An Anthropological Perspective on the Sudden Infant Death Syndrome: A Testable Hypothesis on the Possible Role of Parental Breathing Cues in Promoting Infant Breathing Stability, Part I

Author: McKenna, James Joseph, PhD

Publication info: Pre- and Peri-natal Psychology Journal 2. 2 (Winter 1987): 93-135.

Abstract: None available.

Full Text: Headnote ABSTRACT: This research model moves from a comprehensive review of SIDS research to a consideration of the evolution of human infant development and why we should expect to find that especially in the first year of life, parent-infant sleep contact asserts a significant physiological regulatory effect on the infant's breathing. Prenatal studies of fetal hearing and breathing are reviewed and used to argue that the central nervous system is at birth already sensitized to parental breathing rhythms to which the infant in its "expected" postnatal environment will have access. This perspective shows that important continuities exist between fetal experiences and infant respiratory behavior. Emerging from an integration of clinical, experimental, and developmental studies with an evolutional perspective (which includes cross-species and cross-cultural data) it is hypothesized that access to parental sensory breathing cues (movement, sound, touch, and expelled CO2) ought to help one of many subclasses of infants to override breathing control errors, some of which may be involved in SIDS. This model and the testable hypotheses which emerge from it do not suggest that parent-infant cosleeping necessarily should be recommended, or that it is always safe for the infant; only a careful analysis of the entire constellation of family attributes can determine this. Rather, the paper argues that for some infants certain CNS deficits suspected to be involved in SIDS may interact with nocturnal separation from parental breathing cues (a novel experience for our species) to increase crib death risk and, thus, must be considered as one of the many aspects in the unfolding pathophysiology of SIDS for some victims. This model is offered not as a substitute for the traditional SIDS research model which assumes the primacy of internal breathing control mechanisms, but as a complementary research approach-one in which both internal and external (environmental) factors are thought to interact to affect breathing behavior. This interactional model is justified by a variety of data revealing that aspects of parent-infant physiological synchrony are best understood in terms of the four million years in which parent-infant contact was almost certainly continuous for the infant during the first year of life wherein a high degree of postnatal immaturity is characteristic. INTRODUCTION I suspect that many negative findings would not be so statistically negative if the workers were prepared to see their own work in the light of being one of many contributing causes in a changing causal matrix rather than 'the cause' of a pathological unstable entity.-Emery 1983:132(1) For infants between the ages of one month and one year, sudden infant death syndrome (SIDS), often referred to as cot or crib death, remains the major cause of death. Recognized as a distinct medical entity only as recently as 1963 at the first international SIDS conference, the syndrome continues to be defined as "the sudden death of an infant or young child, which is unexpected by history, and in which a thorough postmortem examination fails to demonstrate an adequate cause of death" (Beckwith, in Bergman, Beckwith, and Ray 1970:18; Wedgewood and Benditt 1965; Valdes-Dapena 1978; Barnett 1980). Since that first meeting, scientists have cooperated in rigorous experimental and longitudinal studies of SIDS (Peterson 1983); yet even after 22 years of persistent and vigorous research, this syndrome still kills annually an estimated 10,000 infants, or approximately two out of 1,000 live births in the United States alone (Tildon, Roeder, and Steinschneider 1983). In the words of one investigator, whose research on the subject spans over 20 years, "There is not yet one positive criterion that can be employed by the clinician to identify the future victim, nor is there yet one positive criterion that the pathologist can use to recognize the subject at autopsy" (Valdes-Dapena 1980:4; Standfast, Jereb, Aliferis, and Janerich 1983). Because infantile defects are so hard to detect both before and after death, an increasing number of SIDS

researchers have come to suspect that the defects involved may be quite "subtle" (Haddad and Mellins 1983) and that there is probably more than one "cause" of sudden infant death. "At a particularly vulnerable period in the infant's life multiple defects may converge," Barnett (1980:13) suggested, either alone or in some combination, leading to death. Perhaps SIDS victims differ structurally, physiologically, and/or immunologically from surviving infants only in degree and not in kind. Thus, Lipsett, a developmental psychologist, has encouraged researchers to consider this possibility: "The environment may fail to instruct the infant by the age of two to four months in how to defend its respiratory passages from occlusion . . . experiential factors conspire with constitutional defects to create crib death risk" (Lipsitt 1981:142). I endorse Lipsitt's view, and in this paper I shall explore it in detail, from an overview of the epidemiological evidence through a review of SIDS research and finally to an anthropological framework in which I shall propose a connected series of new and testable hypotheses. I shall argue that a research strategy that combines anthropological perspectives on the evolution of human infant development and clinical findings on SIDS can help determine with fresh insights which of the infant's experiential factors may be conspiring with other factors, and how and why they do so to create the conditions under which certain forms of sudden infant death are most likely to occur. This paper does not propose any new "causes" of SIDS or any single environmental remedy that will eradicate it. Rather, I shall present cross-disciplinary data pointing to environmental factors possibly related to some SIDS pathogeneses-factors that have not been adequately studied alongside more traditional laboratory and clinical approaches. SIDS has eluded our understanding for over twenty-two years in the face of other major advances in perinatal and neonatal medicine (Peterson 1983). This fact alone suggests the need to supplement the current medical paradigm and to reevaluate the accompanying set of assumptions traditionally employed in its study. Anthropologists have much to offer here. For example, one consequence of reconceptualizing the SIDS research assumptions from an evolutionary point of view, including both cross-species and cross-cultural perspectives, is that the physiological regulatory effects that parents and infants have and can be expected to have on each other are better appreciated. Judging from the recent developmental studies of both human and nonhuman primates (Reite and Field 1985) as well as from the ethnographic data (Konner 1981; Tronick et al., 1985), it appears that in our enthusiasm to view the human infant as a competent, preadapted organism, which clearly it is (Kagan 1984), we have pushed too far the concept of the infant's physiological independence from the parent. For example, one inference that can be drawn from the cross-cultural and evolutionary data is that by having them sleep alone through the night and at very young ages, we may be conditioning urban infants to sleep for artificially prolonged stages of the sleep cycle before their systems are best able to do so (see Konner and Super in press). While not a perspective necessarily argued by sleep clinicians nor by medically trained researchers, nevertheless their data supports this interpretation (see Anders et al., 1983; Paret 1983; Emde et al., 1971). It is significant that SIDS research does not recognize, theoretically or experimentally, that human infants have an evolutionary past. Rather, SIDS research focuses almost exclusively on infants living in urban, Western, and highly industrialized settings, which from an evolutionary point of view, are novel conditions (Lancaster and Lancaster 1982; Konner 1981; Short 1984). Unfortunately, this focus may overlook possible mismatches between the sensory expectations of young infants and the stimulation of most urban care-giving practices. The consequences of this sensory deprivation may not be important to most infants, who are able to respond to environmental disturbances of this sort (Slobodkin and Rappoport 1974; Chisholm 1983; Tronick et al., 1985). But for those infants who begin life with some kind of central nervous system impairment-the number may be as high as 3 to 10 percent of all newborns, according to Kagan (1979:13)-these deprivations may prove to be more significant than previously realized. In this paper I shall propose that there may be a prenatal central nervous system format for postnatal parent-infant breathing reciprocity and that these adaptations are complementary to the human species-specific neurological changes required for speech breathing. I shall begin by describing how fetal liquid breathing, fetal hearing, and fetal respiratory neuronal activity develop at around the same time in utero. I believe that these systems are both structurally and functionally related to a degree

heretofore unappreciated, particularly because fetal breathing is now thought to be a necessary antecedent of postnatal breathing and emerges against the background of a rhythmic auditory (cardiovascular) pacemaker, consisting of a ventricular-induced blood surge heard by the fetus as its mother's blood rushes through her abdominal wall just a few centimeters away from the fetus's ear. Guided by the belief that natural selection operates on the fetus's experiences to make it sensitive to the postnatal environment into which it will be born, my second testable hypothesis is that if given the opportunity to do so, both infants and parents will monitor and respond to each other's vesicular breathing sounds (or lack thereof) and that these sounds are ordinarily supplemented by the infant's feeling the parent's chest rising and falling rhythmically and by CO2 rhythmically expelled by the parent and breathed in by the infant in the parent-infant microenvironment. The result is, I believe, that the rate and form of the parent's, and especially the infant's, breathing will change temporally to become more regular insofar as the infant will be aroused by its parent's sensory cues. I shall present data from different fields showing why these compensating "natural," or evolutionarily old sensory stimuli, can "remind" infants to breathe, should their own internal and developing breathing control systems falter. I shall suggest also that these stimuli may prevent the kind of respiratory collapse associated with some, but not all, cases of sudden infant death. The evolutionary and developmental model proposed here for sudden infant death syndrome is supported by discoveries by linguists, respiratory physiologists, paleoanthropologists, and physiological psychologists who have studied the emergent biological bases of language and, in particular, how and when infants learn to "speech breathe." I shall use these data to frame a general answer to the question of why SIDS is so circumscribed by age and why it has not yet been replicated experimentally in other species. That is, these data also suggest that SIDS may be a human, species-specific malady. I argue that the complexity and extensiveness of the developing intercommunication between the cortex and the brain stem, that enable the changes necessary for infants' voluntary respiratory behavior when they cry and emit noncrying vocalizations, put them in greater respiratory jeopardy than nonverbal species. Specifically, at around two to four months-the same time that infants are at the greatest risk for SIDS-there is a major shift in the extent to which cortically based voluntary breathing and more primitive, brain stem-based, automatic breathing become functionally integrated and shift back and forth using specific neurological conduits. The volitional or purposeful manipulations of airflow rates that infants exhibit at this early age precisely parallel those manipulations required for speech. In fact, crying behavior itself seems to be practice for speech breathing and is mastered before speech begins at seven months of age. The case for a link among these systems that begins in utero points to an evolutionarily based, experientially learned component to breathing, which Lipsett (1981) might endorse. This is supported by studies of the physiology and neurobiology of infantile speech breathing, studies of the difficulties that hearing-impaired persons have in coordinating their breathing and vocalizing, and finally, from what is known about the abnormal crying behavior of SIDS victims. I shall show that the evolutionarily old environmental support systems play an important role in helping human neonates to weather a critical respiratory developmental shift. These systems are relevant to understanding some SIDS pathophysiology. My hypotheses are based on three assumptions that differ from the assumptions ordinarily made by SIDS researchers: (1) The human infant's physiological and social needs are inseparable, because they evolved during at least four million years of expected patterns of parental association and care, which included almost continuous contact with a parent or care giver (Tronick et al., 1985) throughout at least the first year of life and cosleeping and nursing through the night. (2) The human infant's evolved developmental and physiological needs are much less able to change and so change much more slowly than does the behavior of parents who respond to infants in culturally prescribed ways. (3) The infant and its needs are conceptualized in terms of what the infant's evolutionary history suggests to be ideal and not according to both conscious and nonconscious ideologies compatible with our highly complex, industrialized, and recently individualized life style. We may never be able to protect some infants from SIDS. Pathological reports of SIDS victims indicate that the population is exceedingly heterogeneous and that the epidemiological profiles of the circumstances in which

