EVOLUTION AND THE SUDDEN INFANT DEATH SYNDROME (SIDS)
Part II: Why Human Infants?

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Postnatal parent–infant physiological regulatory effects described in the previous paper (Part I) are viewed here as being biologically contiguous with events that occur prenatally, preparing and sensitizing the fetus to the average microenvironment into which the infant is expected, based on its evolutionary past, to be born. Following McKenna (1986), evidence (some of which is circumstantial) is presented concerning fetal hearing and fetal amniotic liquid breathing as they are affected both by maternal cardiovascular blood flow sounds in the uterus and by fluctuating maternal blood sugar levels. These data are linked in turn to the infant's postulated postnatal responsivity to parental sensory cues, including auditory and vestibular respiratory cues that may assist infants as they "learn" to breathe and, for some, to resist a SIDS event.

Data on the respiratory and vocalizing behavior of normal and hearing-impaired persons are used to show that not all forms of human breathing are innate; some forms develop with experience. These data reveal how human infants learn, for example, to coordinate higher and lower brain respiratory nuclei in the context of learning initially to cry with intent and purpose and later to speak. Voluntary, cortex-based breathing emerges at the same time that infants are most likely to die from SIDS, between 2 and 4 months of age. This switch between voluntary and involuntary breathing during both sleep (while dreaming) and wake cycles, which depends on the integration of higher cortical and lower brain stem nuclei, is complex and is possibly the basis of the human species' unique susceptibility to SIDS—a syndrome as yet unrecognized in other species. These human infant vulnerabilities, including delayed maturity, can explain in part why natural selection ought to favor increased infant sensitivity to parental sensory cues provided by a caregiver—stimuli available in the evolving parental care environment that included parent–infant co-sleeping for more than 4–5 million years of human evolution.

KEY WORDS: **SIDS; Speech breathing; Fetal liquid breathing; Hearing-impaired infants; Parental breathing cues**

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INTRODUCTION

This paper continues the discussion begun in the previous paper (Part I) by examining why human infants are especially prepared to respond postnatally to a variety of parental sensory cues, including (as hypothesized earlier in McKenna 1986, and in Part I) parental respiratory cues involving sound, touch, movement, and possibly expelled carbon dioxide gas. It is suggested that when the evolutionary history of parent–infant relationships is considered, parent–infant co-sleeping arrangements should provide the safest possible context within which human infants shift away from strictly lower brain stem, reflexive breathing patterns at 2–4 months of age (during sleep and awake periods) to a system of respiratory control in which higher, cortical nuclei permitting voluntary breathing interact with lower brain stem nuclei. This interaction permits purposeful vocalizations in the form of crying and, later, speech. Speech breathing and the neurological adaptations that underlie these respiratory manipulations are unique abilities that, it is suggested, make human infants especially susceptible to a variety of central nervous system deficits suspected to be involved in some cases of SIDS.

PRENATAL ANTECEDENTS OF POSTNATAL INFANT SENSITIVITY TO PARENTAL BREATHING CUES

To understand the human infant's physiological responsivity to parental sensory contact, reference must be made to the manner in which natural selection, operating on prenatal characteristics, favors a fetus that is adapted both to the immediate uterine environment and to the postnatal

environment into which it "expects" to be born. By beginning with intrauterine life, and thereby considering epigenetic processes, we create a theoretical rationale and basis for predicting which aspects of the developing central nervous system that exhibit structural–functional connections in the womb might well be linked postnatally, and therefore how and why prenatal perturbations affect the infant's ability to respond postnatally to similar or different environmental stimuli or disturbances.

Of special significance to the hypothesis that infants are particularly responsive to a variety of parental sensory breathing cues are recent studies documenting the simultaneous emergence of fetal hearing and amniotic (liquid) breathing at or around 7 months gestational age, although hearing and breathing are not ordinarily functionally or developmentally linked in this way. Henderson-Smart et al. (1983:301) have demonstrated that the brain stem center that regulates breathing is adjacent to the brain stem auditory nuclei, and they suggest that possibly both systems follow a similar time course of structural–functional development. Similarly, by 6 months gestation, the brain stem's vestibular nuclei that process responses to rocking and movement, which are known to promote rhythmic breathing (Korner and Thoman 1972; Korner et al. 1978; McGinty and Hoppenbrouwers 1983), also develop at around 6 months gestational age.

