EVOLUTION AND THE SUDDEN INFANT DEATH SYNDROME (SIDS)

Part III: Infant Arousal and Parent–Infant Co-Sleeping

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This paper extends the evolutionary and developmental research model for SIDS presented in previous articles (McKenna 1990a, 1990b). Data from variety of fields were used to show why we should expect human infants to be physiologically responsive in a beneficial way to parental contact, one form of which is parent–infant co-sleeping. It was suggested that on-going sensory exchanges (touch, movement, smell, temperature, etc.) between co-sleeping parent–infant pairs might diminish the chances of an infantile cardiac-respiratory crisis (such as those suspected to occur in some SIDS cases).

In this article we review recent epidemiological data and sleep research findings on SIDS to show how they relate to evolutionary and cross-cultural perspectives. Results of a preliminary study of the co-sleeping behavior of mother–infant pairs indicate that, with respect to sleep, arousal, and respiratory patterns, co-sleeping mother–infant pairs affect each other in potentially important ways. We suggest specifically that co-sleeping may shorten periods of consolidated sleep among young infants by causing them to arouse more frequently. Moreover, we suggest that partner-induced arousals might help the infant to confront sleep crises more competently. In the long run, these arousals might prevent the premature emergence of prolonged (adultlike) sleep bouts from

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which some infants have difficulty arousing—especially during a breathing pause or apnea.

KEY WORDS: **Sudden Infant Death (SIDS); Mother–infant co-sleeping, synchrony, arousals**

A recent study of the behavioral risk factors associated with sudden infant death found that among the 24 victims included in the sample the behavior with the greatest predictive probability was the degree of difficulty these infants had in awakening (Einspieler et al. 1988). Similarly, Hoppenbrouwers et al. (1989:269) compared the sleep patterns of subsequent siblings of SIDS victims with those of members of a control group. Although they found that similarities between the two study groups "outweighed" the differences, the siblings of SIDS victims tended to awaken less frequently and, once asleep, "exhibited a higher probability of remaining asleep than the controls."

Although the findings that some SIDS victims may have difficulty arousing, or switching from sleep to wakening, are not new (see Guntheroth 1982; Harper et al. 1981; McGinty 1984), until recently there has been no theoretical context within which this issue could be analyzed further. Nor have there been any behavioral mechanisms known that might contribute to our understanding of how infantile constitutional deficits involving arousal might be affected or somehow influenced by the infants' microenvironment either to increase or to decrease the role of arousal in some SIDS pathologies. Recent epidemiological and cross-cultural data on SIDS suggest, however, that SIDS rates are lower in cultures in which parent–infant co-sleeping is the norm. Our own behavioral data on the breathing, sleep, and arousal behavior of co-sleeping mother–infant pairs (McKenna et al. 1990) and several promising hypotheses developed by Hoppenbrouwers and Hodgman (1986) and Sterman and Hodgman (1988) provide a beginning point for a more comprehensive analysis.

This paper will evaluate these recent studies and, where appropriate, integrate them with the evolutionary and developmental research hypotheses described in previous articles (McKenna 1990a, 1990b). Briefly, McKenna argues that because the human infant is less neurologically mature at birth than all other primate infants, we would expect nocturnal physical contact with a parent to provide some physiological benefits. These postulated physiological benefits most likely emerge from parental sensory stimuli, which may function to help high-risk infants to either avoid or more successfully confront certain forms of sleep crises associated with some kinds of SIDS.

Both epidemiological and laboratory SIDS research continues to suggest a multifactorial origin for the syndrome and to find an impressive amount of heterogeneity among SIDS victims (see Hoffman et al. 1988). The physiological interactions that occur between co-sleeping mothers and infants may have been favored by natural selection specifically because they increased infant survivorship over the past 4 million years of hominid evolution, when parent–infant co-sleeping was the norm. Thus, research into the physiological consequences of both solitary and parent–infant co-sleeping is justified. We suggest that to study the sleep, breathing, and arousal patterns of healthy or high-risk infants only in solitary sleeping situations is to ignore the evolutionary context within which infant sleep patterns developed. Moreover, an exclusive focus on solitary sleeping infants most likely has produced an understanding of the development of first-year infant sleep patterns that differ from more universal (species-wide) human patterns, which develop under more "natural" social conditions.

The preliminary data summarized here (see McKenna et al. 1990) represent the first of many steps that will be required to test the hypothesis that, insofar as it may help to protect some infants from SIDS, the sensory-rich microenvironment created by parent–infant co-sleeping is advantageous over solitary infant sleeping arrangements (McKenna 1986, 1990a, 1990b). Microenvironment refers specifically to the immediate area within which the infant sleeps. It includes the concentration of gases (especially CO_2) in the air surrounding the infant's head, ambient as well as blanket temperature affecting infant core temperatures, humidity, and all stimuli (both external and proprioceptive) processed by the infant's nervous system, such as all sounds, sound cessations, movements, touches, or smells made by others with whom the infant is either in proximity or in contact.

We begin with a review of the most recent SIDS epidemiological research and move to a discussion of infant sleep research, SIDS rates in cross-cultural perspective, and a summary of our own preliminary study of mothers and infants sleeping in the same bed (see McKenna et al. 1990). Our findings that co-sleeping mothers and infants communicate and affect each other physiologically throughout the night are discussed in relationship to current SIDS theories that propose a possible link between infantile sleep arousal deficiencies and SIDS among one of several subclasses of SIDS victims.

SIDS EPIDEMIOLOGY RESEARCH UPDATE

Table 1 summarizes the most important SIDS research findings through 1989, some of which will be discussed in more detail below. Compre-

Table 1. Suspected or Implicated Causes of Sudden Infant Death Syndrome

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Neurological, Neurochemical, Respiratory, or Sleep Dysfunction		
Decrease in maturation of quiet sleep	Gould et al. 1988	
Premature maturation of sleep and arousal mechanisms	Sterman and Hodgman 1988	
Hypoxia or hypoxemia	Steinschneider 1972 Naeye 1974, 1976, 1980 Guntheroth 1982, 1983a Kelly 1983	Singer 1984 Tildon et al. 1983 Merritt and Valdes-Dapena 1984 Beckwith 1988
Protracted periods of apnea or increased breathing pauses	Steinschneider 1972 Guilleminault et al. 1975 Guilleminault, Tilkian and Dement 1976 Guilleminault, Souquet et al. 1976 Guntheroth 1982, 1983a Read and Jeffrey 1983 Kelly et al. 1980 Naeye 1974, 1980	Weinstein et al. 1983 Peterson 1983 Bagg et al. 1981 Hodgman et al. 1982 Hodgman and Hoppenbrouwers 1983 Southall et al. 1985 Hoffman et al. 1988 Beckwith 1988 Schwartz 1987 Southall and Talbert 1988 Southall et al. 1982
Deficiency of arousal response to increased CO_2 (hypercapnia) and decreased CO_2	Hunt et al. 1981 Sullivan 1984	Ariagno et al. 1980
Deficiency of arousal system; abnormal arousal levels in brain stem	Guilleminault, Ariagno, Forno et al. 1979 Guntheroth 1977, 1982, 1983a Guilleminault, Ariagno, Korobkin et al. 1979 Harper et al. 1981 Hunt et al. 1981 McCulloch et al. 1982 McGinty 1984	Guilleminault and Coons 1983

