may be defined as sleeping either in contact with another person (in someone's arms, passively touching while lying in bed) or close enough to access, respond to or exchange sensory stimuli such as sound, movement, touch, vision, gas, olfactory stimuli, $CO_2$ and/or temperature. It is not a unitary phenomenon. The sleep environment consists of many different kinds of possible co-factors that may alter infant physiology and consequently influence SIDS risk. Co-sleeping is a condition in which other SIDS risk factors such as overbundling, dangerous (soft or fluffy) bedding, infant sleeping position, environmental temperature and a range of internal conditions may be altered, thereby changing the infant's risk of succumbing to SIDS.

The human infant is one of the least neurologically mature mammals at birth. It experiences the longest delays in both social and biological maturation (1,9–11), a fact not likely to be appreciated without a comparative analysis of mammalian evolution and development (12) and known physiological effects of short-term separation from a caregiver. An astonishing 75% of human brain growth occurs postnatally. As a consequence of its immaturity, the human infant is forced to rely on external regulation and support, especially in the first year of life (13). The extent to which, on a minute-to-minute basis, the infant's most fundamental

physiology such as heart rate, body temperature, breathing, sleep and arousal are likely influenced by the caregiver may be the most important concept for sleep and SIDS researchers to consider.

Infants vary from each other constitutionally and not all respond equally well to the challenges of solitary sleep, as work by both Sadeh and Anders demonstrates. In fact, protesting against solitary nocturnal sleep represents an adaptive mechanism to ameliorate a potentially life-threatening situation—separation from the caregiver. Those infants who protest the most against solitary nocturnal sleep [i.e. the "signalers" to use Anders' term (see below)], are responding adaptively. Certainly, current clinical recommendations concerning infant sleep management reflect the perceived social needs of parents and older children living in western industrial cultures far more than they do the unique psychological and biological needs of the human infant, at least when these needs are examined through an evolutionary lens. Rigid parental expectations as to how their infants and children should sleep contribute to culturally based parent–infant sleep struggles of the kinds described by Sadeh and Anders below. Certainly, the fact that up to 30–40% of children in western industrial countries experience sleep problems suggests that culture and children's psychosocial and biological needs are in conflict.

An evolutionary perspective forces us to consider the potential consequences of the recent shift away from social or co-sleeping arrangements to solitary ones in western industrial cultures, thereby altering the adaptive fit between the human infant's extreme neurological immaturity and the social support environment that presumably made such immaturity possible—or at least safer.

According to Kagan (14), a minimum of 3% of all newborns exhibit some type of central nervous systems deficit, although most of these are minor and resolved quickly without serious consequences. For infants born with particular types of deficits or more serious ones, however, we could speculate that increased postnatal stresses, one form of which may be intermittent rather than continuous contact with the caregiver during sleep, may exacerbate those deficiencies. This, thereby, makes these infants more susceptible to a variety of disorders, one of which may be SIDS. This speculation is based on the deleterious physiological consequences known to afflict nonhuman primates deprived of maternal contact as reported below.

The infant's biology and evolution are inseparable. To define an infant's biological needs is to understand its evolutionary history—specifically, the social and physical context within which the infant's unique biological characteristics (including its vulnerabilities) evolved alongside specific parenting responses. *Infant needs, and parental responses to those needs, constitute a dynamic, co-evolving interdependent system shaped and designed by natural selection to maximize the chances of infant survival and, hence, parental reproductive success.*

In the sections below I address evidence that supports the idea that co-sleeping is old enough to have affected infantile biological characteristics. I also present preliminary results from experiments on the physiological interactions of co-sleeping human mother–infant pairs. In a final section I comment on the recent finding from New Zealand suggesting a positive association between bed sharing and SIDS.

## B. Support for the proposal

### *1) The fossil record*

As early as 3.7 to 4 million years ago, upright locomotion (bipedalism) was already being favored by natural selection (15). At this time, three-and-a-half-foot-tall hominids known as *Australopithecus afarensis* began to exploit the open grasslands of south and east Africa in ways different from the strictly forest-dwelling, ape-like quadrupedal primates from which they evolved. For these more human terrestrial ancestors, an increase in diverse foraging strategies, learned social behavior (including food sharing, carrying and tool-making) developed in parallel with the rapidly expanding neocortex, which tripled in volume during the next three million years of human evolution (16).

To accommodate bipedalism, however, the shape of the pelvis and birth canal changed. These changes made it much more difficult and dangerous for infants to be born, although the survival advantages provided by bipedalism far outweighed the disadvantages. The hominid pelvis broadened and rotated forward, and the ischium (bones at the base of the hips) flattened out and pushed upward a bit to accommodate the hip-femur sockets, effectively diminishing the size of the birth canal. In other words, at approximately two million years ago with the rise of humans (*Homo erectus*) in Africa, and later in Europe and Asia, natural selection favored two conflicting evolutionary trends: increasing brain size (for learning and increased social complexity) and continued structural refinements (smaller birth openings) needed for bipedalism. The adaptive solution and compromise to this apparent evolutionary dilemma was, and continues to be, the birth of exceedingly neurologically immature infants for whom the majority of brain growth will occur postnatally and not in the womb (16). (Our closest living relatives, the chimpanzees, are born with about 45% of their adult brain weight compared to our 25%.)

The evolutionary antiquity of bipedalism is impor-

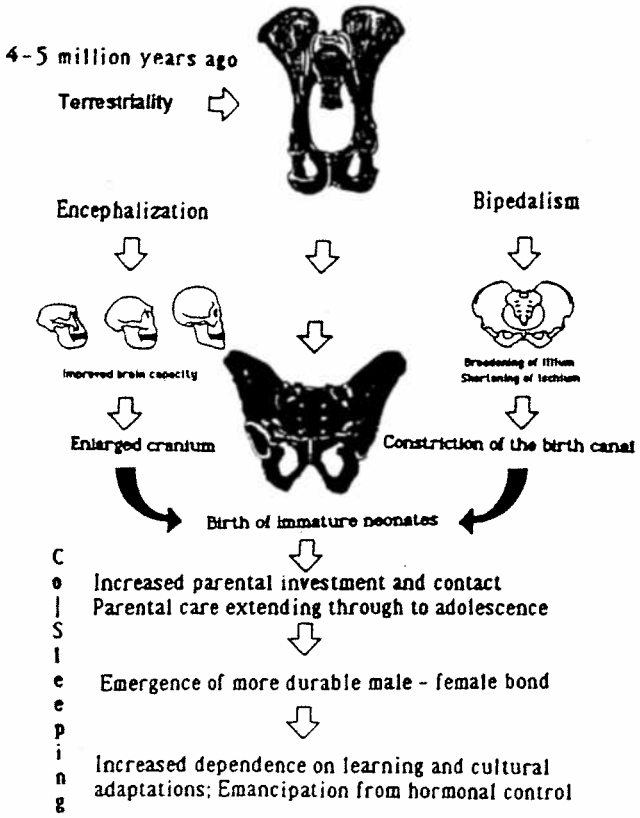



**FIG. 1.** Schematic illustration of the relationships between anatomical changes required for upright locomotion, the birth of neurologically immature infants and the need for prolonged and intense parental contact and care, including co-sleeping.

tant to the co-sleeping argument because it means that within the human lineage, beginning several millions of years ago, nonambulatory, immature hominid infants were being born. Increased parental contact, including longer durations of parent–young co-sleeping, carrying, nursing and protection, is inferred from cross-species, ethnographic and archaeological data, as human infants need, quite literally, to finish their biological and social gestation after birth. It is within this adaptive complex of sustained physical contact with a caregiver that human infant vulnerabilities were offset and the chances of infant survival were maximized.

### *2) Content of mother's milk*

The composition of human milk also supports this extremely old pattern of close and continuous mother–infant contact, including co-sleeping, as suggested by the fossil record. Compared with that of other mammals, human milk is low in fat and protein and relatively high in carbohydrates, especially lactose—a key nutrient needed, among other things, for brain growth. The concentration of lactose in milk is highest among primates whose infants are the least neurologically developed at birth.

Nonprimate mammals, such as lions and several species of deer, who leave their young in nests or burrows and return to them at intervals of 6–12 hours are called the "cache species" (17). Their milk, unlike human milk, is high in fat and protein, allowing the young to be satiated for longer periods of time. The composition of human milk, which provides fewer calories per feeding, indicates the need for more frequent feedings, requiring infants to remain close to their mothers, where they can nurse often. Ethnographic evidence suggests that co-sleeping at night and the carrying of infants in body shawls during the day were used to satisfy the need to keep infants close for feeding. Today, the majority of human beings continues to exhibit this pattern of child care (18,19).

