Researching the Sudden Infant Death Syndrome (SIDS): The Role of Ideology in Biomedical Science

James J. McKenna
Professor of Anthropology
Pomona College
and
Adjunct Clinical Assistant Professor
Departments of Neurology, Child Psychiatry,
and Human Behavior
University of California, Irvine
School of Medicine

MONOGRAPH SERIES OF THE NEW LIBERAL ARTS PROGRAM

The New Liberal Arts (NLA) Program of the Alfred P. Sloan Foundation has the goal of assisting in the introduction of quantitative reasoning and concepts of modern technology within liberal education. The Program is based on the conviction that college graduates should have been introduced to both areas if they are to live in the social mainstream and participate in the resolution of policy issues.

The New Liberal Arts Program has led to significant, new courses and course changes in many colleges and universities. MIT Press and McGraw-Hill are jointly publishing an NLA series of books. These monographs are planned to provide teaching/learning materials for other educational developments. A complete list of the monographs can be found on the last page of this book.

NLA Monograph Series

Co-Directors: John G. Truxal and Marian Visich, Jr.
Production Editor: Carol Galdi

Contents

I. Introduction: The Trouble with SIDS, The Trouble with Science 1

II. SIDS Epidemiology 4

a. When is it SIDS? Why is it so hard to study?
b. How do you "prove" a "cause" for SIDS?
c. Age
d. Sex
e. Geography, Ethnicity, and Seasonality
f. Time of Day
g. Socioeconomics and Age of Mother
h. Characteristics of those at risk
i. Does SIDS run in families?
j. What can be said?

III. SIDS Sleep Research: A Brief Review and Critique 9

a. Breathing during sleep: The possible inability of the infant to arouse to breathe
b. Possible cardiac abnormalities

IV. So, What's the Problem? An Anthropological View of SIDS Research Assumptions 15

a. Infants are not "mini adults"
b. Where's the beef? Toward reconceptualizing infancy using cross-species, paleoanthropological, and cross-cultural data collected by anthropologists
c. What the monkeys tell us
d. What the fossils tell us: Upright posture, fetal encephalization, and the birth of neurologically immature human infants
e. What the cross-cultural (ethnographic) data tell us
f. Where and how infants sleep around the world

V. The Ideology of Separate Parent-Infant Sleep 26

a. Autonomy or interdependence
b. Where did the ideology come from?
c. The suffocation factor

VI. Application: An Anthropological Hypothesis on the Possibel Relationship Between Co-sleeping and SIDS 30

a. Testing the hypothesis: Are SIDS rates lower in co-sleeping societies?
b. SIDS rates in cross-cultural perspective

VII. Physiological Interactions of Co-Sleeping Mothers and Infants: A Preliminary Glimpse 34

a. What we did and why
b. What we found
c. But it doesn't prove anything, does it?

VIII. The Response from the Medical, Social Science, and Sleep Research Communities 42

IX. My Response: Conflict and Congruence (as published) 53

X. Conclusions/Student Study Questions 57

References 60

I. Introduction: The Trouble with SIDS, The Trouble with Science

In a memorable scene from the 1985 film "Terms of Endearment", Shirley Maclaine's character leans her head against the door frame of the nursery as she looks lovingly at her new grandchild sleeping across the room in a crib. Suddenly, she straightens. Her relaxed expression gives way to an attentive and concerned one. She bolts across the room to the crib. With panic on her face she shakes the infant wildly as the baby finally awakens and cries out. Maclaine's face is consumed by relief as she is reassured that her grandchild is still alive and breathing.

Even though the scene could have ended sadly, it provoked a great deal of laughter from the audience. After the film, my friends and I agreed that the reason the scene was so funny was that many of us had experienced exactly the same thing with our own babies. Certainly everyone watching the movie, young and old, seemed to know exactly what was going on and could identify with it. Upon not seeing the infant's chest moving in the crib, Maclaine's character feared that perhaps the infant had already, or was about to die from SIDS.

The sudden infant death syndrome (SIDS), sometimes referred to as cot or crib death, is something many new parents think about, and it is no laughing matter. In fact, in the second half of the twentieth century, perhaps no human malady has remained so successfully resistant to the challenges of so many scientific disciplines. For infants less than one year of age, in the United States alone, SIDS remains the major cause of non-accidental death. Between 5,000 and 7,000 infants a year (approximately 1.5 out of every 1,000 live births) die unexpectedly from this perplexing killer. And while this may not be a large number, the fact that it occurs at all leaves plenty of room for fear. Of course, for the parents and families of lost infants, it is a tragedy of monumental proportions.

As cruel as the unexpected and sudden loss of an infant is, the amount of psychological and emotional devastation parents experience is often made worse by the fact that health professionals are not able to explain why or from what disease their infants died. No one can say, for example, whether the problem was physiological, structural, infectious, metabolic, genetic, anatomical, or some combination of these.

In the face of no explanations, parents can be victimized twice: first by the death of their child and then by overly suspicious law enforcement officers looking for evidence of foul play. Furthermore, by continuing to probe for answers where there are none, parents of SIDS victims can be hurt deeply by insensitive family or friends who continue to speculate "if only" this or that was done for the infant, it may have survived. In the face of so many unknowns, the parents may begin to assume more and more guilt and to believe that there must have been something they could have done to save their child.

The truth is, however, that there is nothing presently known that parents can do which unfailingly can prevent such a death. What's even more frustrating and puzzling is that upon standardized postmortem analysis, SIDS leaves no consistent pathological markers that permit physicians to identify deficient structures or organ systems involved. SIDS is clinically diagnosed only when a thorough postmortem analysis of a sudden and unexpected death of an infant fails to find anything which can actually account for the death; hence it is a diagnosis by exclusion. More than 26 years of persistent, rigorous, and interdisciplinary research involving scientists from all over the world supported by millions of dollars in biomedical research grants have, in the words of one pathologist, "failed to discover even one positive criterion that the pathologist can use to recognize the subject at autopsy" (Valdes-Dapena 1980). It remains the most significant medical mystery of the 20th century. It is a death unaccounted for.

This monograph will review what is known about the SIDS puzzle and some of the factors that make it so difficult to solve. In addition to describing the inherent methodological problems that arise when no consistent clinical markers, either before or after the infant's death, can be identified, I will use my own eight years of experiences in this research area (having moved from biological anthropology, specifically primatology) to comment on the sociocultural factors that play a significant role in shaping and sometimes constraining the SIDS research paradigm. For example, using anthropological rather than biomedical perspectives, I suggest that infants are viewed by SIDS researchers as being much more physiologically independent and biologically more autonomous from the caregiver at the time of their deaths than they actually are. Furthermore, I will argue that this conceptualization of infants affects the way most biomedical researchers think about possible causes of SIDS, how they study infant sleep as it relates to SIDS, and how they come to define "normal" sleep patterns among infants, an issue which is important since so many physiological studies of SIDS use sleep data collected on normal, healthy infants. How SIDS researchers interpret the functional significance of abnormalities, or presumed deficits found during postmortem examinations of SIDS victims, is also based, I will suggest, on a conceptualization of infancy which reflects the Western European cultural experiences of the researcher more than it reflects knowledge of the infant's biology or evolution.

Like all of us, SIDS researchers bring to their work their own world view. Who infants are, what they are like, and what they need are questions not formally considered by SIDS researchers, but certainly how one answers these questions affects their research. For all of us, these ideas are shaped by our cultural values, experiences, ideologies, and education. I will suggest that concepts of infancy which inform the vast majority of SIDS research emerge from within our own highly individualized, urban-industrial culture where infants are encouraged, and thus thought to be, and consequently become, independent from their parents at the earliest possible age.

This world view which informs infant sleep research in general and SIDS research in particular is fundamentally flawed. It fails to consider the three to four million years of evolutionary history of parents and infants in which

human infants came to depend upon almost continuous contact, including co-sleeping, with a caregiver at least during the first few years of life. I will describe briefly research published elsewhere (see McKenna 1986, McKenna and Mosko 1990) which suggests that some of the many different subclasses of infants may be at increased risk for SIDS because they sleep alone in separate rooms away from parents and/or siblings devoid of the sensory cues and stimuli that were found in the environment within which infant sleep, breathing, and arousal patterns evolved. Later I will discuss how this can be researched, some results, and where the difficulties in proving this notion lie.

My own conceptualization of infancy certainly is not removed from bias, either. It, too, must be scrutinized carefully so that its theoretical and ideological bases are made clear and its weaknesses exposed. For example, in my critique of the biomedical research model for SIDS, I choose to emphasize cross-cultural, cross-species, and evolutionary data. My concern is less with what the laboratory reveals about infancy than with parenting and infancy as they evolved and developed in their natural ecological context and, hence, what might be considered a more species-wide or universal perspective. Ultimately, not one perspective but an integration of both should provide the basis for asking new questions and set the stage for new experimental and epidemiological studies of SIDS.

My own research described here is not aimed at discovering the primary causes of SIDS, i.e., what the actual central nervous system deficits are, but rather at delineating possible environmental factors which conspire with infantile deficits to increase the chances that those deficits will find expression in the form of SIDS. I do not propose any new "causes" of SIDS, or how to eradicate it. There are many potential "causes" of SIDS and, hence, most likely there will never be one strategy for preventing all SIDS deaths.

No matter how rigorous and methodologically sophisticated scientific research may be, it always begins with, and perhaps is only as good as, the accuracy of initial conceptualization and assumptions that underlie it. Sometimes these assumptions are made explicit by researchers but mostly they are not. It is common in scientific research for traditions to emerge concerning how a problem such as SIDS should be approached. Assumptions are passed on from one scientist to another. In the process of "passing the baton," especially as research on a topic builds, fundamental assumptions can assume their own orthodoxy and continue to slip by without ongoing critical review (Kuhn 1969). This may be especially true when the basic assumptions fit and support the prevailing cultural values of the society in which the research is being conducted. Unconsciously, like other people, scientists just "assume" certain things. Interdisciplinary communication can be useful here since "outsiders" are much more likely to ask the simplest question--either out of naivete or through a different kind of familiarity with, and knowledge of the subject. An "outsider" has less to lose by questioning the assumptions, held either consciously or unconsciously, in a different research field other than his or her own.

One cannot say at this point, of course, whether or not the new questions I raise concerning SIDS viewed from the vantage point of an

anthropologist will bear any fruit. My own work is in its beginning phase and its validity has yet to be proven. But at very least the contrast between the medical model for SIDS research described here and the anthropological one I propose makes it clear just how significant one's cultural position, experiences, education, or biases as a researcher are in determining the direction that one's own view of science takes. It is a fact of which all of us need constant reminding.

II. SIDS Epidemiology

a. When is it SIDS? Why is it so hard to study?

Epidemiology is the study of the incidence, distribution, and control of disease. This includes the study of factors that seem either to favor or inhibit the expression of disease within particular populations.

The epidemiology of SIDS is complicated by the fact that since SIDS cannot yet be limited to a range of possible causes, pathologists have a difficult time identifying it. Consider that even to standardize a criterion for diagnosing SIDS for local pathologists or coroners, a necessary first step in securing reliable frequency data, a single criterion is ordinarily required. In its absence much confusion arises not only as to what procedures pathologists should employ during postmortem exams, but also over what kinds of data and how much data justify a SIDS diagnosis. While in the United States, formal guidelines for autopsies of suspected SIDS cases exist, they are not always known by pathologists or, even when they are known, they are not necessarily followed. And even where standardized procedures are followed, so similar are some changes in the structures and tissues of the relevant organ systems to other possible causes of death such as accidental asphyxiation, there is not always agreement among pathologists as to whether a SIDS diagnosis is called for. Interpretations can be very subjective. In a major national epidemiological study of SIDS conducted recently by the National Institutes of Child Health, 243 out of 1,000 subjects in the study had to be thrown out because pathologists could not agree on the SIDS diagnosis; and these were all pathologists, keep in mind, who specialized in SIDS, and who were examining the very same infants (see Hoffman 1988).

Biologic changes in the infants' tissues and fluids (metabolic changes) that occur simultaneously with, or at various points in time after death, also make it difficult to know upon autopsy what characteristic is potentially a "cause" of death or simply the result of death. When abnormalities are found in the heart muscles, or in the capillaries of the lungs, or in the brain stem where breathing is controlled, given the functional interdependence of these systems, it is impossible for pathologists to know which may have caused pathological changes in the others--or if only one is the primary cause of death--or if the changes were gradual or sudden.