(continued)

Table 1. Continued

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Petechiae (broken blood vessels) on surface of lungs and general interthoracic vegim caused by central apnea	Guntheroth 1983b	Tildon et al. 1983
Intrathoracic petechiae owing to upper respiratory obstruction	Beckwith 1988 Werne and Garrow 1953	
Respiratory vulnerability during REM sleep	Henderson-Smart and Read 1978 Phillipson 1978	Johnson et al. 1983 Orr et al. 1985
Small, constricted, thickened pulmonary arterioles; increased muscle mass	Naeye 1973 Mason et al. 1975 Williams et al. 1979	Beckwith 1983 Singer 1984
Inability to maintain homeostasis during the developmental period wherein NREM sleep is prolonged and predominates	Gould 1983 Salk et al. 1974	
Respiratory muscle failure owing to muscular immaturity or respiratory paralysis	Jansen and Chernick 1983 Stanton 1984	Beckwith 1988
Overcompliant lung or defective surfactant	Southall et al. 1985 Southall and Talbert 1988	
Leukomalacia or cerebral white matter lesions caused by hypoxemia and inadequate blood circulation to brain (ischemia)	Takashima, Armstrong, and Becker 1978	Beckwith 1983 Haddad and Mellins 1983 Pearson and Brandeis 1983

(continued)

Table 1. Continued

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Abnormal retention of periadrenal fat and adrenal medullatory hyperplasia	Naeye 1976 Naeye et al. 1976	Emery and Dinsdale 1978 Patrick and Patrick 1982
Fatty changes (lipid-laden macrophages) in tegmentum of brain	Gadson and Emery 1976	
Undervascularized reticular formation; gliosis in brain stem	Valdez-Dapena 1988 Naeye 1976 Becker 1983 Takashima, Armstrong, and Becker 1978 Guilleminault, Ariagno, Forno et al. 1979 Takashima, Armstrong, Becker, and Bryan 1978	Guilleminault 1980
Delayed development of the vagus nerve (reduced number of myelinated fibers)	Sachis et al. 1981 Becker 1983	
Relative immaturity of brain stem (dendritic spines rather than more mature bundles)	Quattrochi et al. 1980 Baba et al. 1983 Gunby 1978 Quattrochi et al. 1984	Haddad and Mellins 1983 Pearson and Brandeis 1983
Abnormal auditory evoked potentials as a predictor of SIDS	Orlowski et al. 1979	Stockard 1981 Gupta et al. 1981
Carotid body abnormalities, reduced cell numbers, abnormal "glomic" tissue, or structural abnormalities	Naeye et al. 1976	Dinsdale et al. 1977 Beckwith 1983 Valdes-Dapena 1983
Increased levels of dopamine in carotid bodies	Perrin et al. 1984	

(continued)

Table 1. Continued

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Neck-Throat Abnormalities		
Hypertrophy of laryngeal mucous glands or increased number of mucous glands	Fink and Beckwith 1980 Haddad et al. 1980, 1981	
Hypermobility of mandible causing suffocation, pharyngeal upper airway collapse, or occlusion exacerbated by smaller upper airway	Tonkin 1974, 1975, 1983 Colton and Steinschneider 1980 Thach 1983 Sullivan 1984 Guilleminault et al. 1975 Guilleminault et al. 1986	Guntheroth 1982, 1983a
Elongated uvula	Harpey and Renault 1984	Guilleminault 1984 Guilleminault et al. 1986
Cardiac Abnormalities		
Right ventricular hypertrophy (enlarged right ventricle), indicating hypoxia	Naeye 1973	Beckwith 1983 Williams et al. 1979 Valdes-Dapena 1980a
Prolonged QT interval (time between ventricular contraction and relaxation)	Schwartz 1983, 1987 Southall et al. 1982 Froggatt and James 1973 Verrier and Kirby 1988	Kukolich et al. 1977 Steinschneider 1978 Haddad et al. 1979 Guntheroth 1982
Cardiac and autonomic inactivity leading to arrhythmias	Church et al. 1967	Gunteroth 1983a
Lethal arrhythmias	Schwartz 1976	
Lack of maturational synchrony in right and left sympathetic nerves, leading to increased heart rate	Schwartz 1983	

(continued)

Table 1. Continued

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Lower than normal variation in heart rate	Guilleminault 1988 Schwartz and Segantini 1988 Valimaki et al. 1988	
Metabolism		
Thiamin deficiency, when associated with high carbohydrate intake; general neurochemical defects	Read and Jeffrey 1983 Jeffrey et al. 1985	Tildon and Roeder 1983 Barker et al. 1982 Peterson et al. 1988
Thiamin excess; inability to absorb amounts found in cows' milk	Davis et al. 1983	
Vitamin D deficiency	Hillman et al. 1980	
Anaphylactic shock, allergic reaction to cows' milk (model based on data from guinea pigs)	Coombs and McLaughlan 1983 Guntheroth 1977	Valdes-Dapena 1978, 1983
Hypoglycemia owing to impaired glucogenesis or hyperinsulinism	Swift et al. 1974 Aynsley-Green et al. 1978	Sturner and Susa 1983
Elevated fetal hemoglobin levels	Giulian et al. 1987	Brown 1987
Defective liver enzyme, phosphoenolpyruvate carboxykinase (PEPCK)	Sturner and Susa 1983	Silverstein et al. 1983
Diminished pulmonary surfactant levels	Morley et al. 1984	
Environmental Triggering Factors		
Illnesses (colds, diarrhea, vomiting) in the last two weeks of life	Hoffman et al. 1988	

(continued)