infants the are, likewise, extremely variable. Even assuming that the experimental work currently being conducted (by a team of researchers including myself) will confirm the parent-infant auditory-breathing connection, the hypotheses regarding the role of parental breathing cues may be relevant to only a small subclass of potential SIDS victims, and we still will have to prove that those infants who have access to external breathing cues are less at risk for SIDS. Nonetheless, both biological and social anthropologists are in a particularly strong position to consider aspects of SIDS research in light of their own work. Thus, they are also in a strong position to illuminate important experiential factors (to return to Lipsett's phrase) that may help override the particular defect(s) that cause some kinds of SIDS and that the SIDS medical research paradigm is less likely to discover. This paper will attempt to show, then, why anthropological contributions to this area are needed. At the very least, the evolutionary and cross-cultural data can provide insights into the kinds of environmental factors characteristic of the neonate's and infant's microenvironment that can diminish or exacerbate stress or, in some cases, neutralize the effects of central nervous system impairments. Anthropological theories and methods offer many reasons and much confidence for appraising the puzzle of SIDS from a complementary but nevertheless different direction. Even the kinds of questions that are raised simply by making different assumptions about infants are relevant to understanding this tragic infant malady. CHARACTERISTICS OF THE SUDDEN INFANT DEATH SYNDROME The methodological disadvantages of studying a syndrome that cannot yet be limited to a range of etiological possibilities are enormous. To illustrate, consider that even to standardize the reporting and postmortem diagnosis of SIDS, a single criterion for diagnosis is ordinarily required. Thus, its absence causes confusion about the kind of information that should be secured and how SIDS victims can consistently be identified. Now, however, there are formal guidelines for autopsies of suspected sudden infant deaths (Jones and Weston 1976), and in addition to including socioeconomic, environmental, parental, gestational, and familial data, there are now specific autopsy procedures and checklists. But, even though pathologists have contributed the most to standardizing postmortem diagnoses (Valdes-Dapena 1984), serious diagnostic and methodological difficulties remain (especially see Beckwith 1983). Before their deaths, some SIDS victims are known to have experienced one or several of the following conditons (Barnett 1980; Valdes-Dapena 1980, 1978; Tilden, Roeder, and Steinschneider 1983): * prolonged breathing cessations (apneic attacks) with brachycardia (a slowing of the infant's heart rate to less than 100 beats per minute); * less stable body temperatures; * low APGAR scores (a rating from 1 to 10 assigned to the infant based on its condition and responses at birth); * slower growth rates and weight gain; * quieter, less active temperament; * higher mean heart rates, with less variability during the first two months; * increased Cortisol or stress hormone levels; * longer quiet and active sleep periods, with longer sleep cycles in general and less frequent arousals; * abnormal, high-pitched, biphonated cries. Yet, unfortunately, because so many infants the from SIDS who do not experience any of these conditions whereas others who live do, none of them can be used to predict reliably which babies are at risk for SIDS. But in the minds of many investigators (Froggatt 1983; Beckwith 1973; Schwartz 1981 as cited in Arnon 1983), the single most intriguing clue to understanding the causes of sudden infant death is the syndrome's unique age distribution. No other infant malady, except for infant botulism and possibly another bacterial infection of the intestines-salmonellosis-is so consistently and narrowly delineated by age (Arnon 1983). With some exceptions, neonates up to three weeks of age seem to be immune to SIDS, but shortly thereafter sudden infant death rates increase and peak generally between two and five months, or at around ten weeks of age (Carpenter 1983). Some studies support a death peak at about two months (Krauss 1983), whereas the aggregate data support a peak at around four months, or 18.1 weeks, with a median age of 13.8 weeks (Valdes-Dapena 1980). Analyzing 13 years of data from King County, Washington, Peterson (1983) found that SIDS deaths occurred at an age of between 7 and 270 days, with a median of 90 days. Valdes-Dapena's summary of all SIDS death rates, published between 1975 and 1979, reveals that 90 percent of sudden infant deaths occur before six months of age and that 99 percent of them occur before the infant is one year old (it is exceedingly rare for children over

one year of age to the from sudden infant death syndrome). SIDS rates vary by sex and across cultures, geographic areas, and seasons of the year. For example, the rates are relatively low in Stockholm, Sweden, Israel, the Netherlands, and Czechoslovakia (0.06, 0.31, 0.42, and 0.8 infants, respectively, per 1,000 live births) but are high in Ontario, Canada, Northern Ireland, Great Britain (Oxford Linkage Area), and King County, Washington (3.0, 2.8, 2.78, 2.32 infants, respectively, per 1,000 live births) (Valdes-Dapena 1980:7). The rate for King County, Washington is five times the rate in Sweden, whereas in Hong Kong, according to a recent survey, SIDS appears to be rare, possibly fifty to eighty times less the frequency of Western countries (Davies 1985), the significance of which will be discussed later. A few years ago, it was thought that SIDS rates were decreasing (see Valdes-Dapena 1980), but Peterson (1983) indicated that although general perinatal mortality rates for infants zero to six days of age have decreased significantly, SIDS rates have remained relatively stable. Data from other studies confirm Peterson's findings (Tildon, Roeder, and Steinschneider 1983; Krauss 1984; Bryan 1984). Except for the death rate for SIDS reported for Native Americans-the highest yet reported (7.13 per 1,000 live births)-boys succumb worldwide more frequently to sudden infant death (1.82 per 1,000 live births) than do girls (1.26 per 1,000 live births). Although these figures are based on one study of 525 California SIDS victims, they are consistent with the data from Czechoslovakia, Great Britain, Northern Ireland, and Washington, which show that between 58 and 59 percent of all SIDS victims are males. However, black and Native American males do not significantly differ from their female counterparts with respect to SIDS death rates (Bergman, Pomeroy, Wohl, and Beckwith 1972; Krauss and Barhani 1972; Krauss, Franti, and Barhani 1972; summarized and cited by Valdes-Dapena 1980). Several studies show convincingly that death rates for SIDS are higher among the poor. The rates are higher among blacks, even when socioeconomic status is held constant (Valdes-Dapena 1978,1980; Beal 1983; Froggatt 1983; Krauss 1984; Peterson 1983) and are the lowest among Asians (Davies 1985) and Swedes (Norvenius 1984). In all countries, however, across all cultural and geographic settings, SIDS rates triple during winter or cold months, suggesting to some investigators that in some cases either infectious agents are involved or that infants are, indeed, being overheated by blankets, as Stanton (1984) suggested. The narrow age distribution of SIDS, its resistance to advancing pediatric medicine, which has reduced deaths from other conditions, and the more frequent occurrence of SIDS among boys, the poor, and blacks and in the colder winter months are no less mystifying than the time of day at which infants tend to die and the circumstances surrounding their deaths (as far as they are known). These circumstances are diverse. For example, infants have died from SIDS when their mothers were pushing them in shopping carts or carriages, as their parents held them, or as they slept on their mother's ventrum. But most often, SIDS victims are discovered in their cribs, presumably having died during sleep. Insofar as investigators can tell, infants do not appear to have experienced an agonizing or painful death. Rather, they appear simply to have failed to take the next breath, and always when they are asleep (Lipsett 1981; but see Weinstein and Steinschneider 1983, who question this). It is as if, in the words of Lipsett (1981), some infants simple "forget" to breathe or cannot arouse themselves enough to take the next breath and so to continue the respiratory cycle (Guntheroth 1977, 1982; McGinty 1984). In general, most of these deaths occur out of the sight of the care giver, between midnight and 7:00 A.M. Valdes-Dapena (1980) summarized data from the United States and abroad and found that over 50 percent of the SIDS deaths occurred during an eight-hour period beginning at midnight, whereas 34 percent of the infants died between 8:00 A.M. and 4:00 P.M., with the largest number clustered in the early morning hours, although they might actually have died earlier. Approximately 13.6 percent of the deaths took place between 4:00 P.M. and midnight. And as would be expected, a majority of the deaths (70 percent) occurred at home, 22.3 percent at the hospital (see below), and 6.8 percent en route to a hospital. These data may be skewed in favor of deaths reported outside the home, as such infants are often pronounced dead at the hospital or in the ambulance by medical professionals. While the statistics might suggest otherwise, SIDS occurs extremely rarely in the hospital (Peterson pers. comm.). Three other epidemiological factors are being clarified by a recent cooperative study funded by the National Institute of Child Health and Development.

Although as of this writing the data have not yet been completely analyzed (Barnett 1980, Hoffman 1984), based on data from 400 of 800 SIDS victims from six regions of the country, compared with data on 800 healthy infants, Hoffman (1984) reports that 40 percent of the mothers of healthy infants smoked during their pregnancy, compared with 70 percent of mothers of SIDS victims. Recall that statistics confirm that blacks are three times more likely than whites to have an infant die of SIDS and that black women smoke more during pregnancy than white women do. Intercorrelative tests and multivariate analyses are still needed to sort out these variables (Peterson 1983). Another important question is the occurrence of SIDS among family members, that is, its genetic connection. Peterson (1983:84) points out that although "the risk of SIDS repetition in families is not negligible, at the same time, it is not sufficiently high to discourage future childbearing." For example, Hillman (1984) reports that there is only a 1 percent chance of a sibling dying from SIDS after one has already died from it. Peterson states: "The risk to subsequently born siblings has been estimated at about 20 per 1000" (1983:95). Moreover, data from this NICHD study show that episodic apnea (prolonged but temporary breathing cessation during sleep, leading to depleted oxygen [hypoxia] and excessive carbon dioxide [hypercapnia] or cyanosis [blue-tinged] infants) is "200 times more likely for an infant who does not succumb [to SIDS] than one who does" (Peterson 1983:95; Froggatt 1983; Krauss 1983). These two findings imply to some SIDS researchers that it is futile to focus on samples consisting of so-called near-miss infants, or infants who are thought to need help during apneic attacks (Peterson 1983; but also see Tilden, Roeder, and Steinschneider 1983). Finally, SIDS may be one of the few infant-related reproductive syndromes that give older mothers an advantage over younger ones. Recent data confirm earlier findings that mothers aged twenty or younger are at a higher risk than are older ones of having their child die from SIDS (Standfast, Jereb, Alifens, and Janerich 1983); moreover, these risks increase with each additional child. Maternal age, mothering experience and/or competence, and parity may prove to be more important than previously thought (Sears 1984; Peterson 1983). This also may account for much of the variance between blacks and whites: 44.6 percent of black women have their first baby by age 20, whereas only 19 percent of white mothers do (Statistical Abstracts of U.S., 1984). Thus, the higher proportion of black women smoking during pregnancy and their younger age at their first delivery may together partially account for black infants' being born at a higher risk for SIDS than whites and others are. BACKGROUND, REVIEW, AND CLASSIFICATION OF THE SUSPECTED AND IMPLICATED CAUSES OF SIDS Rumors and pet theories about "the cause" of sudden infant death syndrome (SIDS) come and go like epidemics and leave confusion in their wake. (Chiswick 1985:1193) The accumulating research that we shall discuss does not yet explain the process or processes by which sudden infant death unfolds or offer the consistent epidemiological and pathophysiological data required to predict those infants at risk. One of the most frustrating aspects of SIDS research has been the researchers' inability to replicate and validate precisely one another's findings. Even though it is still not possible to identify which organs and/or which physiological defense systems are involved in SIDS, the data collected during the last 15 years have set the stage for what ValdesDapena (1980) describes as "an evolution of an understanding." It is now believed, for example, that SIDS probably has several causes (multiple etiologies), involving different morphologic, biomechanical, neurochemical, physiologic, and even infectious factors. Also, infants who ultimately die of crib death often begin life with both structural and functional abnormalities and are not as healthy as both parents and physicians once presumed (Valdes-Dapena 1980). The problem, of course, is in determining the range of infantile structural-functional defects and understanding what triggers the final event leading to death in some infants with certain defects but not others. Hypotheses concerning the causes of SIDS abound. For example, during the last two years alone, three very different hypotheses have been proposed, that SIDS may be caused (1) by overheating infants with blankets (Stanton 1984), (2) by excessive levels of dopamine (a neurotransmitter) in the infant's carotid or chemoreceptor bodies (Perrin et al. 1984), or (3) by overcompliant infant lungs (Southall et al. 1985). These theories are based on reliable data. My discussion will concentrate on only those areas of research that have been pivotal and historically important and not on all of the "suspected

causes" listed in Table 1, even though they may be interrelated and may ultimately prove to be important.

