

Maternal Proximity and Infant CO₂ Environment During Bedsharing and Possible Implications for SIDS Research

SARAH MOSKO,* CHRISTOPHER RICHARD, JAMES MCKENNA,
SEAN DRUMMOND, and DAVID MUKAI
*Sleep Disorders Center, University of California Irvine Medical Center,
Orange, California 92868*

KEY WORDS cosleeping; mother-infant bedsharing; CO₂; sudden infant death syndrome

ABSTRACT Sudden infant death syndrome (SIDS) is the leading cause of human infant mortality after the neonatal period in Western countries. Recently, child care practices have been shown to be important in determining infant vulnerability to SIDS. However, very little is known about the impact of parent-infant cosleeping on infant sleep physiology and behavior and SIDS risk. This reflects the failure of Western societal research paradigms to appreciate the human infant's evolutionary history of cosleeping, the recency of the emergence of solitary infant sleeping as a practice and the fact that parent-infant cosleeping is still the preferred sleeping arrangement for the majority of contemporary societies. Incorporating current hypotheses on the mechanisms of SIDS, we have hypothesized that the comparatively sensory-rich cosleeping environment might be protective against SIDS in some contexts. As a first step to characterize cosleeping environments, this investigation is aimed at assessing, in routinely bedsharing mothers and infants, their relative sleeping positions and the potential for sleeping in close face-to-face proximity and for infant exposure to increased environmental CO₂ produced by maternal respiration. The latter is important in that breathing elevated levels of CO₂ can have diverse effects, ranging from respiratory stimulation at low levels to suffocation at very high levels.

Two related laboratory studies were performed. In the first, all-night videotapes of 12 healthy, routinely bedsharing mother-infant pairs were analyzed for sleeping positions and time spent in face-to-face orientation and distances separating their faces. Infants were 11-15 wk old. Mothers predominantly positioned themselves on their sides facing their infants, with the infants placed either supine or on their sides. Mothers and infants slept oriented face-to-face for $64 \pm 27\%$ (S.D.) of non-movement time, with distances less than 20 cm commonly separating their faces. In the second study, concentrations of CO₂ in air were measured in six young women at distances of up to 21 cm from their nares. Peak expiratory CO₂ concentrations remained above 1.0% at distances up to 9 cm and above 0.5% at 18 cm. Both baseline and peak CO₂ levels were further increased at all distances when measured within a partial air pocket created to simulate a bedding environment sometimes seen during bedsharing. We conclude that during bedsharing there is potential for 1) a high degree of face-to-face orientation and close proximity and consequently 2) increased environmental CO₂, as a result of maternal respiration, to non-lethal levels that might stimulate infant respiration.

Contract grant sponsor: National Institutes of Health; contract grant number RO1 HD27482.

*Correspondence to: Sarah Mosko, Ph.D., Sleep Disorders Center, Bldg. 22C, Rt. 23, University of California Irvine Medical Center, 101 The City Drive, Orange, CA 92868.

Received 20 September 1996; revised 2 December 1996; accepted 15 April 1997.

The close proximity would also maximize the sensory impact of the mother on the infant through other modalities. We also suggest that bedsharing may minimize prone infant positioning, a known risk factor for SIDS. *Am J Phys Anthropol* 103:315–328, 1997. © 1997 Wiley-Liss, Inc.

Despite two decades of intensive research to understand and prevent sudden infant death syndrome (SIDS), SIDS is still the number one cause of infant mortality after the neonatal period in Western countries. It is defined as the sudden death of an infant <1 yr of age which remains unexplained after a thorough investigation, including complete autopsy, death scene examination and review of the clinical history (Willinger et al., 1991). There is marked variation in SIDS rates across the world, with highest rates in New Zealand and France (2.3 and 1.9 deaths per thousand live births, respectively) and lowest rates (<0.01) in Greece and Brazil. In the United States, the incidence of SIDS is 1.3 per thousand live births, or over 4,000 cases annually (World Health Organization, 1994).

The etiology of SIDS remains elusive and controversial, although there is a general consensus that the causes are multifactorial, involving both constitutional vulnerabilities and environmental influences. Developmental factors are thought to be involved since SIDS is uncommon before 1 mon and after 6 mon, with a peak occurrence at 2–3 mon of age (Guntheroth, 1995; Willinger et al., 1991). Viable explanations for SIDS must incorporate why most SIDS deaths occur during periods of presumed sleep (Cordner and Willinger, 1995). Currently, there are several lines of evidence suggesting that subtle cardiovascular or respiratory control defects, impaired thermoregulation or deficient arousal mechanisms can each contribute to SIDS deaths (Guntheroth, 1995; Rognum, 1995). Yet, insofar as the mechanisms leading up to a SIDS event may be quite varied and complex, the predominant hypothesis is that there is a final common pathway involving failure to arouse from a prolonged apnea with consequent cardiorespiratory collapse (Harper et al., 1981; Shannon et al., 1977; Guntheroth, 1995).

Perhaps because of the varied and subtle nature of the defects involved in SIDS, extensive post-mortem analyses have failed to elucidate any measureable infant parameters useful in predicting a given infant's level of risk. As a result, prevention strategies have shifted in the last decade to efforts to identify modifiable factors in the infant's environment that could contribute to SIDS risk. In particular, recent epidemiological studies have identified several child care practices associated with increased risk, including maternal smoking and prone infant positioning for sleep (Blair et al., 1996; Mitchell et al., 1991). Of these, the most powerful example is the increased risk associated with prone sleeping (Fleming et al., 1990; Mitchell et al., 1991; Dwyer et al., 1991a, 1991b). Within the last 5 yr, campaigns in several countries to discourage prone positioning have reduced SIDS rates in every instance (Willinger et al., 1994), by roughly one-third to one-half. The mechanism whereby prone sleeping endangers infants is not yet understood, although lethal rebreathing of CO₂ trapped in bedding (Chiodini and Thach, 1993; Kemp et al., 1994) and hyperthermia (Ponsonby et al., 1993) have been implicated by some researchers.