### *3) Studies of parent–infant separation*

Especially for primates, contact with a parent figure has more than social and psychological benefits (20). Physiological regulation of primate infants by caregivers has been dramatically confirmed during the last 15 years by researchers working in several different fields and laboratories (Table 1). In fact, researchers argue that the social-psychological consequences of separation from a caregiver cannot be analyzed thoroughly without also considering the underlying physiological adjustments an infant must make when sensory interaction between them is terminated suddenly. Separation causes changes in the fundamental efficiency of systems not previously thought to be regulated by the presence or absence of a caregiver. For example, when experimentally separated from their caregivers for periods as short as 3 hours, monkey infants can experience significant detrimental effects, such as a decrease in body temperature, a release of stress hormones (ACTH), cardiac arrhythmias, sleep disturbances and compromises to the immune system.

Compared to monkeys, apes and other mammals, human infants are less neurologically developed at birth and develop far more slowly. Thus, physiological regulatory effects of contact should be greater, certainly not less. Only a few studies have examined some of the immediate (short-term) physiological effects of human parent–infant separation. Keefe (21) found that "rooming-in" newborns spent more time in quiet sleep than infants sleeping away from their mothers in the hospital nursery. Another study by Fardig (22) showed that the human newborns placed in incubators lost up to 1.5° of body temperature compared with newborns placed directly (skin-to-skin) on their mothers' stomachs immediately following birth. Like other immature central nervous system (CNS) homeostatic subsystems, infant thermoregulation may not be as efficient in a solitary context (23) as opposed to the evolutionary

**TABLE 1.** *Immediate and short-term physiological consequences of parent–infant separation in monkeys and rats*

| Physiological consequences of separation | Investigator(s) |
|---|---|
| Bonnet or pigtail monkeys | |
| Initial period: increase in heart rate and body temperature | Reite and Snyder 1982 |
| Subsequent period of depressed behavior: decrease in heart rate and body temperature | Reite et al. 1978a, 1978b |
| Increase in cardiac arrhythmias | Seiler et al. 1979 |
| Alterations in heart rate, body temperature, and circadian rhythms | Reite et al. 1982 |
| Disturbances in sleep: increased arousals; increase in REM latency; decrease in time in REM | Reite and Short 1978 |
| Changes in regulation of EEG activity | Short et al. 1977<br>Reite et al. 1982 |
| Alterations in cellular immune response accompanying mother–infant or peer separation | Reite et al. 1981<br>Laudenslager et al. 1982<br>Other references:<br>McKenna 1979, 1982<br>Reite and Capitanio 1985<br>Coe et al. 1985<br>Hofer 1981, 1978, 1983 |
| Squirrel monkey (*Saimiri sciureus*) | |
| Increase in adrenal secretion and plasma cortisol levels | Coe and Levine 1981<br>Coe et al. 1978, 1985 |
| Serum levels of immunoglobulins: decline after 7 days; back to normal in 14 days | Coe et al. 1985 |
| Complement proteins to cortisol diminish | Coe et al. 1985 |
| Lower level of antibody production in response to bacteria (*Escherichia coli*) | Coe et al. 1985 |
| Rats, 2 weeks old | |
| Bradycardia<br>Increased sleep latency<br>Augmented sleep<br>Decrease in REM sleep | Hofer 1981, 1978, 1983 |
| Rats, 10 days old | |
| Fifty percent reduction in brain and heart enzyme (ornithine decarboxylase) due to separation-induced suppression of growth hormone | Butler et al. 1978<br>Kuhn et al. 1978 |

From McKenna, 1986

"expectable" social environment for the neonate and/or infant (24).

### C. Preliminary studies of infant-parent co-sleeping

#### *1) Findings*

In 1986 I argued that a human infant's co-sleeping partner's touch, movement, breathing sounds, temperature and gas ($CO_2$) exchange and sleep vocalizations ought to assert influences on its unfolding sleep, breathing and arousal patterns. My prediction was based in part on data collected by others on prenatal environmental processes against which, I argued, mammal fetuses learn to breathe amniotic fluid before they are born in an environment that presensitizes (or preadapts) infants to auditory and vestibular breathing cues emitted from their parents postnatally (1,25). Recent experimental work on neonatal dogs and pigs has confirmed that auditory stimuli affect infant respiratory rates postnatally (26).

Using standard polysomnographic recording procedures, sleep, breathing and arousal patterns of mothers and their 2–4-month-old infants were recorded simultaneously as they slept in the same bed in the sleep laboratory (2,27,28). The sleep patterns of three mother–infant pairs were also recorded simultaneously in a second study as each pair moved from 2 nights of solitary sleep in adjacent rooms to a third night, when they slept in the same bed. Standard polysomnographic measures of sleep, including electroencephalogram (EEG), electroculogram (EOG), electromyogram (EMG), air flow, respiratory effort and electrocardiogram (EKG), were recorded on each mother and her infant simultaneously on a single polygraph as they slept apart and together over consecutive nights (28,29).

Our preliminary findings revealed that on average, co-sleeping mothers and infants: 1) experience more arousals than they do when sleeping alone (Fig. 2); 2)

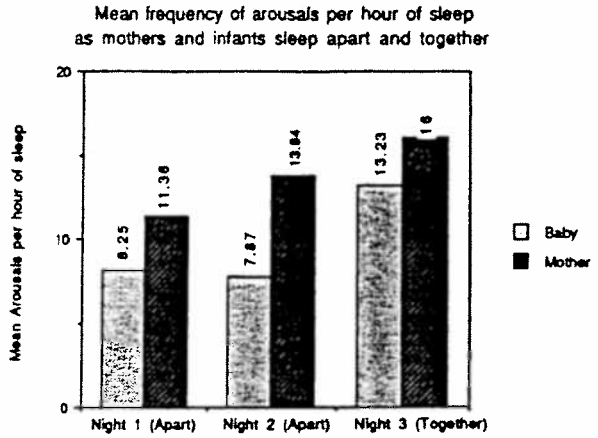


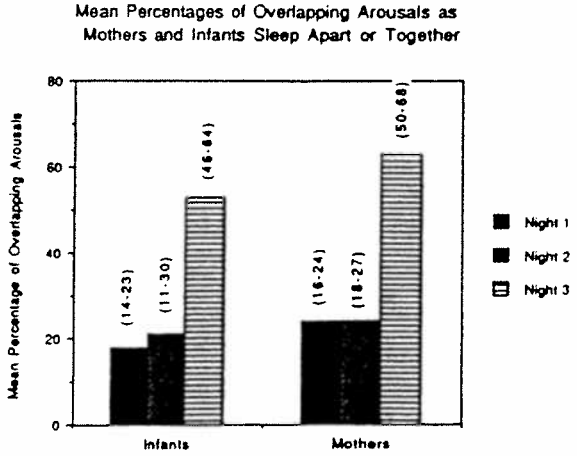

FIG. 2. Mean frequency of arousals per hour of sleep as mothers and infants sleep apart and together. Because the infant's sleep evolved in the context of co-sleeping, infant sleep development may be affected adversely when external arousals are removed from the infant's sleep experience.

exhibit levels of arousal overlap [both longer epochal and smaller physiologically defined transient arousals than would be expected by chance (2,28–30)] (Fig. 3); 3) exhibit more frequent stage shifts (moving from one stage of sleep or awake status to another); 4) spend more time, at the same time, in the same sleep stage or awake status while in the same bed and 5) infants spend less time in nonrapid eye movement (NREM) stages 3 and 4 compared with when they slept alone (Fig. 4). We also found that co-sleeping mothers contributed to the duration of their infants' arousals by using reassurance pats, single touches or their own responses to their infants' arousals. However, sleep latencies and sleep efficiency scores of neither the mother nor the infant were clinically disturbed in the co-sleeping environment. In fact, mothers reported having as much, if not more, sleep than they did at home. Two other interesting findings were that infants were responsible for positioning themselves with respect to their mothers and mostly slept on their sides or backs, diagonally, within just a few inches of her face. Mothers almost always faced their infants during sleep, and vice versa (29).

