An Anthropological Perspective on the Sudden Infant Death Syndrome: The Neurological and Structural Bases of Speech Breathing and Why SIDS Appears to Be a Species-Specific Malady, Part II

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Full Text: Headnote ABSTRACT: This paper extends the evolutionary-based arguments proposed in a previous paper (see McKenna 1987, Part I) but concentrates on why the sudden infant death syndrome is not found among other animal species, and cannot be experimentally replicated, and thus why it appears to be a species-specific, unique human infant malady. Data from studies of the breathing and vocalizing difficulties of normal-hearing and hearing-impaired persons are used to argue that, to a degree unparalleled in other species, certain breathing behaviors are cortically controlled and learned by way of both internal and external auditory processes. While the neurophysiological and neurostructural voluntary-involuntary nerve conduit upon which this system depends is a prerequisite for human language, or more specifically, for speech breathing, it makes human infants more vulnerable to any number of breathing control errors, one of which may be SIDS. Hence, this perspective attempts to explain why SIDS is so circumscribed by age since speech breathing begins to occur as the infant begins to assert increasingly voluntary control over its crying behavior at two to four months when SIDS rates peak. By seven months, well before the infant utters its first words, it has mastered the voluntary pulmonic manipulations required for speech. While making no suggestions about the primary causes of SIDS, this paper like the previous one provides a heuristic model and rationale for new data-based research questions on a malady that has resisted our attempts to understand it for over 20 years. Finally, for some but not all infants, it is suggested that the evolutionary old microenvironment of sustained and continuous parent-infant contact, including cosleeping, provides the safest context within which such a system could evolve. Many investigators studying the sudden infant death syndrome have argued rightfully that the single most intriguing clue to understanding its causes is the syndrome's unique age distribution (see Froggatt, 1983; Beckwith, 1973; Schwartz, as cited in Arnon, 1983). In fact, 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 according to 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, Petersen (1983) found that SIDS deaths occurred at an age of between 7 and 270 days, with a median of 90 days. ValdesDapena's summary of all SIDS death rates, published between 1975 and 1979, reveals that 90% of sudden infant deaths occur before six months of age and that 99% of them occur before the infant is one year old: it is exceedingly rare for children over one year of age to die from sudden infant death syndrome. The highly circumscribed age distribution of SIDS victims is, perhaps, no more intriguing than is the fact that even after more than twenty years of SIDS research no animal model of the syndrome has yet to be produced. This suggests that certain environmental triggering factors in combination with unique human species-specific sets of characteristics may coalesce in ways that are experimentally irreplicable in other animal forms. Guided by knowledge of the restricted age distribution of SIDS victims, in this paper I should like to consider whether or not there are any differences between ourselves and other species, especially with respect to the development of breathing control mechanisms that make human infants more vulnerable to breathing control errors at specific

developmental periods. One set of unique emerging abilities that fits the SIDS developmental age profile and that for the most part has not been included in any discussions of developmental milestones relevant to the SIDS pathogenesis is the process by which the brain's voluntary and involuntary breathing and vocalizing control centers become functionally interdependent during sleep and awake periods. I argue here that our species' unique neurophysiological, in addition to biomechanical, adaptations for language (see Laitman, 1985; Crelin cited in Dorfman, 1985) increase the chance of respiratory errors in certain infants; moreover these adaptations, I will suggest, become apparent around two months of age when the human infant begins to assert increasingly efficient voluntary control over its respiratory behavior, especially in crying and noncrying vocalizations. Such adaptations that are based on links established prenatally between hearing and breathing (see McKenna 1988, Part I) may provide clues to the structural dimensions of some forms of SIDS. SIDS IN A DEVELOPMENTAL CONTEXT Changes in breathing control and breathing during vocalizations must first be viewed in a broad developmental context. And it must be pointed out that because infants are known to pass a number of developmental milestones, SIDS researchers have conjectured that one or several of these developments may be implicated in SIDS pathophysiology. For example, consider that between two and five months of age, when SIDS rates peak, infants experience accelerated neuronal arborealization and myelenization (that is, their neurons acquire a medullary fatty sheath that speeds up interneuronal communication) (McGraw, 1943; Parmalee &Sigman, 1983). It is not simply the brain's accelerated growth, either, that is important but there is also a functional shift as well. For example, higher brain or cortical structures