Table 1. Continued

Observed Abnormality or Condition	*Investigators (Selected Studies)*	*Additional Studies or Critiques*
Lack of breast-feeding and contact with mother during night; co-sleeping, microenvironment	McKenna 1986 Konner and Super 1987 Hoffman et al. 1988 Sears 1985 Maxwell and Maxwell 1979 Davies 1985 Lee et al. 1989 Damus et al. 1988	Cunningham 1976 Beckwith 1979
Overheating	Fleming et al. 1990 Nelson et al. 1989 Stanton 1984	
Carbon monoxide poisoning	Cleary 1984	
Infant botulism *(Clostridium botulinum)*	Arnon 1983 Sonnabend et al. 1985 Cornblath and Schwartz 1976	Cunningham 1976 Valdes-Dapena and Felipe 1971
Sleeping position (prone)	Nelson et al. 1989	Beal 1988 Emery 1988 Brennan et al. 1988
Risks from Diptheria-Tetanus-Pertussis vaccine	Bernier et al. 1982	Griffin et al. 1988 Hoffman et al. 1988
Suffocation in water beds	Bass 1988 Ramanthan et al. 1988	Filardo 1988
Startle disease (hyperplexia)	Vigevano et al. 1989 Kaada 1986 Franciosi 1987	
Suffocation owing to co-sleeping	Luke 1978 Thach et al. 1988 Bass et al. 1986	
Breath-holding or fear, paralysis reflex	Southall, Talbert, Johnson et al. 1985 Kaada 1986	
Intrauterine perturbations	Hoffman et al. 1988	

hensive reviews of all aspects of SIDS research can be found in Bergman (1986), who reports on the history of SIDS research in the United States, and in Golding et al. (1985), Guntheroth (1982), McKenna (1986), Schwartz et al. (1988), and Valdes-Dapena (1980a, 1980b, 1988). As Table 1 illustrates, one of the most frustrating aspects of SIDS research continues to be the inability of researchers to replicate each other's findings.

The most recent, best controlled and designed retrospective epidemiological study (Hoffman et al. 1988) reported on 757 SIDS victims and 1600 control infants divided into groups according to age, race, and birth weight. The samples were drawn from six SIDS research centers scattered throughout the United States. Unfortunately, the NICHD SIDS Comparative Epidemiological Study failed to decide on a common postmortem marker for SIDS. In fact, the difficulty of diagnosing SIDS was underscored when expert panel members could not completely agree on the cause of death of 243 of the 1000 SIDS victims originally included in the study (see Valdes-Dapena 1988 for discussion).

Even so, this study makes significant contributions toward understanding SIDS risk factors. It revealed that about 90% of SIDS victims were less than 24 weeks old. Most SIDS victims generally had low birth weights (less than 2500 g), experienced slower overall (postnatal) growth rates than controls, and were more frequently born to unmarried and poor women who smoked during their pregnancies and who were less than 20 years of age. Interestingly, socioeconomic and behavioral factors, rather than maternal medical or health factors, were more significant predictors of SIDS risks when only maternal risk factors were analyzed (Hoffman et al. 1988). Postnatal risk factors of a small proportion of SIDS victims included mothers reports that prior to their deaths their infants experienced a "stop breathing episode" or turned blue. Most frequently these apnea episodes occurred when the infant was awake. Mixed apneas continued to be associated with some infants before their deaths from SIDS, but more frequently apneas were markers for low-birth-weight infants. Apnea proved not to be a specific risk factor for SIDS.

A significant number of infants who died of SIDS had bouts with diarrhea and/or vomiting and colds within 2 weeks of death. They also experienced droopiness and listlessness during the last 24 hours, as well as increased irritability, respiratory distress, and tachycardia (excessively rapid heartbeats; Hoffman et al. 1988). According to the investigators, all of these factors acted in "secondary fashion" rather than as primary or causal agents.

BREAST-FEEDING AS PROTECTION AGAINST SIDS

One of the important findings from this study is the "lack of breast-feeding" as a risk factor for SIDS.

> The fact that only 9.8 percent of SIDS cases were mostly or only breastfed, compared with 27.7 percent of control A infants (matched according to age) and 22.3 percent of control B infants (matched for race and birth weight), suggests that fewer SIDS infants received the benefit of the protective effect of breast-feeding (Hoffman et al. 1988:26).

Breast-feeding may be responsible for reducing the kinds of infections (those that induce electrolyte imbalances) associated with infants who died of SIDS (Hoffman et al. 1988). In addition, nursing infants are ordinarily fed more frequently than are bottle-fed infants (see Short 1984), necessitating increased maternal contact. More frequent feeding translates into increased sensory stimulation from the mother and potentially increased numbers of arousals, interrupting prolonged nocturnal sleep bouts of both the infant and the mother (Konner 1981; Short 1984). This pattern of sleep, we argue later, is more compatible with the infant's evolutionary past.

With regard to the physiological-immunological benefits of breast-feeding, it may be significant that SIDS rates peak at a time when maternal antibodies (IgG), abundant in the first 2 months of life, are declining, "generally reaching the lowest level at three months of age before the infant builds up its own immunoglobulin to achieve immunological independence" (Huang 1983:593; see also Arnon 1983). Of course, nursing can continue to protect the infant from a host of environmental assaults after this period, because it is through contact with its mother's nipples that the infant has almost a direct line to her enteroimmune system. As Arnon (1983) notes, however, mothers differ biologically in terms of the quantity and types of antibodies. Thus, the emergence of the infant's own functioning immunological system is a relevant developmental milestone when SIDS etiologies are considered.

Arnon's (1983) interpretation of SIDS begins with the observation that the consistent age distribution of SIDS victims is the most important clue to its pathophysiology. Arnon found that the age distributions of those infants who die from infant botulism precisely match the age distribution of SIDS victims. His contention is that human milk contains a maternal antibody (secretory IgA) that agglutinates and destroys the vegetative cells of *Clostridium botulinum,* the botulin bacterium whose ingested spores (unlike those of food-borne botulism) "generate, mul-

tiply, and produce their toxin (one of the most powerful toxins known) in the lumen of the baby's intestines" (Arnon 1983:539). The toxin can then be carried anteriorly to motor nerve endings, causing respiratory muscle paralysis and death. Arnon (1983) observes that some lymphocytes sensitized to foreign bacterial invasion in the mother's gut are known to migrate to the mother's nipple area to produce antigen-specific S-IgA antibodies within 3 days after the mother's contact with the bacteria. For the infant who ingests these immunobodies, this represents a previously unknown enteromammary immune system. Thus the infant who breast feeds is connected both to a lactating woman's intestines and to such immunological factors as lactoferrin, lysozyme, leukocytes, and S-IgA antibodies (Ogra 1979, cited in Arnon 1983).

The extent to which nursing infants were in more or less nocturnal contact with their parents, or if they slept mostly, partially, or at all with their parents before their deaths, was not made clear in the epidemiological SIDS study (Hoffman et al. 1988). These data could be important, however, because nursing on demand throughout the night is the pattern exhibited by modern hunters and gatherers, and mother–infant co-sleeping is quite likely the context in which infant sleep, arousal, and breathing patterns developed throughout most of human evolution (Short 1984). According to this evolutionary perspective, the fewest number of SIDS cases should occur within this microenvironment (McKenna 1986).

WHAT IS NORMAL INFANT SLEEP?