### 2) Interpretation

The finding that co-sleeping mothers and infants exhibit synchronous, partner-induced physiological arousals, although not very surprising, is potentially important because of the suspected relationship between infantile arousal deficiencies and some cases of SIDS. It may be that co-sleeping provides the infant with increased opportunities to practice arousing, thereby becoming more proficient at it. Moreover, these responses increase the overall amount of physiological variation (stage shifting, for example) experienced throughout the infant's nighttime sleep period. Overall, co-sleeping partner-induced arousals may facilitate the synchronous maturity and coupling of cardiorespiratory systems and the various CNS subsystems involved in arousal and/or shift from sleep to wakefulness (Fig. 3). It is possible that these linkages among the infant's physiological subsystems, which interact during arousals, may not occur as easily, as often or as quickly if infants regularly sleep alone (2,29,30).

Our finding that co-sleeping infants spend less time in deep stages of sleep, i.e. stages 3 and 4, and more time in stages 1 and 2 is also potentially important. If these findings are confirmed, it may suggest that solitary sleep environments may accelerate the maturation of deep sleep, possibly before arousal mechanisms are maximally efficient to handle arousals during some physiological crises (1,31).



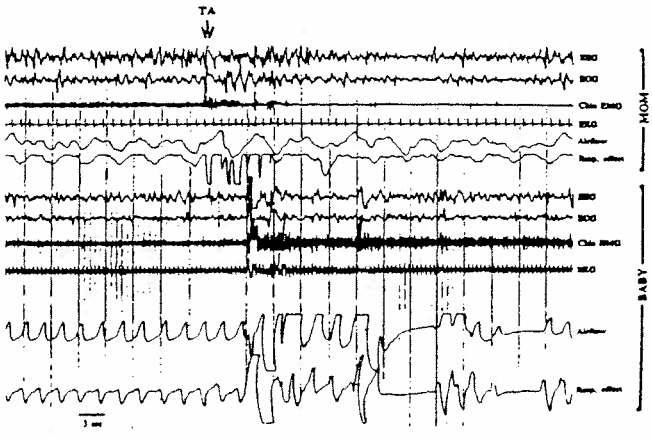




FIG. 3. A. Mean percentage of overlapping arousals as mothers and infants sleep apart and together. These partner-induced arousals often cause a shift from one stage of the infant's sleep to another, thereby promoting more variation. B. Polygraphic recording showing a maternal TA followed and overlapped by an infant TA while co-sleeping. Note the two brief breathing pauses that occur as the infant returns to sleep. Such partner-induced arousals may provide practice in arousing that serves the infant by promoting more successful arousals in response to internally-based respiratory crises.

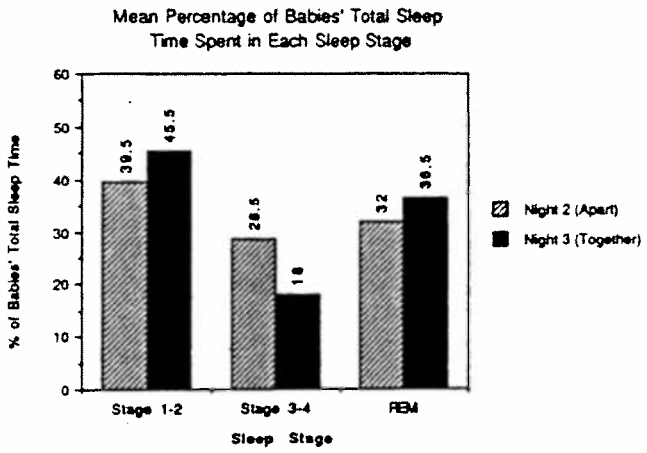



FIG. 4. Mean percentage of babies' total sleep time spent in each sleep stage as a function of environment. It may be that the diminished amount of time spent in deeper stages of sleep from which it is more difficult to arouse is beneficial to infants. (Night 1 is omitted because a signal artifact prevented us from being able to differentiate clearly stage 3–4 for one of our three pairs.)

### D. Does co-sleeping increase SIDS risk? A comment on the New Zealand epidemiological study

No environment is risk free. This may be especially true for human infants because in motoric, communicative, immunological, cognitive and digestive abilities human infants are so undeveloped and dependent on caregivers, and for such a long duration postnatally. Whether the infant sleeps alone in a crib, in the parental bed or with brothers and sisters, safety cannot be taken for granted. In fact, the co-sleeping environment that I suggest is potentially protective of some infants may, under certain circumstances, be dangerous to others, as Thoman suggests below.

Traditionally, sleep clinicians and SIDS researchers have assumed that solitary infant sleep is always safer, preferable and in the best interests of all parents and infants (32). But not all parents or infants are the same, a point made by Thoman in criticizing the co-sleeping perspective below. Certainly, SIDS researchers have never seriously considered the possibility that solitary sleep may itself play a contributing role in some SIDS events, possibly because overlying and/or suffocation has been suggested as the true cause of some deaths diagnosed as SIDS (33,34). Moreover, co-sleeping or bed sharing is often practiced among lower socioeconomic groups, where the known SIDS risk factors, and thus SIDS rates, are almost always substantially higher and where co-sleeping may be practiced under adverse circumstances. This may be the case, for example, in families where adults may be using drugs or infants are sleeping on soft and overly blanketed beds. Recent epidemiological studies of SIDS in New Zealand by Mitchell et al. (35) found four risk factors associated with SIDS: the prone sleeping position, not breastfeeding, smoking during pregnancy and bed sharing. They speculate that bed sharing may induce some SIDS by overheating the infant, or that accidental suffocation may be the actual cause of some diagnosed SIDS.

It is important to keep in mind that these epidemiological studies delineate associations between variables in large populations. They do not reveal, for any given SIDS event, the underlying mechanisms that produced that association among particular individuals. Such studies do not delineate or reveal the physiological or social mechanisms involved, for example, in any particular SIDS event.

Knowing how bed sharing is practiced in New Zealand, for example, especially among the Maori where SIDS rates and all the other risk factors for SIDS (poverty, drug use, maternal depression, smoking during pregnancy) were the highest, is crucial to an understanding of why this association exists. Thermal care (bedding materials, infant dress, blanketing), infant sleep position relative to co-sleeping partners and behavioral characteristics of all the bed sharers, including whether one or both parents were depressed, drink excessively, take drugs or are obese, are all exceedingly important co-factors needed for a full explanation of this association.

Given the kinds of subtle and complex co-factors relevant to each SIDS death, it is not appropriate to conclude that co-sleeping is dangerous across all family circumstances and sleeping conditions, across all cultures or that it cannot protect some infants from SIDS. These findings do not disprove the hypothesis that infant–parent co-sleeping can protect some infants from SIDS. In the next few years, perhaps matched groups of co-sleeping and non-co-sleeping Asian infants can be studied. In these cultures, or cultural subgroups, co-sleeping is the norm and SIDS rates remain the lowest in the world (36–39)—even when they immigrate to non-co-sleeping cultures, although it is not known if they continue to practice co-sleeping (40,41).

Ironically, the electrophysiological studies my colleagues and I are conducting, combined with our analyses of videotapes of infant–mother co-sleeping, are the best way to investigate the effects of social behavior or sleep contact on physiological functioning (and vice-versa)—be they advantageous or potentially deleterious.

## III. PEDIATRIC SLEEP DISTURBANCES: AN OVERVIEW OF SOME CULTURAL ISSUES AND INTERVENTION METHODS PERTAINING TO CO-SLEEPING

**Abraham Sadeh**

Parents struggling with infants and especially young children to get them to sleep and to stay asleep seems

to be an all-too-frequent American preoccupation. Sleep-disturbed infants often spend more time awake at the expense of quiet sleep. Surveys based on parental reports indicate that 20–30% of children have sleep problems, including multiple and/or prolonged night awakenings, or difficulties falling asleep. In fact, some of the major complaints heard by pediatricians are sleep problems that are associated with otherwise healthy and well-developed children (42).

McKenna suggests that frequent arousals may be in the infant's biological best interest. Likewise, he suggests that a later, rather than earlier, developmental consolidation of nighttime sleep might be safer. If these ideas can be supported, it means that clinicians and parents could place less negative meaning to a night waking during the first year of life. Some parents and infants may benefit from a less rigid set of expectations regarding where and how infants should sleep, thereby diminishing both parental and infant distress. There is a need for information on how parents manage their infant's sleep and which management strategies enhance or diminish the amount of satisfaction parents experience as they assist in the development of their infant's sleep behavior.