begin, for the most part, to mediate and predominate over midbrain or hindbrain centers, thus replacing more primitive and less flexible behaviors with more complex and labile ones (Lipsett, 1981, 1982; Morse &Cowan, 1982; Purpura, 1974; Dobbing, 1974). The evidence that much infant learning occurs during this first critical developmental shift can be found in the infant's "increasingly deliberate control of psychomotor activities" and "self-delivery of externoreceptive stimulation coinciding with Piaget's second circular reaction" (Lipsett, 1982:64). The infant's cognitive abilities are clearly emerging at this time. Super (1972) found that at ten weeks of age, infants are capable of recognizing an event twenty-four hours after its occurrence. According to Kagan (1979), this is but one of several kinds of evidence demonstrating that both learning and psychological functioning are assuming a predominant place in the infant's life. For example, between two and three months of age, infants can probably recognize "transformations of congenial experiences," and we know that "structural alterations follow the emergence of the new competence" (Kagan, 1979:14-15). Fleming's (1984) experimental studies of infant respiratory patterns show that the time SIDS rates peak, the infant's respiratory system is becoming more sensitive to internal blood gas levels but, simultaneously, is becoming less stable, relative to its stability in the first few weeks of life when infants seen to be immune from SIDS. Fleming's (1984) data also show that soon after the most critical time period for SIDS, the infant's respiratory system becomes both stable and sensitive to internal state changes. These data indicate that there is a developmental lag in various aspects of respiratory physiological responsivity, integration, and general efficiency. Moreover, not until approximately six months of life is the infant's respiratory system functioning at its most stable, responsive, and efficient level. Insofar as this is true, the idea that external compensatory stimuli or external cues are useful to the infant is strengthened, as I shall argue, especially if we accept Galef's (1981) notion that mammalian infants may lose some physiological integrity or autonomicity once they reengage or attach physiologically to a care giver (McKenna, 1988). The infant's sleep patterns also change quite dramatically during this high-risk period. For example, the infant begins to exhibit a more adult sleep pattern, characterized by as much as 70 to 75% of quiet, or NREM, sleep. This contrasts with the newborn period in which REM or active sleep predominates, reaching as much as 80% of the newborn's total sleep time, compared with 20% or so of quiet, NREM sleep time (Parmalee et al. 1964; Parmalee &Stern, 1972; Emde &Walker 1976). There is much disagreement over which (if either) sleep stage makes infants most vulnerable to SIDS. My reading of the material leads me to conclude that this may be the wrong question to be asking, as sleepstage transitions or indeterminate sleep

seem to place breathing control in more jeopardy (Remmers, 1981). It is also significant that SIDS rates peak at a time when maternal antibodies (IgG), abundant in the first two months of life, are declining, "generally reaching the lowest level at three months of age before the infant builds up its own immunoglobulin to achieve immunological independence" (Huang, 1983:593; also see Arnon, 1983). Of course, nursing can continue to protect the infant from a host of environmental assaults after this period, because it is through contact with its mother's nipples that the infant has almost a direct line to her enteroimmune system. However, as Arnon (1983) notes, mothers differ biologically in the quantity and types of their antibodies. Thus, the emergence of the infant's own functioning immunological system is a relevant developmental milestone when considering SIDS etiologies, especially because SIDS must still be regarded as having different kinds of "causes" and thus the corresponding physiological mechanisms, we can presume, to combat them. THE NEUROLOGICAL BASES OF SPEECH BREATHING With this broader developmental perspective in mind, it is appropriate to consider adaptations unique to humans that clearly place human infants in an especially vulnerable condition-at least insofar as respiratory behavior is concerned. Physiological studies have shown, for example, that the respiratory control of crying and noncrying vocalizations parallels the specific pulmonic manipulations required of humans when they speak (Wilder, 1972) and hence is a practice for speech. The infant completely masters the form of breathing necessary for these vocalizations by seven months of age, well before it utters its first words. However, it is at around two months of age when infants are increasingly at risk for SIDS that these abilities begin to be exhibited. From a biomechanical and neurophysiological vantage point, the phenomenon of speech breathing refers to a series of voluntary, presumably cortex-based manipulations of the respiratory and vocal musculature controlling airflow rates that make speech possible. For example, as compared with vegetative or maintenance breathing, speech breathing essentially limits inspiratory interruptions so as to ensure the maximum use of air for phonation or vocalization as air is expired (Langlois, Balken, &Wilder, 1980). Unlike vegetative or maintenance breathing, speech breathing requires fewer breaths per minute (from an average of 18 breaths per minute