Our research team is interested in whether the social dimension of infants' sleeping environments modifies vulnerability to SIDS. This stems in part from observations that SIDS rates can be low in societies or cultural groups that practice parent-infant cosleeping, such as Japan and Hong Kong (Lee et al., 1989; Davies, 1985; Gantley et al., 1993; Farooqi et al., 1991; Tasaki et al., 1988; Balarajan et al., 1989; Singh et al., 1992). Although additional factors certainly could be involved, that close proximity to parents during sleep contributes to these low SIDS rates is suggested by a recent epidemiological study in New Zealand which found that infants who slept in a room alone were nearly four times as likely to die from SIDS

as infants who shared a room with an adult(s). Furthermore, this protective effect did not generalize to room sharing with siblings (Scragg et al., 1996). The recent increase in SIDS rates in Japan, which has been paralleled by a shift from a tradition of social sleeping to solitary infant sleeping (Watanabe et al., 1994), also supports a protective role of parental proximity during sleep. Yet, almost nothing is known about the cosleeping environment and its impact on infants. This void is no doubt a reflection of a historically recent Western cultural bias favoring the solitary infant sleeping arrangement which has been incorporated almost universally into pediatric and SIDS research paradigms. Such research has failed to appreciate several important concepts. The first is that parent-infant cosleeping must offer some adaptive advantages since it is the sleep environment within which human infant sleep evolved (Lozoff and Brittenham, 1979; Konner, 1981; Lancaster and Lancaster, 1982). Second, since solitary sleeping emerged as a practice only within the last 200–400 yr, failure of infant sleep physiology and behavior to adapt quickly to such a radical change in environment might result in significant vulnerabilities in some infants. Third, among all primates the human newborn is neurologically the most immature at birth, increasing its reliance on the primary caregiver for direct contact and care both day and night (Konner, 1981). Fourth, some form of cosleeping is still the customary arrangement for the majority of the world's cultures (Burton and Whiting, 1961; Barry and Paxson, 1971; Caudill and Plath, 1966; Shand, 1981). In a survey of 127 cultures worldwide from which reliable data on sleeping arrangement were obtained, Barry and Paxson (1971) reported that for 79% of the cultures, infants slept in the parents' room; this involved sharing the same bed or sleeping surface for at least 44% of the cultures. Even within the United States, cosleeping is not an uncommon practice, contrary to popular perception. For example, for infants and toddlers, frequent all-night or part-night bedsharing was reported in 19% of whites, 59% of blacks and 26% of Hispanic families sampled from New York City and Cleveland (Lozoff et al., 1984;

Schachter et al., 1989). Fifth, it has not been appreciated that, without data on contrasting sleep environments, any effects on infants stemming from solitary sleeping, whether positive or negative, also remain unexplored.

Cosleeping can assume diverse forms, depending on the infant's relationship to the cosleeper, the degree of physical proximity and the type of sleeping surface, for example. All forms of cosleeping are probably sensory-rich compared to solitary sleeping, but the greatest contrast in this dimension would be expected with bedsharing with a caregiver where close proximity to and direct physical contact with the caregiver could expose the infant to sensory stimuli involving every modality (auditory, olfactory, tactile, thermal, gustatory and visual). Furthermore, the infant's environmental CO₂ might be increased as a result of a mother's respiration onto the infant at close range. However, any effects of this comparatively sensory-rich environment on infant sleep physiology and behavior remain unexplored. Incorporating current hypotheses on the constitutional deficits involved in SIDS, which include cardiovascular, respiratory, arousal and thermoregulatory defects, we have postulated that, in some contexts, either fundamental sensory features inherent to bedsharing with a parent and/or caretaking behaviors of parents during bedsharing might offer protection against SIDS (Mosko et al., 1993, 1996; McKenna et al., 1990; Richard et al., 1996). The possibility that maternal respiration could elevate the infant's CO₂ environment is important from the standpoint that increased CO₂ can have diverse effects, ranging from respiratory stimulation at low levels to suffocation at very high levels (Schafer et al., 1993; Haddad et al., 1980; Chiodini and Thach, 1993; Kemp et al., 1994). Evidence of respiratory stimulation as a result of bedsharing could be important in the context of compensation for a postulated impairment in respiratory control mechanisms in the etiology of SIDS.

The contrary view, that bedsharing places infants at increased risk for SIDS, has been proposed recently as a result of the New Zealand study, although bedsharing (defined in that study as bedsharing with any-

one) was associated with significantly increased risk only in Maori infants (Mitchell et al., 1993). The cultural variation in their findings was subsequently found to be explained in large part by an interaction of bedsharing with maternal smoking (Scragg et al., 1995). Furthermore, a recent epidemiological study in southern Californian whites, African-Americans, Latinos and Asians failed to find increased risk for SIDS associated with bedsharing, similarly defined (Klonoff-Cohen and Edelstein, 1995). Because of both the important role that other child care practices appear to play in raising or lowering SIDS rates and conflicting ideas among researchers about possible benefits or detriments of cosleeping, an effort to understand how well-defined cosleeping and solitary sleeping environments differentially affect infants is timely and might be critical to the continued development of effective strategies to combat SIDS.

The present studies characterized the cosleeping environment (defined here as bedsharing with the mother) at the peak age of incidence of SIDS by evaluating 1) the mutual body orientations and proximity of routinely bedsharing mother-infant pairs and 2) potential CO₂ concentrations around the infant due to maternal respiration while sleeping face-to-face. The two studies stemmed from an observation that bedsharing mother-infant pairs can spend large portions of the night at close range in face-to-face orientation. This prompted the measurement of their relative sleeping positions and proximity in the first study. That study provides insight into the influence of bedsharing on infant positioning and on the general potential for sensory exchange between mother and infant. The possible impact of the mother's proximity on the infant's environmental CO₂ was evaluated in the second study by measuring air concentrations of CO₂ at distances from women's faces that are within the range at which routinely bedsharing infants were found to sleep. Furthermore, because of the postulated role of lethal CO₂ rebreathing into bedding in the association of prone sleeping with SIDS, we also measured air CO₂ concentrations produced by women when breathing into an air pocket formed by a blanket, as sometimes

occurs around an infant's head while sharing a blanket with the mother.

SUBJECTS AND METHODS

Orientation and proximity of cosleepers

Twelve healthy, breast-feeding, non-smoking mothers, ranging in age from 18 to 37 yr (mean 27 ± 6 (S.D.) yr), were recruited from the Birthing Center at the University of California Irvine Medical Center. All mothers were Latina, because bedsharing is an accepted practice in this ethnic group (Morelli et al., 1992) and to control for potential cultural differences in attitude toward and implementation of bedsharing. All reported that they had been bedsharing with their infants since birth, and this was confirmed by 2 wk of daily sleep logs: for the 10 pairs on which complete data were available, the pairs bedshared an average of 13.6 ± 0.5 out of 14 nights. Mothers were screened for sleep disorders by a detailed sleep history performed by a trained physician, and infants were excluded if the mother's reason for bedsharing related to infant temperament (fussiness). Infants (six males and six females) had 5-min Apgar scores (a general measure of the newborn's condition) of at least 8 (10 is maximum score) and normal developmental histories. They were healthy at the time of testing. Mother-infant pairs coslept in a laboratory bedroom on two nights when the infants were 13 ± 1 wk old (range 11–15 wk). The first night served as an adaptation night, and the second night occurred within two nights of the first. Mother-infant pairs shared a twin-size hospital bed both nights. The measurements reported here derived from all-night infra-red video camera recordings on the second bedsharing night. Monitoring in infants and mothers also included standard non-invasive polysomnographic measures (electroencephalogram, electrooculograms, chin electromyogram, and qualitative measures of airflow and respiratory effort), according to the protocol of a larger study contrasting sleep patterns in solitary and bedsharing environments that has been reported separately (Mosko et al., 1996).