Table 1
Observed Abnormality, Condition and/or Suspected Causes of SIDS 1986

Observed Abnormality or Condition	*Investigator(s) (selected studies)*	*Criticized, Reinterpreted or not Replicated By:*
Neurological, Neurochemical, Respiratory or Sleep Dysfunction		
Hypoxia or hypoxemia	Steinschneider 1972 Naeye 1974, 1976, 1980 Guntheroth 1982, 1983 Kelly 1983	Singer 1984 Tilden et al. 1983 Merrit and Valdez-Dapena 1984
Protracted periods of apnea or increased periodic breathing	Steinschneider 1972 Guilleminault et al. 1975, 1976a,b Guntheroth 1983, Read and Jeffrey 1983, Kelly et. al. 1980 Naeye 1974, 1980	Weinstein and Steinschneider 1983 Petersen 1983 Johnson et al. 1983 Bagg et al. 1981 Hodgman et al. 1982 Hodgman and Hoppensbrouwers 1983 Southall et al. 1982, 1985
Deficiency of the arousal response to increased CO_2 (hypercapnia) and decreased 0_2	Hunt et al. 1981 Sullivan 1984	Ariagno et al. 1980
Inability of arousal system increased tendency to remain asleep—abnormal arousal levels in brainstem	Guilleminault et al. 1979a,b Guntheroth 1977 Harper et al. 1981 Hunt et al. 1981 McGinty 1984	Guilleminault & Coons 1983
Presence of petechiae (broken blood vessels) on surface of lungs and general interthoracic vegim caused from central apnea	Guntheroth 1982, 1983	Tilden et al. 1983 Zebal & Friedman 1984
Respiratory vulnerability during REM sleep	Henderson-Smart & Read 1978 Philipson 1978	Johnson et al. 1983 Orr et al. 1985
Pulmonary arterioles are small, constricted, thickened increased muscle mass	Naeye 1973, Mason et al. 1975 Williams et al. 1978	Beckwith 1983 Singer 1984 Kendeel & Ferris 1977
Inability to maintain homeostasis during the developmental period wherein quiet (NREM) sleep is prolonged and predominates	Gould 1983, Salk 1974	
Respiratory muscle failure due to muscular immaturity	Jansen & Chernick 1983 Stanton 1984	
Overcompliant lung	Southall et al. 1985	
Leukomalacia or cerebral white matter lesions caused by hypoxemia and inadequate blood circulation to brain (ischemia)	Takashima et al. 1978b	Beckwith 1983 Haddad & Mellins 1983 Pearson & Brandeis 1983
Abnormal retention of periadrenal fat and adrenal medullatory hyperplasia	Naeye 1976, Naeye et al. 1976a Emery 1978	Emery & Dinsdale 1978 Patrick & Patrick 1982

Table 1
Observed Abnormality, Condition and/or Suspected Causes of SIDS 1986

Observed Abnormality or Condition	*Investigator(s) (selected studies)*	*Criticized, Reinterpreted or not Replicated By:*
Fatty changes (lipid-laden macrophages) in tegmentum of brain	Emery et al. 1983	
Undervascularized reticular formation, gliosis in the brainstem	Naeye 1976, Becker 1983 Takashima et al. 1978a,b Guilleminault et al. 1979b, 1980	Pearson & Brandeis 1983 Kinney 1984
Delayed development of the vagus nerve (mean number of myelenated fibers less than controls)	Sachis et al. 1981 Becker 1983	
Relative immaturity of brainstem immature, as evidenced by dendritic spines rather than more mature bundles	Quaddrochi et al. 1980 Baba et al. 1983	Haddad & Mellins 1983 Pearson & 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. 1976b	Dinsdale et al. 1977 Beckwith 1983 Valdez-Dapena 1983
Increased levels of dopamine in carotid bodies	Perrin et al. 1984	
Neck-Throat Abnormalities		
Hypertrophy of laryngeal mucous glands or increased number of mucous glands	Beckwith et al. 1983 Fink & Beckwith 1980 Haddad et al. 1980 Haddad et al. 1981	
Hypermobility of mandible causing suffocation, pharyngeal upper airway collapse or occlusion exacerbated by nasophagyngitis or infection; small upper airway	Tonkin 1974, 1975, Colton & Steinschneider 1980 Tonkin 1983; Thach 1983 Sullivan 1984 Guilleminault et al. 1975 Guilleminault et al. 1986	Guntheroth 1983
Elongated uvula	Harpey & Renault 1984	Guilleminault 1984
Cardiac Abnormalities		
Right ventricular hypertrophy (heavier, larger right ventricle) indicating hypoxia	Naeye 1973	Williams 1979 Beckwith 1983 Valdez-Dapena 1980
Prolonged QT interval (time between ventricular contraction and relaxation) reducing electircal stability causing ventricular fibrillation	Schwartz 1983 Smith et al. 1979 Southall 1979 Froggatt & James 1973	Kukolich et al. 1977 Steinschneider 1978 Haddad et al. 1979
Cardiac and autonomic inactivity leading to arrhythmias	Church et al. 1967	

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Observed Abnormality, Condition and/or Suspected Causes of SIDS 1986

Observed Abnormality or Condition	*Investigator(s) (selected studies)*	*Criticized, Reinterpreted or not Replicated By:*
Lack of maturational synchrony in right and left sympathetic nerves leading to increased heart rate	Schwartz 1983	
Metabolism		
Thiamin deficiency, when associated with high carbohydrates intake, and general neurochemical defects	Read & Jeffreys 1983 Read 1978	Tilden & Roeder 1983 Barker et al. 1982
Thiamin excess: inability to absorb amounts found in cow's milk	Davis et al. 1983	
Vitamin-D deficiency	Hillman et al. 1980	
Anaphalactic shock-allergy reaction to cow's milk (model based upon data from guinea pigs)	Coombs & McLaughlin 1983 Guntheroth 1977	Valdez-Dapena 1978, 1983
Hypoglycemia due to impaired glucogenesis and/or hyperinsulinism	Sturner & Susa 1980	Swift et al. 1974 Aynsley-Green 1978
Defective liver enzyme, phophoenolpyruvate carboxykinne (PGPCK)		Silverstein el al. 1983 Sturner & Susa 1980
Diminished pulmonary surfactant levels (abstract)	Haddad & Mellins 1983	Morley et al. 1984
Environmental Triggering Factors		
Reduction in breast feeding frequency and contact with mother during night (co-sleeping) microenvironment	Konner & Super (in press) Maxwell & Maxwell 197 Sears 1985	Cunningham 1976 Valdez-Dapena 1979 Davies 1985
Overheating	Stanton 1984	
Carbon monoxide poisoning	Cleary 1984	Emery 1984
Infant botulism (*Clostridum botulin*)	Arnon 1983 Sonnabend et al. 1985 Cornblath & Schwartz 1976	Cunningham 1976 Valdez-Dapena & Felipe 1971

APNEA AS AN ANTECEDENT TO SIDS AND PROPOSED BRAIN STEM AND ARTERIOLE Abnormalities

Much of the research into the possible structural-functional abnormalities of SIDS victims has focused on the morphology of their neurological tissues, especially the medulla oblongata and the pons (the pontomedallary) region of the brain stem's reticular formation. Situated near the brain's central base at the top of the spinal cord, these medullopontine structures are thought to be a primary respiratory of "pneumotaxic" control center, although the question of a central control is still controversial (Mitchel and Berger 1981; Plum and Leigh 1981). Within its boundaries, the reticular formation, which is composed of different clusters of nerve cells and nuclei, regulates breathing rate, rhythms, respiratory drive, tidal volume (the amount of air moving in and out), and functional residual capacity (FRC) (the amount of air remaining in the lungs after a normal expiration). Current research has shown that these neurological substrates receive incoming nerve signals from a variety of sources, including the lungs through the vagus nerve. The vagus nerve communicates the status of the oxygen and carbon dioxide levels and lung stretch receptors, and signals emanating from at least two important sets of chemoreceptors, the carotid and aortic bodies, both of which also monitor blood carbon dioxide levels. The reticular formation nuclei also direct the respiratory muscles surrounding the lungs (the intercostals) as well as the airway passages of the neck and throat. For example, if oxygen is low and carbon dioxide is high, the reticular formation (and possibly structures located on the spinal cord itself) will drive the diaphragm, sitting below the lungs, via the phrenic nerve (Plum and Leigh 1981:1014), leading to the inspiratory and expiratory behavior needed to maintain proper carbon dioxide-oxygen balance. Other muscles of the thoracic cavity, such as the intercostal muscles surrounding the ribs are also implicated in respiratory behavior and are partially controlled by reticular formation nuclei. The system is, of course, much more complicated than this. As the reticular formation also acts as a major conduit between the higher (forebrain) and lower brain structures and