Moreover, when examining neurological tissue samples of SIDS victims, electromicroscopy permits SIDS researchers to "see" much more than

they ever have seen before; but, unfortunately, it is difficult to know how to interpret much of these data. It is especially hard to say what findings are functionally or clinically significant when the range of natural variation in brain cell morphology among healthy infants is not yet known. If there is no data base against which these more detailed views of cell tissues of the brain can be compared, it is difficult to distinguish true pathologies from harmless variations? What this means is that every unusual finding must be considered suspect when, of course, most probably mean nothing at all.

Another methodological and epidemiological handicap is the fact that SIDS is not known to occur in any species other than humans. Since there is no animal model of SIDS, there can be no experimental manipulations or comparative studies, though it is, of course, highly interesting that as a species human beings possess some constellation of biological characteristics that apparently predispose us toward this type of fatality. But an answer to the question as to why human infants and not other mammalian infants are susceptible to SIDS can only be speculative at this time (see McKenna 1986).

b. How do you "prove" a "cause" for SIDS?

The fact that SIDS researchers are coming to accept the fact that no one hypothesis can account for all SIDS deaths means that proving any one particular hypothesis will be all the more difficult. Among other things, it will be more difficult to prove a successful preventive strategy because "success" will not be measured by the elimination of SIDS but by some possibly small diminution of the overall SIDS rate in particular populations. If just a few babies each year are prevented from dying, I am sure that every researcher would claim 100% success, at least for these infants. But how correct must an explanation be (and the subsequent intervention aimed at preventing death) before it is to be accepted as a partial explanation for some forms of SIDS? And what happens if we discover that a potential preventive strategy for one infant is actually dangerous to some others? The fact that SIDS is still a relatively rare event means that, depending on the absolute number of different kinds of SIDS, a huge number of prospective studies of infants, i.e., studies which follow infants, including control infants, from birth, will be required before dramatic statistical evidence can be marshalled. And, of course, one sobering thought is that since infants come to a SIDS fatality from different physiological, structural, and/or genetic directions, it is highly likely that some infants will never be able to be saved from SIDS. Finally, given the fact that there may be more than one deficit or condition that must coalesce in concert with other deficits at a precise moment in the infant's life to create a SIDS event, being able to put all of these pieces together correctly amounts to a Herculean, if not impossible, task. SIDS can strike any infant, anywhere, anytime, and in any context. It occurs within all industrial cultures studied (though rates vary significantly--see below).

c. Age

Perhaps 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.

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 die from sudden infant death syndrome). Some studies support a death peak at about two months, 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).

d. Sex

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.

e. Geography, Ethnicity, and Seasonality

SIDS rates vary across cultures and geographic areas. 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 1980b:7, see Figure below). The rate for King County, Washington, is five times the rate in Sweden, whereas in Hong Kong and several cities in Japan, according to recent data, SIDS appears to be rare, possibly fifty to eighty times less than the frequency in Western countries (Davies 1985), the significance of which will be discussed later.

In all countries, though, 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) suggests.

f. Time of Day

In general, most of these deaths occur out of the sight of the caregiver, between midnight and 7:00 a.m. Valdes-Dapena (1980a&b) 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.

g. Socioeconomics and Age of Mother

Several studies show convincingly that death rates for SIDS are higher among the poor. But rates are higher among blacks, even when socioeconomic status is held constant (Valdes-Dapena 1978, 1980b; Beal 1983; Froggatt 1983; Krauss 1984; Peterson 1983), and are the lowest among Asians (Davies 1985) and Swedes (Norvenius 1984).

Age of mothers when they first give birth may account for much of the variance between blacks and whites, however; 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 who smoke 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. In any event, it appears that, for all women, SIDS may be one of the few infant-related reproductive syndromes that gives older mothers an advantage over younger ones.

h. Characteristics of those at risk

The most recent, best controlled and designed retrospective epidemiological study (Hoffman et al. 1988) reported on 757 SIDS victims and 1600 control infants divided into groups according to age, race, and birth weight. The samples were drawn from six SIDS research centers scattered throughout the United States. It revealed that about 90% of SIDS victims were less than 24 weeks old. Most SIDS victims generally had low birth weights (less than 2500 g), experienced slower overall (postnatal) growth rates than controls, and were more frequently born to unmarried and poor women who smoked during their pregnancies and who were less than 20 years of age. Interestingly, socioeconomic and behavioral factors, rather than maternal medical or health factors, were more significant predictors of SIDS risks when only maternal risk factors were analyzed (Hoffman et al. 1988). Postnatal risk factors of a small proportion of SIDS victims included mothers' reports that prior to their deaths their infants experienced a "stop breathing episode" or turned blue. Most frequently these apnea episodes occurred when the infant was awake. Mixed apneas continued to be associated with some infants before their deaths from SIDS, but more frequently apneas were markers for low-birth-weight infants. Apnea proved not to be a specific risk factor for SIDS.

One of the important finds from this study is the "lack of breast-feeding" as a risk factor for SIDS. Those babies who were breastfed had less of a chance of dying from SIDS, at least in this study.

A significant number of infants who died of SIDS had bouts with diarrhea and/or vomiting and colds within 2 weeks of death. They also experienced droopiness and listlessness during the last 24 hours, as well as

increased irritability, respiratory distress, and tachycardia (excessively rapid heartbeats) (Hoffman et al. 1988). According to the investigators, all of these factors, however, acted in "secondary fashion" rather than as primary or causal agents.

In another recent study of the behavioral risk factors associated with sudden infant death, it was found that among the 24 victims included in the sample the behavior with the greatest predictive probability was the degree of difficulty these infants had in awakening (Einspieler et al. 1988). Similarly, Hoppenbrouwers et al. (1989:269) compared the sleep patterns of subsequent siblings of SIDS victims with those of members of a control group. Although they found that similarities between the two study groups "outweighed" the differences, the siblings of SIDS victims tended to awaken less frequently and, once asleep, "exhibited a higher probability of remaining asleep than controls." This suggests strongly that infants susceptible to SIDS may suffer from arousal deficiencies. For example, while short breathing pauses and short apneas may be quite normal for infants, those infants who have difficulty arousing to end an apnea, i.e., to take a breath, may be one group of infants who are at increased risk for SIDS, as discussed later.

i. Does SIDS run in families?

Another important question is the occurrence of SIDS among family members, that is, its genetic connection. Peterson (1983:94) 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 child 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).

j. What can be said?

In sum, epidemiological, experimental, and clinical SIDS research continues to suggest that: 1) SIDS has multiple etiologies (origins) and is multifactorial (for any one "cause" there may be more than one factor acting simultaneously to increase risk); 2) SIDS victims represent an extremely heterogeneous population; 3) infants who die from SIDS are not as "healthy" as both physicians and parents once presumed; and, 4) the characteristics that predispose an infant to SIDS most likely develop during intrauterine life.

III. SIDS Sleep Research: A Brief Review and Critique

"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 most recent definition of SIDS is "the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and review of the clinical history" (National Institutes of Health 1991). The most "compelling" general hypothesis proposed to explain the pathophysiology of SIDS is that the infant who dies suddenly has one or more abnormalities in the cardio-respiratory control system which may have their origin in fetal life; infants die of primary cardiac or respiratory failure (National Institutes of Health 1991). What may actually initiate the sequence of events leading to death are, once again, unknown, but a popular current theory is that at least some SIDS victims are unable to terminate a breathing pause or apnea. Some infants may be unable to arouse or awaken themselves from a breathing pause or apnea to breathe. If this is true, the deficiency may lie in the brain stems reticular formation which regulates the interactions between arousal, heart rhythmicity, and breathing patterns, as discussed below. (See Figure 1.)

Table 1.

SIDS Time Line of Influential Research Findings and Events
1963 to 1991

(Note: This chart is not to be read linearly, i.e., one finding does not necessarily supersede or replace those that precede it. Some of these findings below, while important at the time, are no longer thought to be viable explanations of possible SIDS factors.)

1963: SIDS formally defined and recognized as a distinct medical entity: "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."

1963: Presence of petechiae (small point hemorrhages or broken blood vessels on lungs possibly indicating breathing struggles) and heart abnormalities.

1972: Prolonged and frequent breathing pauses and apneas, leading to low oxygen pressure (hypoxemia) injuring brain and/or heart.

1973: Cardiac abnormalities (enlarged right ventricle); also prolonged interval between ventricular contraction and relaxation; lethal arrhythmias; thickened arterioles in lungs indicating possible deficient respiratory drive; hypermobility of mandible causing suffocation.

1974: Impaired glucogenesis; abnormal homeostasis (central nervous system control).

1976: Abnormal amount of fat found on brainstem nucleii; undervascularized brainstem.

1977: Anaphalactic shock--allergies to cows milk; deficiency of chemoreceptors which monitor $C0_2$ and 0_2 levels; arousal deficiency.

1978: Lesions on brain cells caused by inadequate supply of oxygen to the brainstem (ischemia).

1979: Deficiency of arousal to lower $C0_2$ levels.

1980: Vitamin B deficiency; thiamin deficiency.

1982: Risk from DPT vaccine.

1983: Immature brainstem (dendrites); infant botulism; inability to reinitiate breathing after an apnea.

1984: Increased level of dopamine (a neurotransmitter).

1986: Accidental suffocation (overlying).

1987: Elevated levels of fetal hemoglobin.

1988: Lower than normal variations in heart rates; breastfeeding found to reduce risk; problems awakening; deficiency in arousal mechanisms and maturational synchrony between sleep mechanisms and arousal mechanisms.

1990: Overheating infant's body (too many blankets).

1991: New SIDS definition released by National Institutes of Health: "The sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and review of the clinical history."

It is beyond the scope of this monograph to review all of the important diverse lines of SIDS research described in Table 1 (see Guntheroth 1990, McKenna 1986, and Tildon et al 1984 for comprehensive reviews). I would like to concentrate here only on neurological studies of normal and high risk infant sleep and breathing patterns, and possible brain stem abnormalities suspected to be relevant to some cases of SIDS. This subfield of research is the most relevant to my critique of the SIDS research paradigm as well as to my own anthropological research into the natural ecology of human mother-infant co-sleeping. My own research perspective on infant sleep patterns and SIDS contrasts with the more traditional ones insofar as I assume that social or parent-infant co-sleeping is the "normal" infant sleep environment for human infants and that solitary infant sleep represents a recent biological and social deviation with potentially negative consequences for the infant.

a. Breathing during sleep: The possible inability of the infant to arouse to breathe

A SIDS event has never been witnessed from start to finish; therefore, it is not actually known whether infants are actually asleep when they die from SIDS, but it is strongly assumed that they are. This is an important statement and the general belief that infants die in their sleep from SIDS accounts for the thousands of studies conducted on healthy and unhealthy infants in sleep labs as their heart rate patterns as measured by electrical spikes set on a screen and recorded by pen on polygraphic paper (EKG, or electrocardiogram), brain waves (EEG, electroencephalogram), and breathing, to name but a few measures, are observed and recorded polygraphically for comparative analyses.

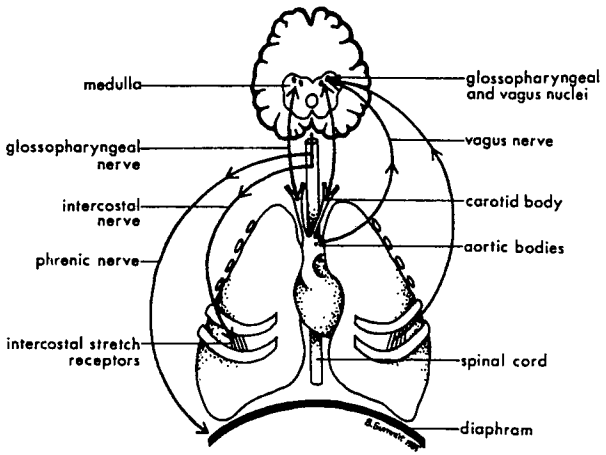


Fig. 1 Neurological and respiratory control structures most frequently studied by SIDS researchers.

With this in mind, much of the research into possible structural-functional abnormalities in infants 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 clusters of cells are thought to be a primary center of breathing control, although the question of a central control is still controversial (see Figure 1). 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 in the lungs and from at least two important sets of chemoreceptors, the carotid and aortic bodies. These are gland-like structures which tell the brain if too much carbon dioxide is in the blood. If so, blowing it out and bringing in fresh oxygen is necessary. The brain signals the phrenic nerve to drive the diaphragm which causes the lungs to expire (get rid of the CO_2) and inspire (bring fresh oxygen into the body).