The developmental neurophysiology of sleep among normal and at-risk infants as well as their respiratory and cardiac patterns are being studied by SIDS researchers from many different disciplines (see Table 1); however, none of these studies have examined infants in the context of co-sleeping. In fact, nowhere in the SIDS literature is there an investigation of the physiological regulatory effects that a caregiver's touch, movement, huddling, temperature, breathing, and sleep sounds can have on the infant's pattern of sleep, quiet breathing, and arousals.

From an anthropological perspective, which incorporates both cross-cultural and evolutionary data, it is particularly significant that no studies of parent–infant co-sleeping exist. Without question, co-sleeping is an evolutionarily ancient arrangement—it is the "environment of adaptedness," to use Bowlby's (1969) description, within which the human infant's sleep, breathing, and arousal patterns evolved. Infant sleep patterns and the neurophysiological patterns that underlie them co-evolved in relation to a high degree of dependence on the caregiver,

including low levels of fat and protein in milk, infantile neurological immaturity at birth, and slow maturity. All of these characteristics necessitated constant physical contact with a caregiver, especially while the infant was sleeping. This statement is not speculation. It emerges from studies of our closest living relatives, the nonhuman primates (see Anderson 1984); from cross-cultural studies of nonindustrialized peoples who continue to sleep with their children (see Konner 1981); and from recent archaeological and paleontological models of hominid evolution (Isaac 1978; Lancaster and Lancaster 1982; Tanner 1981).

Even though the absence of co-sleeping data in the context of SIDS research represents a serious gap in existing knowledge, the issue has only recently been raised. Especially in urban, Western, industrialized societies, parent–infant co-sleeping is not conceptualized as being either natural or even desirable (see Lozoff et al. 1984 for discussion). Co-sleeping is ordinarily discussed in the context of its potential for spoiling or endangering the infant, or for causing parent–child sleep struggles (see Schacter et al. 1989). Under special circumstances, co-sleeping can, and apparently has led to infantile suffocation in urban areas (Bass et al. 1986); this fact makes it even less likely that clinically trained or medical researchers would consider (or even be aware of) the possible benefits of infantile sleep contact with a parent. Fear of jeopardizing the primacy of the conjugal (husband–wife) bond, of violating concepts of parental sexual privacy (Spock 1976), of promoting incest or parental sexual arousal (Ferber 1985), and of violating popular American values of infant independence (Brazelton et al. 1974) are all factors negatively influencing the opinions of both medical and lay communities concerning parent–infant co-sleeping.

Given our cultural context, then, it is not surprising that normative data on the development of infant sleep behavior are derived exclusively from studies of infants sleeping alone, either in sleep laboratories (see Emde et al. 1971) or at home in their cribs (see Anders 1979). Together with the experiences of middle-class Americans, who are not encouraged to sleep with their infants (Lozoff et al. 1984), these data have given rise to a conceptualization of infant sleep that may be at odds with the more universal and ancient human (species-specific) pattern.

Ethnographic data from preindustrial societies in which parent–infant co-sleeping is the norm suggest that the development of long periods of consolidated sleep with minimal numbers of arousals among infants less than a year old is unusual. Super and Harkness (1987) monitored 10 Kipsigis infants living in the Kenyan highlands who regularly sleep with their mothers and found major differences between them and middle-class American infants:

> While American babies increase their longest sleep episode from four to about eight hours during the first four months (satisfying their parent's desire to sleep through the night themselves), the Kipsigis' babies do not show this change. Their longest sleep episode increases very little for at least the first eight months (Konner and Harkness 1987:101).

The studies of !Kung San Bushman infants by Konner (1981) and Konner and Worthman (1980) support these findings, as does the research by Elias et al. (1986, 1987) on La Leche League women in the United States who sleep with their infants.

SIDS RATES IN CROSS-CULTURAL PERSPECTIVE

If natural selection designed the developing human infant's sleep, breathing, and arousal patterns in association with parental contact, as we contend, this perspective gives us an initial basis for postulating (and possibly for better understanding) how and why related control systems might go awry, or somehow function less efficiently when sleep environments diverge from the evolutionarily stable one. If we assume for the moment that all known SIDS risk factors can be held constant, and that no genetic factors predispose some populations more than others to SIDS, we should find lower SIDS rates in societies, or in segments within a society, in which parent–infant co-sleeping occurs.

Cross-cultural data from urban, industrial, Asian countries support this prediction. In Japan, for example, where co-sleeping continues to be the norm (Takeda 1987), current published rates for SIDS are some of the lowest in the world (0.15/1000 births in Tokyo, 1978; 0.053/1000 in Fukuoka, 1986; and 0.22/1000 births in Saga; Tasaki et al. 1988). This finding does not, of course, prove that co-sleeping is protective against SIDS. It may well be that SIDS is underreported in Japan, or that it is misdiagnosed as infantile suffocation. Japanese medical scientists have not participated in international SIDS research studies to the extent that American and European scientists have, so the postmortem procedures they employ to identify SIDS may not be appropriate. Nevertheless, these low SIDS rates deserve explanation and further research.

In 1985, Davies reported on the rarity of SIDS in Hong Kong. He used postmortem diagnostic protocols that, on review for a follow-up study by Lee et al. (1989, see below), were judged comparative to Western diagnostic standards by John Emery, a renowned SIDS researcher from Great Britain. Davies found that even in a context of poverty and overcrowded conditions, where the incidence of SIDS should be high, the

rates were 0.036 per 1000 live births, or approximately 50 to 70 times less common than in Western societies. This finding is even more surprising because nursing is not common (of 175 infants at 2, 4, and 6 months of age, the percentage of infants nursing was 9%, 4%, and 2%, respectively).

Davies proposed that proximity to the parent while the infant is asleep may be one reason why the rates are so low, as well as the typical (prone) sleeping position of Chinese infants. The author asked "whether the possible influences of life style and caretaking practices in cot death are being underestimated in preference for more exotic and esoteric explanations" (Davies 1985:1348)—a viewpoint not unlike that of Taylor and Emery (1988) and Emery (1983), who also implicate, for some English infants, the importance of caregiving environments and other behavioral-socioeconomic factors. A follow-up on Davies's work by Lee et al. (1989) confirms the relative rarity of cot deaths in Hong Kong, finding a slightly higher rate of deaths per 1000 live births (0.3, compared with 0.04/1000 reported by Davies).

A third study confirmed the rarity of SIDS in infants of Asian origin living in England and Wales, particularly infants of mothers born in India and Bangladesh but also infants of mothers with African origins. As the authors point out, Asian women have few illegitimate births, few births at younger ages, and few of them smoke (Balarajan et al. 1989)—all of which seem to reduce the risks of infants dying of SIDS. No mention was made of any possible differences in sleeping patterns that could explain the lower SIDS rate among the Asian subgroup, although it is likely that these infants were sleeping in proximity to their parents.