integrates incoming (afferent) and outgoing (efferent) signals from several major nerve tracks such as the corticobulbar, corticospinal, and reticulospinal pathways, all of which are involved in voluntary and involuntary breathing. In this way, the reticular formation and the specific nuclei thought to promote automatic, involuntary, and rhythmic breathing (that is, the nucleus solitarius ambiguous) are neuronally integrated and constantly communicating with other forebrain, midbrain, and hindbrain regions and must therefore be considered part of a larger network of neuronal centers, referred to as the reticular activating system (McGinty 1983). All together, the reticular activating system coordinates breathing with cardiac output, sleep-wake cycles, and arousal (Kahlia 1981; Plum and Leigh 1981; Mitchell and Berger 1981; Darwish and MacMillan 1984). That deficits in the brain stem reticular formation or adjacent structures could be the cause of some forms of apnea, an infantile sleeprelated breathing disorder that in turn could be linked to sudden infant death, was proposed in landmark papers by Steinschneider (1972) and Naeye (1973). Infantile apnea is a conditon marked by a temporary but sometimes prolonged cessation of airflow during sleep (usually more than 10 or 15 seconds). Depending on its duration or the interval between breaths, apnea can lead to low exygen pressure in the blood (referred to as hypoxia or, if chronic, hypoxemia) and high carbon dioxide (C02) levels (hypercapnia). If severe enough, apnea will lead to a cyanotic condition in which the infant becomes limp, pale, or blue tinged, requiring resuscitation- these are the so-called near-miss infants. Steinschneider (1972) studied five apneic-prone infants from three families, two of whom later died from, it is presumed, apneic attacks and were subsequently diagnosed as sudden infant death victims. His conclusion that apneic infants are greatly "at risk" of dying from SIDS galvanized the research in this area. Since that paper's publication, apneic infants and siblings of SIDS victims have been used extensively in research, in the hopes of finding differences in sleep and breathing abnormalities that could, in turn, be linked to SIDS. Although the original hypothesis of an apnea-SIDS relationship has been supported in some ways (see Guilleminault et al. 1986,1976a and b, 1979a and b, and the near-miss infant has come to be used as a standard research subject for SIDS, the relationship between the two is probably not a simple one. In fact, a number of researchers now argue that apnea does not necessarily precede SIDS (Johnson, et al. 1983), as only an estimated five percent of SIDS victims had apneic attacks before they died. The infant's inability to reinstate or reinitiate breathing once an apneic episode has occurred may be the principal defect, not apnea itself. McGinty (1984) and Guntheroth (1983) stress that most infants are able to overcome apnea and that those who die simply do not wake up (arouse) or are not alerted to the need to take another breath. Guntheroth (1982) feels, however, that the apnea-SIDS connection is still important. McGinty (1984) and McGinty and Hoppenbrouwers (1983) worry that too much attention is being given to apnea and not enough to breathing arousal, stimulation, and external modulation or regulation during sleep. With respect to possible arousal deficiencies, some researchers have found, for example, that near-miss infants show a deficiency in arousing to excessive levels of CO2 (hypercapnia) (remember that when there is too much carbon dioxide in the blood, the chemoreceptors induce inspiration) and in decreasing their O2 (Hunt 1981; Sullivan 1984). Yet, others have not been able to find evidence of diminished responsivity, to either too little oxygen or too much carbon dioxide (Ariagno, Nagel, and Guilleminault 1980). Sullivan (1984) proposed that infants have a natural capacity to smell CO2 and that perhaps the CO2 chemoreceptors in the upper nasal passages do not alert the brain stem to remove the carbon dioxide by breathing (see Widdecombe 1981). Investigations of the reticular system's influence on the arousal system have revealed abnormalities in sleep and waking cycles in infants at risk for SIDS. For example, Harper and associates (1981) demonstrated that compared with the controls, the siblings of SIDS victims (considered by some to be a high-risk group for SIDS; see Peterson 1983; Beckwith 1983) displayed abnormal and unusual sequences of quiet, or NREM, sleep. Recall that NREM, or quiet, sleep is characterized by regular respiration and a slow wave encephalography EEG pattern, whereas active, REM, sleep is characterized by irregular respiration and activated EEG and phasic muscle activity, especially of the eyes. The high-risk infants studied by Harper and associates (1981) had difficulty switching from sleep to waking. Simply stated, and in agreement with McGinty (1984), SIDS victims may have difficulties

not only in waking up but also in waking up to breathe as "normal" or surviving apneic infants might (see also McGinty and Hoppenbrouwers 1983). Thus, SIDS peaks at a time when infants are reorganizing their sleep-wake cycles and moving from those sleep states dominated by REM, or active, sleep to NREM sleep, which, according to Gould (1983), makes them more vulnerable to respiratory instability. Although the relationship among particular sleep stages, breathing control mechanisms, and SIDS is still somewhat problematic, studies continue to implicate the brain stem in one way or another, although the data are frustratingly inconsistent. The possibility that some critical brain stem neurons are simply immature and thus unable to transmit or integrate the respiratory signals needed to produce the necessary response (for example, to reinitiate breathing during apnea) is a promising area of research (see Strang 1977 for a review). For example, Haddad and colleagues (1981) showed that the rate of brain maturity of near-miss SIDS infants was slower when compared with that of controls, as indicated by delayed reorganization of REM and NREM sleep patterns. Baba and associates (1983) observed that there may be a functional imbalance between the maturity of two types of intercellular connections-the dendritic spine synapse (the more common) and the electronic spineless connections between the dendrites that permit, in some cases, lateral intercellular communication. If one kind of connection is mature and the other is not, this condition may "compromise the synchronous synaptic capabilities of the reticular network" (Baba et al. 1983:2791), thereby impeding the transfer of information related to respiratory control during sleep, restoring respiration after apnea. Like Guilleminault and associates (1975), Becker and colleagues (1983) suggest that some added minor stress, such as a respiratory tract infection, may compound the infant's vulnerability to respiratory collapse, as it does not yet have adequate neuronal regulatory control. Quattrochi's finding (1981) that SIDS victims retain these reticular dendritic spines (the vehicles by which messages are passed from one cell to the next), thus indicating neuronal immaturity, supports this perspective. Cardiac and Upper-Airway Abnormalities Cardiac abnormalities (see Haddad and Mellins 1983 for a recent review) constitute another set of possible SIDS causes. For example, the length of time between the heart's ventricular contraction and relaxation has been implicated. Known as the prolonged QT hypothesis (the Q and T refer to the wavelengths on an EKG polygraph recorder) and initially discussed by Froggart and James (1973) but formalized by Schwartz (1983), this hypothesis argues that the heart becomes electrically unstable when the length of time between the contraction and the relaxation of the heart muscles is excessive. Schwartz (1983) (also Froggart and James 1973) observed that ventricular fibrillation could occur, quietly and instantly causing death, an epidemiological characteristic of SIDS. Schwartz (1983) indicated that death could occur as a result of the different maturation rates of the heart's right and left sympathetic nerves; that is, the heart rate and the improper timing increase, and thus, so does the heart's instability. Once again, however, Steinschneider (1978) and Haddad and associates (1979), to name but a few studying this, did not find this phenomenon in near-miss infants who later died of SIDS or in living high-risk infants, and thus this explanation also is not satisfactory. Among the structural-anatomical systems that have been linked to SIDS are the laryngeal, pharyngeal, and general upper-airway structures (Sullivan 1984, Guilleminault et al. 1975). For example, Tonkin (1974) suggested that SIDS infants display hypermobility of the mandible, which may cause a collapse or constriction of the airway and produce hypoxia, cyanosis, and then death. Colton and Steinschneider (1980) relate this hypothesis to the SIDS victim's biphonated cry. This structural condition could be exacerbated by nasopharyngitis, or an infection of the upper airways. Continued low subglottal pressure, exacerbated by low functional residual capacity (FRC)-or the amount of air remaining in the lungs following expiration-could lead to pharyngeal collapse. Some researchers have found an excessive number of mucous glands in the laryngeal region, with some being hypertrophied, adding to the possibility that infants die from upper-airway suffocation or excessive secretion that prevents airflow (Beckwith et al, 1983, Fink and Beckwith 1980, Haddad et al. 1980, 1981). The Botulism, Excessive-Thiamin, and Endocrine-Disorder Hypotheses, and Possible Carotid Body Abnormalities From an altogether different perspective, Davis, Icke, and Hilton (1983) studied 233 SIDS victims and 46 controls and decided that SIDS may be caused by the inability of the two to five-month-old infant to

absorb serum thiamin, a naturally occurring vitamin that is found in human milk but that is ten times more concentrated in formula cow's milk. Related to these findings, at least insofar as they concern breast versus formula feeding and SIDS, are two studies, by Coombs and McLaughlan (1983) and Arnon (1983). Coombs and McLaughlan (1983) return to an idea originally reported years earlier: in some cases, infants can die quickly and quietly from anaphalactic (allergic) reactions during sleep, following the accidental inhalation of cow's milk allergens that, through the release of excessive histamines from blood blasophils, immobilize the respiratory apparatus. Arnon's (1983) interpretation of SIDS begins with the observation that the age distribution of SIDS victims, so delineated and consistent, is the most important clue to its pathophysiology. Arnon found that the age distributions of those infants who die from infant botulism and of those who survive the infection precisely match the age distribution of SIDS. The preventive role of breast-feeding in SIDS is still questioned, with some arguing that breast-feeding is preventive and others contending that there is little difference in the rates of SIDS among bottle-fed and breast-fed infants (Beckwith 1979, Valdes-Dapena and Felipe 1971). Although I shall address the role of breast-feeding in the context of nocturnal arousal later in more detail, Arnon's contention is that human milk contains a maternal antibody (secretory Ig A) that functions to agglutinate and destroy the vegetative cells of Clostridium botulinim. The botulin bacteria whose ingested spores (unlike those of food-borne botulism) "generate, multiply, 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 arteriorly to motor nerve endings, causing respiratory muscle paralysis and death. Arnon (1983) observed that some lymphocytes sensitized to foreign bacterial invastion in the mother's gut are known to migrate to the mother's nipple area to produce antigenspecific S-Ig A antibodies within three 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 to both a "lactating woman's intestines" and immunological factors such as lactoferrin, lysozyme, and S-Ig A antibody (Ogra 1979; cited in Arnon 1983). Arnon's point is that such a breast-feeding system fights an array of rapidly developing and potentially fatal infant bacteria, as well as preventing the anaphalactic shock that may accompany premature exposure to other foods, regardless of whether that food is liquid or solid (see also Jeliffe and Jeliffe 1978). Arnon concluded by stating that the failure to find evidence of the botulism organism in SIDS may reflect geographic or methodologic differences or intraspecies differences pertaining to maternal antibody populations. This may explain why both toxins and organisms were found in only 41.7 percent (10 out of 24) cases of SIDS (see also Sonnabend et al. 1985). Finally, endocrinological and metabolic disorders, such as hypoglycemia and the instability of enzyme production needed for glucogenesis, have been implicated as contributing causes of SIDS. For example, the liver enzyme phosphenolpyrule carboxykinase was found in low amounts in SIDS victims (Sturner and Sosa 1983; Silverstein et al. 1983). Moreover, Aynsley-Green (1978) and Swift and colleagues (1974) argue that the sugar needed for a variety of metabolic functions was found in dangerously small amounts in high-risk infants, because of hyperinsulinism, the pancreas's production of too much insulin, removing excessive amounts of sugar from the body. A review by Tilden and Roeder (1983) pointed out the difficulties of assessing the status of biologically pertinent fluids in dead bodies and discussed the inconsistencies in the metabolic and endocrine characteristics thought to lead to SIDS. They indicated, too, that currently the endocrinological or metabolic factors, perhaps related to SIDS, have not received the attention necessary to standardize research techniques. Another line of research has concentrated on possible defects in the carotid bodies, the two oval-shaped structures budding off each carotid artery (one on each side of the neck) involved in blood gas homeostatis. The carotid bodies are glandlike tissues considered to be major chemoreceptors that help maintain respiratory rhythm and a proper 02-C02 balance. Naeye and associates (1976b), Takashima and associates (1978a), and Cole, Lindenberg, and Gallato (1979) found fewer cells and abnormal cells in the carotid body of SIDS victims. Most recently, Perrin and colleagues (1984) found higher levels of the neurotransmitter dopamine in the carotid bodies of SIDS victims. However, although nobody has denied the importance of the carotid bodies in helping