Fetal liquid breathing is practice for postnatal infant breathing. It 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). In fact, Jansen and Chernick 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" (1983:460). 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% of the time, beginning at around 30–31 weeks gestation, although its frequency varies greatly among infants and may occur initially at around 21 weeks. Though the fetus's breathing is variable and different from postnatal breathing in some important ways (e.g., 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 et al. (1978a, 1978b, 1980) monitored patients for up to 24 hours at a time during the last 10 weeks of pregnancy and found that the frequency of the fetus's breathing varied

from hour to hour but peaked around 2–3 hours after the mother ate a meal. Patrick and colleagues also found that the fetus's breathing began to increase from 4 to 7 A.M., 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 (1983) point out, premature infants are therefore at a distinct "disadvantage" because, among other reasons, they are unable to practice breathing *in utero* to the degree that, in many cases at least, they become competent in organizing respiratory behavior.

Although investigators strongly suspected that fetal breathing occurs among many mammals, they were less certain whether the brain stem, in a way similar to its function in postnatal breathing, actively directs and controls this behavior prenatally (McKenna 1986). This question was tentatively answered by Chernick et al. (1973a, 1973b), who mapped the proposed respiratory centers in "exteriorized" fetal sheep while their mothers were anesthetized. They demonstrated that stimulating the fetus's medulla oblongata (the presumed control center for postnatal breathing) could induce respiration. Their work also showed that the respiratory center nerves (though different in distribution, of course, in the fetal and the fully developed lamb) mature around the same time that fetal breathing begins and thus should be thought of as part of the system that controls its onset (Jansen and Chernick 1983).

Bystrzycka and co-workers (cited in Jansen and Chernick 1983) were the first to record the medullary respiratory neurons directly and to document the relationship between early fetal breathing and the discharge of specific neurons in the fetus's brain stem clearly. They showed that "inspiratory neuronal discharges were found in phase with fetal breathing in half of the fetuses studied" and, most important, that "even when the fetal subjects experienced apneas (temporary breathing cessations), tonic discharges of inspiratory neurons continued" (Jansen and Chernick 1983:455). This situation parallels what occurs during some instances of apneas among humans (Darwish and McMillan 1983). Jansen and Chernick (1983:455) suggest that fetal lambs may experience apneas in part owing to an "ineffective breathing rhythm generator" that helps to coordinate and drive sequential breathing. Patrick et al. (1978a, 1978b, 1980) also indicate that some kind of respiratory pacemaker may be important in coordinating fetal breathing. It is here that auditory cues, enhanced by vestibular stimuli, may play a role and help to explain, for instance, why the lamb fetuses that Chernick describes suffered from apneas even while their respiratory neurons continued to fire. This finding is important, I believe, for understanding how infants initially develop *in utero* a sensitivity to external auditory cues that can affect their breathing rates later in life.

WHAT THE FETUS HEARS IN THE UTERUS

The mother remains the initial and exclusive source of auditory stimuli for the fetus because her abdominal wall acts as a barrier to almost all external environmental sounds lower than that of a passing train (about 115 dB; see Bench 1968). Walker et al. (1971) found that background noises inside the human uterus reach approximately 95 dB, rising to rhythmic peaks of about 110 dB around 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 et al. 1971, cited and discussed in 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, placenta, and uterus (Figure 1).

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 Smith and Steinschneider's (1975) findings are considered. These researchers examined newborns 24–48

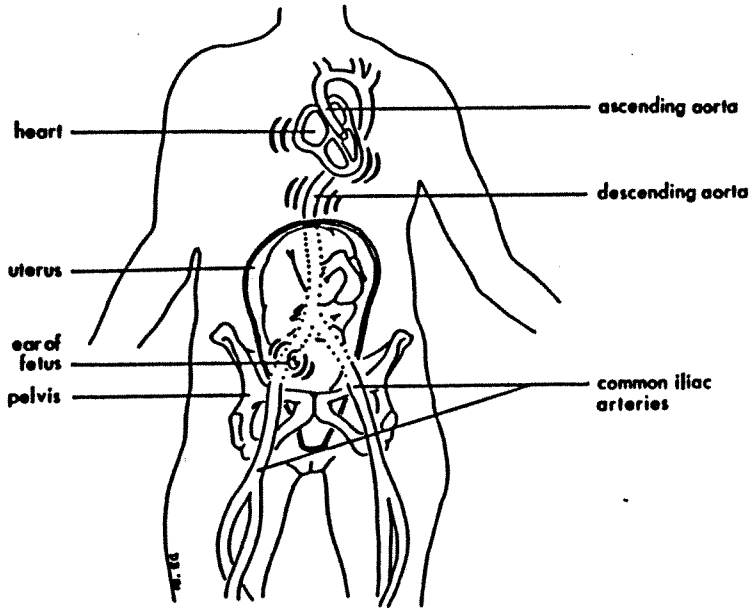

Figure 1. Prenatal auditory experiences of the fetus: how the fetus monitors the heart rate sounds of its mother (from McKenna 1986).