Infants were maintained on their usual feeding and sleeping schedules, with mothers performing all caretaker interventions

ad libitum. Mothers were blind to all experimental hypotheses and instructed to put their infants to bed as they would at home. Mothers retired at their usual time, an average of 55 min after the infants, and monitoring was terminated after mother and infant had awakened the next morning at their usual times. Only the bedsharing portion of the night was used in the present analysis. Sleeping positions were recorded in both infants and mothers as supine, prone, or on the side. Differences in time spent in the three positions were tested first by the Friedman Two-Way Analysis of Variance, followed by Wilcoxon Matched-Pairs Signed-Ranks Tests. Anytime a mother and infant were facing each other the distance between their nares was measured on the video monitor screen and extrapolated to the actual face-to-face distance by a standard distance marker (provided by a large digital clock) in the video field. The reliability of this technique was verified (to within ± 3 cm) by measuring objects of known dimension from the video field. The amount of time each pair spent at given face-to-face distances was computed to the nearest minute. Times when either member of a pair were moving could not be scored reliably and were excluded from the analysis.

CO₂ concentrations at varying distances from the nares of women

To assess the potential for infant exposure to CO₂ from the mother while bedsharing, concentrations of CO₂ in air were measured in a separate group of six healthy, awake women (aged 29 ± 4 yr) at varying distances from their nares in sitting and reclining positions. All measurements were done in the absence of infants so that CO₂ levels would not be contaminated by infant respiration. Subjects were instructed to breathe normally through their noses. CO₂ concentrations were measured by mass spectrometry (Perkin-Elmer 1100 Medical Gas Analyzer) at a sampling rate of 45 ml/min and recorded for digital storage at 10 samples/sec. All recordings were performed at the same location within a well-ventilated laboratory.

While sitting, CO₂ concentrations were measured at distances of 3 to 21 cm from the

nares at 3 cm increments by holding the sampling port in the line of expired air. At each distance, 30- to 45-sec readings were obtained and regression analysis was applied over this range to assess the relationship of distance to CO₂ concentration. Then reclining measurements were taken which were an outgrowth of the observation that, while mother-infant pairs coslept and shared a common blanket, the blanket sometimes appeared to form a partial air pocket around the infant's head (Fig. 1). For pairs sleeping face-to-face in close proximity, this raised the question of whether such a pocket could, by trapping the mother's exhaled CO₂, further alter the infant's CO₂ environment. For the reclining measurements, the sampling tube was attached to the nose of a doll which approximated the body size of a 3-mon-old infant. The woman and doll were placed on their sides in face-to-face orientation with the doll's face in the line of expired air. CO₂ measurements were made at nose-to-nose distances of 9, 15 and 21 cm. Two sets of measurements were made at each distance. The first 2 min of recording were without an applied blanket; for the second 2 min, a loose weave cotton blanket was laid across the woman and doll, as shown in Figure 1, covering up the woman's neck and about one-third of the doll's head. A 2×2 within-subjects design Analysis of Variance (ANOVA) was used to analyze mean CO₂ concentrations obtained over 30 sec samples taken before and after blanket application. The two within-subjects factors were presence or absence of the blanket and the three nose-to-nose distances. An interaction effect also was evaluated.

This research was approved by the Human Subjects Review Committee of the University of California, Irvine.

RESULTS

Orientation and proximity of cosleepers

The period of cosleeping, from the beginning to the end of bedsharing, averaged 468 ± 63 (S.D.) min (range 324–543 min). Non-movement time (NMT) accounted for 92% of this or 432 ± 53 min (range 299–485 min).

Mothers showed a strong preference for side sleeping while bedsharing (Table 1). All but one mother spent the majority of the



Fig. 1. Drawing showing cosleeping simulation performed with toy doll to measure the effect of a partial air pocket formed around an infant's head on CO₂ levels produced by the mother's respiration. The dotted line represents the blanket.

TABLE 1. Sleeping positions (% NMT)

	Side	Supine	Prone
Mothers	74 ± 19	23 ± 18	3 ± 6
Infants	49 ± 41	40 ± 40	11 ± 29

NMT, non-movement time. All entries reflect group means (±S.D.).

time on their side. A Friedman Two-Way ANOVA indicated a highly significant positional effect ($P < .0001$), and the differences in %NMT between each of the three positions were also highly significant (Wilcoxon Matched-Pairs Signed-Ranks Tests: side vs. supine, $P = .004$; supine vs. prone, $P = .005$; side vs. prone, $P = .002$). Only three mothers ever slept prone, ranging from 2% to 19% NMT.

Both side and supine positioning predominated in infants (Table 1). When just two position categories were used (prone vs. non-prone), a Friedman Two-Way ANOVA revealed a highly significant positional effect ($P = .004$). A significant overall posi-

tional effect persisted when the supine and side positions were categorized separately ($P = .034$). However, individual position comparisons (Wilcoxon Matched-Pairs Signed-Ranks Tests) revealed non-significant trends in the differences between the prone and side position ($P = .059$) and the prone and supine position ($P = .075$) and no discernible difference between the supine and side position ($P = .722$). Only two infants were ever placed prone by the mother, for 31% NMT in one case and 100% NMT in the other. The latter case was especially atypical in that the mother placed the infant prone with its entire upper body propped up on a pillow, whereas no other mother ever used a pillow under the infant. Exclusion of this infant's data from the analyses greatly increased the significance level of the Friedman test ($P = .009$) and also resulted in highly significant differences between the prone and both side ($P = .008$) and supine ($P = .011$) positions. There was no clear relationship between the prone position in mothers and infants, insofar as the mother who kept her infant prone all night slept on her side the entire time. None of the infants ever changed position on their own (except for head turning).