Data from other industrial societies, among which at least some general comparisons of SIDS rates can be made, also tend to support the general hypothesis that increased nocturnal contact between the parent and infant may reduce the chances of SIDS among some infants. For example, in cultures in which infants are less likely to have their own room or in which infants are more likely to be in close proximity to a parent during the night, SIDS rates tend to be lower. Rates are relatively low in Stockholm (Sweden), Israel, the Netherlands, and Czechoslovakia (0.06, 0.31, 0.42, and 0.8 infants, respectively, per 1000 live births) and high in Ontario (Canada), Northern Ireland, Great Britain (Oxford area), and King County, Washington (3.0, 2.8, 2.78, 2.32 infants, respectively, per 1000 live births; Valdes-Dapena 1980b:7). The rate in King County, Washington, is five times the rate in Sweden.

Even under the best of circumstances, SIDS is difficult to diagnose. Because it is relatively rare, and because postmortem procedures for identifying SIDS are not necessarily standardized internationally, it is difficult to interpret differences in SIDS rates across cultures. Since par-

ent–infant co-sleeping is hypothesized to be relevant only to some subclasses of potential SIDS victims, proving the hypothesis becomes even more difficult.

Even within a society it can be difficult to show a correlation between co-sleeping and reduced incidence of SIDS. Consider, for example, the results of one of the very few studies of sleeping arrangements in the United States (Lozoff et al. 1984). In their study of parent–infant co-sleeping behavior among urban Americans in New York City, Lozoff et al. found that 35% of poor urban whites and 79% of poor urban blacks routinely slept with their children, who ranged in age from 6 months to 4 years (beyond the peak age for SIDS). If the hypothesis is correct, why are the SIDS rates for black Americans in New York City higher than those for any other group if parents in the former group are more likely to sleep with their infants? The benefits of co-sleeping in this particular situation may be obscured by the fact that black mothers ordinarily have their infants at a younger age ($\leq$ 20 years), smoke during their pregnancies, live in impoverished conditions, are less likely to be married, and may lack access to education on both parenting and prenatal care (Statistical Abstracts 1984). All of these factors, or some of them at least, may override the possible benefits of co-sleeping. All of them are known to increase the chances of an infant dying from SIDS (Hoffman et al. 1988).

SLEEP, BREATHING, AND AROUSAL PATTERNS IN CO-SLEEPING MOTHER–INFANT PAIRS

Even though the co-sleeping hypothesis cannot be confirmed by the fact that SIDS rates are low in societies in which parent–infant co-sleeping is the norm, the data serve as an important baseline from which additional research questions can be asked. In order to discover mechanisms by which co-sleeping could affect the acute and long-term patterns of nocturnal infant sleep, arousal, and breathing—data essential to advance the hypothesis—we began a preliminary investigation in which five mothers (< 30 years old) and their full-term healthy infants (2–5 months old) slept overnight in the sleep lab in the same bed (see McKenna et al. 1990). Throughout the night we monitored their breathing, heart rates, and brain activity. Continuous electroencephalograph (EEG), electrooculograph (EOG), chin electromyograph (EMG), and chest strain gauge recordings were made using standard, noninvasive methodology (Rechtschaffen and Kales 1968).

Polygraph recordings were scored for sleep stages in 30-second ep-

ochs according to accepted criteria. The Rechtshaffen and Kales (1968) system for young adults was used for the mothers, whereas the scoring system for 3-month-olds developed by Guilleminault and Souquet (1979) was used for the infants. Identification of sleep–wake states in both scoring systems depends on three simultaneous parameters: EEG, EOG, and chin EMG. Five sleep stages are defined in adults: REM (Rapid Eye Movement) plus four stages of non-REM (NREM) sleep delineated as Stages 1, 2, 3, and 4. In the 3-month-old infant, only three sleep stages are defined: REM, Stage 1–2, and Stage 3–4 (see McKenna 1990 for further discussion). Another major difference between the infant and adult systems is the higher voltage criterion for delta waves (>150 uV) in the infant. In the present study, the adult Stages 1 and 2 were combined to obtain total light NREM sleep and Stages 3 and 4 were combined to obtain total slow wave sleep (McKenna et al. 1990).

The epochal system of sleep-stage scoring assigns to each 30-second epoch either Wakefulness (W) or one of the stages of sleep based on the predominant (>50%) sleep/wakefulness pattern occupying that epoch. Although awakenings of 15 seconds or longer (i.e., epochal awakenings—EWs) are automatically identified by the epochal system, shorter arousals occupying less than 50% of an epoch are not. Because we are interested in all arousal phenomena in sleep, we quantified these subepochal or transient arousals (TAs) as well. Carskaden et al. (1982) defined a TA as any clearly visible EEG arousal lasting ≥ 2 seconds but not associated with any change in sleep stage. We have omitted this exclusion criterion in the scoring of TAs; in our research, all short-lived arousals were quantified regardless of the sleep stages that preceded or succeeded them. TAs among infants were typically indicated by either an abrupt increase in the predominant EEG frequency or a sudden burst of distinctly higher voltage slow waves. In the mothers, TAs were evidenced by an increase in EEG frequency (to alpha or beta) often accompanied by bursts of K-complexes or sharp waves. By far the majority of TAs in all infants and mothers were accompanied by signs of arousal on other channels: for example, a change in EOG pattern (to slow rolling eye movements or blinking), an increase in chin EMG amplitude, or a change in pattern of respiration (Figure 1).

Overlap of Sleep–Wake Stages in Co-Sleepers

Using this epochal stage scoring system we computed for each co-sleeping mother and infant the percentage of time each individual spent in the same stage of sleep or wakefulness (waking after sleep onset) as the other member of the pair. We called these times of corresponding

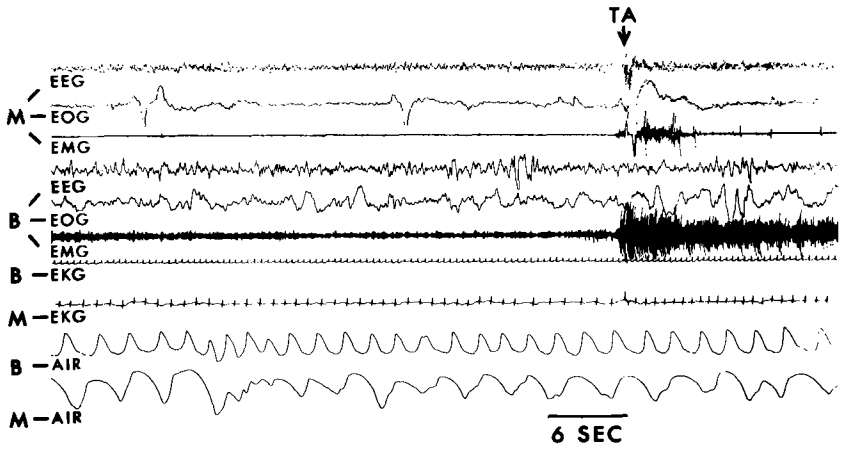

Figure 1. Polygraphic recording showing overlapping transient arousals (TAs) in one co-sleeping mother (M)–baby (B) pair (from McKenna et al. 1990).

sleep–wake stages Simultaneous Activity Time (SAT). Total SAT averaged 46% for the mothers (range 43–48%) and 44% for the infants (range 43–45%).