regulate respiratory stability and rhythm, Valdes-Dapena (1983), Dinsdale et al. (1977), and Beckwith (1983) all had problems replicating Naeye's findings of carotid abnormalities in SIDS victims, thus placing these findings in dispute (see also Haddad's and Mellins' 1983 findings). For example, Pearson and Brandeis (1983) studied nine infants who died from SIDS and 13 diseased controls. They found no carotid abnormalities in the SIDS sample; they therefore doubt that the carotid body plays any role in SIDS pathophysiology. ESTABLISHING A THEORETICAL BASIS FOR SIDS RESEARCH Table 1 and the epidemiological data discussed earlier reveal the extent of heterogeneity found in the SIDS population and illustrate why most researchers suspect that SIDS is caused by many, interacting factors, although these factors are not expressed to the same degree or found in all victims. An overview of SIDS research indicates that SIDS researchers make certain assumptions about infant development. For example, they assume that the human neonate-infant's developing physiological systems, including its respiratory system, are not influenced enough by the social and physiological interaction with a parent figure to call into question those SIDS studies that monitor, for instance, high-risk infants sleeping and breathing alone. Likewise, the postmortem analysis does not take into account that a highly immature neotenous creature is being examined; rather, the analysis is directed toward isolating from their immediate microenvironment any defects, or sets of defects, found in the tissues or organs of the dead infants. In addition, the postmortem analysis does not usually consider, at least in detail, that the infant was both physiologically and psychosocially dependent on the care giver at the time of death and, thus, that the seriousness of such defects may depend on how the caregiver's behavior mitigated or exacerbated them. No studies, for example, have yet looked at the physiological effects of human infants sleeping with their parents, the context in which, for at least three million years, the infant's developmental system evolved (Konner 1981). These observations are not meant to imply that SIDS researchers have not been interested in, or aware of, the possible socioenvironmental correlates of SIDS (see Valdes-Dapena 1977, 1980). Also, it is not implied that because of expense, technological considerations, or the like this line of inquiry cannot be justified. It does mean that important complementary lines of research have not yet been explored adequately, and it is here that anthropologists can contribute. The advantage of an evolutionary and cross-cultural approach that considers developmental processes simultaneously with clinical findings is that it offers a guiding theory thus far missing in SIDS research. Such a method offers a basis for predicting how infants who differ constitutionally can be expected to respond to environmental perturbations. So far, SIDS research has not enunciated its assumptions about the human infant's developmental systems or explained the ones that can be inferred. The evolutionary approach maintains that in order to understand how an infant's developmental or physiological systems can go awry, we must first consider that natural selection not only adapts the fetus to its prenatal (uterine) environment but also prepares it for postnatal events. The evolutionary approach thus forces us to recognize the kinds of conditions into which infants "expect" to be born and to confirm these "expectations" by means of cross-cultural and anthropological data. Over the long term, natural selection has, of course, adapted the development of the highly neotenous, or slow-maturing, human primate to the expected environmental conditions by which its maturing systems are cued and protected from assault. By applying the rules of epigenesis to long-term evolution, we can create a theoretical basis for predicting which structuralfunctional systems that were linked prenatally will be linked postnatally and how and why certain prenatal perturbations should affect infants' ability to respond postnatally to similar or different environmental disturbances. A TESTABLE HYPOTHESIS LINKING THE EMERGENCE OF FETAL HEARING AND BREATHING In this section I shall argue that natural selection has produced developmental continuities between the fetus's respiratory experiences and its postnatal respiratory behavior and that the parent's hypothesized ability to drive, or at least affect, the infant's breathing has prenatal structural and functional antecedents. I shall use research on a variety of species to justify my contention that the maternal cardiovascular rhythms processed by the fetus's auditory, vestibular, and metabolic pathways can provide a reference point by which postnatal parent-infant respiratory interdependence, if given the chance to grow, can be predicted (see Hofer 1981; Chisholm and Heath, in press). If it can be

demonstrated, for example, that the infant's breathing is stabilized by the parent's breathing sounds and rhythms (a hypothesis that I am currently testing with a team of pediatric researchers), then perhaps some of the breathing control defects that some SIDS victims exhibit before death may be stabilized or overridden simply by access to the parent's vesicular cues. Of special significance to this perspective are recent studies documenting the simultaneous emergence of the fetus's hearing and amniotic (liquid) breathing at or before seven months gestational age, although heretofore they have not been functionally or developmentally linked in this way. Henderson-Smart et al. (1983:801) did discover, however, that the brain stem center that regulates breathing lies close to the brain stem's auditory nuclei and that it is possible that both systems follow a similar prenatal and postnatal time course of structural and functional development. Similarly, the brain stem's vestibular nuclei that organize responses to rocking and movement are well developed by six months of gestation and may also serve to prepare the infant to respond by breathing more regularly to stimuli available or potentially available in the postnatal environment (Ornitz 1983). That is, the fetus begins to practice breathing against the background of prenatal uterine rhythms, including the movement created by the mother's pelvic blood flow through her abdomen. The mother's falling glucose levels thus physically induce the fetus's breathing, and an auditory pacemaker or cue becomes part of the process by which the fetus, and later the infant, learns to breathe. The Fetus's Liquid Breathing As Practice for Postnatal Breathing Although we currently know little about the development of the respiratory control mechanisms at the cellular level, the fetus's (liquid) breathing has now been documented among so many mammalian species (monkeys, rabbits, cows, and humans) that "it is considered an important stage of mammalian fetal development, including human fetal development" (Jansen and Chernick 1983:440). Indeed, Jansen and Chernick (1983:460) argue that "it is no longer appropriate to speak of the initiation of breathing at birth . . . postnatal breathing may instead be viewed as a continuation of the process begun in utero." The fetus's breathing is recognized experimentally when there is an inward movement of the fetus's chest wall associated with an outward movement of its abdomen. From several ultrasound studies of human infants, this movement is estimated to occur approximately 40 percent of the time, beginning at around 30 to 31 weeks gestation, although its frequency varies greatly among infants and may occur initially at around 21 weeks. Although the fetus's breathing is variable and different from postnatal breathing in some important ways, (as for example, in its apparently extremely low sensitivity to its mother's blood gas levels) there is ample evidence from both animals and humans that the fetus's breathing responds to changes in the mother's endocrinological and metabolic state. For example, Patrick and associates (1978a, 1978b, 1980) monitored patients for up to 24 hours at a time during the last ten weeks of pregnancy and found that the frequency of the fetus's breathing varies from hour to hour but peaks around two to three hours after meals. Patrick and associates found that the fetus's breathing also began to increase from 4 A.M. to 7 A.M. in the early morning when the mother's glucose levels were falling rapidly. In this way, a prenatal form of the fetus's circadian breathing rhythm becomes tied to its mother's rhythm (Patrick et al. 1980). As Darwish and McMillan (1984) pointed out, premature infants are therefore at a distinct "disadvantage" because, among other reasons, they are unable to practice breathing in utero to a degree that, in many cases at least, they become competent in organizing respiratory behavior. Although investigators strongly suspected that fetal breathing occurred among many mammals they were less certain as to whether the brain stem, in a way similar to its function in postnatal breathing, actively directed and controlled this breathing behavior prenatally. This question was tentatively answered by Chernick et al. (1973a, 1973b), who used stereotaxic coordinates to map out the proposed respiratory centers in "exteriorized" fetal sheep while their mothers were anesthetized. He demonstrated that stimulating the fetus's medulla oblongata (the presumed control center for postnatal breathing) could induce respiration. His work also showed that the respiratory center nerves (although different in distribution, of course, in the fetal and fully developed lamb) matured around the same time that fetal breathing began and thus should be thought of as part of the system that controls its onset (Jansen and Chernick 1983). Bystrzycka and coworkers, as Jansen and Chernick (1983) reminded us, were the first to

record directly the medullary respiratory neurons and to document more clearly the relationships between early fetal breathing and the discharge of specific neurons in the fetus's brain stem (Jansen and Chernick 1983:499). They showed that "inspiratory neuronal discharges were found in phase with fetal breathing in half of the fetuses studied" and, most importantly, that "even when the fetal subjects experienced apneas (temporary breathing cessations), tonic discharges of inspiratory neurons continued." This situation parallels what occurs during some instances of apneas among humans (Darwish and MacMillan 1984). Chernick (1983) suggests that "exteriorized fetal lambs may experience apneas, in part, due to an ineffective breathing rhythm generator" that helps coordinate and drive sequential breathing. Patrick and colleagues (1978a, 1978b, 1980) and Jansen and Chernick (1983) indicated that some kind of respiratory pacemaker may be important to coordinating fetal breathing. It is here that auditory cues, enhanced by vestibular stimuli, may play a role and help explain, for instance, why the "exteriorized" lamb fetuses that Chernick described suffered from apneas even while their respiratory neurons continued to fire. That is, once "exteriorized," the fetuses had no pacemaker by which respiratory behavior was organized. This finding is important, I believe, to understanding how infants initially develop in utero a sensitivity to external auditory cues that can affect their breathing rates later in life. The idea that fetal hearing and breathing are functionally linked depends on evidence showing not only that the human fetus actually breathes but also that the fetus's hearing abilities are advanced enough to process and distinguish sounds in the uterine environment when and if they are available. With respect to the second point, we have known for quite some time that "cochlear function is demonstrable as early as the fifth fetal month by which time both middle and inner ear structures have reached full adult size" (Eisenberg 1983; also see Ehret 1983). The cochlear region is the part of the middle ear that first receives incoming auditory signals. Although the auditory cortex structures of the higher brain (that is, the inferior colliculus and medial geniculate nuclei that are involved in more complex sound meaning and location integration) are not myelinated or as mature as the peripheral auditory structures (Parmalee and Sigman 1983), the human fetus's ear is capable of responding to sound. To illustrate, Birnholz and Benneceraf (1983) examined 236 fetuses between 16 and 32 weeks gestational age to explore fetal audition through high-resolution ultrasound imaging, in order to determine when the fetus could respond through eye blinks to a specific vibroacoustic stimulus. They found that the human fetus's auditory system is a "functionally interactive sensation" by as early as the beginning of the third trimester. The acoustic stimulator was pressed onto the mother's abdominal wall, and though it emitted an output of 110 db (an intensity approximately equal to that of a subway train), once through the abdominal wall, the estimated sound intensity was a soft 15 db (a loudness approximately equal to rustling leaves or gentle breathing sounds). The fetus's eye blink responses indicated that the sound was being processed and responded to, at an earlier age than had been assumed previously. Fetal Hearing and Uterine Sound Several other researchers have discussed similarities and differences between intrauterine and extrauterine environments and their possible importance to infants (Dreyfus-Brisac 1974; Lawson, Davin, and Turkewitz 1977). Walker and colleagues (1971) showed that in regard to sound, the mother remains the exclusive and initial source of auditory stimuli for the fetus, as her abdominal wall acts as a barrier to almost all external environmental sounds lower than that of a passing train (about 115 db) (see also Bench 1968). Walker, Grimnade, and Wood (1971) found that background noises inside the human uterus reach approximately 95 db, rising to rhythmic peaks of about 110 db about 0.03 seconds after the contraction of the mother's left ventricle. This is the amount of time required before the surge of freshly pumped blood reaches the uterus and, hence, the sound reaches the fetus's ear (Walker, Grimnade, and Wood 1971; cited and discussed by Hofer 1978, 1981; Morse and Cowan 1982). Thus, although it may be impossible for the fetus to hear a clean, repetitive maternal heartbeat, as Salk (1962) once argued, it can monitor the rhythmic peaks of blood passing through the vessels and arteries of the mother's abdomen and uterus. The idea that the fetus's hearing its mother's blood flow rhythms may facilitate its postnatal integration of auditory cues with respiratory behavior seems less speculative when considering Smith and Steinschneider's (1975) findings. They examined newborns 24 to 48 hours old and found a relationship