The reticular formation nuclei also direct the respiratory muscles surrounding the lungs as well as the airway passages of the neck and throat. Other muscles of the chest cavity, such as the intercostal muscles surrounding the ribs, are also involved in respiratory behavior and are partially controlled by reticular formation nuclei.

The system is, of course, much more complicated that this. The reticular formation also acts as a major conduit between 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.* All together, the reticular activating system coordinates breathing with cardiac output, sleep-wake cycles, and arousal (Kahlia 1980; Plum and Leigh 1981; Mitchell and Berger 1981; Darwish and MacMillan 1984).

That deficits in the brainstem reticular formation or adjacent structures could be the cause of some forms of apnea (long intervals between breaths) 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 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, apnea can lead to low oxygen pressure in the blood

(referred to as *hypoxia* or, if chronic, *hypoxemia)* and high carbon dioxide (CO_2) levels (*hypercapnea*). If severe enough, apnea will lead to a cyanotic or oxygen deprived 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 hopes of finding differences in sleep and breathing abnormalities that could, in turn, be linked to SIDS. If the '70s could be described as the decade in which apneas, caused by some deficiency in the brainstem that also negatively affected the infant's heart, was thought to be the cause of SIDS, the '80s and '90s could be described as the decades in which arousal deficiencies and not apneas were thought to be linked to SIDS.

Although the original hypothesis of an apnea-SIDS relationship has been supported in some ways 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 argue now that apnea not necessarily precede SIDS, as only an estimated 5 percent of SIDS victims had apneic attacks before they died. The most recent epidemiological study supports this view (see Hoffman et al. 1988).

The infant's inability to reinstate or reinitiate breathing once an apneic episode has occurred may be the principal defect, not apnea itself. Researchers 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. 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 CO_2 (hypercapnea) (remember that when there is too much carbon dioxide in the blood, the chemoreceptors induce expiration). Yet, others have not been able to find evidence of diminished responsivity, to either too little oxygen or too much carbon dioxide. Sullivan (1984) proposed that infants have a natural capacity to smell CO_2 and that perhaps the CO_2 chemoreceptors in the upper nasal passages do not alert the brainstem to remove the carbon dioxide by breathing (McKenna 1986).

Investigations of the influence of the reticular system 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 controls, the siblings of SIDS victims (considered by some to be a high- risk group for SIDS) displayed abnormal and unusual sequences of quiet, or NREM, sleep. Recall that NREM (non-rapid eye movement), or quiet sleep, is characterized by regular respiration and slow wave encephalographic EEG pattern, whereas active, REM (rapid eye movement) 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, SIDS victims may have difficulties not only in waking up but also in waking up to breathe as

"normal" or surviving apneic infants might. It may be significant that the chance of an infant dying of SIDS peaks at a time when infants are reorganizing their sleep-wake cycles and moving away from those sleep states dominated by REM (rapid eye movement) or active sleep to NREM sleep (stages 1-2, 3-4), which may make them more vulnerable to breathing control errors.

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 brainstem 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. 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 spineless nerve connections between 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 ability of the nerves to signal the brain that the next breathing cycle (inhalation then exhalation) is needed, especially after the baby has experienced a long pause since the last breath.

Becker and colleagues (1983) suggest that some added minor stress, such as a cold, may compound the infant's vulnerability to respiratory collapse, as it does not yet have completely efficient control over its breathing at this age. 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.

b. Possible cardiac abnormalities

Heart 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, where each heart beat is shown as a spike on the paper), a concept 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) observed that ventricular fibrillation or rapid incomplete contractions, could occur, quietly and instantly causing death. 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, the heart's instability. Once again, however, other researchers do 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 entirely satisfactory.

IV. So, What's the Problem? An Anthropological View of SIDS Research Assumptions

All of the studies described in Table 1 and above are scientifically important and make it possible for other researchers, including myself, to move forward making new interconnections. My criticisms of SIDS research, then, have more to do with some of the underlying assumptions these researchers make about infants, for the possibility of missed alternative explanations of their results, and for research that isn't done, rather than with technical aspects of the work itself.

a. Infants are not "mini-adults."

Consider, for example, that SIDS researchers 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 with whom its body was designed to sleep 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 neurologically immature 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 caregiver at the time of death and, thus, that the seriousness of such defects may be depend on the extent to which sensory stimuli, either present or absent in the infant's microenvironment, inhibits or exacerbates 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 (McKenna 1986).

The evolutionary approach suggests that in order to understand how an infant's developmental or physiological systems can go awry, we must first consider whether or not the developmental setting of the infant, which includes the nature of its physical and social relationships with its caregivers, is the same as what it was during the millions of years of its evolution at which time natural selection produced an adaptive fit between the special needs of infants and parental responses to these needs, maximizing the chances of infant survival. The evolutionary approach forces us to consider the differences between the socioecological and developmental context into which the infant expects to be born--given its evolutionary past--and the conditions provided. A "mismatch" for infants may significantly affect the efficiency by which developing breathing, arousal, and sleep patterns are expressed. The evolutionary record and archaeological record of human evolution and, indeed, the historical record all indicate that until very recently, say 200 years ago, the human infant's body was

almost always in continuous physical contact with a caregiver. This contact, is it argued, is necessary to help compensate for the infant's lack of maturity at birth.

b. Where's the beef? A re-conceptualization of infancy using cross-species, paleoanthropological, and cross-cultural data collected by anthropologists.

These contentions, and my criticisms in general, are supported by three lines of anthropological research: 1) cross-species studies of nonhuman primates which demonstrate dramatic short-term physiological consequences of separation from contact with a caregiver; 2) the fossil record of human evolution that shows, for at least four million years, the birth of relatively helpless and slow developing infants in need of continuous contact and carrying (including co-sleeping); and 3) cross-cultural data indicating that, with the exception of urban-industrial, western European societies, all people around the world sleep either in direct physical contact or in close proximity with their infants throughout the first few years of life. The data and most of the generalizations presented below represent a summary of research conducted by anthropologists. Although the application of this research to the problem of SIDS is my own, the points of information can be found in any introductory text to biological, cultural, or archaeological anthropology (see Campbell 1986, for example).

c. What the monkeys tell us.

During the last fifteen years, several laboratories around the country have demonstrated that, in addition to suffering devastating, long-term psychosocial effects of separation from their mothers and siblings at birth, infant primates face other serious challenges when their primary caregivers are suddenly removed. If, as psychobiologist Myron Hofer states, "neural traffic" in the form of sensory exchange with the caregiver, is abruptly terminated, the animal's own physiological system must adjust to an entirely new (novel) situation. For example, experimenters have found that, upon separation from attachment figures, monkey infants experience a loss of body temperature, the release of stress hormones, cardiac arrhythmias, and sleep disturbances, as well as immunological compromises (a loss of antibodies) -- altogether changes in physiological systems which were not thought to be significantly affected by the presence or absence of contact with another (see Table 2).

Compared with monkeys and apes, human infants are more vulnerable and neurologically more immature at birth. Hence, there is good reason to think that forms of parental contact, or the lack of it, will have equal, if not more, physiological consequences. Could nocturnal separation during sleep deprive human infants of sensory cues that promote breathing stability? The general hypothesis, as discussed below, proposes that perhaps, for some subclass of SIDS victims, solitary sleep or changes in the infant's body induced by sensory isolation from the parents, conspires with infantile deficits to increase risk.

This hypothesis seems more viable when we consider that human infants are born with only 25 percent of their total adult brain weight; our

closest living relative, the chimpanzee, is born with at least 45 percent. Not only are humans the least developed primate at birth, they are dependent for a longer period of time. Monkeys and apes are born with muscles capable of clinging to their mother's ventrum; but human infants need to be carried for months owing to delayed locomotor development (see McKenna 1986). It almost goes without saying that all monkey and ape parents sleep with their young (Anderson 1984). Surely, this is an evolutionarily stable pattern rooted not only in our primate, but in our mammalian, past. Could it be that sensory cues, such as touching, movement, hearing breathing sounds, exchanging carbon dioxide and/or smells, and the temperature exchanges which occur while huddling, somehow affect physiological processes during sleep? The monkey and ape data suggest that this possibility is worth serious consideration.

d. What the fossils tell us: Upright posture, fetal encephalization, and the birth of neurologically immature human infants.

The fossil record on the origin and evolution of human upright posture (bipedalism) is another important line of evidence which, at first glance, would seem to have very little to do with the biological needs of contemporary human infants, or whether parent-infant co-sleeping is beneficial and can possibly help some infants to resist a SIDS event. Yet, as our hominid ancestors moved gradually from the trees to the ground in Africa some 3 to 4 million years ago, important anatomical changes in the human pelvis necessary for upright walking occurred which placed an upper limit on the size of the fetus-infant's head at birth. Not only, then, did bipedalism free the hands for carrying objects and making and using tools but, in combination with another hominid trait -- larger brains -- it transformed the developmental status of human infants at birth. That is, in order to be born safely, hominid-human infants had to be born in an extremely immature or "altricial" condition -- a physiological condition wherein physical contact began to significantly influence the infant's body temperature, heart and growth rates, sleep and arousal patterns, and other fundamental physiological systems such as the immune system (see McKenna 1986, 1990 for extensive review).

Table 2.

Immediate and Short-Term Consequences of Parent-Infant Separation in Monkeys and Rats

(Medical Anthropology, Vol. 10, 1986, J. McKenna)

Physiological Consequences of Separation	Investigator(s)
Bonnet or pigtail monkeys	
Initial period: increase in heart rate and body temperature	Reite and Snyder 1982
Subsequent period of depressed behavior: decrease in heart rate and body temperature	Reite et al. 1978a, 1978b
Increase in cardiac arrhythmias	Seiler et al. 1979
Alterations in heart rate, body temperature, and circadian rhythms	Reite et al. 1982
Disturbances in sleep: increased arousals; increase in REM latency; decrease in time in REM	Reite and Short 1978
Changes in regulation of EEG activity	Short et al. 1977 Reite et al. 1982
Alterations in cellular immune response accompanying mother-infant or peer separation	Reite et al. 1981 Laudenslager et al. 1982 Other references: McKenna 1979, 1982 Reite and Capitanio 1985 Coe et al. 1985 Hofer 1981, 1978, 1983
Squirrel monkey (Saimiri sciureus)	
Increase in adrenal secretion and plasma cortisol levels	Coe and Levine 1981 Coe et al. 1978, 1985
Serum levels of immunoglobulines: decline after 7 days; back to normal in 14 days	Coe et al. 1985
Complement proteins to cortisol diminish	Coe et al. 1985
Lower level of antibody production in response to bacteria (*Escherichia coli*)	Coe et al. 1985
Rats, 2 weeks old	
Bradycardia Increased sleep latency Augmented sleep Decrease in REM sleep	Hofer 1981, 1978, 1983
Rats, 10 days old	
Fifty percent reduction in brain and heart enzyme (ornithine decarboxylase) due to separation-induced suppression of growth hormone	Butler et al. 1978 Kuhn et al. 1978

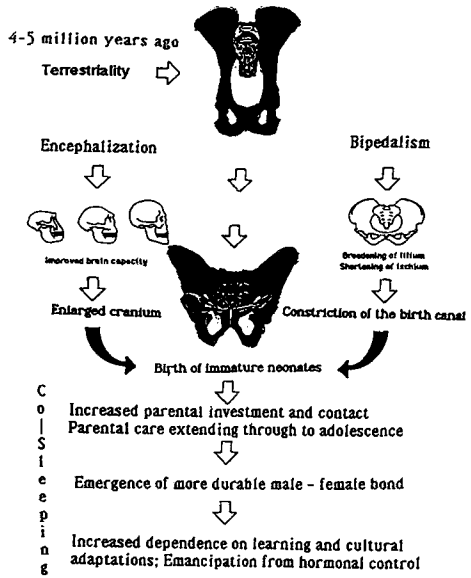


Fig. 2 The relationship between upright (bipedal) posture and the birth of neurologically immature infants in need of intensive contact and care including co-sleeping. As our ancestors descended from the trees some four to five million years ago and began to exploit a terrestrial (ground-based) African niche, the pelvis changed in ways that prevented easy deliveries. An increase in brain size (encephalization) including fetal brain size relative to body size meant more complex social behavior including tool-using and making and a general emphasis on learning. Bigger brained fetuses made birth even more difficult and dangerous. The solution to this evolutionary dilemma was, and continues to be, the birth of neurologically immature infants who are born with only 25% of adult brain volume. Immediate and sustained parental contact and care including co-sleeping was, of course, required to maximize the chances of infant survival. Co-sleeping is very old, indeed.