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 asleep faster, and generally cried less often than did infants born to mothers with higher heart rates. Similarly, Reppert and Schwartz (1983) found that a mother rat's REM and NREM sleep cycles affected the circadian rhythms of her offspring. Smith and Steinschneider's (1975) findings implicate fetal–maternal rhythmic experiences in an understanding of postnatal central nervous system development. These data also agree with Chisholm's (1983) findings on the effect of maternal high blood pressure on infant temperament and with the study of Schmidt et al. (1980) on the effect of heartbeat sounds during the active sleep of premature infants.

All of these studies and their implications recall Salk's (1961, 1962; Salk et al. 1974) hypotheses that suggested a relationship between a mother's preference for carrying her infant on her left side and the calming influence of her heartbeat on the infant's disposition. According to Salk (1961, 1962), the mother's 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 neonatal weight gain exceeding that of nonexposed infants. Because a higher birth weight is associated with a better chance of survival (Williams et al. 1977:8–9), such auditory stimulation could be adaptive.

But does the uterine environment in fact permit 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) demonstrates 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 the effects of rhythmic sounds on infants).

If fetal hearing and breathing are functionally linked, there must be evidence demonstrating not only that the human fetus actually breathes in the womb, 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 month, by which time both middle and inner ear structures have reached full adult size" (Eisenberg 1983:239; 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 gen-

iculate nuclei, which are involved in complex assignment of meaning to sound and location integration) are not as mature as the peripheral auditory structures (see Parmelee and Sigman 1983), the human fetus's ear is capable not only of responding to sound, but also of recognizing and later preferring to hear its mother's voice (DeCasper and Fifer 1980).

Birnholz and Benacerraf (1983) examined 236 fetuses between 16 and 32 weeks gestational age through high-resolution ultrasound imaging to determine when the fetus could respond (by blinking its eyes) 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 (1983:517). The acoustic stimulator was pressed onto the mother's abdominal wall, and although 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 only 15 dB (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.

Thus, the human fetus and infant, at a very early point in their prenatal and postnatal development, respectively, can hear and respond to acoustic rhythms, possibly even breathing rhythms. This fact is not surprising, because human vocalizations are the most complex of any mammal, both in regard to the control of the respiratory muscles that enable speech (Whitehead 1983) and in regard to the role that hearing plays in enabling the proper vocalizations to be formulated (Laufer 1980; Lieberman 1984). Moreover, 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 to make the kinds of respiratory responses that they made against the background of cardiovascular auditory stimuli and movements they received *in utero*.

SIDS AND BREATHING RESEARCH: THE APNEA CONNECTION AND POSSIBLE BRAIN STEM ABNORMALITIES

According to Bergman (1986), the relationship of the cardiorespiratory system to SIDS was galvanized basically by two kinds of studies: those that found small hemorrhages (petchiae) in the thymus area or thoracic cavity, indicating negative chest pressure presumed to be caused by the

infant's unsuccessful attempt to either inhale or exhale (see Beckwith 1988), and those that showed a relationship between hypoxemia and/or apnea and the eventuality of the infant dying of SIDS (see Naeye 1973; Steinschneider 1972). The apnea–SIDS connection is at this point more problematic than is the possibility of some infants dying from an upper respiratory obstructive apnea (Beckwith 1988)—a condition associated with the presence of petchiae on autopsy. The possibility remains, however, that through physical contact the chances of a loss of structural potency that may induce obstructed apneas may be reduced or somehow modified enough to aid the infant in overriding a fatal event.

Most 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 pontomedullary) region of the brain stem's reticular formation (see Figure 2). Situated near the brain's central base at the top of the spinal cord, these medullopontine structures are thought to be a primary respiratory or "pneumotaxic" control center, though the question of a central control is still controversial (Mitchell 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 and rhythm, 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: the lungs (through the vagus nerve, which communicates the status of the oxygen and carbon dioxide levels and the lung stretch receptors) and 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 (situated 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 (Figure 2), are also implicated in respiratory behavior and are partially controlled by reticular-formation nuclei. It is important to point out that 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 tracts, such as the corticobulbar, corticospinal,