between prenatal maternal rhythmic stimulation and neonatal arousal. Infants born to mothers with low heart rates slept for longer periods of time, fell alseep faster, and generally cried less often than did infants born to mothers with higher heart rates. Similarly, but among rat pups, Reppert and Schwartz (1983) found that the mother's REM and NREM sleep cycles affected the circadian rhythms of her offspring. Recall that one theory contends that SIDS infants characteristically have a difficult time arousing from apnea attacks in order to begin breathing again (Harper et al. 1981). Therefore, Smith and Steinschneider's (1975) findings implicate fetal-maternal rhythmic experiences in understanding some postnatal central nervous system development. These data agree with Chisolm's (1983) data on the effect of maternal high blood pressure on infant temperament and with Schmidt and associates' (1980) study of the effect of heartbeat sounds during the active sleep of nonintervened premature infants. All of these studies and their implications recall Salk's (1961, 1962, 1974) hypotheses that suggested a relationship between the mother's heartbeat and her preference for carrying the infant on her left side, and the calming of the infant's disposition. According to Salk (1961, 1962), the mother's preference for carrying the infant on her left side, thereby giving it access to her heartbeat rhythms-a soothing and comforting sound to which the infant had "imprinted" during gestation-could reduce the infant's anxiety. Salk (1961) showed that postnatal exposure to a simulated heartbeat (that is, auditory stimulation) led to a neonatal weight gain exceeding that of nonexposed infants. Because a higher birth weight is associated with a better chance of survival (Williams, Spence, and Tideman 1977:8-9), such auditory stimulation could be said to be adaptive. The most serious objection to Salk's hypothesis is whether the uterine environment in fact permitted the infant to hear the mother's heartbeat. As we have just seen, the background blood flow noises in the maternal abdomen itself mask the sounds of a beating heart (Hofer 1981). Moreover, Brackbill (1973,1975) demonstrated that infants will calm down in response to almost all forms of repetitive stimulation and not just to simulated heartbeats. But this last point, though contradicting Salk's contention, reinforces the notion that auditory rhythms do, nevertheless, significantly affect the growing infant (see Schmidt 1975 for a review of rhythmic sound effects on infants). The possible relationship between the fetus's hearing and breathing and its postnatal respiratory behavior can be discussed in another way that more directly incorporates evolutionary considerations. Let us assume that natural selection shapes and influences the maturational or developmental rates of the fetus's sensorimotor systems to meet the demands confronting it at birth and before, irrespective of whether those demands are endogenous, exogenous, or both (Blurton-Jones 1983; Sameroff and Chandler 1975). We would expect that those sensorimotor systems (sucking, for example) most critical to the infant's or fetus's survival would develop the most quickly and, by means of neuron maturation rates, function the most efficiently. Not in any obvious way, at least, can either the fetus or the infant use its precociously developed hearing: an infant cannot, for example, alert its mother to escape from predators. Nevertheless, the fact that the human fetus and infant, at a very early point in its prenatal and postnatal development, respectively, can hear and respond to acoustic rhythms, possibly breathing rhythms, is not surprising. Human vocalizations are the most complex of those of any mammal, both in regard to the control of the respiratory muscles that enable speech (Whitehead 1983) and to the role that hearing plays in permitting the proper vocalizations to be formulated (Laufer 1980, lieberman 1984). Moreover, as I shall argue later, an infant's sensitivity to external parental breathing cues is the safest possible context for the necessary neurological transition from a strictly automatic breathing control system to one that, to a large extent, requires both voluntary-purposeful breathing and automatic breathing. The overall picture of infants' auditory sensitivity at birth suggests that they are prepared, if not yet able, to continue making the kind of respiratory responses that they made against the background of cardiovascular auditory stimuli and movements they received in utero. THE CASE FOR POSTNATAL PARENT-INFANT BREATHING RECIPROCITY To appreciate the possible importance of the external sensory stimuli to helping override breathing control errors, we should remember that perhaps for as long as three months after birth, "sensory stimulation alone in the absence of blood gas changes (i.e. oxygen/C02) regularly initiated rhythmic breathing in the human fetus" (Jansen and Chernick 1983:466). The

data on which this statement is based should force us to consider more carefully both the extent to which sensory stimuli in the infant's microenvironment influence the infant's breathing stability and the circumstances in which and the extent to which the infant's respiratory system is protected from environmental disturbances. One thing is clear. Regardless of the presence or absence of what might be considered compensatory external stimulation, the basic anatomy and physiology of the neonatal respiratory system must be at least sufficient to permit the infant to breathe on its own, or surely it will die. Interestingly, during the first few weeks of life, the full-term newborn seems to have a natural immunity to SIDS, possibly because of a gasping reflex that promotes oxygenation during periods of asphyxia. Soon afterwards, however, according to Guntheroth (1983), the infant loses this reflex, becoming more vulnerable to breathing control errors (apneas, periodic breathing) and, in fewer cases, to complete respiratory collapse (that is, failure of the respiratory drive). Galef s (1981) comparative evolutionary perspective on mammalian parentinfant relationships predicts this period of vulnerability, though not necessarily with respect to respiration. Unflattering as it may be, he compares mammalian infants with parasites, who lose some structural and functional integrity once attached to a host. That is, once the human infant begins to engage, or, more appropriately, reengage, with its mother-becoming increasingly dependent on the nature and timing of her care (Field 1985) while simultaneously losing reflexive behaviors to higher-brain (labile) behaviors as its development proceeds-it also loses some of its structural and physiological integrity. It thus becomes vulnerable to neurological control system errors, especially during these early, critical transitions or developmental shifts. This is the same viewpoint espoused by Lipsett (1981, 1982) around which the arguments made in this paper revolve. It is also clear that the infant's form and neurological bases of breathing change dramatically during its first year of life. For example, the newborn breathes extremely rapidly, completing about 87 breaths per minute, which by the end of its first year will drop to about 47 breaths per minute. In contrast, during vegetative (maintenance) breathing, the average adult breathes between 16 and 20 breaths per minute (Langlois, Balken, and Wilder 1980; Bunn and Mead 1971). In the beginning, the infant's breathing is shallow and depends mostly on diaphragmic movements rather than rib muscle (intercostal) movement. Respiratory behavior is primarily reflexive and based not on the infant's voluntary contribution, but on chemical receptors and respiratory neuronal centers in its brain stem's reticular formation and possibly on nuclei in the spinal cord itself (Mitchell and Berger 1981). At the age when the infant is at the highest risk for SIDS, the control of its respiratory behavior is shifting to include voluntary or volitional and presumably cortex-based breathing as, for example, noncrying, purposeful vocalizations, and controlled crying behavior, as I shall discuss in more detail later. According to Fleming's (1984) physiological studies, this period of shifting neurological control coincides with the less overall stability of those structures that integrate the chemical signals related to proper carbon dioxide and oxygen levels. It is at this time, it is hypothesized, that external sensory cues may prove to be the most useful to some infants. The relationship between auditory stimuli and respiration has not been explored to the extent that, for example, auditory cues and heart rate have (Schmidt 1975; Brackbill 1975) or that the role of tactile and vestibular stimulation has (Barnard 1981). There is, however, abundant clinical and experimental evidence indicating that very young infants can detect and then respond to rapidly changing sounds, although there is no direct evidence, of which I am aware, that human breathing sound per se can effectively drive infant breathing. (This is a question currently being studied by a team of researchers, including myself, by monitoring the breathing of mothers and infants as they breathe and sleep in the same bed.) But the evidence does suggest that two- to five-month-old infants can detect and respond to sounds within the decibel range of human breathing and, indeed, to a variety of repetitive and rhythmic sounds in general (Brackbill 1973; Salk 1962; Schmidt et al. 1980; Laufer 1980; Morse and Cowan 1982). Eisenberg's studies (1976, 1983) demonstrate that infants respond especially to speechlike sounds, such as the synthetic vowel sound ah at 60 dbs, and that this sound is processed at what can be considered high levels of the auditory system. In other studies, Brackbill (1973,1975) found that intermittent auditory stimulation tended to raise infants' arousal levels. More pertinent to the question raised here is Bradford's (1975) finding that normal

infants display an acute sensitivity to and respond by breathing to decibel levels ranging between about 15 to 30 dbs, the decibel range of human vesicular breathing sounds. These infants most consistently responded to repetitive mechanical clicks at an average of 15 dbs. Relative to other mammals, it is clear that human infants, in addition to displaying auditory precocity, have also evolved an ability to detect subtle changes in the intensity of sound frequencies or pitch (Fobes and King 1982; Eisenberg 1983). Fobes and King (1982) found that when compared with other mammals and other primates, a human primate's "best frequency"-a designation referring to the sound level that humans can hear most easily and respond to most quicklyis a low-frequency level. This makes sense when one considers that language is our primary communicative system, on which our survival, and thus our fitness, has depended for a considerable period of time. This kind of communication system demands attention to soft, rapidly changing tones and pitch, as well as to vocal, respiratory, and social cues related to turn taking during interactions (Laufer 1980; Brazeleton et al. 1974; Field 1985). It is obvious that in the environment in which infants evolved, auditory cues did not occur in isolation away from tactile, vestibular, thermal, and gaseous sensory signals. All of these signals, especially the tactile, gaseous, and vestibular ones, directly enhance auditory cues. Recall that the physical proximity and mother-infant contact or contact with sibling care givers (Tronick et al. 1985) evolved concomitantly with delayed infantile maturity. Infants must be carried for at least the first year and must rely on continuous rather than periodic nursing, owing to the relatively low fat and protein content of human milk (Blurton-Jones 1972). This means that even if the continuouscontact-and-carrying model that has been criticized by Tronick et al. (1985, in press) is not the only model possible for human infant care throughout evolution, the infant will still be in close contact with a care giver's breathing sounds and also with a variety of important alternating rhythmic and arrhythmic stimulation, not the least of which is the infant's feeling the locomotor movement of its care giver. Of course, when sleeping, the infant will feel the rhythmic chest movement of its parent's respiratory behavior as well as disruptive sleep movements and activities. Rocking and Infant Breathing The effects of rhythmic rocking and movement (vestibular stimulation) on the human infant undoubtedly have been recognized since prehistoric times, but their precise physiological and social correlates, especially with respect to how they promote breathing, have been delineated only during the last 20 years or so (Baker and McGinty 1977; McGinty 1983; Barnard 1981; Chisholm 1983; Ornitz 1983; Korner and Thomen 1972). Gregg, Huffner, and Korner (1976) suggested that for human infants, rocking stimulation may prove to be more helpful than tactile stimulation is in soothing and alerting them, because of its effect on their reticular activating system. According to studies by McGinty (1984), McGinty and Hoppenbrouwers (1983), and Donnelly (1984), it is clear that vestibular stimulation directly augments reticular formation neuronal firing "in parallel" with respiratory behavior in many mammalian species including human (McGinty and Hoppenbrouwers 1983). McGinty (1984) reported that kittens' rapid eye movement (REM) sleep stage was altered and increased in duration when they were rocked. In another study, McGinty and Hoopenbrouwers (1983) describe kittens whose sleep tended to be synchronized by a rocking stimulus and whose respiration increased when the rocking frequency reached one-half the baseline frequency of the kittens' quiet sleep respiration rate. In other words, the kittens' breathing was more strongly influenced by an external pacemaker when the rocking movement was about the breathing rate of an adult cat, an adult who breathes much more slowly than a kitten does and who is likely to be in contact with it. It has been demonstrated in humans that the kind of vestibular movement provided by the parent's chest movement (particularly when the parent and infant sleep together) also can affect the infant's breathing patterns. For example, Korner and colleagues (1975; Korner 1981) described the positive results of placing apnea-prone premature infants on oscillating water beds that when set at a movement of between 12 and 14 oscillations per minute-the approximate breathing rate of an adult-the rates of apnea per sleep hour in seven of the eight infants studied were reduced by 13 to 48 percent. Most exciting is Thomen's recent preliminary success in stabilizing the breathing patterns of some high-risk infants by placing in their cribs mechanically breathing teddy bears that provide a constant source of the rhythmic vestibular stimulus that most closely resembles the parent's chest