Because the progression of sleep stages through the night has an inherent organization that could contribute to these high percentages of Simultaneous Activity Time, and because we were interested in determining whether co-sleeping would influence the amount of synchrony in sleep–wake stages, we also computed SATs produced by pairing each mother with every other infant with whom she did not sleep. Starting with the first epoch of recording time of each mother and infant to be compared, we computed SATs for the 20 non–co-sleeping pairs. The mean SAT in mothers paired with other infants averaged 29% (range 18–30%), and for infants paired with other mothers the average SAT was 28% (range 17–43%). The increase in mean SAT in mothers paired with their own versus other infants is significant, as is the increase in SAT in infants paired with their own versus other mothers (based on the results of a *t*-test, $p < 0.0004$; McKenna et al. 1990).

We also calculated the percentage of simultaneous overlap for each sleep–wake stage separately. Mean Simultaneous Activity Time for mothers paired with their own children was higher for every sleep–wake stage than it was for mothers paired with other infants. The difference reached statistical significance only for Waking After Sleep Onset (WASO), which refers to intrasleep waking time ($p < 0.0001$). The same was true for SAT comparisons of individual sleep–wake stages of intants paired with their own mothers and with other mothers. Stage

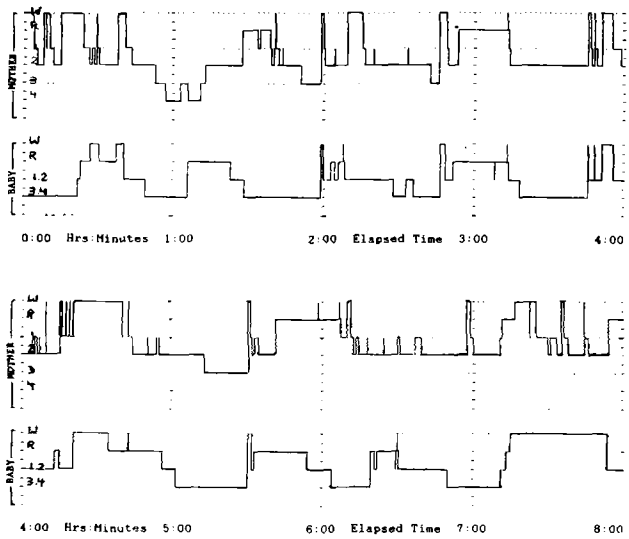

Figure 2. Sleep-stage histogram of co-sleeping mother and infant. Mother's histogram shows five adult sleep stages: Stages 1–4, and R (REM). Infant's histogram shows three sleep stages: 1–2, 3–4, and R (REM). W = Awake.

1–2 showed the smallest increase in SAT with co-sleeping for mothers and infants alike. Our small sample size may explain the lack of statistical significance in the face of an apparent trend in co-sleepers toward greater simultaneous overlap in all sleep stages, especially stages 3–4 and REM.

Synchrony of sleep–wake stages in co-sleepers is also strongly suggested by examination of histograms showing the occurrence of simultaneous and similar progression of sleep–wake stages (Figure 2).

Arousal Patterns in Co-Sleepers

We also investigated the temporal relationship of arousals in co-sleeping mothers and infants. Epochal Awakenings and Transient Arousals in each member of a pair were evaluated for overlap with EWs and TAs in the other. In 45% (75) of the mothers' combined total of 167 EWs, the infants had an overlapping EW. In most of these cases (67 EWs) the infants awoke in the same epoch, and in the remaining 8 cases the infant was already awake from a previous EW. When mother–infant pair #4 is excluded (because the infant had only 2 EWs all night compared to 12–36 EWs in each of the other four infants), 50–60% of individual mothers'

EWs overlapped an EW in their infant. On average, infants remained asleep through 55% of their mothers' EWs.

In contrast, mothers remained asleep through an average of only 11% of their infants' EWs, perhaps revealing greater maternal sensitivity to infant arousals. Infants averaged 20±10 EWs (range 2–36). Of the 101 EWs recorded for all infants, 90 (89%) overlapped with a maternal EW. Of those 90 overlapping infant EWs, the mother most often (67 cases) had an EW in the same epoch, whereas in 23 cases the mother was already awake from a previous EW. Again ignoring mother–infant pair #4, we found that 83–97% of each infant's EWs overlapped an EW in the mother (for infant #4, who had only 2 EWs, this value was 100%).

These temporal relationships in Epochal Awakenings far exceed chance. When records of mothers were paired epoch-by-epoch with those of infants with whom they did not sleep, the relative frequency of total maternal EWs overlapping EWs in the infant was only 9%, and for total infant EWs this value was only 23%. When compared to values obtained in co-sleeping pairs, the differences are highly significant ($p < 0.0001$).

Transient Arousals also showed high frequencies of temporal overlap with both TAs and EWs in the other member of the dyad. Mothers averaged 58±12 TAs (range 28–76). Of the 290 TAs scored across mothers, 39% were accompanied by a TA in the infant within 5 seconds and 10% overlapped an infant EW. Infants averaged 78±5 TAs (range 52–115). Of the 388 infant TAs, 29% were accompanied by a maternal TA within 5 seconds and 37% overlapped a maternal EW. Infants showed no arousal phenomena (i.e., no TA or EW) for 51% of maternal TAs, whereas mothers showed no arousal phenomena for only 34% of infant TAs, again suggesting perhaps greater maternal sensitivity to infant arousals than vice versa.

When Transient Arousals and Epochal Awakenings are combined, 48% of the maternal arousals were associated with some type of arousal in their infants, and 71% of the infant arousals were associated with some type of maternal arousal (see McKenna et al. 1990).