It is strange to think that our locomotor system forced a change toward an unprecedented amount of parental contact and care that far exceeds the amount given by other mammal parents to their young, but such is the case. Consider the manner in which locomotor anatomy affects primate obstetrics -- specifically how easy or difficult it is for an infant to be born. Compared with the quadrupedal monkeys and apes who locomote using all four limbs, an upright stride requires that the ilium or hip bones broaden and rotate forward so that our abdominal muscles can attach to the tops of them, structurally supporting our internal organs as we stand. At the same time the ischium, the bone at the floor of the pelvis, must flatten out a bit and push upward to accommodate the hip-femur socket which connects the legs. This also permits the smaller gluteal muscles to stabilize the hips while walking.

The overall result of these changes is that for both men and women the size of the pelvic opening diminishes making the human birth process much more difficult than it is among the quadrupedal monkeys and apes whose pelvises are longer than they are wide. Labor is much shorter among monkeys and apes and birth-related (obstetric) infant mortalities are lower than they are among humans -- at least for those of us who give birth without the possibility of medical intervention if it proves necessary.

But here is the real problem. Not only were pelvic openings being re-shaped making them smaller within evolving human lineages to accommodate bipedalism, but the adult brain sizes were simultaneously becoming substantially larger. In fact, when measured in terms of brain weight relative to overall body size, except for dolphins and elephants, human beings have the largest cranial capacities of all the mammals. The paleoanthropological (fossil) evidence supporting this fact includes hundreds of hominid skulls, skull caps, and skull fragments from all parts of Africa, as well as hundreds, if not thousands, of vertebral columns and pelvises (see Fleagle 1988 for review).

The oldest bipedal hominid fossil found by Johansson and White at Hadar, Ethiopia, dates to 3.6 million years ago. It was astonishing to paleontologists (a group known to become ecstatic by finding just a small fragment of a bone) that over 40% of this ancient skeleton was preserved and successfully excavated. "Lucy" (*Australopithecus afarensis*) as she came to be called, definitely walked upright; but in many ways she surprised anthropologists because she resembled contemporary apes more so than she looked like the more recent and advanced African hominids or, later, modern humans. For example, her 360 cubic centimeter brain was no larger than that of a contemporary chimp or gorilla (modern humans have a brain volume measuring approximately 1400 to 1500 cubic centimeters). Moreover, Lucy's long arms and dental-facial characteristics seemed more ape-like than human.

But discoveries of more recent hominids from South and East Africa, especially those discovered by the Leakey family at Olduvai Gorge in Tanzania, prove that by two million years ago the newer part of the mammalian brain, the neocortex, began to expand quite considerably. In fact, between 3.6 and .5 to 1.5 million years ago, the human brain almost tripled in volume, reaching between 1000 and 1250 cc's.

The neocortex consists of the two cerebral hemispheres on each side of the brain -- they represent parts of the brain that permit language and culture. Anthropologists suggest that during this time period natural selection began to emphasize learned behavioral adaptations, including the abilities to manipulate the environment through technology (tool-using and making) and increasingly complex sociality, rather than adaptations achieved primarily through instinctual, or more genetically-based behaviors. In other words, at or around 1 to 2 million years ago, which witnesses the rise of our own genus, Homo, (specifically Homo erectus) in Africa and Europe (see Foley 1987), natural selection favored two seemingly conflicting evolutionary trends: increasing encephalization (brain size) on the one hand, and on the other continued structural refinements with further anatomical accommodations to bipedalism effectively diminishing the size of the birth canal.

The adaptive compromise to this evolutionary dilemma was, and continues to be, the birth of exceedingly neurologically immature infants for whom 75% of their brain growth will occur post-natally (after birth) and not in the womb. To illustrate, paleoanthropologist Chris Stringer causes mothers to wince when he points out that in order for human fetuses to be as neurologically developed at birth as either a monkey or ape infant, it would have to be nourished in the womb for up to three or four additional months -- not exactly a happy proposition for mothers. However, if vulnerable infants are not being housed and nourished in the womb, but develop outside of it, someone has to care for them. Hence, in order to maximize the chances of an infant surviving, significant time and energy must be devoted to infant-child care by mothers, fathers, and the groups to which they all belong. Increased parental contact, including co-sleeping, carrying, feeding and general bodily care (increased parental investment) is necessary as infants quite literally finish their biological and social gestation. Interestingly, dental eruption patterns found on 1 million year old fossil children show that, like modern children, molars were erupting at between 8 and 12 years of age -- a sure signal that at this early point in evolution maturation was not much faster than it is today (Fleagle 1988). Taking care of newborns as they "extero-gestate," a word used by anthropologist Wenda Trevathan, and then caring for slow-maturing children, does not come cheaply -- neither then nor now.

In sum, around two million years ago, along with bipedalism, our hominid ancestors evolved significantly larger brains. Bipedalism had the effect of reshaping the pelvis in ways that made the birth of larger brained babies difficult and dangerous. The adaptive response was to give birth before fetal brains became too large, threatening mother-infant survival, i.e., to hold off the majority of brain growth until after birth. For these reasons, anthropologists argue that intense and prolonged parental contact and care toward infants, including parent-infant co-sleeping, had to evolve early in our evolution. In fact, not only did evolving hominid and later human primates exhibit the natural mammalian tendency for prolonged care of the young, including co-sleeping, but the fossil and archaeological record indicates that hominid adaptations built on, and substantially extended this care as infants were born increasingly less mature and took longer and longer amounts time to develop (Lancaster and Lancaster

1982). Human parents are the only primates, indeed the only mammals, to extend care of offspring through to and including the juvenile period (Lancaster and Lancaster 1982).

It is clear, then, that for at least two million years human infant nocturnal sleep patterns co-evolved alongside sensory stimulation provided through contact with the caregiver. Since co-sleeping behavior per se does not, of course, fossilize, we must reconstruct what actually happened between parents and infants with a certain amount of caution. But through "best guess" inferences and reference to both contemporary monkey and ape behavior and cross-cultural data (see below), it is clear that there could not have been any alternative to the very ancient pattern of co-sleeping with infants. Thus, parent-infant co-sleeping must be considered the evolutionary stable and the evolutionarily "expectable" pattern of contemporary human infant sleep. It has both a long history and prehistory.

e. What the cross-cultural (ethnographic) data tell us

Relationships between environments, anatomy, and behavior of contemporary monkeys, apes, and people help anthropologists reconstruct models of the possible lifestyles and adaptations of our fossil ancestors, as discussed above. But another critical line of evidence for these human evolutionary reconstructions is how noninudstrial, non-urban peoples, indeed most people in the world live today. It's just too easy to assume that what you or I, or people in our own society, do or think (in this case about infants) is what everyone else in the world does or thinks. But nothing could be further from reality. In fact, this way of thinking is ethnocentric. Especially studies of non-industrial hunting and gathering societies such as the Kung Bushmen of the Kalahari in South Africa (described by Shostak 1981) are extremely useful since, for 99% of our existence as a species, *all* human beings lived as these modern hunter/gatherer/collectors do--in small bands of related peoples using a simple technology. The hunting and gathering lifestyle is the context within which contemporary human needs and behavior evolved and most likely, in some ways, the lifestyle to which human beings are best adapted.

There is another reason why the cross-cultural data are so important, especially for understanding how infants "expect" to sleep, given their evolutionary past. It is far too easy for us as members of a particular culture to assume that the way we think about infant sleep is exactly how everyone else in the world thinks, and where we place infants for sleep is the same place where other people do. But this ethnocentric way of thinking is false. Compared with the rest of the world, it is clear that American parents, and western Europeans in general, provide their infants with a unique, if not novel, sleeping experience. Infants either sleep alone or are encouraged to do so, even when they communicate their unwillingness to do so. Clinical psychiatrists label an infant's biological or emotional inability to conform to our own sleep standards as an "infant sleep pathology." If popular culture and our own personal experiences are to be trusted, it is clear that as soon as possible infants are placed in their own sleeping quarters and, of course, they are encouraged to sleep as often and as much as they can--the "Shhh, the baby is sleeping" or the "Shhh,

you'll wake the baby approach." These approaches apparently do not accept the notion that, if they need to sleep, infants will do so in the middle of a rock concert. When an infant begins to sleep through the night there is cause for celebration. Quite clearly, the "good" baby is the one who sleeps the most and is the quietest at the earliest possible age -- the very characteristics that are associated with some SIDS victims. Not-so-good infants obviously are those who, for whatever reason, cannot or will not conform to the ideological construction of "normal infant sleep" that we in industrial America have constructed for them. The truth is that the ones who protest the most may well be those who are best adapted. These are infants who are capable enough to determine the environment in which they will profit the most.

The cross-cultural data are invaluable because they remind us that, insofar as evolution is concerned, industrial societies such as our own are "new" and that caregiving practices such as separate parent-infant sleeping reflect the immediate values and social needs of their members -- and not some "natural" evolutionarily old pattern. These practices do not necessarily reflect infant needs; in fact, they may be, as suggested here, in conflict with them. Infant sleep management clearly reflects the perceived social best interest of the parents rather than the biological best interest of the infant.

Urban industrial culture has a tendency to obscure, for example, the close biological connectedness that infants have with their caregivers -- the physical nature of the relationship is rather hidden from view by clothing, baby bottles, decorated cribs, blankets, and something we call etiquette. Nobody necessarily is to blame for this, for surely the biological relationship between an infant and its mother living in the United States is not as obvious as it is in cultures where it is normal to see a mother's nipples accessible and exposed as her infant rides skin-to-skin, tied by a shawl around her torso. In our society overt reminders of this biological connectedness are considered by some to be rude or disgusting as, for example, when mothers nurse their infants in public and are criticized for doing so. Indeed, when one observes parent-infant interactions in non-industrial cultures, the "physicality" especially of the mother-infant relationship becomes patently clear and an objective and honest reminder as to how things were for all people everywhere up until very recent historic periods with the industrial revolution.

f. Where and how infants sleep around the world

The Human Relations Area File (HRAF) is a compendium of ethnographic data which includes, among other things, information on sleeping arrangements of societies around the world. Anthropologists use this database to make ethnographic or cross-cultural comparisons in subjects ranging from forms of witchcraft or marriage customs to how people marry, get circumcised, or make a living. There are literally thousands of coded pieces of data on almost every culture in the world that has been written about. Barry and Paxton (1971) surveyed 90 of these societies for which there were sleep data and found that in 71 of them infants slept in the same bed or floor mat with their parents; only in the United States and Europe were babies found sleeping in separate rooms. In my own random survey of 27 cultures from the HRAF, all were found to

maintain some form of close physical contact with their infants throughout the night (see McKenna 1986:49-50) (see Table 3).

Table 3.