### A. Causes of pediatric sleep disturbances

The most common causes of pediatric sleep disturbance in children older than a year may be problems in parent–child interaction. Patterns of daytime physical contact or proximity, bedtime rituals, feeding style and, ironically, co-sleeping itself are also associated with sleep problems. For example, several studies indicate that "reactive" co-sleeping, i.e. temporary co-sleeping that occurs in response to ongoing sleep disturbances or temporary problems (i.e. frightening or traumatic experiences), tends to exacerbate childhood sleep problems (43–46). If parents perceive that co-sleeping constitutes a negative situation to begin with, and it represents a last resort measure in dealing with ongoing sleep problems, conflicts are likely to continue, especially if the parents may see themselves as exhibiting deficient parenting skills or see deficiencies in their infant who refuses to sleep alone or without intervention.

On the other hand, when co-sleeping occurs from birth as an elective sleep management strategy and if parents feel comfortable or positive about the practice itself—even when the infants awaken frequently—the infant's sleep pattern is not necessarily perceived by the parents as being a problem (43,47). Social and psychological factors clearly play an important role in determining how parents perceive that a problem exists and for whom.

### B. Parental intervention

We studied two different treatment processes in 50 sleep-disturbed infants: the "checking" and "co-sleeping" intervention methods. Sleep-disturbed infants were infants and toddlers aged 9–24 months, whose parents were referred to our Sleep Disorders Center for help because of their infants' sleep disturbance. Sleep for these infants was characterized by an average of 4.3 night awakenings each night, which is more than what is generally described in other clinical studies. The "checking" intervention consisted of limited involvement by a parent. The parent was instructed to approach the crying or protesting child every 5 minutes and to restore the child to a sleeping position before leaving the room. A modified "co-sleeping" intervention was also used, whereby one parent slept near the child's bed or crib in a separate bed for a full week, with no physical or social interaction with the child during the night (48).

A decrease in the amount of night waking and an increase in infant sleep time followed both types of intervention. When infants did wake up, some from each of the two groups learned to return to sleep without signaling by crying. Improvement in sleep during the intervention was characterized by a decrease in night wakings of more than one night waking per night on average, and an increase of more than 3% in sleep percent (the amount of time the infant spent in actual sleep from sleep onset to rise time). A follow-up conducted 6 months after the conclusion of the intervention, however, revealed that more than 50% of the children in both groups had experienced relapses subsequent to environmental or social changes and stresses, suggesting that, perhaps, sleep-disturbed children have special vulnerabilities or possibly increased tendencies to develop sleep problems in response to environmental stressors.

Although in our study a limited and specific kind of co-sleeping was found to be helpful in improving sleep in sleep disturbed infants, co-sleeping may be more influential during a particular developmental phase. At an early age, co-sleeping is reported to affect sleep–wake state regulation and organization. Newborns who sleep with their mothers in the same room spend more time in quiet sleep and less time crying and in indeterminate sleep compared with newborns who sleep in a separate room (21). However, a parent sleeping with a newborn is very different physiologically and socially from a parent sleeping with an 8-month-old—or later, with a toddler. Undoubtedly, the effects of co-sleeping change with development.

Of course, our research has focused primarily on healthy children, but sleep disturbances can also be caused by undiagnosed or subliminal physiological

conditions, such as milk allergies, ear or respiratory tract infections or other health problems. "Difficult" temperaments and lack of self-soothing capacity have also been considered contributing factors in infant sleep behavior (49–51). This means that regardless of environmental factors, there will likely always be some infants for whom sleep disturbances occur. I suspect that McKenna is right in suggesting that, at best, co-sleeping will only benefit some of the infants at risk for SIDS.

### C. Are night awakenings true sleep disturbances?

One interesting question raised by McKenna is whether we should continute to consider night awakenings as examples of sleep disturbances, rather than as normal patterns of infant or childhood sleep. I agree that the question of what constitutes a natural course of development of sleep–wake patterns during the first year of life and how to distinguish between the best interest of the infant and that of the parents (waking or sleeping through the night) deserves further investigation. It has been commonly accepted by clinicians that because the first 6–9 months of life constitute a period of rapid changes in sleep–awake patterns, parents should not consider intervention prior to the end of that period. However, recent studies (52,53) have focused on strategies that reduce the number of nocturnal awakenings beginning at birth, which may have adverse consequences in some cases if McKenna's concerns regarding the potential risks of solitary, consolidated infant sleep are correct.

## IV. SOLITARY OR SOCIAL SLEEP AMONG INFANTS AND CHILDREN: THE RELATIONSHIP ASPECTS OF SLEEP

**Thomas F. Anders**

### A. Parental concerns

Sleep problems have become the most frequent complaints of parents during well-baby visits (32). Several recent books advise parents how to solve their infant's sleep problems (32,54), but few studies provide the kind of data needed to shape these recommendations. Although sleep problems have traditionally been viewed as problems of individuals, as discussed in the previous section, regulation of sleep and waking states has both individual and relationship components.

When do parental concerns become sleep problems and why? What are the antecedents and consequences of concerns and problems? McKenna's work suggests that we re-examine the cultural meanings that parents and researchers place on nighttime awakenings and reconsider the role that values and cultural expectations play in creating the problems themselves. He does not question the fact that sleep problems exist, but suggests that, depending on the infant or child's age, both parents and professionals may be interpreting awakenings in an unnecessarily negative way and promoting unrealistic expectations that not all infants can, or necessarily should, meet (55).

### B. Patterns of solitary infant sleep

The vast majority of studies on infant sleep disturbances rely on maternal reports rather than on observations of actual sleep–wake behaviors or relevant parent–infant interactions. Time-lapse videosomnography shows that infants, during the first year of life, may awaken briefly one or more times during the night without disturbing their parents. Two-thirds of them return to sleep without crying, i.e. "self-soothers"; one third remain awake and finally arouse their parents, i.e. "signalers" (55–57).

"Self-soothers" were found to be more likely to use a sleep aid, such as a pacifier or their fingers, to assist them in falling asleep. Moreover, self-soothers were generally put into their cribs awake at bedtime and were able to fall asleep on their own, a pattern that is repeated after an awakening in the middle of the night. Older infants were more likely to be put into their cribs awake at bedtime and older infants were more likely to be able to make use of a sleep aid.

In our sample, 21 infants were videotaped for 2 nights at 3 weeks and 3 months of age and followed up at 8 months. Being asleep before being put into the crib at 3 weeks of age was common, especially at the beginning of the night. After an awakening in the middle of the night, however, even at this young age, there was an increased likelihood of an infant's being returned to the crib still awake. Thus, videotapes revealed that 3-week-old infants possess the capacity to fall asleep on their own at this age. It appears, therefore, that for the most part cultural norms and parent–infant interaction influence the way in which infant sleep patterns are expressed, irrespective of whether we want to evaluate these achievements as being positive or as potentially detrimental to the health of some infants—a possibility that McKenna raises. That cultural norms, rather than knowledge of the infant's evolutionary history (or biology alone), dictate how, when and if infants should sleep through the night, alone or with another, is an empirically significant observation worthy of further exploration.

### C. Summary

McKenna's perspective on infant sleep is both exciting and novel. It recognizes the importance of social, emotional and physiological regulatory mechanisms upon which the infant–caregiver relationship builds and infant survival depends—a viewpoint underlying

much of my own work (58,59). This combination of evolutionary, crosscultural and developmental data calls attention to the heretofore neglected importance of sensory exchanges between caregiver and infant in ways that developmental models alone simply don't. Given the negative physiological consequences of primate mother–infant separations and research showing that human parental contact affects the maintenance of infant homeostasis, it is reasonable to assume that sleep problems may arise from insufficient or inadequate dyadic regulation (55). Moreover, McKenna's (1) suggestion that, in a co-sleeping microenvironment $CO_2$ concentration from the mother's airstream may alter the infant's breathing environment, thereby inducing more rhythmic breathing, is in itself an idea that merits further study.

Our studies do not answer the question of why some infants or children seem to need special nighttime comforting, and we still need to know more about the relationship between attachment and waking interactions. A more rigorous definition of a problem sleeper is also needed, especially in light of the complex evolutionary arguments and potential consequences McKenna proposes. Only future research of both co-sleeping and solitary sleeping can answer these questions and those raised by McKenna and by our own research. Already it is clear, however, that nighttime interactions and sleep–wake regulation, whether in a solitary or social setting, should be of special interest to clinicians. Fundamental psychological, sociocultural and biological processes constantly interact, just as they did throughout the evolution of our species. They are embedded within the context of parent–infant relationships, one aspect of which is sleeping arrangements.