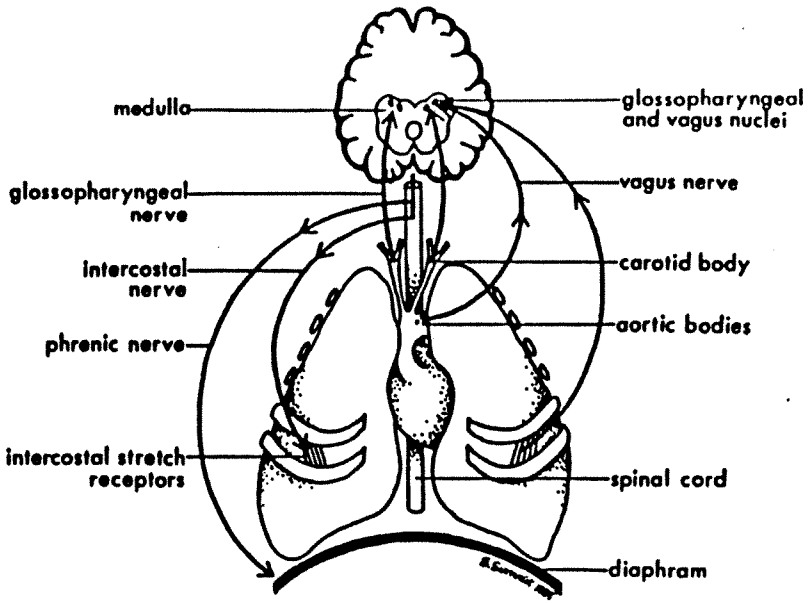

Figure 2. Structural, neurophysiological, and morphological systems most frequently examined in SIDS research (after McKenna 1986).

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 ambiguus) 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*. The reticular activating system coordinates breathing with cardiac output, sleep–wake cycles, and arousal (Darwish and McMillan 1983; Kahlia 1981; Mitchell and Berger 1981; Plum and Leigh 1981).

In landmark papers, Steinschneider (1972) and Naeye (1973) proposed that deficits in the brain stem reticular formation or adjacent structures could be the cause of some forms of apnea, which in turn could be linked to sudden infant death. Infantile apnea is a condition 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, the cessation of airflow can lead to low oxygen pressure in the blood (referred to as *hypoxia* or, if chronic, *hypoxemia*) and high carbon dioxide (CO_2) 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 apnea-prone infants from three families; two of these infants 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 research in this area. Since that paper's publication, apneic infants and siblings of SIDS victims have been used extensively in research, in the hope 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. 1976a, 1976b, 1979a, 1979b, 1986) 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), since only an estimated 5–7% of SIDS victims had apneic attacks before they died. The most recent epidemiological study does not reveal a strong association between apnea and SIDS; but although the apnea–SIDS connection remains unclear, it is still "arguable" (Hoffman et al. 1988). It is clear, though, that the occurrence of apneas neither permits predictions regarding which infants are at risk of dying from SIDS nor provides explanations for the majority of SIDS incidences.

Breathing control errors and possible brain stem postmortem markers of SIDS were also implicated in the pioneering studies of Naeye (1976) and Naeye et al. (1976). For example, they showed that SIDS victims had enlarged or hypertrophied pulmonary arterioles, that is, increased muscle mass. From this finding, they inferred that SIDS victims undergo chronic hypoxia, or underventilation, before death, because of abnormalities in the brain stem–directed respiratory drive. Like Steinschneider, Naeye and his colleagues suggest that the reticular formation's breathing control mechanisms may be defective and so may contribute to the apnea preceding SIDS. In addition, because the respiratory drive is deficient, the heart must work harder to compensate, which causes arterial thickening. Beckwith (1983) points out, however, that only one study in five confirmed Naeye's specific finding of increased pulmonary muscle mass, and research reported by Haddad and Mellins (1983) and Pearson and Brandeis (1983) likewise failed to replicate this finding (also see Singer 1984).

Becker (1983), Quattrochi et al. (1980), and Takashima et al. (1978a, 1978b) found in SIDS victims a significant number of astrocytes, the star-shaped neuronal cells in the brain stem's reticular formation (especially in the nucleus tractus solitarius, a primary respiratory control region), which were marked by extra fibers and/or small tumors. These

abnormalities constitute a condition referred to as *astrogliosis*, or brain stem gliosis, and conceivably could interfere with diverse vagal pulmonary nerve signals as well as interrupt signals from the carotid bodies (the structures that monitor the blood's CO_2 levels) to the brain through the glossopharyngeal nerve (see Figure 2).

Other proposed brain stem and nerve abnormalities leading to SIDS include leukomalacia, or white matter lesions in the brain stem, probably caused by inadequate blood flow (ischemia) to the brain; fatty changes and tissues among brain stem neurons, including a retention of fat; an undervascularized reticular formation and damage to lateral and dorsal regions (Becker 1983); and less mature or less myelinated vagus nerve fibers, compared with those of controls (Sachis et al. 1981).