Where Infants Sleep and With Whom:
A Cross-Cultural Survey

Culture	Where Do They Sleep?	Upon What Do They Sleep?	Comments
Bemba	Until 2 or 3 years with parents	Wood beds	
Santal	Until 13, with parents	String beds (khatias)	
Tarahumara	Infants & children with parents	Summer, open canvas winter, wood beds	Sleep in clothes
Ngoni	Infants with parents, children	Finely woven reed mats	In huts; older children with grandparents
Nahane	In skin hammock hung within mother's reach	Animal pelts and/or dry grass	Cabins or tents
Pukapuka	Infants & young children between parents	Floor; mats of coconut leaf or pandamas on coral pebbles	
Klamath	All children with parents	Twined or sewn mats or pelts; pillows of grass	
Ifugao	Until 3 or 4, with parents; then in same-sex groups	Raised wool	
Papago	Infants with mothers, young siblings	Mats of cactus fiber	Men in Big House; pubescent girls with grandmothers
Semang	Infants in same hut with parents	Mattress of split bamboo	Same-sex groups at puberty; separate quarters
Tlingit	Infant with parents		Sleeping sections in single structure
Gond	Infant with mother in narma-lon	Mats	Boys sleep on aghali
Yurok	Infant with parents	Floor of hut	Men & boys in sweathouse
Toradja	Infant in crib, in same room as mother & inlaws	Mats on floor, with pillows	
Ganda	Infant with parents	Bed, with bark cloth	Teenagers and single adults in same-sex groups

Table 3. (Continued)

Culture	Where Do They Sleep?	Upon What Do They Sleep?	Comments
Flores	Infant with parents	Bamboo sleeping bench	Children with friends in samesex group
Lau	Infant with owner & wife	Pile of mats; Tongan tapa as covers	Boys move to other huts at 7
Maori	Families sleep together; several to a hut	Special sleeping huts for summer and winter	
Bushmen	Infant with parents	Scooped-out area in	At 7 girls sleep with grand-parent
Yap	Infant with parents until 2 or 3, sometimes in hanging basket		At 5 child moves from parent's room
Garo	Infant with parents	Mats from bark of trees	Bachelor quarters
Katab	Mothers with infants, husband, siblings	Mattress of rags	Bachelor and maiden co-sleep dorms
Mauri	Infant with parents	Sleeping mat, Kathari mattress of rags	
Tiwi	Children with parents	Mats of paper bark	
Cuna	Infant with mother in same hammock; father in same room	Handmats--cotton of ciba tree covered with plantain leaves	
Tzeltal	Youngest infants on parents' mats		At 5 sleep with same-sex sibling
Bhil	Infant with mother		

It is not simply a question of where infants sleep, either, but how their bodies respond physiologically as they sleep in social vs. solitary environments that obviously has some implications. Consider this possibility: Without sensory interruptions from parents, solitary sleeping infants learn to sleep for longer periods of time during single sleep bouts than infants who sleep socially. If the infant's physiology enables it to sleep efficiently, but does not yet enable it to arouse efficiently, these long uninterrupted sleep bouts could prove dangerous. There is some data suggesting that when infants sleep next to, and have access to, their mother's nipples throughout the night, they wake up frequently to nurse (see Konner and Worthman 1980, cited in McKenna 1986). Anthropologists Charles Super and Sarah Harkness monitored the sleep patterns of co-sleeping Kipsigis infants in the Kenyan Highlands and found major differences between these social sleeping infants and middle class American infants. They found that 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) study and Konner and Worthman's (1980) study of San Kung Bushmen infants of the Kalahari in sub-Saharan Africa support these findings, as do the studies by Elias, Nicolson and Konner (1987), who studied La Leche League women in the United States who sleep with their infants. These works suggest that when infants are in contact with another person throughout the night, physiological mechanisms work against their sleeping for prolonged and uninterrupted periods of time at young ages before biologically they may be best able to handle such sleep. At very least, it appears that infants who regularly sleep with parents do not develop sleep patterns which are comparable to solitary sleeping infants.

V. The Ideology of Separate Parent-Infant Sleep

a. Autonomy or interdependence?

If, as I claim, the ideology justifying separate sleeping arrangements for parents and infants also constrains biomedical research on infant sleep and investigations into SIDS, it deserves some further attention. Interestingly, the practice itself is probably no more than a couple of hundred years old, at best, and is mostly a western European phenomenon which began in urban and industrial settings. The more extreme negative attitudes about co-sleeping found presently in American culture cannot be separated from our inherent concern and advocation of individualism, autonomy and independence as compared with caregiving practices which promote dependence, group harmony or interdependence.

Child psychologist Jerome Kagan suggests that early socialization practices that promote autonomy and individual rather than group effort and responsibility lead many adolescents to conclude that their future social and material success depends on their personal abilities and maturation. Kagan (1984) suggests that from the time the child is but a few months old most parents try to instill an "independent frame of mind" in their children -- a

seemingly necessary prerequisite for becoming a happy, successful adult in urban western society. In his best selling *Baby and Child Care*, pediatrician Benjamin Spock advises parents that children can sleep in their own rooms from the time they are brought home from the hospital and he urges parents to make sure that they do.

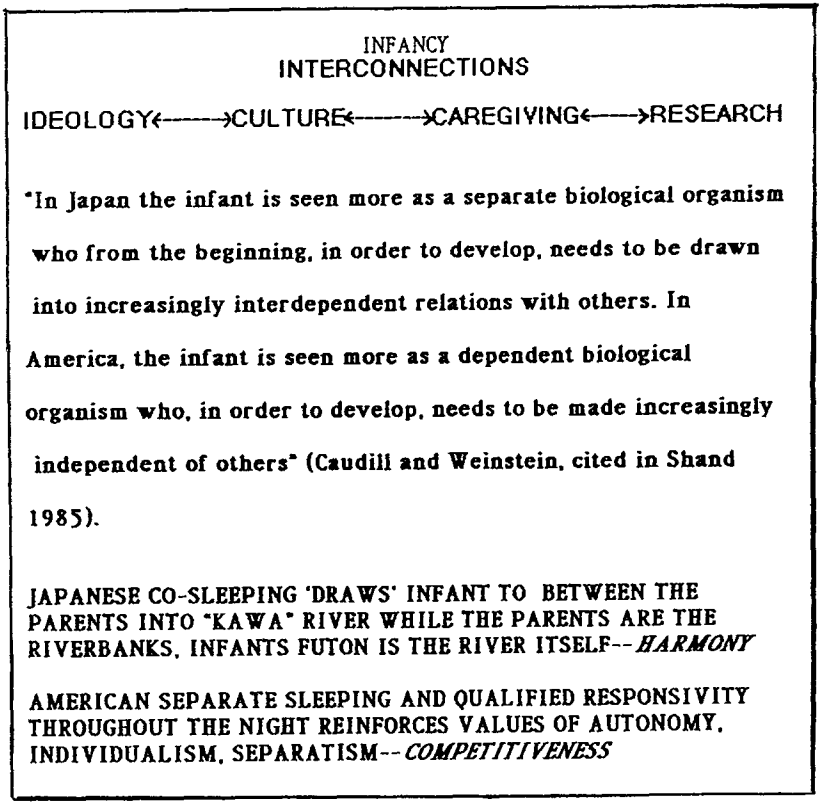


Fig. 3 Infancy interconnections: This figure illustrates the interconnections between cultural ideologies, including values and socialization, infant caregiving patterns, and research questions and strategies.

Interdependence and group harmony are two values positively sanctioned in Japan where co-sleeping occurs in large measure to create a social environment which fosters the very kind of socioemotional dependencies which Americans attempt to inhibit or suppress. According to Christopher (1983:70, cited in McCrary 1988): "One monkey that does perch on the back of nearly all Japanese is a deeply ingrained feeling that individual gratification is possible only in the group context -- a feeling which, like the taste for dependence, clearly

stems from childhood experiences." The fact that American children are taught to be self-reliant and display their individuality (and, thus, learn to sleep alone) and Japanese children are taught to "harmonize with the group" (and, hence, sleep with their parents) is best understood by the very different attitudes and values Japanese and American parents have concerning the "nature" of the infant at birth. Caudill and Weinstein, cited in Shand (1985) explain:

> *In Japan the infant is seen more as a separate biological organism who from the beginning, in order to develop, needs to be drawn into increasingly interdependent relations with others. In America, the infant is seen more as a dependent biological organism who, in order to develop, needs to be made increasingly independent of others.*

The Ideology of Solitary Infant Sleep as "Normal / Safe / Good Sleep: Where Did It Come From?

1. Laws and proclamations passed from the 16th through the 18th century protecting infants from infanticide--purposeful overlying in conditions of poverty

2. 17th century "Moralists" and concerns of the Catholic Church in getting teenage daughters out of parents bed to protect from fathers sexual abuse

3. The emergence of the importance of the "conjugal bond" at the expense of the child-parent bond, early 17th into 18th century Europe and France

4. The emergence in the 18th century of "romantic love"

5. 17th and 18th European socio-political changes re-defining patriarchy and the socio-sexual privacy of the family / husband-wife from the public

6. American values of individualism, separateness, autonomy--"the "All American Infant" idea— our own socialization and experience

References: Aries 1962, Flandrin 1979, Kellum 1974, Stone 1977, Vinosky 1987

Fig. 4

b. Where did the ideology come from?

Where our attitudes about co-sleeping actually come from is a question that cannot be answered in any simple way. Our beliefs about the practice stem from a diffuse confluence of historical, socioeconomic, ideological, and religious factors, including a concern for children's safety, that carries over in distorted way from historical events in Europe centuries ago. For example, moralist philosophers writing in 17th century France were concerned with the sexual "purity" of children. Incest between teenage children and parents who slept together became a concern, particularly during the 17th and 18th centuries. The Catholic Church advocated separate sleeping places for older children when tales of incestuous relationships between pubescent daughters and their fathers were making their way into the confessionals (Flandrin 1979). Moreover, the rise of the notion of romantic love and the preeminence of the conjugal (husband-wife) bond over the parent-child relationship which also occurred in western cultures at around the same time (see Stone 1977) also influenced attitudes concerning parent-infant co-sleeping. The infant-child came to be seen as a competitor who threatened the sanctity of affection and love between the parents--a view not too unlike what later became formalized in Freudian psychology as the Oedipus complex.

c. The suffocation factor.

A common reason given by contemporary pediatricians and health officials as to why co-sleeping should not be practiced concerns fears of "overlying" or suffocating the infant -- a reason that may have some historical connection to the fact that in England, Germany, and France during the 17th century, proclamations were written and laws were passed threatening parents with jail should they be caught in the same bed with their infants. These laws were aimed at preventing infanticide -- a serious problem among the extreme poor of England and France during this period. Not able to nutritionally support more than one child at a time, many parents claimed to have accidentally rolled over, killing their infants while sleeping with them. When conditions of resource deprivation exist, infanticide has been a form of birth control practiced when an infant is born before the previous one is weaned. Infanticide by suffocation was a serious problem in urban centers of Paris, London, and Germany during the 16th, 17th, and 18th centuries (see Aries 1962 and Flandrin 1979), enough to see much legislation outlawing infants less than two years of age from their parents' bed (see Kellum 1974).

While it is, of course, possible to suffocate an infant by rolling over on it, especially by irresponsible parents desensitized by drugs or alochol, under normal circumstances it is very difficult to do so and probably no more likely than an infant strangling itself in a crib (see McKenna 1986 for discussion). Infants are very strong and vocal when their oxygen is threatened (see Woolly 1945) and, besides, if infant suffocation had ever been a serious problem for our species, given the fact that our species practiced co-sleeping until approximately 200 years ago, none of us would be here today because our ancestors would have died as the result of co-sleeping. Certainly the fear of overlying or suffocating

the infant during co-sleeping is exceedingly prevalent in the United States but it is a fear that is disproportionate to the likelihood of it ever occurring. It is a fear exacerbated by myth, socialization, and ideology, not facts.

Finally, in the United States, baby care books are mostly written by men who have long advocated separate sleep for parents and infants (see Spock 1976). Too much dependence by infants and toddlers on parents during the night, thereby limiting the infant's growth toward autonomy, as in the form of self-soothing and self-regulation of sleep, remain major worries, as does the misplaced fear that infants will be traumatized by hearing the sounds and possibly seeing their parents' love-making (see Robertiello 1975). While issues of sexual privacy may be important to parents in some cultures more than others, nowhere has it been shown, certainly not in the ethnographic data, that the sights or sounds of lovemaking by parents have any damaging effects on infants whatever.

In the face of so much authority arguing against co-sleeping, and in view of the work schedules of urban parents who may require more consolidated sleep than a co-sleeping environment may provide, solitary sleep environments for American infants, as early in the infant's life as possible, seems to be an acceptable if not convenient caregiving practice.

In sum, contemporary infant sleep researchers and scientists concerned with SIDS live in a culture where co-sleeping is assumed to be physically dangerous for the infant, potentially deleterious for the infant's psychological and socioemotional development, and simply not "natural." Furthermore, it is highly unlikely that either sleep or SIDS researchers as infants slept with their own parents. Within this cultural context it is not surprising then that questions concerning whether co-sleeping could have any significant beneficial effects on infants, or if some infants might need nocturnal contact with parents more than others, or if, in fact, co-sleeping could make it more difficult for the SIDS deficit to find expression, are questions not likely to be asked.

It is due to the power and ubiquity of this unchallenged ideology, and the assumptions about the unnaturalness of parent-infant co-sleeping, that among the thousands of infant sleep papers published during the last twenty years or so there exists not one single physiological study of the effects of parental contact (social sleeping) on the infant's sleep, breathing, and arousal -- the stable microenvironment within which, for well over four million years, these physiological systems evolved.

VI. Application: An Anthropological Hypothesis on the Possible Relationship Between Co-sleeping and SIDS

The intersection of cross-cultural, cross-species, and evolutionary perspectives on the parent-infant sleep patterns discussed above suggests that solitary nocturnal sleep devoid of parental sensory cues is a novel environment for the human infant. Given that SIDS has many different causes, and that for at

least some SIDS victims multiple factors conspire to increase SIDS risk, it is hypothesized that the natural sensory stimuli which occur during parent-infant co-sleeping and which induce small transient arousals in infants may help some infants to resist a SIDS event. As discussed earlier, this hypothesis emerges from developmental studies of nonhuman primates who show significant negative physiological consequences upon short-term separation from their caregivers. It also emerges from preliminary data on the sleep patterns of infants who sleep with their parents who do not achieve "adult-like" consolidated sleep levels, i.e., sleeping for prolonged uninterrupted periods, until well after the first year of life. In comparison, solitary-sleeping infants in the United States can achieve adult-like sleep levels as early as four months and certainly by a year. The hypothesis described below has the most significance to the SIDS research showing that some infants who died from SIDS experienced arousal deficiencies, or problems in awakening, before their deaths.

a. Testing the hypothesis: Are SIDS rates lower in co-sleeping societies?