to about 10 to 14 per minute in adults) and a larger residual volume, or air remaining in the lungs after expiration or vocalization (Whitehead, 1983; Forner &Hixon, 1977). Speech breathing requires the lungs to increase their air pressure (pulmonic pressure), and even while exhaling, the glottis must hold back some air in order to maintain adequate subglottal pressure to manipulate the vocal cords (Wilder, 1972; Langlois, Balken, &Laufer, 1980). These behaviors also permit manipulations of the oropharyngeal muscles, the tongue, the lips (Langlois, Balken, &Wilder, 1980), and the larynx-the throat structure that converts the relatively steady flow of air from the lungs up through the trachea into a series of bursts or puffs of air (Lieberman, 1967). Compared with the more finite, generally less versatile call systems of other animals, human language is distinguished as much by what it accomplishes (it permits a finite number of meaningless utterances to be recombined into an almost infinite number of meaningful sentences and phrases) as by its unique anatomical-structural and neurophysiological correlates, all of which could make infants more vulnerable to breathing control errors. First among these accomplishments is the extent of volitional or voluntary control of respirations, which may be unparalleled in other species (Tenny &Bartlett, 1981). Moreover, speech production depends on the unique 90 degree articulation of the oral cavity with the pharyngeal cavity, which is elongated in humans, as compared with that in other primates, and serves as a resonant chamber. Laitman's (1985) evolutionary reconstruction of the hominid upper respiratory tract indicates that these anatomical adaptations have existed for only about 300,000 to 400,000 years, making language before this period improbable (also see de Grolier, 1983). Among other primates angle of articulation of the mouth and throat are more obtuse. Among humans, as the nasal, oral, and pharyngeal cavities can be closed off from one another and combined with a shortened, musculated tongue, rapid vowel and consonant sounds are possible (Hill, 1972; lieberman, 1967; Laitman, 1985; Laitman &Crelin, 1980). All of these structural preadaptations evolved at the same time and perhaps were made possible by the migration of the foramen magnum (the great openings at the base of the skull that connects with the skull and the vertebral column) from the back of the skull to the center, a necessary structural

accommodation for upright, bipedal locomotion (see Tobias, 1985). Of course, the adaptations of the mouth and throat alone were not sufficient for language but were accompanied by cortical expansion and greater complexity pertaining to left-hemisphere lateralization. The expansion of humans' brains led to more complex social adaptations and greater dependence on learning, tools, and technology-that is, humans' cultural behavior, of which symbolic communication is a part (see Lancaster &Lancaster 1982). As is well known, as the neocortex expanded during hominid evolution, infants matured at slower and slower rates, thus becoming more dependent physiologically and socially on their caregivers for longer periods of time. In part, the constricted birth opening of the human pelvis, another accommodation to bipedalism, forced the birth of relatively less-developed infants, at least in terms of brain weight and locomotor abilities. At around two months of age, the infant begins to be able to switch back and forth between voluntary and involuntary, or automatic, breathing. Hollien (1980) reported that neonates' reflexive cries, snorts, and cooing begin to give way to more controlled, elongated cries and, as Wilder's (1972) data demonstrate, to an accompanying breathing pattern in which inspiratory time is shortened while expiratory time is lengthened, thus maximizing the amount of air available for phonation. This change in the respiratory cycle is a synergistic interplay of the thoracic, oropharyngeal, and laryngeal muscles, which are volitionally or purposefully manipulated to alter airflow rates and the volume of air retained in the lungs. The infant is now able to make noncrying vocalizations that are neither vegetative (hiccuping, coughing) nor emotional (laughing or crying) but purposeful (Langlois, Balken, &Wilder 1980). These cortically based vocalizations make it easier to understand why, when their infant is around three months old, some mothers and/or care givers can discriminate their infant's specific cries. To a limited extent, infants can change the frequency, pitch, and duration of their cries to communicate specific needs, as, for example, to be fed, comforted, carried, and so forth. But the data on infant acoustic signal specificity at this very young age are, admittedly, controversial (see Murry &Murry, 1980 for a review). Even though there is much disagreement over how tightly linguists should tie crying and noncrying vocalizations to speech itself, or how functionally specific these acoustic signals really are at three or four months of age, there is little disagreement that infants can volitionally control breathing at very young ages or that this is a prerequisite for speech and that it is learned behavior. Lennenberg asserts that "the regulation of air pressure for speech itself is ... apparently fully developed by the time the infant begins to babble in utterances of multiple syllables, roughly during the sixth or seventh month of life" (1967:82). Laufer's study on the temporal regularity of prespeech indicates that "vocalizations