movement (Turkington 1984). This discovery that external rhythmic cues, in this case movement, influence infant's breathing patterns implies that there is continuity in the ways that mothers' biorhythms physiologically regulate their offspring both prenatally and postnatally, a point of view confirmed by recent research on parents and infants (Reite and Field 1985). For example, in regard to the general difficulties that premature infants experience, Korner (1981) suggests that a defective zeitgeber, or rhythm giver, may well contribute to the disorganization of the premature infant's behavior. This perspective is supported by a great deal of research conducted and discussed by Dreyfus-Brisac (1974), Barnard (1981), Hofer (1981), and reviewed by Jansen and Chernick (1983) and Field (1985). The fact that vestibular stimulation helps in the development of some fetal and infant systems is not surprising, because, like the auditory and respiratory nuclei, the vestibular nuclei located on the brain stem develop structurally and functionally very early prenatally. For example, the large neurons of the vestibular nuclei are functional at 21 weeks gestation, and by six months of age, according to Ornitz (1983), the vestibular system is well advanced. That both prenatal and postnatal vestibular stimuli may help develop the infant's motor skills has been documented. Masi (1979) found that the vestibular stimulation of preterm infants improved their sensorimotor functioning relative to those of unstimulated controls. Chisholm and Heath (in press) explain this finding by reminding us that during at least three to four million years of hominid evolution, human fetuses developed in the context of the vestibular stimulation received as their mothers foraged. Thus, Chisholm and Heath hypothesized, the fetus is preadapted for high levels of postnatal maternal locomotion, as humans are a "carrying," as compared with a "cache," species, that is, infants are not kept in nests when their mothers or fathers forage for food but are transported. Most interesting is that the human infant's vestibular system is functioning at its "highest level of reactivity" after the first few months of life (Ornitz 1983:527), the very time period that infants are at greatest risk of dying from SIDS. In other words, at the time that their respiratory control system is the most vulnerable, infants may be most sensitive to respiratory augmentation through external vestibular and auditory stimuli-cues regularly available in the infant's expected microenvironment if parents and infants sleep together throughout the night. THE PHYSIOLOGICAL EFFECTS OF TOUCHING Touching also stimulates breathing, and of course, it is through the skin that infants "feel" and sense movement, particularly if their heads are stationary. Although I shall not review here the extensive literature on the effects of tactile stimulation (Smerglio 1981, 1983; Suomi 1982; Montagu 1978), suffice it to say that in the short run touching has significant physiological effects (Schwartz and Rosenblum 1983; Hofer 1983), and in the long run it may be crucial to healthy socioemotional development such as reducing the likelihood of aggressive behavior (Prescott 1972a, 1972b). Specifically with respect to breathing, Kattwinkel and associates (1977) demonstrated that rubbing an infant's feet for about five minutes at 15 to 30 minute intervals reduced both the duration and frequency of apneas. And most infants who experience apneas resume breathing when they are touched or are touched and picked up, although some infants require more acute therapies (Southhall et al. 1985). Although the longitudinal deprivation studies of nonhuman primates conducted at the University of Wisconsin, beginning with Harlow's classic surrogate (wire versus terrycloth) mother studies, clearly documented infants' need to cling to a warm and soft object-to feel "contact comfort"-these studies concentrated on the long-term psychosocial effects of loss and not their short-term physiological effects (see McKenna 1979 for a review). The relative effects of, for example, tactile deprivation versus auditory versus vestibular specific sensory stimulation loss were not specifically monitored. During the last 15 years or so, however, studies of the physiological correlates of loss, including the deprivation of tactile contact with other animals (particularly mothers), have revealed dramatic and important findings. Studies being conducted in the labs of Hofer, Reite, and Levine (Reite and Field 1985), most especially, have shown that in order to understand the form and consequences of separation (primarily mother from infant) for the infant, we must know how the infant's body changes physiologically after separation; thus, we must know how through contact the mother physiologically regulates her infant's temperature, metabolic rate, hormone levels, enzyme production, antibody titer, sleep cycle, heart rate, and respiration so as to promote her infant's health and survival. (Table 2) Together, these

data disclose the overall impact of immediate separation and remind us that when human (urban) infants are regularly separated, for example, for nocturnal sleep (which can be regarded as an evolutionarily novel situation), there is no reason to think that their physiological systems are not also affected. For example, Fardig (1980) found that for 17 mothers and babies, radiant-heated cribs could not maintain the mean skin and core temperature of human newborns placed on their mothers' bare chests for skin-to-skin contact, even when the ambient temperatures were equivalent. The difference, she suggested, may be the production of stress hormones such as the Cortisol produced by the infant when separated, which causes a drop in its body temperature. Studies using telemetry on a variety of macaque monkey species (Bonnets, Pigtail, and Rhesus) indicate that, when separated from their mothers, primates as old as four to six months of life also lose body temperature and can experience disturbances in sleep, with decreased REM sleep periods (Reite and Short 1978), changes in EEG activity (Short, Iwata, and Reite 1977), alterations in cellular immune responses (Reite, Harbeck, and Hoffman 1981), and increases in cardiac arrhythmias (Seiler et al. 1979). It has been demonstrated in squirrel monkeys that separation increases their adrenal (stress) secretions and plasma Cortisol levels (Coe and Levine 1981) and decreases their ability to combat pathogens because of decreases in immunoglobulins (after seven days). Lower levels of antibodies against certain bacteria strains have also been found (Coe et al. 1985). When two-week-old rat pups were separated from their mothers, they immediately experienced a drop in growth hormone levels, concommitant with a corresponding drop in a brain enzyme (ornithine decarboxylose, ODC) needed for the synthesis of several brain proteins (Butler, Suskind, and Schamberg 1978; Kuhn et al. 1978). Apparently, a drop in these two substances was not related to the nutritional or body temperature changes associated with separation, as they had been taken into account. Much to the researchers' surprise, the drop in growth hormones seemed to be related to some unknown aspect of the pup's interaction with its mother; her presence seemed to be important to the release and/or production of the growth hormone (see Hofer 1981 for a further discussion). Reite and colleagues (1985) think that this finding may help explain the "failure to thrive syndrome" defined by Powell, Brasil, and Blizzard (1967) in regard to human infants deprived of physical affection. These infants gradually lose weight, and many of them die, even though they are fed a diet sufficient to sustain life. Much life the collective physiological data on the effects of parentinfant separation among primates, this finding implies that food intake alone is not enough to guarantee normal weight gain and survival in the absence of physical (and, one presumes, vestibular and auditory) stimulation. For other mammal infants, but especially primate infants, shortterm separations lead to physiological consequences. This conclusion forces us to consider the possible effects of nocturnal separation on human infants who, in Western and urban societies, regularly sleep apart from their parents in separate rooms. It is ironic that the human primate infant, which is born the most immature neurologically, develops the most slowly, and is thus more, rather than less, dependent physiologically on its care giver, is the only primate that, in urban settings, is expected to sleep alone and at a much younger age. Monkey and ape parents would no doubt regard this as a form of child abuse, as do peoples living in preindustrial societies when told of parent-infant sleeping arrangements in urban settings (Konner and Super, in press). The question here is whether it is easier for an infant's respiratory, immunological, or some general central nervous system defects to appear in microenvironments from which species-specific auditory, tactile, and vestibular cues are missing. The question is not whether solitary infant sleeping arrangements cause SIDS, because surely they do not. Rather, it is whether, as Lipsett phrased it, the microenvironments of some small class of potential SIDS victims lack such stimuli and so conspire with neonatal or infantile defects, thus creating favorable conditions in which SIDS can occur. For some apnea-prone infants, such as those with excessively compliant rib cages that collapse, as Southall and associates (1985) discovered, parent-infant cosleeping and all the available "compensatory" stimulation probably cannot help. But many infants appear to have more "subtle" defects (see Haddad and Mellins 1983) that may be overcome in microenvironments that more closely approximate the conditions in which the human infant's physiological and developmental systems evolved and that, including cosleeping,

more closely approximate the prenatal sensory experiences discussed earlier. Parent-Infant Thermal and Gas Exchange Galef (1981) reminds us that a parent-infant huddle reduces the participants' surface-to-volume ratio and thus conserves energy. Surely this is relevant to Fardig's (1980) study of infants in radiantheated cribs and infants in their mother's arms. The exchange of heat between and its conservation by parents and infants while sleeping together or in a ventral-ventral embrace are other examples of a process and a set of stimuli denied to infants encouraged to sleep alone for as long a period as convenient for the parents and at as early a developmental age as possible. Such deprivation is, once again, perhaps not serious for the "healthy" infant, but it is well known that ventilation depends on environmental (ambient) and body temperature (Lahiri and Dalaney 1975); in fact, temperature is also one of the most important external modulators of sleep, especially REM sleep (McGinty 1984). Excessive cooling of an infant's body, even by a few degrees, will depress its expiratory neuron firing and diminish its respiratory effect. Thus, the protection that a cosleeping parent's body warmth offers is perhaps not to be considered insignificant, although there are surely other ways to compensate for the parent's contribution to maintaining the infant's resting temperature. When a parent sleeps with or near an infant, it is likely, at least for much of the night, that not only will their body heat be exchanged but also will their expelled carbon dioxide gases. Recently, Sullivan (1984) suggested that during REM sleep, infants can smell carbon dioxide, in part because of the presence of carbon dioxide chemoreceptors in the infant's nasal mucosa (see also Widdicombe 1981). If this is so, then in the microenvironment created by its parent, the infant could respond to the parent's exhaled carbon dioxide by breathing in some of it. That is, the infant's upper nasal chemoreceptors may receive enough of its parent's CO2 to increase the chance of a brain stem-directed inspiration. Moreover, Guz (1977) discussed recent evidence that C02-sensitive receptors lie in mammals' respiratory tract, possibly the lungs, which generates reflexive breathing (cited by Tenney and Bartlett 1981). The addition of CO2 to air inspired by dogs increased their respiration frequency because of a decrease in their expiration duration. If these findings are generalized to humans, proximity to the parent in this way should increase the likelihood of the infant's taking the next breath, should its other internal respiratory signals falter. Obviously, this gaseous exchange occurs simultaneously with touch, movement, heat exchange, scents, and auditory stimuli. This is only one of many factors in the infant's evolutionarily expected environment that, if operating alone, undoubtedly is insufficient to promote continuous rhythmic breathing; but in concert with other stimuli it may help to reduce the likelihood of some forms of respiratory failure, at least, the infant's evolutionary history suggests that it should. PARENT-INFANT COSLEEPING: THE RELEVANCE OF IDEOLOGY TO FORMULATING SIDS RESEARCH QUESTIONS Parent-infant cosleeping is the evolutionarily old arrangement, the "environment of adaptedness," to use Bowlby's (1969) phrase, during which the environmental respiratory cues delineated here are most likely to be found. Support for this statement is drawn from studies of parent-infant sleeping patterns among nonhuman primates (Altmann 1980; Anderson 1984), cross-cultural data and ethnographic materials (Konner 1981; Super and Harkness, in press; Munroe, Munroe, and Whiting 1980), and various models of hominid evolution that emerge from both archaeological and paleontological data (Lancaster and Lancaster 1982; Isaac 1978; Tanner 1981). But it is not my intent to use these diverse lines of inquiry to argue that all urban infants should, therefore, sleep in the same bed with their parents or to suggest that if they did, SIDS would be eliminated, because clearly the epidemiological data show that it would not. Because there have been many misconceptions and much misinformation regarding parent-infant cosleeping that has affected SIDS research assumptions and strategies, this issue needs to be addressed. In fact, the widespread belief in urban Western societies that parent-infant cosleeping is dangerous and unhealthy for all concerned (Thenevin 1975) may explain why there is not one sleep study with which I am familiar, or could locate, that examines sleep-wake cycles, REM and NREM sleep-stage durations, and the other physiological processes that occur when an infant sleeps with a parent. This belief may also be the reason that SIDS researchers have never seriously considered whether parent-infant nocturnal separation is in any way related to some SIDS pathogeneses. Except for a few studies (Sander et al. 1970; Korner et al. 1974; Anders