One way to test the hypothesis is to determine if SIDS rates are lower in hunting and gathering societies or third world countries in which co-sleeping is regularly practiced. But because SIDS is so rare, and its diagnosis so difficult (as discussed earlier), especially in preindustrial societies in which high infant mortality from all causes exists, such comparisons are almost meaningless. It would be ideal if we could use Barry and Paxson's survey of the 90 societies in which 71 co-sleep, or my own survey (Table 3), to look for SIDS rates, but the data are not there either. In some ways, only affluent countries such as our own have the luxury of studying a syndrome which kills so few infants.

If we could locate subgroups within our own culture who practice parent-infant co-sleeping and compare SIDS rates against non-co-sleeping subgroups, this might be a place to start. But even within our own society it can be difficult to demonstrate an inverse correlation between co-sleeping and reduced SIDS rates because socioeconomic and other epidemiologically important factors may differentiate the groups. For example, Lozoff et al. (1984) found that in New York City, 35 percent of poor urban whites and 79 percent of poor urban blacks routinely slept with their children -- though most of the children in the samples were beyond the peak age for SIDS. Nevertheless, if my hypothesis is correct, and if we can assume that shared sleeping includes infants, there should be slightly lower SIDS rates among urban blacks in general. But this is not the case. The SIDS rate for New York City blacks is actually higher than it is for whites. In this case, the hypothesis cannot be tested by these data because the hypothesized benefits of co-sleeping may be negated by the fact that compared with poor white mothers, black mothers are more likely to be single, have their babies at an earlier age, live in more extreme poverty, smoke during their pregnancies, and lack access to prenatal care. All of these are known to be important epidemiological factors associated with mothers whose infants have an increased chance of dying from SIDS. Only if all these epidemiological factors were the same between white non-co-sleepers and black co-sleepers could we hope to test the hypothesis.

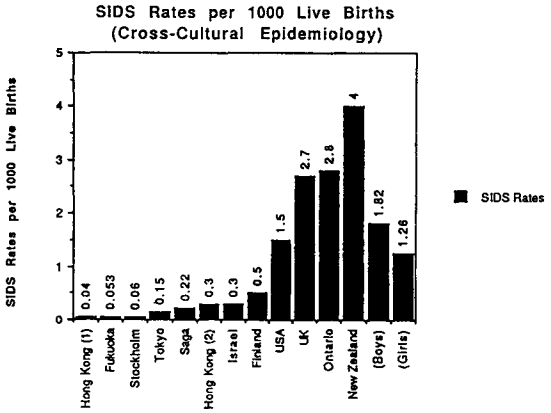


Fig. 5 The first eight cities, which have the lowest SIDS rates, are urban societies wherein co-sleeping is the norm. The USA, UK, Ontario, and New Zealand practice separate sleeping.

b. SIDS rates in cross-cultural perspective.

More promising, but still not perfect, is testing the hypothesis in an urban-industrial setting in which parents practice co-sleeping and the procedures for identifying SIDS upon autopsy are comparable to western procedures. In Japan, for example, where co-sleeping continues to be the norm (Takeda 1987), current published rates for SIDS are some of the lowest in the world (0.15/1000 births in Tokyo, 1978; 0.053/1000 in Fukuoka, 1986; and 0.22/1000 births in Saga; Tasaki et al. 1988). This finding does not, of course, prove that co-sleeping is protective against SIDS. It may well be that SIDS is underreported in Japan, or that it is misdiagnosed as infantile suffocation. Japanese medical scientists have not participated in international SIDS research studies to the extent that American and European scientists have, so the postmortem procedures they employ to identify SIDS may not be appropriate. Nevertheless, these low SIDS rates deserve explanation and further research.

In 1985, Davies reported on the rarity of SIDS in Hong Kong, another urban society in which co-sleeping is the norm. He used postmortem diagnostic protocols that, on review for a follow-up study by Lee et al. (1989, see below), were judged comparative to Western diagnostic standards by John Emery, a renowned SIDS researcher from Great Britain. Davies found that even in a

context of poverty and overcrowded conditions, where the incidence of SIDS should be high, the rates were .036 per 1000 live births, or approximately 50 to 70 times less common than in Western societies.

Similar to my own hypothesis, Davies proposed that proximity to the parent while the infant is asleep may be one reason why the rates are so low. The author asked "whether the possible influences of life style and caretaking practices in cot death are being underestimated in preference for more exotic and esoteric explanations" (Davies 1985:1348) -- a viewpoint not unlike that of Taylor and Emery (1988) and Emery (1983), who also implicate, for some English infants, the importance of caregiving environments and other behavioral-socioeconomic factors. A follow-up on Davies's work by Lee et al. (1989) confirms the relative rarity of cot deaths in Hong Kong, finding a slightly higher rate of deaths per 1000 live births (0.3, compared with .04/1000 reported by Davies).

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

c. More questions.

It should be clear by now that no hypothesis about SIDS will be easy to test. The methodological difficulties discussed earlier will not go away. The reduction in SIDS rates that occurs, especially in co-sleeping Asian cultures reported above, does not adequately test the co-sleeping hypothesis, but only helps to raise questions and to further justify exploring the hypothesis. For example, if contact protects some infants, exactly what does the contact do? In other words, do infants who sleep with parents sleep differently? Do they mimic or somehow copy their sleeping partner's breathing? Do they awaken more frequently? Do they spend more time in light sleep (stage 1-2) and less time in deep sleep (stage 3-4) from which arousal is most difficult? Or do infant co-sleepers spend less time in rapid eye movement (REM) sleep when respiration and breathing in general are less stable? Is it possible that none of these things change but that proximity to infants permits parents to better monitor their infants' physiological status? Do parents notice and awaken to breathing silence?

VII. Physiological Interactions of Co-sleeping Mothers and Infants: A Preliminary Glimpse

a. What we did and why.

Once a theoretical justification for a particular theory is in place and the specific hypothesis formulated, the investigator has the responsibility of testing it. But what can be done in this situation, given the difficulties of comparing SIDS rates within co-sleeping and non-co-sleeping societies? Moreover, knowing as we do that SIDS likely has several different causes, all of which stem not from one localized abnormality but from a complex or "suite" of diffuse central nervous system deficits, how can the hypothesis be tested?

Certainly, there is no way to test the hypothesis directly, at least for now. Millions of dollars of biomedical research grants would be needed to secure two populations for a prospective study, plan the study, and hire personnel. Ideally, two groups of newborns (infants who sleep with their parents and those who don't) could be recruited and all subjects matched for socioeconomic status, ethnicity, age of mother, health, and so forth, and monitored throughout the first year. SIDS rates should be lower in the co-sleeping group if the hypothesis is correct. For valid statistical results, we would need a minimum of 5000 infants in each group; however, 10,000 in each group would be better. Since a new research hypothesis is never rewarded with funding until some preliminary data validate it, or at least some part of it (a kind of "catch-22" in American scientific research), what kind of preliminary study could we conduct to earn some respectability for the general hypothesis? A theoretical position, even if it is strong, will never convince skeptical scientists, especially SIDS scientists who are used to hearing about a new hypothesis on the "cause" of SIDS every week.

My colleague on the project is Dr. Sarah Mosko, a psychobiologist and a licensed polysomnographer at the University of California, Irvine Medical Center, Department of Neurology, where I also hold a position. This means that she is an expert in reading and interpreting sleep data collected on polygraph machines. We decided that one step we could take which does not test the SIDS hypothesis directly, but, nevertheless, is relevant to it, is to demonstrate 1) physiologically significant changes in the sleep experiences of solitary and social sleeping infants and 2) that the changes we do find (presuming they are real) are pertinent to understanding suspected infantile deficits thought to be involved in some SIDS instances.

A critical part of our hypothesis is the expectation that infants will have a different experience physiologically when they sleep with another person. However, at the time we proposed this project, there was not one single published paper on the subject. The fact that none existed is yet another indication of the complete acceptance of the idea that solitary sleeping environments are perfectly normal for human infants.

Some of the more specific questions we hope to answer include: What kinds of physiological changes in arousals and breathing and heart rates do parents and infants have as a result of contact with each other throughout the

night? Perhaps the kind of sensory communication we have postulated above, i.e., auditory stimuli such as breathing sounds, smells, sudden movements, breathing movements of the chest, and/or touch, reduces the number of breathing pauses, or increases arousals in infants, or simply prevents infants from sleeping in deep stages or from having apneas? Perhaps mothers and infants spend a great deal of their night in the same sleep stage at the same time? Certainly, if nothing of clinical interest or significance can be physiologically documented, the justification for the hypothesis could be questioned.

We applied for and received a small grant and recruited five healthy mothers and their healthy infants (all between 2 and 4 months of age, the peak age for SIDS) to spend one night in the sleep lab, sleeping together in the same bed. Each pair came on a different night. We knew that the study would be difficult because, from a technical standpoint, no sleep study before had attempted to hook two persons up to the same polygraph machine using ten simultaneous channels. The difficulties caused by signals crossing from each partner were very real, i.e., the mother's chest movement signal effecting the infant's separate signal and vice-versa. Amplitudes had to be checked and re-checked and adjusted to eliminate signal artifacts. It was difficult, but the difficulties were overcome.

While the mother and baby slept in one room Dr. Mosko and I were in a different room overseeing the recordings being made continuously on the polygraph machine throughout the night in front of us. Signals from the sleeping pair were transmitted via thin strain gauges gently taped to their skulls, chins, cheeks, and chests. Five signals on each mother and each infant (ten per pair) were recorded simultaneously: EEG (electroencephalograms needed to record brain wave activity to score sleep stages or awake status); EOG (electroculograms needed to record eye movement also important to sleep stage scoring); EKG (electrocardiograms which monitor heart rates); and several small non-invasive electrodes on the subjects' chests to detect and record diaphragmic or respiratory behavior (breaths per minute) (see Figure 6).

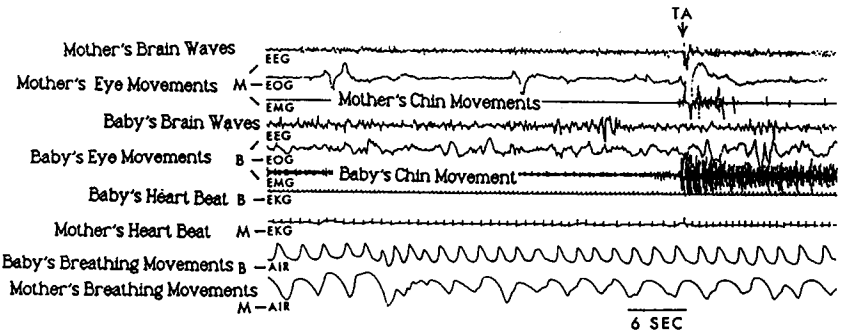


Fig. 6 In this segment of time the mother has a small arousal (a transient arousal, TA) as indicated by a change in her brain waves, eye movement waves and her chin position. Within a second or so of mother's arousal the infant opens its mouth, or

moves it laterally and its heart rate increases -- it also experiences an arousal. The possible significance of this event is that insofar as the infant is concerned, it may not have happened had the infant been sleeping alone. In this case the mother's behavior interrupted, or somehow changed the state of the infant. It is one example of a naturally occurring sensory exchange between co-sleeping mothers and infants.

In and of itself this event probably isn't particularly significant. But the accumulation of these kinds of events over many nights of co-sleeping (recall that this is the evolutionary stable context for infant sleep) may well set the infant on a very different overall developmental trajectory of its sleep patterns from what occurs when throughout the first year of life the infant sleeps regularly alone. For example, it may well be that over the long run these small interruption experiences by the infant, due to mother's presence, may prevent it from staying too long in a deep stage of sleep (stage 3-4) from which it is difficult for the infant to arouse to breathe, should the infant experience a breathing crisis.