displayed by the human infant during the first six months of life reflect greater systematicity than was previously thought... they seem to reflect a type of central programmed regulation ... of cortical or subcortical origin" (1980:305). Wilder (1972) described the attempts by crying, of four- to six-monthold infants, to control the elastic recoil forces of expiration "either to prolong the expiratory phase (as in crying) or to reduce subglottal pressure of relaxation pressure becomes too great" (Wilder, 1972:149). These attempts to control airflow and subglottal pressure were indicated by the presence of subcycles on the polygraph recordings. Wilder explained these breathing spurts: "Because his motor control is immature, the infant overshoots his target when attempting to check expiratory movements, and there is momentary expansion giving rise to a subcycle. Such overshooting is common during the acquisition of other motor skills, such as reaching and grasping" (1972:149). Wilder's (1972) data and explanation of the infant's attempt to control airflow during crying and noncrying vocalizations is important because it implies that (1) there is an experientially based or learned component in infant respiratory behavior as it relates to vocalizations, and (2) insofar as this is true, during the period of time in which infants are at increased risk for SIDS, there is a shift toward greater functional interdependence between higher-brain cortical structures that permit voluntary control of breathing and lower-brain stem structures that control automatic breathing. Voluntary-Involuntary Breathing Interconnections We have good evidence regarding when infants begin to assert voluntary control over their breathing during vocalizations (Wilder, 1972; Hollien, 1980). But we do not know how infants (or adults for that matter) switch back and forth between voluntary and involuntary breathing cortical nuclei so quickly and frequently or whether during REM

sleep, when infants dream and sometimes vocalize, cortical-based breathing control errors are more likely. Remmers (1981:1199) is right in saying that trying to separate voluntary from involuntary breathing control sometimes "leads to a platonic search for reality behind the shadows." Yet, however difficult this search may be, it is clear that the voluntary control of respiratory behavior required for language is extensive-a control that, as we have seen, begins at the same time that infants are at the greatest risk for SIDS. Insofar as SIDS does not occur among other species, this quantitative distinction separating humans from other animals may be worth examining, specifically because Arnon (1983) has commented that no hypothesis concerning SIDS is viable unless it can explain the syndrome's restricted age distribution. As reviews by Plum and Leigh (1981) and Mitchell and Berger (1981) reveal, most of what we know about voluntary breathing is from experimental studies of mammals (sheep, dogs, cats) with vocalization or communicative control systems quite different from our own and from clinical studies of human adults with serious breathing disorders caused by tumors and/or strokes (see Plum &Leigh, 1981). The general picture is that voluntary and involuntary respiratory signals travel separately along primarily two but sometimes three ascending and descending nerve tracts. Two of these three nerve tracts, the corticobulbar and the corticospinal, project diffusely into the neocortex by way of the thalamus connecting with the lower brain stem (pontomedullary) structures as well as with the spinal cord itself, as does the third nerve tract, the reticulospinal (Mitchell &Berger, 1981). It is known that a tumor on the corticobulbar nerve tract can eliminate voluntary breathing, whereas damage to the automatic brain stem structures will not necessarily prevent it, all of which indicate that the forebrain and, especially, the cortical areas near the motor area may influence breathing in important ways (McGinty, 1984; Harper, 1984), as may the spinal cord itself (Leigh &Plum, 1981; Mitchell &Berger, 1981). Especially when infants dream and breathing control may vacillate between the voluntary and involuntary systems, or when REM sleep (which might include some voluntary breathing) becomes indeterminate or NREM sleep, the efficiency of intercellular communication between these two systems may be critical. The kinds of findings and interpretations of Baba and associates (1983) and Quattrochi and associates (1980) may be relevant to hypothesizing what may go awry during such sleep-stage transitions. For example, the processes by which neurostructures mature synchronously to permit, or fail to permit, rapid voluntary-involuntary switching must represent a significant challenge to the two- to five-month-old infant, either sleeping or awake. Recall that this is the developmental stage in which a range of psychomotor skills (reaching, grasping, Piaget's second-stage reactions) are beginning to be mediated by cortical structures, a time period representing the infant's first critical developmental shift (Lipsett, 1981). It thus seems fair to suggest that the neocortex plays a similarly important role in mediating voluntary breathing during some sleep periods (see Remmers, 1981) and that learning may be involved. But unlike the development of other psychomotor skills at this time, the neocortex does not ever replace or even dominate the lower brain stem control structures; rather, cortical-brain stem structures share breathing control and remain functionally