## V. INFANT SLEEP RESEARCH AND ECOLOGICAL VALIDITY

**Evelyn B. Thoman**

A new note has been sounded challenging the notion of "ecological validity", a concept first introduced by Brunswick in 1955 and used extensively by researchers interested in the ontogeny of sleep. McKenna's theory, which is based on an anthropological perspective and rooted in evolutionary theory, argues that co-sleeping (with mother) was the sleeping condition that prevailed throughout recent evolutionary history and, therefore, solitary sleeping conditions are not the most appropriate context for obtaining basic information on sleep ontogeny during the infant's first year.

For sleep researchers, McKenna offers a new and different definition of the concept of "natural ecology" and, hence, differing requirements for data considered to have ecological validity—at least at the species-wide level. He extends his argument with the proposal that co-sleeping should be more facilitative of the infant's development than solitary sleeping. Finally, he speculates that co-sleeping could possibly provide protection from SIDS to some infants.

I have no disagreement with McKenna over the fact that, during the evolutionary process in humans and other mammals as well, infant–parent co-sleeping was the prototypical behavior. It is also apparent that this practice changed only very recently in certain cultures, and changed most dramatically in western culture. Furthermore, I agree that, in view of the evolutionary history of co-sleeping, it is reasonable to ask whether co-sleeping might provide greater facilitation of neurobehavioral development than solitary sleeping for some infants. The basic questions then become: Under what circumstances? For whom? For how long? Individual differences among infants are great, especially with respect to sensory thresholds and, thus, the effects of environmental stimulation on their sleep patterns. Individual differences among parents are also great, including their goals for their infant's developmental course and the nature of the adaptations they can and want to make as parents. Thus, although I accept the notion that co-sleeping has evolutionary origins and that it should be very useful to investigate this form of early social interaction, I would urge more caution with respect to McKenna's assumptions and generalizations derived from the evolutionary evidence.

### A. Potential benefits and risks: A breathing teddy bear perspective

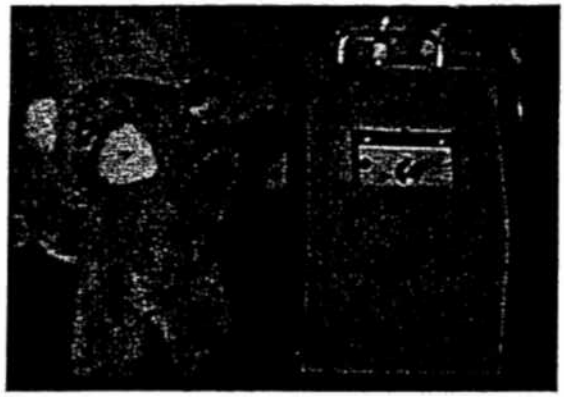
Before taking a stance on the controversial conceptual issues, I should like to give a brief description of research we have carried out, which is basically consistent with McKenna's view of the possible advantages of co-sleeping for infants. The studies have focused on premature infants, who were not designed by the evolutionary process to survive. During the preterm period, these fragile infants may show physiological decompensation, even from social stimulation. Any stimulation must be very carefully titrated for them. In order to accommodate to their vulnerability, as well as to individual differences in sensitivity to stimulation, we have developed a "surrogate companion" for preterm infants (60–62), which is made available to them but not imposed on them. It is designed to offer some of the tactile and kinesthetic and rhythmic stimulation McKenna suggests as being important for normal infants.

The surrogate is a Teddy bear, made to "breathe" by means of a specially designed pump (Fig. 5). The bear breathes quietly, in a smooth, sinusoidal fashion that mimics the breathing of a healthy, normal infant. The bear is individualized for each baby, not only

because it is optional, but also because the rate of breathing of the bear is set to reflect the breathing of the baby it is with.

In our studies of the bear as a "companion" for preterm infants, we have found that infants are dramatically responsive to it. Because of its placement in the isolette, the baby can make contact with the bear or move away. Premature infants are able to use their motor activity to find, touch and cuddle with the bear. Those with a breathing bear (in comparison to infants with a nonbreathing bear or no bear) show increased contact and shorter latencies to make contact over a period of 2 weeks. Infants who have had this opportunity for stimulation from 33–35 weeks conceptual age show more quiet sleep both during the preterm period and also during the early postterm weeks after discharge from the hospital. In addition, their respiration in quiet sleep shows greater regularity. Thus, the experience with the breathing bear apparently facilitates neurobehavioral development in these fragile infants at a time when social interchange must be limited and can even be overwhelming for them.

Clearly, this research suggests further investigation of McKenna's hypothesis that a real-live companion could be appropriate for more mature infants. However, it also highlights my concern for the generalization that co-sleeping may be the optimal circumstance for infant sleeping or that it is possibly safer than separate sleeping arrangements. Even for some infants more mature than prematures, it may be inappropriate. Thus, I seriously question the assumption made by McKenna that because a behavior pattern (e.g. co-sleeping) has survived the evolutionary process, it must be best for all individuals in modern western culture. Can those infants with specific vulnerabilities be identified?



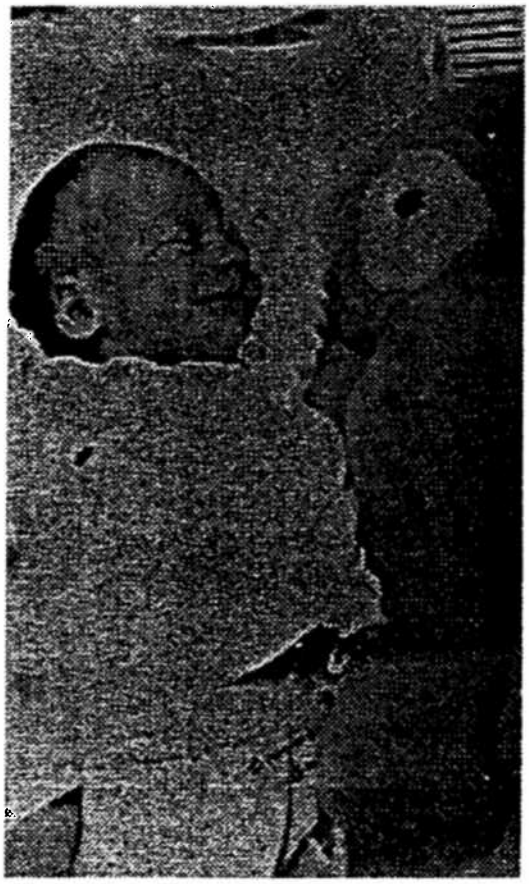


FIG. 5. The breathing Teddy bear and its sleeping companion.

### B. Ecological validity of social versus solitary infant sleep

The assumption that social sleep is the ecologically valid sleep situation is primary to McKenna's inference that SIDS might be diminished if co-sleeping were practiced (1). However, early evolutionary processes were neither kinder nor gentler than they are today. For example, in contrast to his assumption, it could reasonably be argued that during evolution, co-sleeping served to eliminate some of the "less fit" infants, say, those with fragilities or deficiencies that made them less able to respond vigorously to smothering and laying-over type experiences, which can occur during co-sleeping. Thus, there is reason to question the assumption that the co-sleeping environment is optimal because it is "natural".

There is a more serious reason to question the assumption that only co-sleeping conditions can provide ecologically valid measures of infants' sleep. McKenna argues that an ecologically valid approach to the study of infant sleep must include reference to how infant sleep develops in environments that most closely approximate the environments within which sleep behavior and physiology evolved, i.e. the co-sleeping environment. But this is not the way the concept of ecological validity has been, and continues to be, defined in research. The generally accepted meaning of ecologically valid study is that the research procedures permit description of the subjects behaving in their real world, without intervention. After its introduction by Brunswick in 1955, the concept of ecological validity was discussed and welcomed in the literature. Over subsequent years, it has become the accepted approach to "naturalistic" behavioral study up until the present.

Willems (63) characterized the goals very simply: "Ecological methods reveal, and discover, natural occurrences; they answer the question: What exists or what goes on here?" (p. 36). There are no criteria implied for whether conditions are "natural" other than that they are not contrived by the researcher. The emphasis is on "what is there in the behavioral world *unaltered by the investigator*? (p. 37). Thus, it is important to have measurement" (p. 38). While recognizing the importance of evolutionary processes in molding the sleep patterns of infants in past eons, the researcher is obligated to describe behavior as it is functioning in the here-and-now environment. Thus, if most infants sleep alone in western culture, description of solitary sleep is ecologically valid for western culture. Data obtained must be representative of any population for generalization to that population.