Infants faced (i.e. face oriented toward) the mother for an average of 83% NMT (range 14–100%). All but two infants faced the mother for a large majority ($\geq 69\%$) of the night, and seven faced the mother for the entire night. The infant with the minimum NMT facing the mother (14%) was the one who was prone all night. Virtually all of the time that infants were on their side bodily facing the mother their faces were oriented toward the mother. Only one infant was ever placed on its side so that it bodily faced away from the mother (for 39 min or 9% NMT). While supine, infants also remained predominantly oriented toward the mother. Of the nine infants who slept supine part or all of the night, the average fraction of supine time that the infant remained with its head turned to face the mother was 80% (range 0–100%), and six of the infants maintained that orientation for 100% of supine time.

On average, mothers spent almost as much time facing their infants ($73 \pm 21\%$ NMT) as did the infants facing the mothers. All but

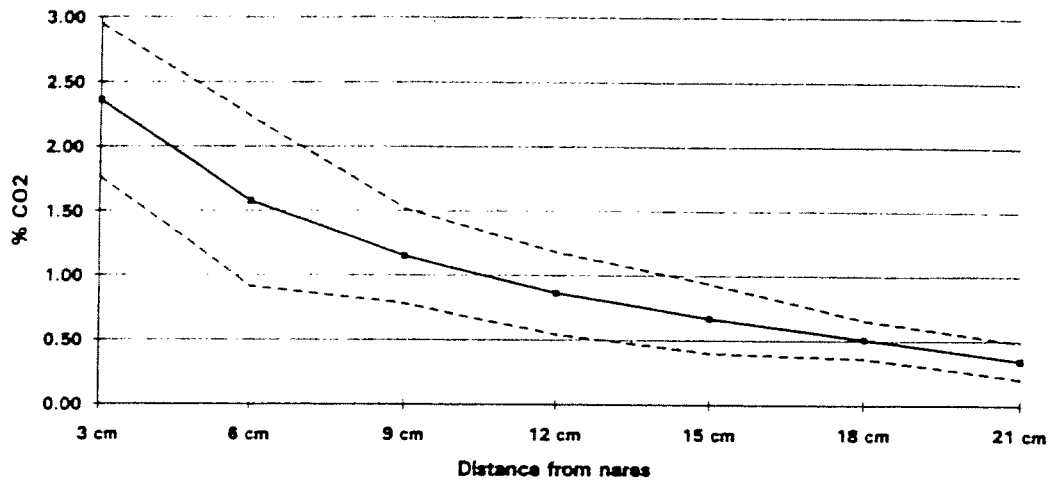


Fig. 3. Mean peak expiratory CO₂ concentrations, with 95% confidence limits, at distances of 3 cm to 21 cm from the nares for women in the sitting position ($n = 6$).

sured at the nose of a doll separated 9, 15 or 21 cm from the nares of the women. This elevation in CO₂ by trapping expired CO₂ was especially pronounced at distances of 15 and 21 cm (Fig. 4). Because identification of individual CO₂ peaks and troughs was not always possible for the reclined position, especially after blanket application, quantification of the blanket's effect was achieved by calculating the mean CO₂ concentration over representative 30 sec samples of continuous CO₂ measurements, one before and one after blanket application at each distance (Fig. 5). At least 30 sec was allowed to elapse after blanket application to allow the CO₂ concentrations to stabilize before taking a sample. Without the blanket, the group CO₂ means were 0.41%, 0.28% and 0.20% at 9, 15 and 21 cm, respectively, and they increased to 0.75%, 0.69% and 0.51%, respectively, with the blanket. This means that as a result of blanket application, the doll's average CO₂ exposure from the women's respiration increased by 83% at 9 cm, by 146% at 15 cm, and by 155% at 21 cm. The effect of the blanket was highly significant ($F = 34.37$; $P = .002$), and the effect of distance was also significant ($F = 5.11$; $P = .030$), but there was no significant interaction effect ($F = 0.39$; $P = .685$). With the blanket, inspiratory (or trough) CO₂ levels remained above 0.2%, and sometimes appreciably higher than this, for the majority of

the 30 sec sample at all three distances for all subjects with one exception. The highest peak levels attained occurred at 9 cm and were between 1.5 and 2.0% CO₂.

DISCUSSION

The findings indicate that routinely bed-sharing mother-infant pairs can sleep oriented face-to-face for large portions of the night, with distances less than 20 cm commonly separating their faces. Our Latina mothers preferentially slept on their side facing their infants. Infants were preferentially placed in non-prone positions by mothers (side or back), and they also typically faced the mother the majority of the night. Most pairs slept oriented face-to-face for the large majority of the recording. Furthermore, all of the pairs slept for portions of the night at distances less than 20 cm separating their faces. The measurements of CO₂ concentrations at distances of 3 to 21 cm from the nares of women demonstrate that, even at 21 cm, there is potential for appreciably increased environmental CO₂ as a result of a mother's breathing.

Since all mother-infant pairs were Latino and routinely bedshared at home, the generalizability of these results to other cultural groups is not known since cultural attitudes toward bedsharing vary greatly (Lozoff and Brittenham, 1979). For example, bedsharing is a more common practice among Latin

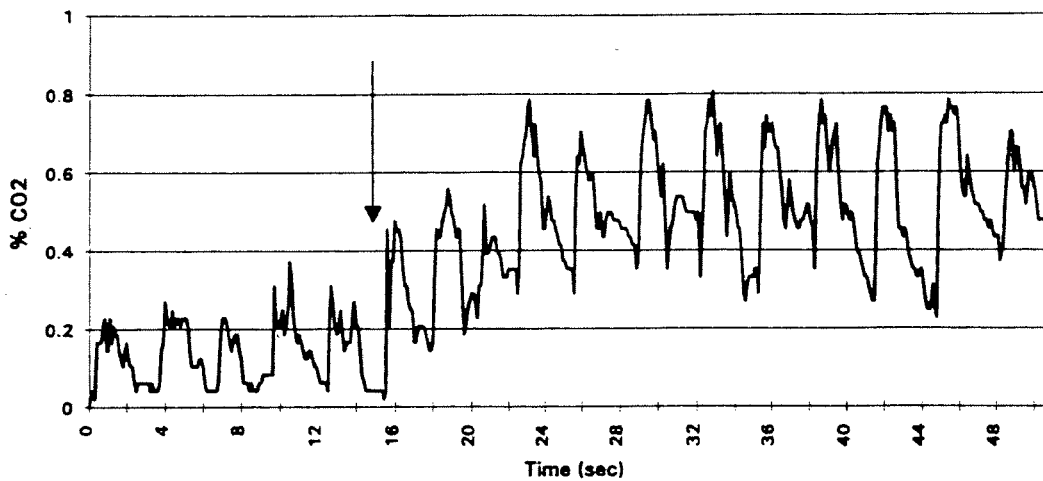


Fig. 4. Example of continuous CO₂ recording, measured at the doll's nose, 21 cm from one reclining woman's nares. The blanket was applied at the arrow.