interdependent throughout an individual's life. Interestingly, it is at the other end of the life span that involuntary-voluntary interconnections pertinent to breathing may give rise to clinical breathing disorders. Consider, for example, that many older men have serious sleep apneas which are associated with cognitive defects, and thus, we might presume them to be cortical defects (Guilleminault et al., 1976a), though there are other structural problems involved as well (McGinty et al., 1982). The collective data on respiratory behavior patterns of crying and noncrying vocalizations suggest that they are, in part, learned abilities (Wilder, 1972; Laufer, 1980). And the neuroanatomical studies confirming the two distinct areas controlling respiratory behavior, that is, the voluntary and the involuntary system (see Mitchell &Berger, 1981), help us appreciate how and why environmental factors may promote respiratory stability (see McKenna 1987, Part I). For example, recall that Smith's and Steinschneider's (1975) and Chisholm's (1983) studies both argue empirically that prenatal experiences can affect postnatal respiratory activity. As discussed earlier, a strong circumstantial case can be made for the existence of important functional relationships between hearing and breathing, breathing and movement, and hearing, breathing, and vocalizing. If this is correct then a deficit in one of these systems should

change the efficacy of one or all of the other systems. For example, deaf infants who cannot hear either themselves or others breathe and thus do not receive any environmental auditory cues should exhibit breathing defects, most likely in vocalizing. If such infants also do not receive any vestibular cues, they should be at greater risk for SIDS than normal hearing infants are. Moreover, infants with serious neurological disabilities that interfere with their cognitive development and that could also hamper the ease with which they learn to control and maintain proper air pressure and respiratory flow rates should vocalize differently than do healthy infants. Another way to state this is that age-related breathing disorders may also be manifested simultaneously with both learning and cognitive defects and by differences in how normal and abnormal infants cry and later speak. Many of these predictions are borne out. For example, we have known for quite some time that before their deaths, a significant number of SIDS victims cry abnormally, and so the pitch, amplitude, tempo, and latency of cry responses are proving to be helpful in diagnosing a range of genetic and neurological infantile disorders. Colton and Steinschneider (1980) studied the cries of siblings of SIDS victims and discovered that they cried at a higher average pitch and for a shorter duration and had a longer latency period between pain stimuli and cry responses than did controls. Similar to hearing-impaired adults and as predicted by the model proposed here, both the siblings of SIDS victims and the SIDS victims themselves before death exhibited more vocal gliding, biphonation, or voicing during crying bouts. When compared with normal infants, the cries of a child who later died of SIDS were shorter in duration, weaker, accompanied by glottal voicing, and higher in pitch (Stark &Nathanson, 1975). According to Colton and Steinschneider (1980), vocal tract constriction (exacerbated by upper respiratory-tract infections) produces biphonation and/or voicing and could be responsible for some SIDS deaths. Similarly, Lipsett's theory (1981) that infants must learn to breath implies that SIDS victims have not learned how and when to maintain the proper pulmonic or subglottal pressure required not only to coordinate vocalizing and breathing (as demonstrated by abnormal cries) but also to prevent the kind of pharyngeal collapse proposed by Tonkin (1975) in yet another hypothesis regarding some SIDS deaths. Breathing and Vocalizing of the Hearing Impaired Perhaps a more dramatic confirmation of the linkage between hearing and breathing and the experiential or learned bases of speech breathing and the auditory and vocal processes is provided by research conducted on the respiratory patterns and language acquisition of the hearing impaired. Whitehead's (1983) study, for example, reveals that the severely hearing impaired are the most unintelligible speakers, in part because they are not able to learn, presumably through hearing, how to coordinate inspirations and expirations with linguistic patterns. Moreover, the glottal valving of the airstream is inappropriate and inefficient, producing too much air wastage per vocal utterance. Sometimes unintelligible deaf speakers lost three times more air per syllable than did normal controls (Whitehead, 1983). According to both Whitehead's (1983) and Forner's and Hixon's (1977) experimental data, hearing-impaired persons initiate speech at much-too-low lung volumes, and generally while speaking they maintain lower-than-required functional residual capacities (the total amount of air remaining in the lungs after expiration). Because they maintain only half the amount of air in their lungs that normal speakers do, hearing-impaired persons must apply greater muscular pressure, which, as Whitehead pointed out (1983), actually works against respiratory (lung) recoil forces. Thus the speech of the hearing impaired continues beyond the functional residual capacity of the lungs that support it (Whitehead 1983, Forner &Hixon 1977). These findings illustrate that the inability to hear prevents individuals