Small strain gauges sensitive to electrical activity or movement are taped onto various parts of the mothers' and infants' bodies; one is placed on the skull, one below the eyes, one near the chin, one near the heart, and the last one near the diaphragm (mid-chest) on each person. Simultaneously, as is seen in this figure, each signal (a total of ten for each mother-baby pair) is transmitted by each wire to the polygraph machine situated in another room where ten pens lined up one next to the other record varying movements or electrical activity onto paper moving along a conveyer belt. These electrical signals direct the form of the resulting graphics and permit us to distinguish between different physiological states as, for example, when one or both of the partners awaken, or move in or out of one of three sleep stages: stage 1-2, 3-4, or rapid eye movement (active) sleep.

As is the case here, these signals permit us to detect simultaneous or overlapping arousals among the co-sleeping pairs. Such simultaneous, or partner-induced arousals, may be important for the infant as discussed in the text.

b. What we found.

In a strange way our study documented the obvious: co-sleeping partners have an effect on each other as they sleep together. Wow! What a surprise! (Sometimes science is like this.) Movements, vocalizations, touches, and all the other kinds of sensory exchanges which we predicted could make a difference in the sleep patterning, arousal level, breathing, and progression of

sleep between the sleeping partners were demonstrated. For example, in our first study we found that each partner could induce small physiologically defined arousals in the other (within a time frame of 2 seconds) thereby inducing a shift from one sleep stage to another as well as changes in breathing and heart rates following the arousal. This was true for both the mother and the infant. Sometimes short breath-holding episodes (small apneas) overlapped between mothers and infants as if each momentarily mimicked what the other was doing (see Figures 7a and b). Simultaneous epochal arousals -- arousals longer than a minute -- were documented. One of several kinds of epochal awakenings involves nursing wherein both partners are awake for some number of minutes together (usually 5 to 20). At frequencies different than would be expected by chance, both epochal and transient arousals of mothers and infants overlapped temporarily (see Figure 8) (see McKenna et al. 1990, and McKenna and Mosko 1990 for all the results and more extensive discussion).

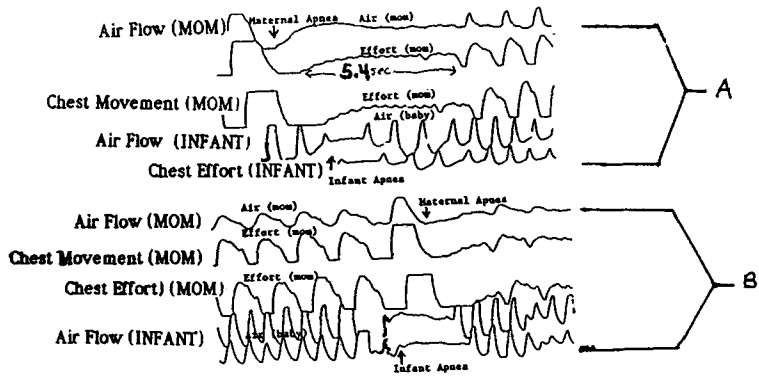


Fig. 7 Overlapping breathing pauses of co-sleeping mother and infant.

In the first series of polygraph recordings marked (A) the mother initiates a breathing pause and within a second or so her co-sleeping infant does likewise. In the series of recordings below (B) the reverse occurs: the infant initiates a brief breathing pause and within a second the mother follows. It is difficult to say with 100% certainty that one partner "caused" the event in the other. But an analysis of the data collected before and after these events suggests that these events are related. The most important point illustrated by these and other data collected in this study is that in a variety of ways co-sleeping mothers and infants respond to each other physiologically throughout the night. In almost all stages of sleep their bodies seem to be sensitive to what the other is doing. The accumulation of these and other kinds of responses may have important short and long term clinical significance which needs to be studied further.

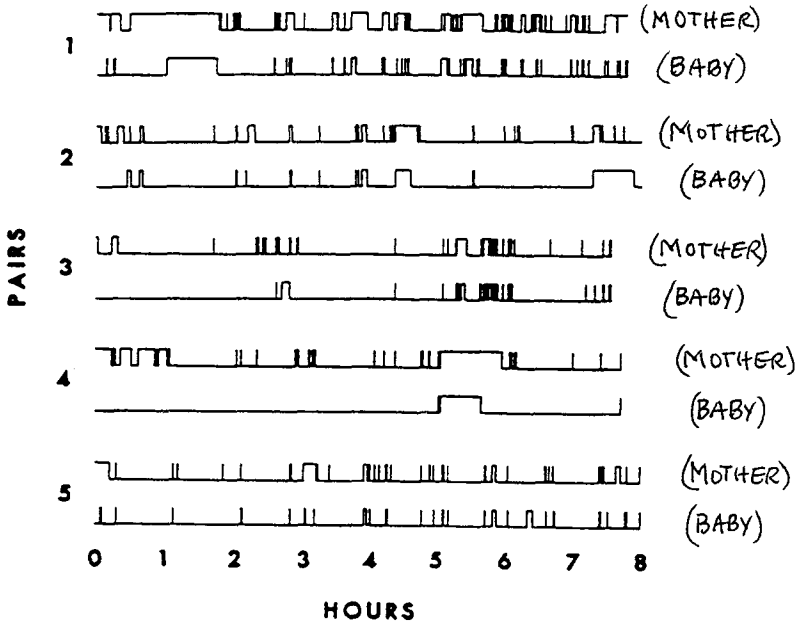


Fig. 8 Sleep-wake transitions of co-sleeping mother-infant pairs over an eight-hour period.

Each pair (1-5) is a co-sleeping mother and infant. The line indicates whether each partner was awake (when line is at the top) or asleep (the line travels along the bottom) over an eight-hour period. The spikes represent one minute of awakening. Notice how many times the mother and infant awaken at the same moment, i.e., look for instances in which the mother's spike matches the infant's, and the times when they were awake together. For example, look at pair 4. Notice that the infant awoke only once to noise and, of course, mother was awake, too. This computer-generated illustration shows much synchrony between co-sleeping mothers and infants.

(From: American Journal of Physical Anthropology, 1990, Vol. 83, pp. 331-347, McKenna, et. al.)

Ideally, if we had been able to have the mothers and infants stay over for two or more consecutive nights for this first study as we did in the second study (see below), we could have compared their sleep and arousal patterns in separate vs. social sleeping situations. Nevertheless, we figured that if there was something of importance that occurs during co-sleeping between parents and infants, very likely we could measure it regardless of the experimental limitations we faced. And, indeed, we did.

We also found in this first study that when sleeping together, mothers and infants show a great deal of homogeneity as to when or at what point they enter various sleep stages; that is, even though the infant's sleep system is not yet adult-like, when sleeping together mothers and infants are either awake in the same sleep stage sometimes for as much as 40 to 50 percent of their sleep time, though we do not know what the precise regulatory mechanisms other than partner-induced arousals may account for this. Let me explain further. Using 30-second "epochal" scoring systems for sleep-wake stages, we computed for each co-sleeping mother and infant the percentage of that individual's sleep period time (total minutes from first falling asleep to final morning awakening) spent simultaneously in the same stage of sleep or wakefulness as the other member of the pair. We called these times of corresponding sleep-wake stages Simultaneous Activity Time (or SAT) as shown in Figure 9. It averaged 46 percent for the mothers (range 43-48 percent) and 44 percent (range 43-45) for the infants.

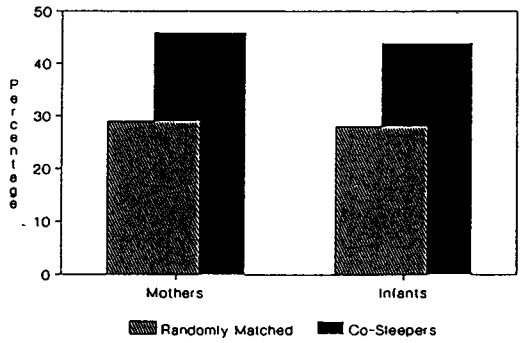


Fig. 9 Simultaneous Activity Time of co-sleeping and non co-sleeping (randomly matched) mothers and infants. Simultaneous activity means that the mother and infant were in exactly the same sleep stage, or were awake, at the same time. This graph shows that when mothers and infants slept in the same bed, they shared more time in the same sleep or awake period than each of them did if their status was randomly compared on a minute-to-minute basis with other infants, or mothers, with whom they did not sleep.

(From: American Journal of Physical Anthropology, Vol. 83, pp. 331-347, McKenna, et. al., 1990.)

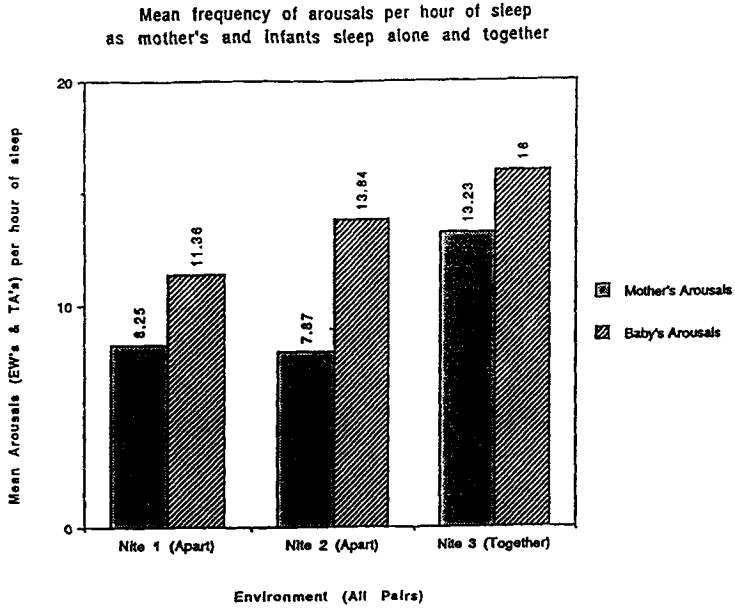
Now you may be wondering, how do we know that this "homogeneity" or simultaneous activity time is not simply the result of chance? That is, if you and I fall asleep in separate rooms, would not much of our sleep status compared minute-to-minute throughout the night be "simultaneous activity time" simply because sleep follows some inherent biologically-based progression -- stage 1 to 2 to 3 to 4, and them REM? Obviously, our scores do not, in this situation, reflect any kind of effect that we are having on each other; our SAT score simply reflects chance as well as the expectable biological characteristics of sleep. You would be right.

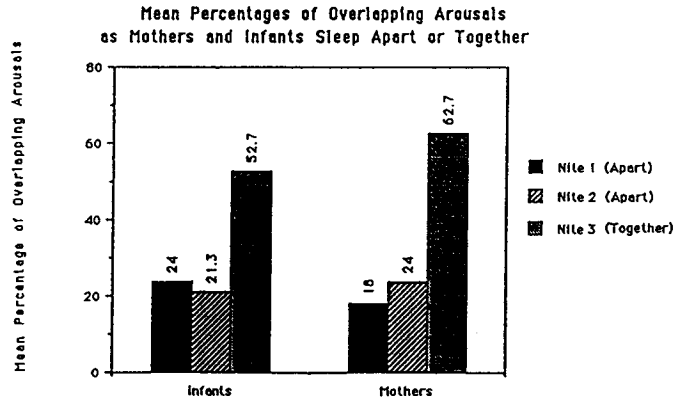
In order to determine, then, the extent to which there *is* some mutual regulatory influence that co-sleeping partners have on each other, we matched infants with mothers with whom they did not sleep minute to minute, and mothers with infants with whom they did not sleep and calculated the simultaneous activity time scores for each. As Figure 9 reveals, there was a significant difference in the amount of time true co-sleepers spent in SAT compared with the randomly matched non-co-sleepers. The difference was statistically significant at the .01 level. The regulatory effect of co-sleeping, for now, at least, seems real.

In a second study we just completed we had better experimental controls. The data proved even more dramatic and supportive of our predictions that infants have very different physiological experiences when they sleep with a parent. In this study, three mother-infant pairs spent three consecutive nights in the sleep lab. The first night the mother and infant slept in separate rooms as both were monitored physiologically as described above. The second night they also slept apart, in separate rooms, but the third night they slept in the same bed, also monitored physiologically. The co-sleeping night served as our control. Though not all of the data are analyzed yet, Figures 10a and b show some of the changes we are seeing as infants move from the solitary situation (nights 1 and 2) to the social sleeping environment (night 3). Our hypothesis concerning changes in sleep architecture and arousal synchrony in a social sleeping situation is being confirmed. But, of course, our work is in its beginning phases and much more work is planned.

c. But it doesn't prove anything, does it?