1978; Anders et al. 1983; Emde et al. 1971), the clinical picture of the normative development of infant sleep behavior is derived from studies of infants sleeping alone in sleep laboratories. These data, in addition to the experiences of middle-class American parents who do not sleep with their infants, have given rise to a conceptualization of infant sleep that may be at odds with what infants have actually experienced throughout prehistoric and most of historic time (Konner and Super, in press) and with what might be a more species-specific infant sleep profile (Raphael 1976; Anders 1979; Carey 1975). This may be relevant for understanding the relationship between parentinfant contact and infant arousal patterns-needed arousal for infants suffering from some reticular formation defect. For example, ethnographic data from preindustrial societies show that the duration of infants' nocturnal sleep-wake patterns are altered significantly when the infants sleep next to, and have access to, their mother's nipples throughout the night (Konner 1981; Konner and Worthman 1980). Super and Harkness (as discussed by Konner and Super, in press) monitored ten Kipsigis infants (living in the Kenyan highlands) during 24-hour sleep periods 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 parents' 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's (1981) and Konner and Worthman's (1980) student of Sun Kung Bushmen infants support these findings. "Overlying," or the accidental suffocation by a parent rolling over on the infant, is most frequently cited by pediatricians as a reason not to have infants in the same bed (in addition to other reasons, such as spoiling, and trauma at seeing the parents have sex; see Spock 1976), and undoubtedly this fear contributes to the idea that parent-infant cosleeping is not a legitimate issue to explore. Overlying is, of course, possible, but it is not likely, just as strangulation by defective cribs, while known to have happened, also is not common (Smialek et al. 1977). As Konner and Super pointed out, soft mattresses, as opposed to hard mats, may change the overall safety picture of parent-infant cosleeping. Nevertheless, even day-old infants will struggle violently and protest vocally in response to obstruction of their air passages (Woolley 1945). In addition, parents are exceedingly alert to an infant's sounds of distress, providing that chemical substances such as alcohol and drugs do not limit this sensitivity, and so smothering is only a remote possibility (Konner and Super, in press). Furthermore, the possibility of overlying does not diminish the benefits for the infant of sleeping with a parent. Indeed, if overlying were a serious problem, our species would probably not be here today to study it. Are SIDS Rates Lower in Countries Where Cosleeping Is Practiced? An easy way to test the major hypotheses argued here would be to determine whether SIDS rates were lower in societies in which cosleeping is practiced. But SIDS is so rare, its diagnosis is so difficult amidst the many perinatal and neonatal causes of infant death in the preindustrial societies that practice cosleeping, and the duration of ethnographic studies is so short, that this is impossible. Barry and Paxson's (1971) survey of 90 societies, of which cosleeping is or may be practiced in 71, would be ideal, but for obvious reasons, data on SIDS in these societies are not available. Comparisons are better for urban societies that have standardized their reporting and diagnosis of SIDS, but even these can be difficult. For example, SIDS rates are lower in Sweden, the Netherlands, Switzerland, and Israel, where in some segments of society, infants sleep with their parents or in the same room. Moreover, the infant mortality rates in general (rather than just for SIDS) are extremely low in Japan, an urban country in which cosleeping occurs (Caudill and Plath 196) but I could not secure figures for infant deaths from SIDS, though I would hypothesize that SIDS rates are significantly lower there. The most important recent finding that supports the predictions considered in this paper are based on a five-year survey of cot deaths in Hong Kong, a culture in which cosleeping is likely, in part because of the overcrowded conditions. So rare is sudden infant death there (0.036 per 1,000 live births, or approximately 50 to 70 times less common than it is in Western societies), even though nursing is not common (out of 175 infants at two, four, and six months, the percentage of breast-feeding was 9 percent, 4 percent, and 2 percent, respectively, cited by Davies 1985:1347) that the investigator wonders "whether the possible influences of life style and caretaking practices on cot death are being underestimated in

preference for more exotic and esoteric explanations" (Davies 1985:1348). Davies' question follows his consideration of the recent findings by SIDS researchers, and although he does not conclude that sleep patterns alone are responsible, he uses these data to ask new questions about the importance of infant microenvironments. Davies also reasons that putting infants down to sleep supine, rather than prone, may help prevent their suffocating, an idea refuted by Orr and associates (1985). In this case, crowded living conditions and prenatal care (the mothers are older when their first child is born, and they do not smoke during their pregnancy) are also suggested as reasons for the rarity of SIDS in Hong Kong. Astonishingly little is known, but much is presumed, about American infants' nighttime sleeping arrangements. It is fair to guess that most American infants sleep alone. If, or how often, they are brought into their parents' bed is unknown, and because parents apparently feel guilt when or if they do bring their infants into their beds, it is difficult to secure reliable data. One recent investigation comparing the parent-infant cosleeping patterns of urban American blacks and whites is instructive. It is one of the few studies on this subject and it illustrates some of the potential difficulties associated with testing various kinds of SIDS theories that, even if correct, may not for other reasons be supported by the data. For example, Lozoff and colleagues (1984) found that blacks more often than whites sleep with their infants. Out of 150 "well child" appointments at pediatric facilities, 35 percent of the white and 70 percent of the black families "routinely" slept with their children six months to four years of age (Lozoff et al. 1984). Although this sample includes infants past the most vulnerable or highest-risk period for SIDS, by extension it could be inferred that because black mothers sleep in the same bed with their infants more frequently than do whites, SIDS should be reduced in this subgroup of Americans, but the opposite is true. As already observed, black infants are at a higher risk for SIDS. But although cosleeping may protect some infants from respiratory difficulties, more important are the facts that more blacks than whites smoke during their pregnancies and have their infants at a younger age, thus offsetting the possible benefits of cosleeping. Even if it can be proved that parents can somehow drive the breathing of their infants, the epidemiological and experimental data on SIDS indicate that only certain kinds of infants would be protected. Perhaps if all the known perinatal risk factors were recognized and infants slept in close proximity to a parent for the first year of life, they would be at the least risk for SIDS. SUMMARY AND IMPLICATIONS FOR RESEARCH This paper presents several testable hypotheses on the possible relationships between the development of hearing and breathing, both prenatal and postnatal, and considers what these hypotheses imply for SIDS research. My purpose is to call attention to the need for SIDS researchers and anthropologists who I hope will become interested in this area, to examine the contexts in which human infantile defects may "conspire," as Lipsett phrased it, with sensory experiences, or the lack thereof, to make their expression easier or more difficult. I have used the important studies on the immediate physiological effect of parent-infant separation among nonhuman primates, cross-cultural and evolutionary perspectives, to argue that nocturnal parent-infant separation among humans represents a behavior pattern that is evolutionarily novel to our species. Although this is obviously not critical to most infants, it may be critical to infants with central nervous system impairments. If this is true (and only new research strategies conducted on living human and nonhuman primates can find out), I propose that there may be a connection between parent-infant sleeping arrangements and some SIDS pathogeneses. I am not the first to suggest such a relationship between SIDS and the solitary sleeping arrangements of urban infants (Maxwell and Maxwell 1979; Sears 1984; Konner and Super, in press). Except for Konner and Super, who present an anthropological perspective on SIDS complementary to my own but with a different emphasis, this is the first paper to bring together relevant and recent research findings on both prenatal and postnatal developmental processes to link hearing and breathing systems with anthropological, SIDS, and neurological studies in order to formulate testable hypotheses. The human infant is conceptualized here as an organism whose social and physiological needs are inseparable, having coevolved over three to four million years in a caregiving microenvironment which, among other things, includes prolonged periods of parent-infant contact, one form of which is cosleeping. Another guiding theory here, around which the interpretation of

clinical and experimental data on SIDS revolves, is that natural selection operates on the fetus (in utero), preparing and sensitizing it to the evolutionarily-old and stable environment into which the fetus expects to be born. From this perspective, I argue that experimental findings point to a prenatal developmental link between hearing and breathing that has implications for postnatal infantile respiratory behavior. Specifically, I suggest that it is not simply coincidence that fetal liquid breathing (including neuronal firing) and the fetal middle ear develop simultaneously and that both of these systems emerge against, and are affected by, cardiovascular auditory rhythms in the maternal abdomen. It is hypothesized that this prenatal experience prepares the fetus to respond to repetitive or rhythmic auditory breathing cures and movement (of the chest) given by the parent, a question presently being tested by the author. Moreover, a variety of work on the effects of vestibular and tactile stimulation on infants and recent findings about CO2 chemoreceptors in the infant's nose and lungs suggest even more strongly that by sleeping in proximity or contact with a parent, less physiologically versatile infants can benefit; it may be that parental breathing behavior can offer an external compensatory support to certain infants, helping them to override some, but surely not all, defects involved in respiratory collapse. Anthropologists need to become more familiar with laboratory and clinical SIDS research while medical researchers can enrich their approach to this problem by incorporating valuable insights that the evolutionary and cross-cultural perspective so familiar to anthropologists and psychologists can provide. This paper purports to link these two research perspectives to move us toward a complementary and one would hope, a more comprehensive assault on this tragic infant malady. AuthorAffiliation James Joseph McKenna, Ph.D. AuthorAffiliation James Joseph McKenna is an Associate Professor of Anthropology at Pomona College, Claremont, California, and an Adjunct Clinical Assistant Professor in the Departments of Pediatrics, Child Psychiatry and Human Behavior at the University of California School of Medicine, Irvine, California. This article is reprinted in modified form with permission from Medical Anthropology: Cross-Cultural Studies in Disease and Illness, 1986, Vol. 10, No. 1. The original paper has been shortened here and divided into two parts, the second of which (PART II) will be published in Vol. 2, No. 3 of this journal, together with a complete Bibliography.

Publication title: Pre- and Peri-natal Psychology Journal

Volume: 2

Issue: 2

Pages: 93-135

Number of pages: 43

Publication year: 1987

Publication date: Winter 1987

Year: 1987

Publisher: Association for Pre&Perinatal Psychology and Health

Place of publication: New York

Country of publication: United States

Journal subject: Medical Sciences--Obstetrics And Gynecology, Psychology, Birth Control

ISSN: 08833095

Source type: Scholarly Journals

Language of publication: English

Document type: General Information