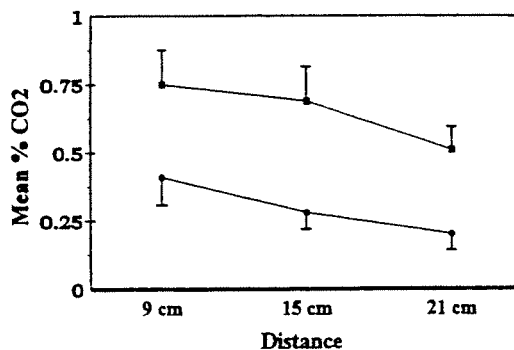


Fig. 5. Mean (\pm S.E.M.) concentrations of CO₂ over 30 sec samples taken before (circles) and after (squares) blanket application at three distances from the nares of reclining women.

Americans and African Americans than among whites in the area of California from which our subjects were drawn (Klonoff-Cohen and Edelstein, 1995). Differing cultural attitudes might impact the degree of physical proximity parents exhibit in bedsharing with an infant, although no figures exist describing the physical proximity or behavior of bedsharing parents and infants for other cultural groups. That the sample size in our study was not large also limits the generalizeability of the findings, although bedsharing in most conceivable contexts would seem to hold some degree of potential for close face-to-face sleeping.

The twin size bed used in our study raises the question of whether the relative body positions and degree of infant-mother proximity we measured resulted from bed size. The observations that infant position was usually managed by the mothers during periods of breast-feeding and that mothers enclosed their infants within their arm(s) for large portions of the night (unpublished observations) argue against a passive effect of bed size. Breast-feeding places infants and mothers in extremely close proximity and in face-to-face orientation, both of which are often maintained afterward since infant and mother commonly fall back to sleep with the infant still attached to the breast. [Note also that breast-feeding, which is thought to confer a protective effect against SIDS through mechanisms not yet identified (Hoffman et al., 1988), appeared to position infants' faces generally in the line of the mothers' expired air.] Together with observations that bedsharing increases nocturnal breast-feeding (McKenna et al., 1997) and that breast-feeding mother-infant pairs who do not routinely bedshare also typically sleep close together and face-to-face when they do bedshare (Richard et al., 1996), this suggests that breast-feeding contributes importantly to the mutual orientation and proximity of mother and infant during bedsharing. The relative body positions and degree of physical proximity that non-breast-feeding

mother-infant pairs would attain while bed-sharing remain to be explored.

Although the age at which infants are first able to change body position is unclear, there is general agreement that at 3 mon of age sleeping position is still largely managed by the caregiver, except perhaps when infants are placed in the more unstable side position (Hassall and Vandenberg, 1985; Engelberts and de Jonge, 1990). The facts that the mothers in our study almost never placed infants on their sides bodily facing away from themselves and the infants never turned over on their own suggest that the high degree of face-to-face orientation we measured was in large part determined by the mother. However, the observation that even when supine the infants' heads were turned to face their mothers on average 80% of the time suggests that infants too preferentially orient toward their mothers. Other evidence exists to support this notion. By 2 weeks of age, breast-fed infants preferentially orient (by head turning) toward odors from their mothers' breast and axillary regions during sleep as well as waking (Cernoch and Porter, 1985; Russell, 1976; Schaal et al., 1980; MacFarlane, 1975).

Atmospheric concentrations of CO₂ were measured at the same distances from the nares of women as commonly separated the noses of mother-infant pairs while bedsharing. By sampling CO₂ in the line of expired air, we attempted to assess the maximum potential for infant exposure to CO₂ from a cosleeping mother, on the basis of 1) the short ranges at which mothers and infants sometimes slept and 2) the observation that the infant's face is typically positioned in the line of the mother's expired air for breast-feeding. CO₂ was significantly elevated at all distances measured. For women in the sitting position, peak CO₂ concentration averaged 2.36% at 3 cm; it remained above 1% at 9 cm, was approximately 0.5% at a distance as great as 18 cm, and was still about eight times room air at 21 cm.

We noted that mothers and infants often slept together under a common blanket, and since infants typically were positioned lower in the bed than the mothers (i.e., infant's head close to the level of the mother's breast), a potential emerged for the blanket to form

an air pocket, around the infant's head, into which the mother might breathe. This condition was of particular interest given recent evidence that accumulation of lethal levels of CO₂ in bedding might contribute to some cases of SIDS (Chiodini and Thach, 1993; Kemp et al., 1994). Using a doll to simulate an infant, we found that a blanket partially covering the doll's head could trap CO₂ from a reclining woman's breathing. Both trough (inspiratory) and peak (expiratory) CO₂ concentrations were raised by the blanket's presence at the measured distances of 9, 15 and 21 cm. Using the mean CO₂ concentration over 30 sec intervals as an index of the doll's CO₂ exposure from the women, we found that the blanket produced a highly significant increase in CO₂ exposure. With the blanket, the group mean CO₂ concentration over the 30 sec interval was highest at 9 cm (0.75% CO₂), dropping to 0.51% CO₂ at 21 cm. Of note, with the blanket, trough CO₂ levels remained above 0.2% for the majority or all of each sample for five of the six subjects, even at 21 cm. These CO₂ levels indicate that infants could be breathing slightly hypercapnic levels of CO₂ continuously for extended periods, if such a pocket were formed. It is noteworthy that the CO₂ concentrations observed with the blanket are similar to inspiratory CO₂ levels measured in infants when "mother and infant were close together and partly under covers" in a recent preliminary investigation from another laboratory (Sawczenko et al., 1995).

The literature on infant responses to hypercapnic exposure suggests that the concentrations of CO₂ we found, with and without the blanket, have the potential to significantly affect infant respiration. Most ventilatory response studies to CO₂ have been performed on premature and term neonates exposed to relatively high ($\geq 3\%$) concentrations of CO₂. However, using steady state 2% CO₂, Schafer et al. (1993) found that minute ventilation increased by 17–33% in infants aged ≤ 18 mon old, and in infants aged ≤ 4 mon Haddad et al. (1980) reported an average increase of 33%. In both studies, the ventilatory response largely resulted from increased tidal volume (not measured in our study), with little change in respiratory frequency. Studies in younger infants

suggest that even lower concentrations would result in ventilatory changes. Steady state concentrations as low as 0.5% CO₂ increased minute ventilation significantly and converted periodic breathing, thought to be indicative of respiratory instability, to a normal breathing pattern in term newborn and premature infants (Kalapesi et al., 1981; Cross et al., 1953; Katz-Salamon et al., 1991). Together, these studies demonstrate that the infant respiratory system is sensitive to very low concentrations of CO₂ well within the range of values we measured, even without the enhancement caused by the blanket. At the other, high end of the CO₂ spectrum, steady state breathing of concentrations of $\geq 4\%$ is reported to often result in infant arousal (Katz-Salamon et al., 1991; Brady et al., 1985; Ariagno et al., 1980). We never observed such high levels during any measurements.