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 et al. (1981) show that the rate of brain maturity of near-miss SIDS infants was slower than that of controls, as indicated by delayed reorganization of REM and NREM sleep patterns. Baba et al. (1983) observe 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. Like Guilleminault et al. (1975), Becker and Thoman (1983) suggest that added minor stress, such as a respiratory tract infection, may compound the infant's vulnerability to respiratory collapse because it does not yet have adequate neuronal regulatory control. The finding by Quattrochi et al. (1980) 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, as does the recent epidemiological study that shows a strong association between respiratory infections and SIDS 2 weeks prior to the deaths of many infants (Hoffman et al. 1988).

WHY ARE HUMAN INFANTS AT RISK OF DYING FROM SIDS?

As far as we know, SIDS is unique to humans; it appears to be species-specific. The fact that no animal model of SIDS has yet to be produced

suggests that it is important to consider both the unique delayed developmental trajectory of the human infant, whose brain is approximately 25% of its adult weight at birth, and possibly species-specific differences in the development of sleep, arousal, and respiratory behavior in humans and their underlying neurological bases. With respect to breathing control mechanisms, we can ask: What seems to make human infants particularly vulnerable to control errors during a particularly restricted age of between 1 and 6 months?

The process by which the brain's voluntary and involuntary breathing and vocalizing control centers become functionally interdependent during sleep–wake transitions, during dreaming episodes, and during infant awake periods represents one set of emerging human abilities that not only fits the SIDS age distribution but reminds us that the human infant faces severe respiratory control challenges not necessarily encountered by other species. At the age at which infants are at the highest risk of dying from SIDS, they begin to deliver purposeful vocalizations and to manipulate voluntarily the amplitude, pitch, and tempo of their cries.

Most striking is that by 7 months, well before the infant utters its first words, it has mastered the control and coordination of its vocalizations and breathing—or speech breathing, as it is called (Wilder 1972). And, as Laitman and Crelin (1980) demonstrate, both the lowering of the larynx and the elongation of the pharyngeal cavity occur at this time as well.

Physiological studies have shown 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. 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 that control airflow rates and make speech possible. For example, unlike vegetative or maintenance breathing ("quiet breathing" in Figure 3), speech breathing essentially limits inspiratory interruptions to ensure the maximum use of air for phonation or vocalization as air is expired (Langlois et al. 1980). Speech breathing requires fewer breaths per minute than quiet breathing (from an average of 18 breaths per minute to about 10–14 per minute in adults) and a larger residual volume, or amount of air remaining in the lungs after expiration or vocalization (Forner and Hixon 1977; Whitehead 1983). Speech breathing requires the lungs to increase their air pressure (pulmonic pressure), and, even during exhalation, the glottis must hold back some air to maintain adequate subglottal pressure needed to manipulate the vocal cords (Langlois et al. 1980; Wilder 1972). These behaviors also

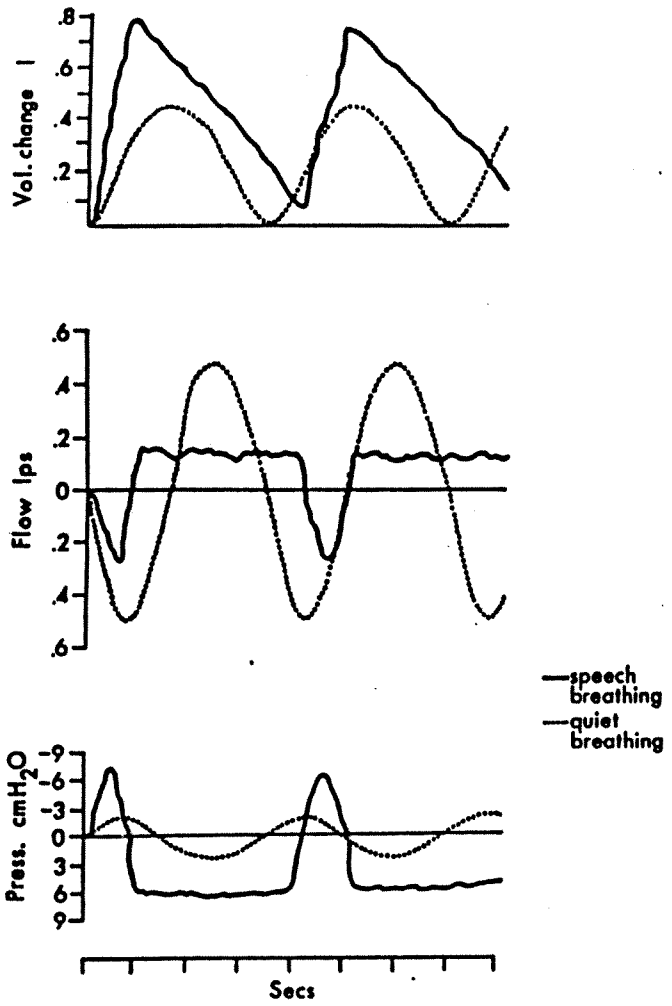

Figure 3. Schematic representation showing changes in volume, pressure, and air flow during speech breathing and quiet (maintenance or vegetative) breathing (from McKenna 1986; after Langlois et al. 1980).