The issues I have raised concerning the uses of the terms "natural", "ecological validity" and "optimal" are more than quibbles. The concepts we use provide the framework for our scientific logic.

My collaborators and I have claimed ecological validity for our own studies of infant sleep over the past two decades. Our procedures have included prolonged (7-hour) direct behavioral observations, which made it possible to assess measurement reliability for the sleep/wake state variables and developmental changes over the early weeks of life. As examples, we found that instability in infants' state characteristics during the early weeks predicted developmental dysfunction at later ages (64). Also, infants who showed stable sleep patterns when they were alone showed stable waking state patterns when they were with their mothers (65). Thus, the measures of sleep obtained are reliable and valid for the population of infants studied.

Most recently, we have developed an automated procedure that permits continuous monitoring of an infant's sleep without instrumentation of the baby. In this way, the sleep of infants can be recorded for 24-hour periods in the home with minimal intervention. The procedure for the home monitoring system (HMS) uses a pressure-sensitive mattress pad, from which a single-channel recording from respiration and body movements is obtained. It is possible to code the signals for active sleep, quiet sleep, active–quiet transitional sleep, sleep–wake transition, wakefulness and periods out of the crib (66,67). Our studies using the HMS have also demonstrated measurement reliability, developmental changes and rhythmicity in state parameters during the early postterm weeks in both premature infants and fullterms. In addition, state patterns during the early weeks are predictive of later developmental status (68). It is reasonable to conclude that this procedure is also reliable and valid for study of the population of infants from which the samples were selected—that is, solitary sleeping infants in western culture, sleeping in their typical environment with a minimal "system disturbance" introduced by the research methodology.

### C. Summary

There is no disagreement among us that co-sleeping is a phenomenon that merits investigation in terms of the nature of the potential effects on the early development of sleep and the mother–infant relationship. I would agree that it is reasonable to hypothesize that, in some ways and for some parent–infant pairs and for some duration of time, co-sleeping may facilitate development during the first year of life, as McKenna suggests. But I do not accept the argument that co-sleeping is necessarily optimal for all infants and I do not agree that there is only one way to define what is

"natural", or ecologically valid. What is "natural" in one family and in one culture and in one era may not be natural, or valid, in a different environment at a different time. Accordingly, I contend that the study of either social or solitary sleep can be considered ecologically valid if the conditions for sleeping are those that are typical for the families studied. Each behavior pattern occurs in its own ecological niche. McKenna's ideas are most important for reminding us that results from studies of solitary-sleeping infants apply only to solitary-sleeping infants.

## VI. CO-SLEEPING AND THE SUDDEN INFANT DEATH SYNDROME

**Vicki L. Schechtman**

### A. The sudden infant death syndrome (SIDS)

The sudden infant death syndrome (SIDS) is the leading cause of postneonatal infant mortality in North America (69,70). Victims of SIDS are apparently healthy infants, predominantly 2–6 months of age (71,72), who are typically discovered dead in their cribs following a sleep period. Autopsies on SIDS victims demonstrate no apparent cause of death.

### B. Cardiovascular differences in infants who succumb to SIDS

Because SIDS by definition has no clear predictive symptoms, is a postmortem diagnosis of exclusion and has a relatively low incidence, obtaining physiological data from SIDS victims prior to their deaths is very difficult. In a major collaborative study aimed at identifying possible physiological abnormalities in SIDS infants, prospective 24-hour recordings of respiratory movement and ECG were performed on almost 7,000 normal full term infants (73). Sixteen of these infants subsequently died and were diagnosed as SIDS victims by autopsy. Because six of these SIDS victims were recorded on two occasions at least 1 month apart, 22 recordings of infants who later died of SIDS were obtained. With the aid of an automated infant sleep state classification system based on cardiac and respiratory measures (74), several differences have been identified between recordings of SIDS victims and recordings of age-matched control infants.

Prior to 1 month of age, SIDS victims exhibited significantly higher heart rates during all sleep–waking states than age-matched controls. During REM sleep, SIDS victims had higher heart rates than controls in both the first and second months of life. Although as a group these SIDS victims differed significantly from control infants, most SIDS victims had heart rates within the normal range. Thus, this difference does not help predict which individual infants will die of SIDS. Nonetheless, our findings provide a valuable piece of information: because differences in heart rate were apparent as early as the first month of life in infants who later died of SIDS, the factors predisposing some infants to SIDS seem to be encountered at a very early age.

Furthermore, SIDS victims showed diminished heart rate variation at the respiratory frequency (75,76), and lower frequencies during periods of rapid eye movement (REM) sleep and waking (76). These differences were significant even after the heart rate differences were factored out. This reduction in heart rate variation may reflect a difference in autonomic response patterns or the reduction in motility identified by other groups in infants at risk for SIDS (77,78).

Infants who subsequently died of SIDS also showed significantly fewer central apneic pauses in both sleep states (79) (Fig. 6). This difference appeared only during the second month of life. This finding suggests a difference in respiratory control immediately preceding the epidemiological period of maximal risk for SIDS that was not present earlier. This change may, therefore, be associated with other changes in the control of respiration, which make some infants vulnerable to SIDS during this developmental window.

### C. Environmental antecedents of SIDS

Although many physiological differences have been identified between groups of infants who subsequently died of SIDS and infants who survived infancy, the fact remains that no predictive differences have been found for individuals. Furthermore, the vast majority of infants at increased risk for SIDS, such as infants who have suffered an apparent life-threatening event and subsequent siblings of a SIDS victim, are physiologically similar to SIDS victims (80–84), despite the fact that the vast majority of these infants do not die of the syndrome. Therefore, environmental factors play the most likely role in determining which infants will ultimately die. Epidemiological findings, including the increased risk of SIDS during the colder months (85–89), also implicate environmental factors. Co-sleeping introduces a variety of additional factors into the infant's microenvironment, all of which may have consequences for SIDS risk.

Many researchers agree that SIDS is a multifactorial disorder and that SIDS victims do not share a common etiology, although "failure to arouse" from sleep to cope with homeostatic challenges may be in the final common pathway. During the neonatal period, respiration is largely controlled by brainstem centers, as

are most bodily functions. As forebrain connections develop over the first few postnatal months, the cortex acts to modulate brainstem reflexes with coordinated responses mediated by higher neural centers. It has been proposed that SIDS deaths may occur in response to various respiratory challenges encountered during this critical transition period when brainstem reflexive respiratory control has begun to be suppressed, but before forebrain regulatory systems are fully mature (90–92). If some SIDS deaths are, in fact, triggered by respiratory challenges during a particularly vulnerable (transition) developmental period, any factor that reduces the likelihood or severity of respiratory challenges or that increases the infant's tolerance to these challenges may reduce the infant's vulnerability to SIDS.

### D. How co-sleeping might reduce risks

Infant respiration is very responsive to temperature, mechanical and chemical stimuli (93–97). In all three of these respects, the co-sleeping environment may be a favorable one.

#### *1) Temperature*

Infants have a limited capability of behaviorally manipulating their thermal environment. Thus, they often remain heavily bundled throughout the night or may be uncovered for long segments of the night, regardless of ambient temperature. Increases in environmental temperature can increase both the frequency and duration of apneic pauses in premature infants (98–99). It has been postulated that hyperthermia, due to overwrapping, may be the cause of many SIDS deaths (100–103). Co-sleeping with an adult, who is capable of manipulating the thermal environment as needed, could increase the likelihood that the infant's environmental temperature would remain within a safer range throughout the night. Thermoregulation in infants and its possible link to SIDS will be discussed in greater detail by S. Glotzbach (below).

#### *2) Movement*

Mechanical stimuli also play a role in infant respiratory drive. In addition to exerting positive influences on the health and physical development of premature infants (104,105), rocking and other types of movement have been shown to reduce the frequency of apneic pauses in infants (106). The constant movement associated with parental respiration and gross body movements, as suggested by McKenna (1), may be a stabilizing force for vulnerable infants, especially during the period of maximum vulnerability.