Breathing Behavior in Co-Sleepers

Our preliminary respiration data reflect hand counts in 60-second epochs throughout the night. Because the technology required to record and store respiratory data appropriate for more sophisticated analyses was not available to us, only gross measures of respiration were possible. Hand counts were based on definitions of respiratory cycles devised by Richards et al. (1984). Table 2 shows mean respiration rates collapsed

Table 2. Number of Breathing Cycles per Minute for Co-sleeping Mothers and Infants

	Pairs	*Mean*	*Standard Deviation*
Pair 1	Mother	13.19	1.97
	Infant	28.91	5.186
Pair 2	Mother	15.45	1.97
	Infant	32.73	6.103
Pair 3	Mother	16.28	1.712
	Infant	31.99	4.966
Pair 4	Mother	14.20	4.692
	Infant	30.42	3.735
Pair 5	Mother	13.0	1.6
	Infant	27.3	3.45

Range of Mothers 13–16
Range of Infants 28–32

across sleep–wake stages for each mother–infant pair, with infant rates averaging about twice the maternal rates. When average breaths per minute (BPMs) were calculated as a function of sleep–wake stage, the infant rate was highest in waking and lowest in Stage 3–4, as expected (Figure 3).

Figure 4 graphs mean respiration rate at 30-minute intervals for one

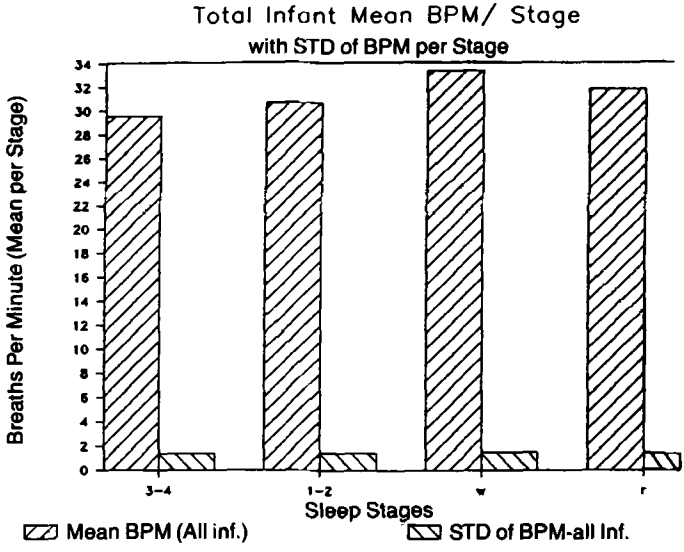

Figure 3. Co-sleeping infants' breaths per minute (BPMs) by sleep stage.

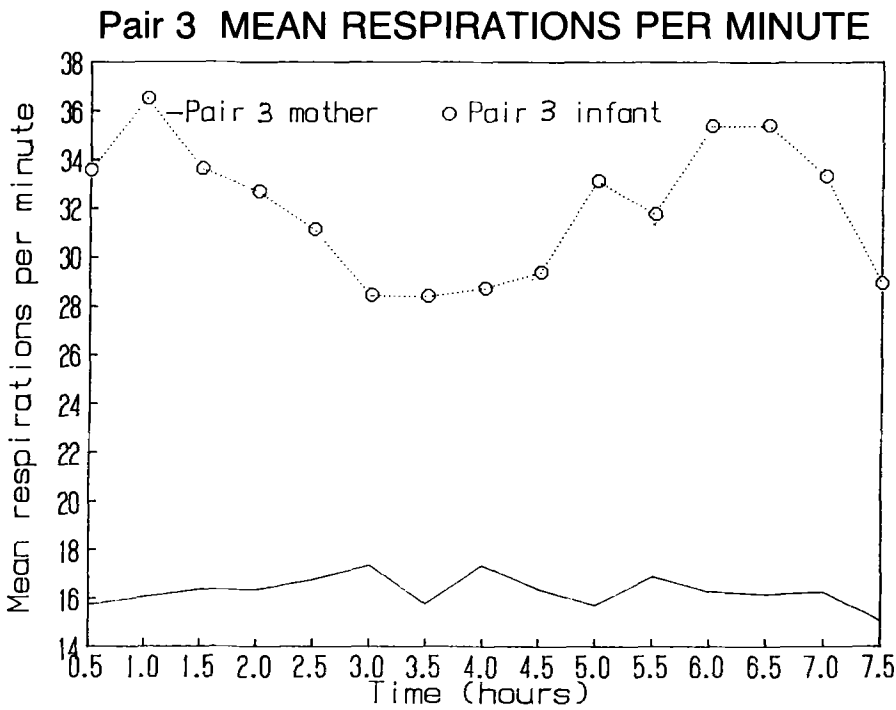

Figure 4. Mean respiration rate per minute recorded every half-hour for co-sleeping mothers and infants.

mother–infant pair. Because the repeated data samples from each individual are not independent, the use of simple correlational techniques to investigate the relationship in rates across time between mothers and infants would not be appropriate. In a future study, respiration and cardiac data will be digitalized to permit valid analysis of the interactions between co-sleepers.

For infants, we defined a breathing pause or apnea as a cessation of air flow and respiratory effort lasting at least 2 seconds. Table 3 presents the mean frequency and duration of breathing pauses as a function of sleep–wake stage. The REM stage showed the highest frequency but shortest duration of breathing pauses, and the reverse was true for Stage 3–4. Rarely did breathing pauses exceed 10 seconds, and very few obstructive apneas of any duration were observed. The small number of infants in this sample, their age range, and the high degree of variation among subjects in breathing pauses precludes meaningful comparison with published data from normal infants sleeping alone.

An anecdotal yet potentially important observation from our prelim-

Table 3. Frequency and Duration of Infant Breathing Pauses

	Frequency (per hour)		*Duration (in seconds)*	
	mean	*s.d.*	*mean*	*s.d.*
Stage 1–2	11.5	± 6.0	5.3	± 2.4
3–4	7.9	± 10.1	6.9	± 3.6
REM	19.4	± 15.8	4.5	± 2.3
WASO*	12.1	± 8.1	5.1	± 2.4

*Waking After Sleep Onset

inary investigation concerns a number of occasions of overlap in mother and infant apneas. Figure 5a shows a maternal apnea followed by an overlapping infant apnea, and Figure 5b shows an infant apnea followed by an overlapping maternal one. In these two examples, neither partner had exhibited an apnea 30 minutes before or after these events, which suggests that the events might be related.

The most important findings from these preliminary studies are *(a)* the high degree of temporal synchrony in arousals among co-sleeping mother–infant pairs and *(b)* the fact that more homogeneity (simultaneous activity time) is evident in the sleep–wake patterns of co-sleeping mother–infant pairs than can be explained by chance. Because arousals from sleep impact a variety of physiological systems, including respiratory and cardiac activity, as our research progresses we expect to find that co-sleeping has measurable influences on these and other systems as well. The physiological regulatory effects of co-sleeping also could be mediated through other mechanisms, such as responsivity to the other's touch, radiant temperature, movements, expired gasses, and breathing sounds.