permit manipulations of the oropharyngeal muscles, the tongue, the lips (Langlois et al. 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).

Speech breathing is learned before speech itself in the context of the infant asserting increasingly efficient voluntary control of its crying; this assertion is based on studies of the changing biomechanical bases of

infant breathing and vocalizing during the first year of life (see Laufer 1980; Lenneberg 1967; Lieberman 1967; Michelsson and Wasz-Hockert 1980; Prescott 1980) and on studies of the hearing, breathing, and vocalizing patterns of the hearing-impaired. Shortly after birth, the newborn's breathing shifts from shallow, diaphragmatic–abdominal-based breathing to a deeper breathing made possible by maturing chest and rib muscles (the internal and external intercostals), by positional changes in the ribs relative to the spinal cord (Langlois et al. 1980), and by the rapidly increasing number of alveoli in the lungs needed for oxygenation (Thurlbeck 1975). The dramatic nature of these changes is reflected by the fact that the infant's breathing rate drops from an average of 85 breaths per minute in the first month of life to as low as 42 breaths per minute by 13 months. This figure compares with between 17 and 21 breaths per minutes for adults (Langlois et al. 1980).

At around 2 months of age, the infant begins to be able to switch back and forth between voluntary and involuntary, or automatic, breathing (Figure 4). Hollien (1980) reports 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 and expiratory time is lengthened, thus maximizing the amount of air available for phonation. This change in the respiratory cycle is a synergistic interplay of thoracic, oropharyngeal, and laryngeal muscles, which are volitionally or purposefully manipulated to alter airflow rates and the volume of air retained in the lungs.

Wilder (1972:49) illustrates the extent to which the control of breathing during the infant's crying episodes is learned. She describes the attempts of 4- to 6-month-old infants, by crying, to control the elastic recoil forces of expiration "either to prolong the expiratory phase (as in crying) or to reduce subglottal pressure if relaxation pressure becomes too great." These attempts to control airflow and subglottal pressure were indicated by the presence of subcycles on the polygraph recordings. Wilder explains these breathing spurts as follows: "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 data and explanation of the infant's attempt to control airflow during crying and noncrying vocalizations are important because they imply that (*a*) there is an experientially based or learned component in infant respiratory behavior as it relates to vocalization, and (*b*) 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