#### *3) Altered chemical environment*

The increased $CO_2$ content of the co-sleeping infant's microenvironment (resulting from parental expiration

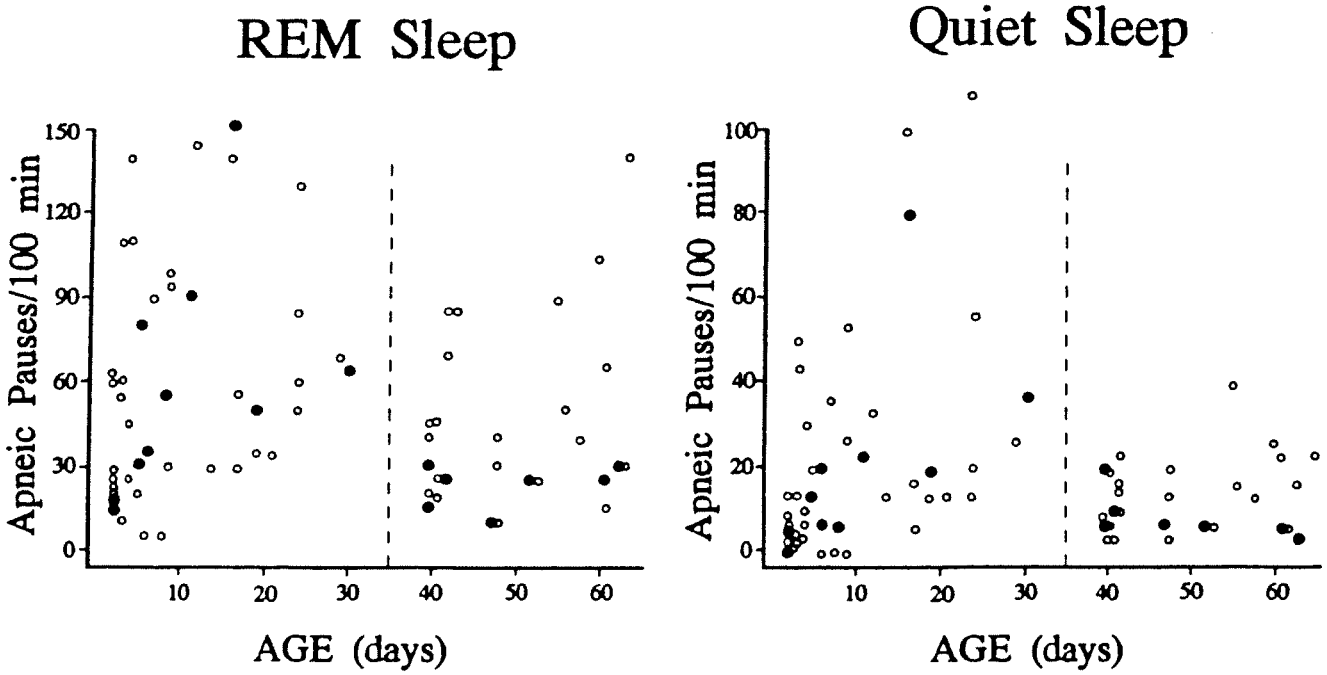



FIG. 6. Apneic pauses of 4–30-second duration per 100 minutes in each sleep state in SIDS victims (filled circles) and control infants (open circles). After 1 month of age, the SIDS victims showed significantly fewer respiratory pauses than age-matched control infants. (Figure reprinted from Schechtman et al. 1991.)

of air with a $CO_2$ content slightly higher than that of normal room air) may increase respiratory drive in infants.

### E. Airway obstruction in SIDS and co-sleeping

Petechiae identified on the lungs and thymus of a large number of SIDS victims (107–110) suggest that, in many cases, the terminal event may have been associated with an obstructive apnea. The frequent arousals associated with co-sleeping, as seen in McKenna's preliminary results, might also reduce the occurrence of obstructive apneas or, as he suggests, provide infants with additional experiences in the task of arousing more efficiently.

### F. Summary

SIDS risk likely represents a continuum, some infants being at high risk and others at very low risk. Environmental factors most likely play a role in determining which infants will ultimately succumb. These infants at highest risk for SIDS may well benefit from a co-sleeping environment.

## VII. CO-SLEEPING AND INFANT THERMOREGULATION

Steven F. Glotzbach

### A. Co-sleeping: New questions

Investigations on the development of sleep, temperature regulation and biological rhythmicity in infants have almost exclusively involved solitary sleeping infants. Co-sleeping presents a new and important context for investigating interactions between temperature and sleep in infants. Because of the interactions between sleep and temperature and the many potential avenues by which temperature could play a role in SIDS (70,111), it is important to examine the thermal impact of co-sleeping vs. solitary sleeping environments on sleep and arousal. These data are especially timely because Mitchell et al. (35) report an association between bed sharing and increased risk of SIDS in New Zealand, suggesting that co-sleeping may overheat the infant.

### B. Preterm infants, diurnal cycles and co-sleeping

In the solitary sleeping environment, infants are deprived of many cues that were present in utero. The situation for preterm infants, who are at increased epidemiological risk for SIDS compared to full term infants (41), may be worse—they are not only denied the full benefit of the uterine environment to term pregnancy, but also may spend 3–4 months in an intensive care nursery (ICN). There is great spatial and temporal variability in light in the intensive care nursery, with no clear diurnal pattern. At most sites, ambient and supplemental lighting regimes preclude any diurnal periodicity in light intensity (112). Although these infants can be considered to be solitary sleepers during their hospitalizations, the noise and interventions in the nursery provide continual stimuli to the infants. There has been concern that this stimulation may interfere with the development of sleep during a critical period of central nervous system maturation.

What is known about the development of the diurnal cycle of body temperature? The preterm infant has a very small diurnal fluctuation in body temperature (113,114), the amplitude of which increases with increasing age. In many of these infants, a servo-control probe attached to the abdomen or back is used to maintain the infant's body temperature at a fixed level. Given the small amplitude body temperature rhythm seen in the preterm infant, it is of interest to discover how the neonatal intensive care unit influences the evolving temperature rhythm and, hence, other developing systems, such as sleep and wakefulness. It would be of great interest to evaluate the effect of co-sleeping on the development of the core temperature rhythm in both preterm and full term infants, which has been studied only in solitary sleeping infants (114,115). Factors that influence the development of the core temperature rhythm are likely to affect the maturation of other systems, such as sleep.

### C. SIDS and body temperature

In addition to the analysis of temperature as a marker of the circadian system, it is important to consider thermoregulatory homeostasis and the impact of environmental temperature on sleep in co-sleeping vs. solitary sleeping infants. It is well known from numerous studies on adult humans and animals that (a) thermoregulation is influenced by both sleep and circadian factors, (b) body temperature is regulated at a lower level during NREM sleep than during wakefulness, (c) during REM sleep there is a marked inhibition of thermoregulation and (d) body temperature and the thermal environment are important determinants of arousal state distribution. However, there is a paucity of data on the interaction of temperature, thermoregulation and sleep in infants.

In the normal full-term infant, thermoregulatory effector mechanisms are operating soon after birth. In particular, intact heat-loss systems have very impor-

tant implications for SIDS. Unfortunately, little data are available on the changes in these effector systems during sleep state transitions or how the response characteristics change in REM sleep compared to NREM sleep. Specifically, much more information is needed on thermoregulation during REM sleep in infancy. In fact, there is some evidence that thermoregulation in infants may not be as inhibited during REM sleep as it is in adults (116). This question may be very important considering the relatively high percentage of REM sleep in the first postnatal weeks.

It is important to examine changes in the characteristics of the thermoregulatory system during the postnatal period from 2–4 months, when the occurrence of SIDS is highest. Wailoo et al. (117) studied the changes in rectal temperature during the night in 67 normal infants at 3–4 months of age. Recordings were done in the home, the infants were in a solitary sleeping environment and data were normalized to time of sleep onset. Rectal temperature fell rapidly, from about 37–36.4°C within 90 minutes of sleep onset, and started to rise before the end of the night. Additionally, the fall of rectal temperature was related more to sleep onset than to the time of day.

The range of rectal temperatures recorded in these normal infants was from 35.9–38.5°C (96.6–101.3°F). Furthermore, the time course of the decrease in body temperature appears to depend minimally on the environment, indicating a well-organized, endogenous temperature rhythm. An infant in a warm room, who also had twice the insulation as a control infant, showed an identical profile of rectal temperature vs. time compared to the control infant, who was in an "ideal" environment (118). In the warm environment, regulation of body temperature was achieved by increasing evaporative water loss from the forehead and by exposing the head and limbs to enhance heat loss.