It is difficult to extrapolate from steady state experiments to the dynamic bedsharing environment where CO₂ exposure could vary appreciably over relatively short periods of time. However, the air pocket simulations elevated CO₂ to sufficient levels for sufficient durations to probably increase minute ventilation and perhaps to reduce periodic breathing in infants. Data are very limited on which to base predictions about the effect of exposure to the more intermittent and bolus increases in CO₂ which probably also occur during bedsharing in the absence of an air pocket effect. One study showed that a 10 sec bolus of 7% CO₂ acts a powerful respiratory stimulus in pre-term infants (Rigatto et al., 1991). Literature on infant exposure to smaller boli (of shorter duration or lower concentration) of CO₂ is completely lacking with the exception of one preliminary report in term infants ≤ 2 wk old that raising inspired CO₂ to 2–4% "for a few seconds" produced transient increases in ventilation (Fleming et al., 1988). That breathing could be influenced by bolus exposure to low CO₂ concentrations can be argued on the premise that the respiratory system is designed to maintain stability by responding very rapidly to small changes in blood gases. For oxygen, this sensitivity is exemplified by the peripheral chemoreceptor-mediated decrease in ventilation which has

been measured in the first 10 sec after a *single* breath of 100% O₂ in 1–3 mon old infants (Parks et al., 1991). As Rigatto et al. (1991) have also pointed out, experiments designed to assess the responsiveness of the respiratory system to very brief CO₂ challenges would probably be more meaningful as measures of chemoreceptor sensitivity than the steady state and rebreathing methods now in use which utilize artificially prolonged and high levels of hypercapnia. Experiments also are lacking that address the effect of repeated, short exposures to hypercapnia. The only data available are in adults. Repeated 2 min challenges with 5% CO₂ separated by 5 min of room air produce consistent increases in ventilation but with rapid changes in ventilatory strategy not seen when the challenges are separated by 15 min (Gozal et al., 1995). Two other dynamic aspects of the local atmosphere of the bedsharing environment also need to be tested: the potential effects of reciprocal lowering of the oxygen content on infant respiration and the contribution of the infant's own breathing to local concentrations of CO₂ and oxygen.

These results have several implications for current ideas on the mechanisms of and risk factors for SIDS. First, the results support that bedsharing, at least when practiced with breast-feeding, may minimize prone infant positioning, a known risk factor for SIDS (Willinger et al., 1994). Second, it is important to distinguish the bedsharing infant's potential exposure to CO₂ produced by the mother from conditions thought by some to cause apparent SIDS through suffocation by rebreathing (Kemp et al., 1993; Bolton et al., 1993). In contrast to the intermittent and low level CO₂ exposures suggested by our measurements, lethal rebreathing is believed to occur as a result of entrapment of expired gases while sleeping prone and face down, leading to progressively higher CO₂ levels. Such a pattern was not seen in our study, even with the blanket simulations we performed in which the women's bodies could also have acted to reduce dispersal of CO₂ away from the doll. Furthermore, because both mothers (Mosko et al., 1997) and infants (Mosko et al., 1995) arouse more frequently during bedsharing, associ-

ated body movements should act to aerate the bedding and prevent excessive CO₂ accumulation. It should also be acknowledged that our results do not reflect on conceivable "worst case" scenarios such as might occur if an infant were trapped beneath non-porous bedclothes or even under an adult's body. However, intermittent exposure to low levels of CO₂ during bedsharing, as we measured, might compensate for a respiratory control defect, thought by many to lead to SIDS (Schwartz et al., 1988), by stimulating ventilation and suppressing periodic breathing. Third, some researchers have proposed a role of arousal deficit in the pathogenesis of SIDS (Harper et al., 1981; Hoppenbrouwers et al., 1989; Kahn et al., 1986; McCulloch et al., 1982). Bedsharing might minimize SIDS risk by enhanced multi-modality sensory stimulation associated with sleeping in close proximity, thus compensating with external stimulation for deficient intrinsic arousability. Lastly, the simple fact of the caregiver's proximity during bedsharing and opportunity for close monitoring could be advantageous to an infant at risk for SIDS, as suggested by the nearly fourfold increase in SIDS risk found in association with solitary sleeping when compared to room sharing with adults (Scragg et al., 1996).

In summary, we conclude that, in breast-feeding mothers-infant pairs, routine bed-sharing may minimize prone infant positioning. Furthermore, such pairs can remain face-to-face and in close proximity for large portions of the night, potentially exposing the infant to elevated levels of CO₂ generated by maternal respiration. The concentrations of CO₂ to which infants could be exposed are within the range known to stimulate infant respiration and minimize periodic respiration. However, in situ studies, correlating quantitative measurements of infant respiration with the amount of CO₂ inhaled by infants, the mother's proximity and bedding conditions, are needed to further assess the potential impact of bedsharing on infant breathing. Studies also are needed to evaluate the role of breast-feeding in the relationship of bedsharing to infant sleep positioning. Additionally, future studies should include infants at high risk for SIDS since their ventilatory responses to

CO₂ may be blunted compared to low risk infants (McCulloch et al., 1982; Shannon et al., 1977).

ACKNOWLEDGMENTS

We thank Dr. Raouf Kayaleh for his assistance and the use of his equipment, Dr. James Ashurst for his statistical support, and Dr. Ronald Harper for his editorial comments.