from learning not only how to formulate particular sounds but also how to control and coordinate the voluntary, cortex-based respiratory behavior that underlies such sounds. The data in this field of inquiry underscore the important functional relationship among hearing, breathing, and vocalizing, as discussed earlier, and support the idea that a functional deficit in one of these systems can affect the efficacy of the others. The sensory systems of hearing and breathing are interrelated in other ways as well. For example, when summarizing his own and others' work, Ornitz (1985) observed that vestibular dysfunctions, though associated with other disorders, also are associated with some language disorders and "certain types of hearing loss" (Ornitz, 1983:521). For example, Seeman (1969, in Ornitz) found that 25% of children with delayed speech

development suffered a vestibular dysfunction that led to differences in coordinating the speech muscles. It is known also that hypoxemia, or chronically low oxygen levels, results in the neuronal deterioration of both the peripheral and the internal hearing structures and that likewise in low birth weight infants (less than 1,800 g) a significant correlation was found among cyanotic attacks, spastic dysplagia, and hearing loss (McDonald 1969, cited in Ornitz 1983). The respiratory distress syndrome clinically defined and introduced by Spector and colleagues in 1978 includes hemorrhaging in the inner ear, which usually leads to death, but for one child in their study, it led to significant hearing loss (Spector et al., 1978). Finally, Guilleminault, Tillian, and Dement (1976b) studied clinical disorders of older patients with sleep apneas and found that these apneas were associated with both memory and cognitive defects-another reminder that during periods of cortical involvement during sleep, the breathing control of those individuals with some kinds of learning defects is jeopardized. Adults (usually men) do not, of course, die of SIDS. But perhaps-unlike those infants whose systems are still immature-in adults, other compensating arousal systems are mature enough to ensure that at some point they will breathe before cyanosis, whereas the compensating checks of infants' respiratory systems are not yet working efficiently enough to permit recovery, as Fleming's (1984) data suggest. SUMMARY When integrated with clinical findings on SIDS, an evolutionary perspective on the human infant also offers insights as to why SIDS seems to be a species-specific malady, why it occurs at between two and four months, and why it cannot be replicated experimentally among other mammals. For example, studies of the respiratory and crying behavior of normal and hearing-impaired human infants show that experience plays a role in how human infants (but not necessarily other species) learn to breathe. That is, the data indicate that at the time that infants are at greatest risk for SIDS, the infant's neurological preadaptations for language or speech breathing first become apparent. At two to four months, studies reveal that the infant learns to assert voluntary control over respiration underlying crying and noncrying vocalizations-voluntary, cortex-based manipulations that precisely replicate and provide practice for speech breathing, which is mastered by the infant by seven months of age, well before the first word. The degree to which humans switch back and forth between voluntary and involuntary breathing during sleep and wake cycles (or cortex and brainstem nucleii) is unparalleled in other forms, but it suggests a system that is complex, experientially based, and subject to a variety of control errors, not the least of which is a lack of maturational synchrony in nerve centers or conduits connecting these two interdependent respiratory centers. Yet, these unique adaptations can explain, in part, why we should expect that natural selection might enhance infantile sensitivity to compensatory auditory, vestibular, and gaseous breathing cues; these are all stimuli reliably available in the microenvironment in which the infant evolved, but which for certain times in the infant's day, are missing in urban environments when infants sleep separately from parents. The perspective and hypotheses presented here consider the potential physiological regulatory effects human parents have on their highly neotenous infants; and, insofar as we may have underestimated these regulatory effects, new questions can be asked about how the respiratory system can go awry. Finally, when an evolutionary and, indeed, cross-cultural view of the infant is considered, it would appear that in our enthusiasm to recognize the human infant's competence and preparedness to become competent, we have pushed the notion of the infant's physiological independence too far-or at the very least, pushed it out of line with what the infant's evolutionary history suggests to be the case. An evolutionary perspective offers many new research questions, helps us to reexamine underlying assumptions of SIDS research, and potentially can move us closer to better understanding the multiple internal and external factors that coalesce to create the conditions within which SIDS is most likely to occur. References REFERENCES Altmann, J. (1980). Baboon mothers and infants. Cambridge: Harvard University Press. Anders, T.F. (1978). Home recorded sleep in two- and nine-month-old infants. Journal of the American Academy of Child Psychiatry, 17, 421-432. Anders, T.F. (1979). 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