A primary reason for studying the maturation of thermoregulatory responses in infants is that temperature may play a key role in SIDS mechanisms. In some infants, there may be an intrinsic problem of body temperature control. Naeye et al. (119) found that some SIDS victims had experienced more spontaneous bouts of both hypo- and hyperthermia compared to control infants, and Kahn et al. (120) have noted that many infants considered to be at higher risk for SIDS are observed to sweat more than normal infants. It is also well known that temperature can modulate sleep state distribution, with decreased sleep outside of thermoneutrality and a relatively lower percentage of REM sleep as ambient temperature deviates from thermoneutrality. Warm temperatures in the upper thermoneutral zone could actually enhance REM, the importance of which will depend on the status of thermoregulatory control in infants during this sleep state. Warm temperatures have also been observed to result in more apneic periods and in reduced upper airway patency.

Many SIDS victims are reported to have had a mild illness just prior to their death. Either fever or sleep deprivation resulting from the illness could increase the number of obstructive apneic events, as was reported recently by Canet et al. (121), and could result in increased postdeprivation sleep and an increased arousal threshold. The arousal threshold to thermal stimuli during sleep in infants is currently under investigation (122), although preliminary results do not show a significant difference in arousal (during quiet sleep) to cool or warm temperatures in SIDS siblings vs. control infants. However, it is clear that there are many potential routes by which temperature could influence SIDS via the interaction of temperature with sleep, respiratory control and arousal mechanisms (70,111).

All of the studies reviewed have been conducted on solitary sleeping individuals. Using heat transfer theory, we can contrast the co-sleeping vs. the solitary sleeping environment to evaluate some of the possible effects of temperature on sleep in these two situations. First, the solitary sleeping environment is a relatively simple system, with a two-component interaction between the infant and his or her surroundings. Second, this scenario is characterized by a stable or slowly changing thermal environment. Third, the infant's insulation, or "bundling level", could be considered fixed at bedtime, with little or no feedback. However, some qualifications of these remarks are necessary. There is some thermal contact during the night when the mother or caregiver feeds the infant. Additionally, Wailoo et al. (123) found that over 75% of infants 3–4 months of age disturbed the parents at least once during the night. Moreover, the waking pattern was related to the thermal environment of the infant: the higher the insulation or room temperature, the more often babies awoke their parents.

About two-thirds of the infants who aroused their parents were found sweating. It is possible that if a baby can't arouse in response to a high environmental temperature (or if a parent can't respond), then the infant may be at higher risk for SIDS. In contrast, the thermal features of the co-sleeping environment provide more complex interactions, with the parents, infant and surroundings influencing each other. Moreover, there will be dynamic thermal stimuli as the position of the parents changes relative to the infant. The parents can also provide immediate protection to the infant from inappropriate thermal environments, i.e. by regulating room temperature.

### D. Summary

Several points warrant emphasis. There will likely be more thermal variability for the infant in the co-sleeping environment, which could be good or bad. Variability could be good by keeping the infant "aroused", but might be bad if arousals were too frequent or caused significant sleep disruption. Just as McKenna argues that infants may benefit from "learning to arouse", it could also be argued that consolidated sleep provides the opportunity for infants to improve their ability to respond to homeostatic challenges during sleep. Infants in the co-sleeping environment may have more problems keeping cool, and it would be of interest to examine the relationship between the core body temperature rhythm and sleep architecture in the co-sleeping vs. solitary sleeping infant. Finally, it is important to remember that in either solitary or co-sleeping environments, temperature interacts with other systems and is an important modulator of sleep distribution and breathing. From a thermal standpoint, however, it is not possible at present to conclude whether co-sleeping modifies sleep architecture or the risk of SIDS.

## VIII. SYNOPSIS

Human infants are different from other mammals insofar as the central nervous system is exceedingly undeveloped at birth. Developmental studies support the contention that their bodies have been designed by natural selection to be highly responsive to contact with a caregiver on whom—and for a considerable period of time—the infant's survival depends.

McKenna puts forth several different but interrelated proposals concerning the evolutionary history of parent–infant sleep and hypothesizes that for some possibly small subclass of SIDS-vulnerable infants, parent–infant contact throughout the night (either directly through physiological mechanisms or indirectly through parental manipulation) may help them to override challenges that could otherwise result in SIDS. Paleoanthroplogical and archaeological studies of human evolution, integrated with crosscultural data on human behavior, and evolutionary theory itself can serve as an unbiased beginning point for conceptualizing biomedical problems—especially those like SIDS that remain enigmatic.

To understand all aspects of infant sleep ontogeny and the ways in which the environment shapes both short- and long-term sleep patterns, all of us agree that studies of infant–parent co-sleeping are important and necessary. Thoman reminds us that solitary sleep studies are important to cultures where infants continue to sleep in solitary environments and where early sleep independence is encouraged for a variety of cultural reasons. She disagrees with considering co-sleeping as more "natural" than solitary infant sleep, at least for the populations for which solitary sleep is the norm.

Others of us question the validity of clinical models and experimental data on the development of infant sleep in the first year of life because these data are constructed exclusively from solitary sleeping infants. McKenna contends that no matter how culturally appropriate solitary infant sleep studies may be and no matter how nonintrusive studies of solitary sleeping may be, by virtue of separating the infant from a co-sleeping partner, these studies will always miss important evolutionary-based social, psychological and physiological benefits accruing to infants. These potential benefits can only be elucidated if new studies are undertaken comparing normative data from solitary-sleeping infants with co-sleeping infants. Moreover, the possibility exists that western attitudes underlying biomedical research into infant sleep and definitions as to what constitutes "normal" infant sleep give rise to unrealistic expectations about where and how infants "should" sleep. The extent to which overly rigid, culturally-based parental attitudes and the attitudes of health professionals themselves create the conditions within which parent–infant sleep struggles emerge is a question pediatric sleep researchers need to consider.

The relationship between co-sleeping and SIDS, as McKenna proposes, is the most problematic issue raised here. However, preliminary physiological data on co-sleeping mothers and infants in the laboratory provide a beginning point for a variety of new questions pertinent to future research that could address the hypothesis more directly. For example, biological rhythms, arousal patterns (frequency, timing), sleep architecture, cardiac integrity (rate and variability as well as coherence between infant and maternal activity), respiratory rate and variability, thermoregulation including parental interventions, infant body positions (prone vs. supine), infant motility patterns and infant oxygen saturation as infants sleep in solitary vs. co-sleeping environments are all important parameters in need of further investigation.

Based on his anthropological research, McKenna argues that cultural values advocating individualism, autonomy, and independence—and caregiving strategies that are believed to promote these values—are an inherent part of the underlying assumptions found in both SIDS and infant sleep research. The question is, does it really matter? In disagreement with Thoman, he suggests that these unrecognized assumptions have not only steered researchers away from conceptualizing

the extent and significance of the biological dependence of the human infant on the caregiver, but also prevented us from obtaining a more comprehensive, species-wide (universal) picture of infant sleep ontogeny that is important for understanding infant health, regardless of their cultural ecologies. In our enthusiasm to view the infant as a competent organism (which, clearly, it is), he maintains that we have pushed too far the notion of the infant's physiological independence from the caregiver, thereby confusing its preparedness to adapt with actual adaptation, taken here to mean the assumption of the infant's physiological autonomy (1,30). Separate sleeping arrangements, advocated by health professionals for all parents and infants, are but one of the many manifestations of this view, according to McKenna.

Although we enjoy tremendous medical advantages over nonindustrial western people, the link between the infant's biological status and co-evolved patterns of parental care cannot be understood only by examining infancy in its contemporary biocultural context. Hunting, collecting and foraging and, indeed, parent–infant co-sleeping represents the evolutionary context within which modern humans were sculpted and designed biologically and psychosocially for well over 95% of our existence as a species. Mismatches between recent cultural changes in child care practices and the more slowly changing biological needs of infants that emerged throughout millions of years of human evolution may emerge as the source of many physiological and psychological disorders.

Evolution never promised us a rose garden, to stretch Thoman's perspective just a bit, but it can't be all wrong either. Future research on co-sleeping promises to reveal some unexpected new relationships tying parental behavior and infant physiology together in unanticipated ways. Moreover, there is consensus here that co-sleeping research will elucidate new insights into developmental norms and offer an important opportunity to compare co-sleeping and solitary-sleeping data. Conceivably, this research direction may yield data relevant to one of the most significant medical mysteries of the twentieth century, SIDS, for which biomedical research models have yet to prove adequate.

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