LITERATURE CITED

- Ariagno R, Nagal L, and Guilleminault C (1980) Waking and ventilatory response during sleep in infants near-miss for sudden infant death syndrome. *Sleep* 3:351-359.
- Balarajan R, Raleigh VS, and Botting B (1989) Sudden infant death syndrome and postneonatal mortality in immigrants in England and Wales. *Br. Med. J.* 298:716-720.
- Barry H III and Paxson LH (1971) Infancy and early childhood: Cross-cultural codes. 2. *Ethology* 10:466-508.
- Blair BS, Fleming PJ, Bensley D, Smith I, Bacon C, Taylor E, Berry J, Golding J, and Tripp J (1996) Smoking and the sudden infant death syndrome: Results from 1993-5 case-control study for confidential inquiry into stillbirths and deaths in infancy. *Br. Med. J.* 313:195-198.
- Bolton DP, Taylor BJ, Campbell AJ, Galland BC, and Cresswell C (1993) Rebreathing expired gases from bedding: A cause of cot death? *Arch. Dis. Child.* 69:187-190.
- Brady JP, Chir B, and McCann EM (1985) Control of ventilation in subsequent siblings of victims of sudden infant death syndrome. *J. Pediatr.* 106:212-217.
- Burton RV and Whiting JWM (1961) The absent father and cross-sex identity. *Merrill-Palmer Q.* 7:85-95.
- Caudill W and Plath D (1966) Who sleeps by whom? Parent-infant involvement in urban Japanese families. In R Levine (ed.): *Culture and Personality*. Chicago: Aldine, pp. 25-154.
- Cernoch JM and Porter RH (1985) Recognition of maternal axillary odors by infants. *Child Dev.* 56:1593-1598.
- Chiodini BA and Thach BT (1993) Impaired ventilation in infants sleeping facedown: Potential significance for sudden infant death syndrome. *J. Pediatr.* 123:686-692.
- Cordner SM and Willinger M (1995) The definition of the sudden infant death syndrome. In TO Rognum (ed.): *Sudden Infant Death Syndrome: New Trends for the Nineties*. Oslo: Scandinavian University Press, pp. 17-20.
- Cross KW, Hooper JMD, and Oppe TE (1953) The effect of inhalation of carbon dioxide in air on the respiration of the full-term and premature infant. *J. Physiol.* 122:264-273.
- Davies DP (1985) Cot death in Hong Kong: A rare problem? *Lancet* 2:1346-1349.
- Dwyer T, Ponsonby A-L, Gibbons LE, and Newman NM (1991a) Prone sleeping position and SIDS: Evidence from recent case-controlled and cohort studies in Tasmania. *J. Pediatr. Child Health* 27:340-343.
- Dwyer T, Ponsonby A-L, Newman NM, and Gibbons LE (1991b) Prospective cohort study of prone sleeping position and sudden infant death syndrome. *Lancet* 337:1244-1247.

- Engelberts AC and de Jonge GA (1990) Choice of sleeping position for infants: Possible association with cot death. *Arch. Dis. Child.* 65:462-467.
- Farooqi S, Perry IJ, and Beevers DG (1991) Ethnic differences in sleeping position and in risk of cot death. *Lancet* 338:1455.
- Fleming PJ, Gilbert R, Azaz Y, Wigfield R, and Stewart AJ (1990) Interaction between bedding and sleeping position in the sudden infant death syndrome: A population case-control study. *Br. Med. J.* 301:85-89.
- Fleming PJ, Levine MR, Cleave J, and Long A (1988) Developmental respiratory patterns: implications for control. In RM Harper, and HJ Hoffman (eds.): *Sudden Infant Death Syndrome*, New York: PMA, pp. 291-305.
- Gantley M, Davies DP, and Murcott A (1993) Sudden infant death syndrome: Links with infant care practices. *Br. Med. J.* 306:16-20.
- Gozal D, Ben-Ari JH, Harper RM, and Keens TG (1995) Ventilatory responses to repeated short hypercapnic challenges. *J. Appl. Physiol.* 78:1374-1381.
- Guntheroth WG (1995) *Crib Death: The Sudden Infant Death Syndrome*. Armonk, N.Y.: Futura.
- Haddad GG, Leistner HL, Epstein RA, Epstein MAF, Grodin WK, and Mellins RB (1980) CO₂-induced changes in ventilation and ventilatory pattern in normal sleeping infants. *J. Appl. Physiol.* 48:684-688.
- Harper RM, Leake B, Hoffman H, Walter DO, Hoppenbrouwers T, Hodgman J, and Sterman MB (1981) Periodicity of sleep states is altered in infants at risk for the sudden infant death syndrome. *Science* 213:1030-1032.
- Hassall IB, Vandenberg M (1985) Infant sleep position: A New Zealand survey. *N.Z. Med. J.* 98:97-99.
- Hoffman HJ, Damus K, Hillman L, and Krongrad E (1988) Risk factors for SIDS: Results of the National Institute of Child Health and Human Development SIDS Cooperative Epidemiological Study. *Ann. N.Y. Acad. Sci.* 533:13-30.
- Hoppenbrouwers T, Hodgman J, Arakawa K, and Sterman MB (1989) Polysomnographic sleep and waking states are similar in subsequent siblings of SIDS and control infants during the first six months of life. *Sleep* 12:265-276.
- Kahn A, Picard E, and Blum D (1986) Auditory arousal thresholds of normal and near-miss SIDS infants. *Dev. Med. Child Neurol.* 28:299-302.
- Kalapesi Z, Durand M, Leahy FN, Cates DB, MacCallum M, and Rigatto H (1981) Effect of periodic or regular respiratory pattern on the ventilatory response to low inhaled CO₂ in preterm infants during sleep. *Am. Rev. Respir. Dis.* 123:8-11.
- Katz-Salamon M, Hertzberg T, and Lagercrantz H (1991) CO₂-sensitivity in newborn and young infants tested by the rebreathing method. *Biol. Neonate* 59:126-132.
- Kemp JS, Kowalski RM, Burch PM, Graham MA, and Thach BT (1993) Unintentional suffocation by rebreathing: A cot death scene and physiologic investigation of a possible cause of sudden infant death syndrome. *J. Pediatr.* 122:874-880.
- Kemp JS, Nelson VE, and Thach BT (1994) Physical properties of bedding that may increase risk of sudden infant death syndrome in prone-sleeping infants. *Pediatr. Res.* 36:7-11.
- Klonoff-Cohen H and Edelstein SL (1995) Bed sharing and the sudden infant death syndrome. *Br. Med. J.* 311:1269-1272.
- Konner MJ (1981) Evolution of human behavior development. In R Monroe and B Whiting (eds.): *Handbook of Cross-Cultural Human Development*. New York: Garland STPM, pp. 3-52.
- Lancaster JB and Lancaster CS (1982) Parent investment: The hominid adaptation. In D Ortner (ed.): *How Humans Adapt: A Biocultural Odyssey*. Washington, D.C.: Smithsonian Institution Press.
- Lee NNY, Chan YF, Davies DP, Lau E, and Yip DCP (1989) Sudden infant death syndrome in Hong Kong: Confirmation of low incidence. *Br. Med. J.* 298:721.
- Lozoff B and Brittenham G (1979) Infant care: Cache or carry. *J. Pediatr.* 95:478-483.
- Lozoff B, Wolf AW, and Davis NS (1984) Cosleeping in urban families with young children in the United States. *Pediatrics* 74:171-182.
- MacFarlane AJ (1975) Olfaction in the development of social preferences in the human neonate. *Parent-Infant Interaction*. Ciba Found. Symp. 33:103-117.
- McCulloch K, Brouillette RT, Guzzetta AJ, and Hunt CE (1982) Arousal responses in near-miss sudden infant death syndrome and in normal infants. *J. Pediatr.* 101:911-917.
- McKenna JJ, Mosko S, Dungey C, and McAninch J (1990) Sleep and arousal patterns of cosleeping human mother-infant pairs. A preliminary physiological study with implications for the study of sudden infant death syndrome (SIDS). *Am. J. Phys. Anthropol.* 83:331-347.
- McKenna JJ, Mosko SS, and Richard CA (1997) Bedsharing promotes breast feeding. *Pediatrics*, in press.
- Mitchell EA, Scragg R, Stewart AW, Becroft DMO, Taylor BJ, Ford RPK, Hassall DCH, Barry BMJ, Allen EM, and Roberts AP (1991) Results from the first year of the New Zealand cot death study. *N.Z. Med. J.* 104:71-74.
- Mitchell EA, Stewart EA, Scragg R, Ford RPK, Taylor BJ, Becroft DMO, Thompson JMD, Hassall IB, Barry DMJ, Allen EM, and Roberts AP (1993) Ethnic differences in mortality from sudden infant death syndrome in New Zealand. *Br. Med. J.* 306:13-16.
- Morelli GA, Rogoff B, Oppenheim D, and Goldsmith D (1992) Cultural variation in infant's sleeping arrangements: Questions of independence. *Dev. Psychol.* 28:604-613.
- Mosko S, McKenna J, Dickel M, and Hunt L (1993) Parent-infant cosleeping: The appropriate context for the study of infant sleep and implications for sudden infant death syndrome (SIDS) research. *J. Behav. Med.* 16:589-610.
- Mosko S, Richard C, and McKenna J (1995) Infant sleep and arousals during bedsharing. *Pediatr. Pulmonol.* 20:340 (abstract).
- Mosko S, Richard C, and McKenna J (1997) Maternal sleep and arousals during bedsharing with infants. *Sleep* 20:142-150.
- Mosko S, Richard C, McKenna J, and Drummond S (1996) Infant sleep architecture during bedsharing and possible implications for SIDS. *Sleep* 19:677-684.
- Parks YA, Beardsmore CS, MacFadyen UM, Pallot DJ, Goodenough PC, Carpenter R, and Simpson H (1991) The effect of a single breath of 100% oxygen on breathing in infants at 1, 2 and 3 months of age. *Am. Rev. Respir. Dis.* 144:141-145.
- Ponsonby A-L, Dwyer T, Gibbons LE, Cochrane JA, and Wang Y-G (1993) Factors potentiating the risk of sudden infant death syndrome associated with the prone position. *N. Engl. J. Med.* 329:377-382.
- Richard C, Mosko S, McKenna J, and Drummond S (1996) Sleeping position, orientation and proximity in bedsharing infants and mothers. *Sleep* 19:685-690.
- Rigatto H, Kwiatkowski KA, Hasan SU, and Cates DB (1991) The ventilatory response to endogenous CO₂ in preterm infants. *Am. Rev. Respir. Dis.* 143:101-104.
- Rognum TO (1995) *Sudden Infant Death Syndrome: New Trends in the Nineties*. Oslo: Scandinavian University Press.
- Russell MJ (1976) Human olfactory communication. *Nature* 260:520-522.