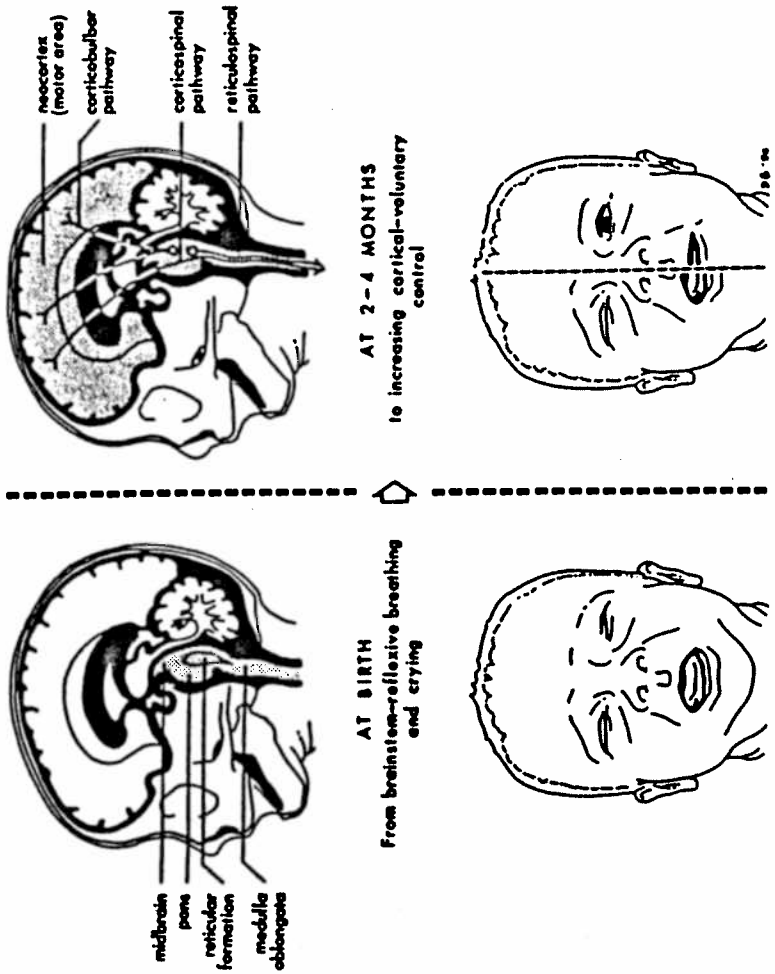

Figure 4. Shifting respiratory control at 2–4 months postnatal age showing the emergence of shared brain stem–cortical breathing control necessary for speech breathing vis-à-vis crying (from McKenna 1986).

permit voluntary control of breathing and lower brain stem structures that control automatic breathing.

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) is replaced by intermediate NREM sleep, the efficiency of intercellular communication between these two systems may be critical. The kinds of findings and interpretations of Baba et al. (1983) and Quattrochi et al. (1980) may be relevant to hypothesizing about what might 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 2- to 5-month-old infant, either sleeping or awake. The neuronal conduction that involves different parts of the brain, one can speculate, makes breathing control susceptible to errors. Unlike the development of other psychomotor skills, the development of breathing skills never involves the replacement or even domination of the lower brain stem control structures by the neocortex; 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 disorders. For example, many older men have serious sleep apneas that are associated with cognitive defects, and thus we might presume them to be cortical defects (Guilleminault et al. 1976a), though there may be other structural problems involved as well (McGinty et al. 1982).

Voluntary and Involuntary Breathing: How and When Do They Occur in Humans?

Remmers may be right when he states that trying to separate voluntary from involuntary breathing control "leads to a platonic search for reality behind the shadows" (1981:1199). But even so, the kind of cortically mediated control of breathing 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, particularly because Arnon (1983) has commented that no hypothesis concerning SIDS is viable unless it can explain the syndrome's restricted age distribution. 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, nor do we know whether cortical breathing control errors are more likely during REM sleep, when infants dream and sometimes vocalize.

As reviews by Mitchell and Berger (1981) and Plum and Leigh (1981) reveal, most of what we know about voluntary breathing derives from experimental studies of mammals (sheep, dogs, and cats), which have vocalization or communicative control systems quite different from our own, or from clinical studies of human adults with serious breathing disorders caused by tumors or strokes (see Plum and 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 (Figure 5). Two of these 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; the third nerve tract, the reticulospinal, begins in the lower brain and descends into the spinal cord (Mitchell and Berger 1981). It is known that a tumor on the corticobulbar nerve tract can eliminate voluntary breathing, whereas damage to automatic brain stem structures will not necessarily prevent it, which indicates that the forebrain and, especially, the cortical areas near the motor area may influence breathing in important ways (Harper 1984; McGinty 1984), as may the spinal cord itself (Mitchell and Berger 1981; Plum and Leigh 1981).

If there are important functional relationships between and among hearing, breathing, and vocalizing, a deficit in one of these systems might well change the efficacy of one or both 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 expectation 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.

Some of these predicted associations hold. We have known for quite some time that before their deaths, a small number of SIDS victims cried abnormally (but see Hoffman et al. 1988); thus the pitch, amplitude, tempo, and latency of cry responses are proving to be helpful in diag-

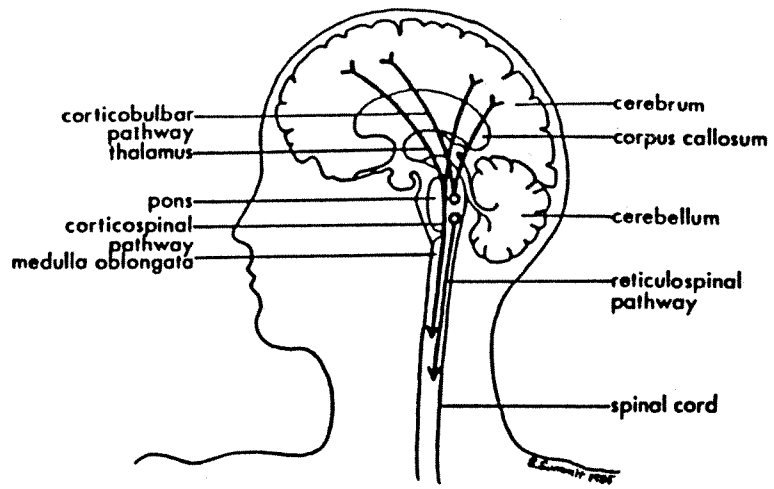

Figure 5. Neurological bases of voluntary–involuntary breathing control and their interconnecting nerve conduits (from McKenna 1986).