CO-SLEEPING, AROUSAL DEFICIENCIES AND SIDS: SOME POSSIBLE CONNECTIONS

For many years it seemed that the link between infant apneas and SIDS was strong. More recent work suggests that the infant's inability to reinitiate breathing after first arousing once an apneic episode has occurred may be the principal defect, not apnea itself. In other words, neither infantile breathing pauses nor apneas are necessarily precursors to SIDS.

Hoppenbrouwers and Hodgman (1986) recently proposed that the inability of infants to arouse to breathe may represent an adaptive fail-

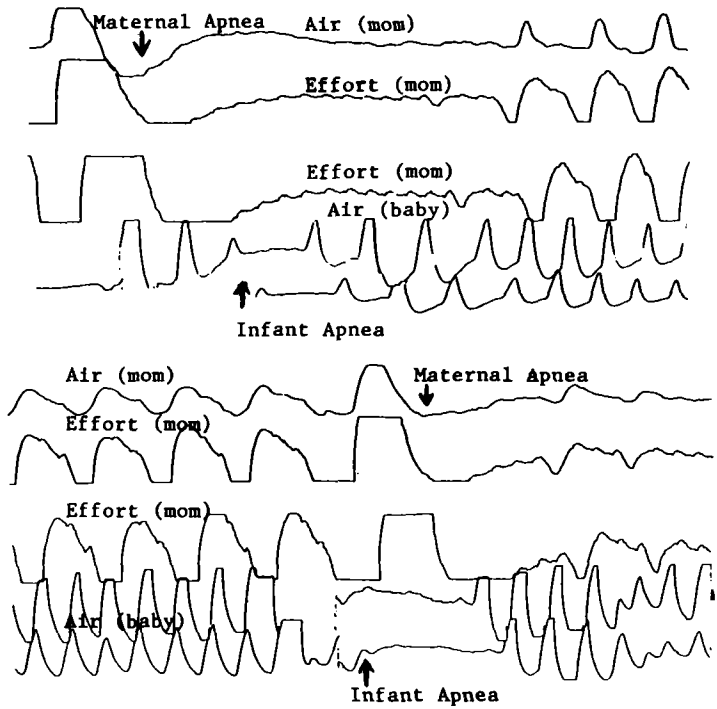

Figure 5a, b. Polygraphic tracings of synchronous breathing pauses of co-sleeping mother–infant pairs.

ure, possibly caused by a depressed cortex, that compromises breathing. This condition may lead to hypoxia and death for some infants. Sterman and Hodgman (1988:56) have elaborated on the above notion and suggest that "the early maturation of neural substrates for sleep and waking could result in a higher threshold for arousal at a critical period in autonomic regulatory maturation." They suggest that adultlike sleep patterns, which include prolonged sleep bouts without awakenings, may develop prematurely in some infants in the absence of corresponding maturational levels achieved by the arousal system, which functions to awaken infants to breathe during respiratory and other sleep crises. Clearly, the work by Harper et al. (1981) and Einspieler et al. (1988), and the epidemiological study by Hoffman et al. (1988), support this perspective.

Our finding that both shorter and longer arousals in each partner overlap at rates significantly different than chance suggests that co-sleeping partners induce arousals in each other. These physiological interactions in co-sleeping situations may illustrate some of the microen-

vironmental and behavioral mechanisms that can prevent the premature emergence of uninterrupted and prolonged sleep bouts—for example, the premature maturation of quiet sleep leading to higher thresholds of arousal, which may account for some SIDS events (Sterman and Hodgman 1988). Recall that infants who regularly sleep with their parents continue to awaken during the night until well after the first year (Elias et al. 1986; Super and Harkness 1983, cited in Konner and Super 1987). It is possible that the on-going sensory stimuli offered by co-sleeping partners may help to prevent infants from experiencing the kind of cortical depression Hoppenbrouwers and Hodgman (1986) speculate may precede breathing control errors leading to SIDS in some infants. Partner-induced arousals may in fact act to compensate for deficient, internally controlled arousals in the infant, when and if they are needed, but more research on the effects of co-sleeping on both normal and high-risk infants is needed.

If wakefulness does constitute a strong stimulus to breathing, and failure to awaken from sleep may jeopardize breathing (Harper et al. 1981; Hoppenbrouwers and Hodgman 1986), it seems important to understand what behaviors and traits evolved to foster wakefulness among infants given the contemporary socioenvironmental conditions. At this point we can merely make the co-sleeping hypothesis proposed by McKenna (1986) more specific; we cannot prove it. Based on these preliminary data, it is a research question that deserves further investigation.

CONCLUSIONS

Investigators into SIDS cases in which infants are out of contact with the caregiver at the time of death should begin with several realizations. First, prolonged and uninterrupted sleep bouts in the first year of life are at odds with what the human infant's vulnerable and slow-developing central nervous system was designed to experience. Second, solitary, nocturnal sleeping behavior represents an exceedingly recent, culturally induced change that contrasts in significant ways with the developmental setting within which the infant's respiratory, arousal, and sleep systems evolved. Third, we should not presume that all infants confront this environmental perturbation with the same amount of constitutional versatility, or that solitary sleeping is necessarily in the psychosocial or biological best interest of the infant. Although infants sleeping alone may be in the parents' best interest, and accords with the primacy of the conjugal bond and other values in Western societies, it is not necessarily in the infant's biological best interest.

As we have shown here, evolutionary data provide a less culture-

bound view of the subjects we are attempting to understand, and these data also provide a scientifically based foundation to ask important new questions that challenge traditional SIDS research assumptions. Although our contention that parent–infant co-sleeping offers a potentially important infantile microenvironment for human infants may not be applicable to all infants, it may be to some.

> Many parents in urban societies where both parents work have been pushing the adaptive capacities of the human infant to the limit by arranging infant care which proceeds in exactly the opposite direction from that suggested by the infant's continued need for physiological support during the first year of life, and to some extent later. Many studies now, however, are demonstrating that an adequate period of symbiosis or dependency in the first year is the best preparation for later independence and autonomy on the part of the infant. But these lessons, while shown in present research, are seriously challenged by the direction in which our urban society is moving, and it will be difficult for Americans in particular to accept the idea that we must relearn something from our more primitive past which we have forgotten or which has been lost in the rush of civilization and technology. (Call 1986:57)

Our research cannot reveal the solution to this problem. But by considering the infant's evolutionary past, we can begin to separate the social best interests of the parent from the biological and social best interests of the infant, and by doing so propose new research questions on SIDS. The fact that evolution, paradoxically, may place parent–infant interests in conflict is but one of many cultural predicaments our species presently must face. How we choose to resolve these dilemmas will have some important consequences. For now, as is the case here, we need to know if prevailing cultural practices of promoting solitary infant sleep play any role at all, for any infants (however small their number), in the pathophysiology of SIDS.

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