- Sawczenko A, Galland B, Young Y, Ring W, and Fleming PJ (1995) Night time mother-infant interactive behavior and physiology: A longitudinal comparison of room sharing versus bed sharing ("co-sleeping"). *Pediatr. Pulmonol.* 20:341.
- Schaal B, Montagner H, Hertling E, Bolzoni D, Moyse A, and Quichon R (1980) Les stimulations olfactives dans les relations entre l'enfant et la mere. *Reprod. Nutr. Dev.* 20:843-858.
- Schachter FF, Fuchs ML, Bijur PE, and Stone RK (1989) Cosleeping and sleep problems in Hispanic American urban young children. *Pediatrics* 84:522-530.
- Schafer T, Schafer D, and Schlafke ME (1993) Breathing, transcutaneous blood gases, and CO₂ response in SIDS and control infants during sleep. *J. Appl. Physiol.* 74:88-102.
- Schwartz PJ, Southall DP, and Valdes-Dapena M (1988) The Sudden Infant Death Syndrome. Cardiac and Respiratory Mechanisms and Interventions. *Ann. N.Y. Acad. Sci.* 533.
- Scragg RKR, Mitchell EA, Stewart AW, Ford RPK, Taylor BJ, Hassall IB, Williams SM, and Thompson JMD (1996) Infant room-sharing and prone position in sudden infant death syndrome. *Lancet* 347:7-12.
- Scragg R, Stewart AW, Mitchell EA, Ford RPK, and Thompson JMD (1995) Public health policy on bed sharing and smoking in the sudden infant death syndrome. *N.Z. Med. J.* 108:218-222.
- Shand N (1981) The reciprocal impact of breast-feeding and culture form on maternal behavior and infant development. *J. Biol. Sci.* 13:1-17.
- Shannon DC, Kelly DH, and O'Connell K (1977) Abnormal regulation of ventilation in infants at risk for sudden-infant syndrome. *N. Engl. J. Med.* 297:747-750.
- Singh H, Gill PJS, Soni RK, and Raizada N (1992) Sleep pattern and nightly awakening in healthy infants. *Indian Pediatr.* 29:1373-1377.
- Tasaki H, Yamashita M, and Miyazaki S (1988) The incidence of SID in Saga Prefecture (1981-1985). *J. Pediatr. Assoc. Jpn.* 92:364-368.
- Watanabe N, Yotsukura M, Kadoi N, Yashiro K, Sakano M, and Nishida H (1994) Epidemiology of sudden infant death syndrome in Japan. *Acta Paediatr. Jpn.* 36:329-332.
- Willinger M, James LS, and Catz C (1991) Defining the sudden infant death syndrome (SIDS): Deliberations of an expert panel convened by the National Institutes of Child Health and Human Development. *Paediatr. Pathol.* 11:677-684.
- Willinger M, Hoffman MA, and Hartford RB (1994) Infant sleep position and risk for sudden infant death syndrome: Report of meeting held January 13 and 14, 1994, National Institutes of Health, Bethesda, MD. *Pediatrics* 93:814-819.
- World Health Organization (1994) World Health Statistics Annual, 1993. Geneva: WHO.