nosing 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 infants in control groups. 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 the cries of 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 and Nathanson 1975). According to Colton and Steinschneider (1980), vocal tract constriction (exacerbated by infections of the upper respiratory tract) produces biphonation and/or voicing and could be responsible for some SIDS deaths, although the most recent epidemiological study by Hoffman et al. (1988) failed to find a significant association between abnormal cries and SIDS deaths. Similarly, Lipsitt's (1981) theory that infants must learn to breathe implies that some SIDS victims may not have 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 (McKenna 1986).

The Relationship among Hearing, Breathing, and Vocalizing: Evidence from the Hearing-Impaired

A more dramatic illustration of the linkage between hearing and breathing and the experiential or learned bases of speech breathing is provided by research conducted on the respiratory patterns and language acquisition of the hearing-impaired. Whitehead's (1983) study, for example, reveals that severely hearing-impaired speakers are the most difficult to understand, in part because they are not able to learn how to coordinate inspirations and expirations with linguistic patterns. Moreover, the glottal valving of the air stream is inappropriate and inefficient, producing too much air wastage per vocal utterance. Sometimes deaf speakers lose three times more air per syllable than do normal controls (Whitehead 1983).

According to both Whitehead's (1983) and Forner 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 as that held by normal speakers, the hearing-impaired must apply greater muscular pressure, which actually works against respiratory (lung) recoil forces (Whitehead 1983). Thus, the speech of the hearing-impaired continues beyond the functional residual capacity of the lungs that support it (Forner and Hixon 1977; Whitehead 1983).

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 discussed earlier, and these findings also support the idea that a functional deficit in one of these systems can affect the efficacy of the others.

Ornitz (1983) has observed that vestibular dysfunctions, though associated with a number of other disorders, also are associated with some language disorders and "certain types of hearing loss" (Ornitz 1983: 521). For example, Seeman (1969, cited in Ornitz 1983) found that 25% of children with delayed speech development suffered vestibular dysfunction that led to differences in coordinating the speech muscles. It is also known that hypoxemia, or chronically low oxygen levels, results in the neuronal deterioration of both the peripheral and the internal hearing structures; likewise, in low-birth-weight infants (less than 1800 g), a

significant correlation was found among cyanotic attacks, spastic diplegia, and hearing loss (McDonald 1969, cited in Ornitz 1983). Guilleminault et al. (1976a) studied clinical disorders of older patients (usually men) 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 deficits is jeopardized. Adults do not, of course, die of SIDS. But perhaps in adults—in contrast to infants, whose systems are still immature—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 at a level efficient enough to permit recovery, as Fleming's (1984) data suggest.

SUMMARY AND CONCLUSION

In this paper I propose that especially infants born with central nervous system deficits should benefit physiologically from contact with a parent or caregiver throughout the night. I do not address here how well, or if, this research model fits with the most recent epidemiological findings on SIDS or with some preliminary results of our own study of the sleep and breathing patterns of co-sleeping mother–infant pairs. This fit will be discussed in a paper in the next issue (Part III). My purpose here has been to provide the developmental and evolutionary background against which at least some new SIDS research questions could be formulated. By seriously considering as I do here the evolutionary background and the biology of the human infant, including both its unique delayed developmental trajectory and its neurophysiological adaptations for speech breathing and language, one can better appreciate how its physiological systems might go awry when both constitutional deficits and environmental factors coalesce. Moreover, the novelty, the evolutionary recentness, and the likely social, psychological, and biological disadvantages of solitary sleep patterns of young infants can be understood more fully.

This model does not suggest that separate parent–infant sleeping causes SIDS; rather it suggests that much circumstantial and indirect evidence indicates that social sleep and its effect on arousal and the emergence of infant sleep patterns in general are more likely to help an infant to resist or combat a SIDS event than are solitary infant sleep environments. It is time to question in a serious way some of the assumptions we make about what "cannot" be involved in SIDS; after all, SIDS is now believed to have multifactorial origins (Schwartz and Seg-

antini 1988), and this alone has major implications for how we research its origins and evaluate explanations of its causes. A rethinking of the SIDS puzzle must begin by rethinking the biological and social conceptualizations of the individuals who are tragically victimized. It is in this area that the anthropological perspective discussed here has the greatest chance of being useful.

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