The preliminary studies we have completed in the sleep lab certainly do not prove anything about SIDS. But they do show that co-sleeping mothers and infants assert potentially important influences on each other's sleep experiences, including sleep stage duration and progression, arousals, and breathing -- all issues intensively studied by sleep researchers for purposes of discovering something about SIDS pathophysiology.





Figs. 10a and b These figures show that the night in which mother and infant slept in the same bed, night 3, both experienced more arousals. (During nights 1 and 2, infant slept alone.) The graph shows the extent to which arousals overlapped or occurred within ± 2 seconds of an arousal in the other. This amounts to a form of temporal snychrony in arousals which has the effect of changing the sleep architecture, or amount of time infants stay in certain stages of sleep. Over time, these kinds of interactions may lead to an overall different development of the infant's sleep if they occur consistently in a social context. These data were collected by Drs. James McKenna and Sarah Mosko.

The fact that we have excellent data showing that arousals between co-sleepers can be induced by either sleeping partner indicates that we now have a beginning point for discussions concerning possible social mechanisms that provide infants with "practice" in arousing to breathe before a life-threatening event requires it. Should this prove to be an ability that some infants lack, future studies may demonstrate that a social sleeping partner can help the baby to learn this response. Of course, we do not yet know that partner-induced arousals or practice by the infant in arousing to breathe will diminish the likelihood of a SIDS event, but this is an important question that emerges from the new data. It is a possibility that we can consider and think about and test what might have been missed had we never considered the social sleeping issues. There are others, too, raised by this new data, all of which would not be available had we not challenged the accepted ideology that says that solitary sleeping infants are "normal" sleeping infants, so there is no need to wonder how infants would sleep in some other situation.

From an evolutionary perspective it is not surprising to find that across diverse environmental contexts infants and mothers are highly responsive to each other's sensory cues, or that they are able to communicate with each other throughout the night. Recall that natural selection undoubtedly favored both mothers and infants who responded quickly and effectively to each other since to do so protected mothers' substantial reproductive investment and led to infant survival. The infant must certainly play its part, too; it is born with sensory systems primed to be responsive to its caregiver's cues. Together the caregiver and the infant make an adaptive fit.

VIII. The Response from the Medical, Social Science, and Sleep Research Communities

a. The first step is to publish a theoretical rationale

Before the studies of co-sleeping mothers and infants described above were completed, I authored a theoretically-based monograph entitled "An Anthropological Perspective on the Sudden Infant Death Syndrome (SIDS): The Role of Parental Breathing Cues and Speech Breathing Adaptations." As might be inferred from my earlier discussion, my purpose was to put forth a series of new, testable hypotheses relating to different aspects of the SIDS puzzle that scientists from a variety of fields could explore.

Obviously, cross-cultural studies of infants, field and laboratory studies of monkeys and apes, and human evolutionary, including paleontological data are not materials covered in medical education or in the classes SIDS researchers such as pathologists and epidemiologists take in medical school. Beginning with a new conceptualization of the infant, I knew it was necessary to lay out a theoretical justification for the type of approach to SIDS that I was advocating before anyone would, or perhaps could, take these issues seriously. The manuscript was published in 1986 as a special issue of the journal *Medical*

Anthropology: Cross Cultural Studies in Disease and Illness, Volume 10. As is standard practice for scientific journals the manuscript was anonymously refereed by three scientists. Because SIDS is such an emotional and important issue, and the content of my manuscript so interdisciplinary and somewhat critical of the prevailing SIDS research paradigm, the editors invited eleven international known scientists, seven of whom published and conducted research on SIDS, to respond in writing to my paper. Their comments and my response to them were published simultaneously with the monograph.

I want to share parts of their reactions, both favorable and not so favorable, as a way of illustrating the kinds of important issues that are raised when scientists from different backgrounds and fields consider the same problem but "see" it from the knowledge and traditions of their own discipline. The abstracted commentaries that follow reflect a wide range of opinions as to whether my hypotheses seem tenable and/or testable. Two preeminent SIDS researchers discuss the implications, or the "drawbacks", of the research hypothesis, i.e. their concern that parents of SIDS victims might feel that their infants might not have died if they had not been sleeping alone. Recall my own worry about this very issue as stated in the beginning of this monograph (I respond to this issue in detail at the end).

b. A summary of my hypotheses and assumptions

My main hypothesis is that the sensory stimuli infants receive from a co-sleeping partner should counteract, or somehow help to override, some of the deficits involved in some of the very different kinds of SIDS incidents. Hence, parent-infant co-sleeping should reduce SIDS rates (however slightly) when and where it is practiced.

I consider the following assumptions to be the most important ones underlying my research and the ones that significantly differentiate my perspective from the more traditional biomedical one. These include:

1) Parent-infant co-sleeping represents the species-wide or "normal" expectable environment for nighttime sleep for human infants while solitary nocturnal infant sleep represents a recent historical deviation from the evolved pattern; in fact, an infant sleeping alone in a separate room represents a situation of sensory deprivation.

2) The human infant's evolved developmental and physiological needs (such as the need for contact with a caregiver) are much less able to change and so change much more slowly than do caregiving behaviors by parents who respond to infant needs in ways deemed appropriate by fast-changing cultural values, ideologies, and conditions.

3) The biological and social needs of parents and infants are not the same; what is in the infant's biological and social best interest (such as co-sleeping) is not necessarily in the biological or social best interest of the parent.

4) Not all infants are the same biologically; those that are born with nervous system deficits, or less biological versatility, will respond less positively when developmental settings deviate significantly from evolutionary expectable ones, i.e., when they find themselves sleeping alone, as compared with another person.

5) Infant needs should be conceptualized initially in terms of what the evolutionary and cross-cultural data suggest to be ideal and not according to either conscious or nonconscious ideologies compatible with our highly individualized (urban-industrial) lifestyle.

c. *The responses and commentaries below originally appeared in a special issue of Medical Anthropology: Cross-Cultural Studies in Health and Illness. They are abstracted here, in alphabetical order, and with permission of Gordon & Breach, publishers.*

Donald Brenneis, Ph.D., Socio-linguistics, Socio-cultural anthropologist.
Pitzer College, Claremont, CA 91711, U.S.A.

> Reading McKenna's paper as a linguistic anthropologist, I found it to be innovative, thorough, careful, and highly suggestive, not only about SIDS but also in relation to human communicative potential and practice. I will comment briefly on several related aspects of the paper.
>
> First, McKenna very effectively forces a reconceptualization of both SIDS and the methodological assumptions underlying its study by locating the phenomenon so squarely within an evolutionary framework. That contemporary urban society with all its implications for parent-infant interaction is a recent and limited case is indisputable; the fact that such societies may post particular challenges for infants can no longer be neglected as a potential factor in SIDS. . . .

Dr. Justin Call, Chief of Child Psychiatry
University of California, Irvine, School of Medicine

> . . . The findings and suggestions in this study will be quite challenging to our urban culture and to traditional notions of what is best for the baby. Pediatricians have now for several generations been taught to advise separate sleeping arrangements for infants and their parents. Many parents in urban societies where both parents work have been pushing the adaptive capacities of the human infant to the limit by arranging infant care which proceeds in exactly the opposite direction from that suggested by the infant's continued need for physiological support during the first year of life, and to some extent later. Many studies now, however, are demonstrating that an adequate period of symbiosis or dependency in the first year is the best preparation for later independence and autonomy on the part of the infant. But these lessons, while shown in present research, are seriously challenged by

the direction in which our urban society is moving, and it will be difficult for Americans in particular to accept the idea that we must relearn something from our more primitive past which we have forgotten or which has been lost in the rush of "civilization" and technology. It is quite likely that other physiological disturbances, i.e., rumination and other eating disturbances, sleep disturbance, the irritable infant syndrome, failure to thrive, certain forms of child abuse, and disturbances in affecting development, including mood and attachment disorders of infancy, can best be understood within the general framework which McKenna has provided for our better understanding of SIDS.

This monograph should elicit appreciation and response by researchers in all of the fields that have previously engaged in or contemplated such engagement in this stubbornly challenging problem to human mortality, and should engage emerging new professions concerned with developmental science in consideration of these assumptions and the investigation of these newly conceptualized questions concerning the etiology and prevention of SIDS and other physiological disorders of infancy.

James S. Chisholm, Professor of Anthropology
Department of Anthropology, University of New Mexico

McKenna's novel approach to SIDS in a masterpiece of synthesis and is bound to be widely cited as an exemplar of "the new medical anthropology" -- by which I mean a medical anthropology informed by the integrated evolutionary/developmental/cross-cultural perspective he employs. A special value of his work is in showing how stultifying traditional disciplinary boundaries can be, and by implication, suggesting how we might all benefit from teaching our medical students the basics of hominid evolution and evolutionary and developmental theory. To more fully understand health and disease we require ultimate and ontogenetic explanations as well as more proximate ones. As an irreverent evolutionary biologist colleague once put it, "the trouble with medicine is that doctors are just applied biologists." Even if McKenna should turn out to be wrong on any particulars, his illustration of theory building in medicine is a significant contribution.

. . . As McKenna emphasizes in both word and his own research efforts, it has not yet been demonstrated that fetal breathing is affected by maternal circulatory rhythms. A partial test of this proposition, and its relevance to the etiology of SIDS, might come from determining whether or not placenta previa or other abnormalities of implantation or position result in a higher incidence of SIDS. In other words, one prediction from McKenna's hypothesis might be that when the fetal ear is not close to the descending aorta or iliac artery the fetus will be less well entrained to maternal rhythms, and thus at greater risk for later breathing irregularities. McKenna does suggest that deaf infants should

be at greater risk for SIDS, but one problem with looking at the incidence of SIDS in deaf children is that if they are also deaf in utero they might well adapt by becoming more sensitive or responsive to uterine vestibular or tactile cues for breathing, becoming sufficiently entrained through alternate pathways so that there will be no greater risk for SIDS. On the other hand, it fetal hearing is normal, but because of fetal position the fetus cannot hear maternal circulatory sounds so clearly, there may be less tendency to become more sensitive/responsive to other maternal cues for breathing.

David P. Davies, M.D.
Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong

. . . The thesis Dr. McKenna puts forward to explain the etiology of cot death is refreshingly very different from those currently propounded in most clinical medical journals. His holistic approach to the problem, using evolutionary and cross-cultural perspectives which bring together studies from social, medical, and biological sciences, is, to me at least, a new and highly original approach to the problem of cot death etiology, viewing it as more a consequence of a biological deviation rather than a result of discrete pathophysiological mechanisms, which seems to be more the approach emerging out of current clinical and laboratory research studies. His paper emphasizes how easy it is for us to become blinkered to other perspectives as we cocoon ourselves within our own narrowed disciplines. Most important is Dr. McKenna's setting of the developmental scene in which cot death occurs--a few million years of evolutionary adaptations where pre- and postnatal systems responsible for respiratory control, the most important of all early biological systems concerned with species survival, are heavily dependent on sensory cues from both within and without the young developing organism. . . .

. . . Reasons for the rarity of cot death in Hong Kong remain unexplained, but some clues are, I believe, emerging out of Dr. McKenna's paper. When viewed in the context of current "conventional" hypotheses in the clinical literature concerning its epidemiology and possible etiological pathophysiological mechanisms I originally arrived at several paradoxes and contradictions: breastfeeding is very uncommon here; respiratory infections very common; living conditions for large sectors of the population unacceptable by many Western standards in terms of both living densities and quality of abode -- three factors that, on current ideas, might anticipate a high incidence of cot death. I speculated in my original paper that perhaps some other factors are able to "overcome" this potential vulnerability. For example, there is a tradition in Hong Kong for mothers to lie their babies supine for fear of suffocation. There is a slightly lower incidence of very prematurely born babies, who are well recognized to

be overrepresented in cot death statistics. Perhaps life style, methods of looking after babies, and, in particular, overcrowded living conditions are even protective. Babies are virtually never left alone. There is never the question raised "When can I put the baby in his own room" -- an important milestone in the lives of many young babies in many Western countries. Living conditions simply cannot allow this. I wondered whether overall closer contact with the sleeping baby would lessen chances of sudden death. Ambient noise is probably higher. In this context it is worthwhile recalling the close proximity of brain stem centers that regulate breathing to the auditory and vestibular nuclei and reticular formation, connections between which might be important to continue what is initiated in fetal life to help the baby "learn" to breathe regularly after birth.

Over the past year I have received many letters from people who have lived and worked in South East Asia and whose own experiences have also led them to the conclusion of the infrequency of cot death in this part of the world. This had also made them wonder about the modern trend in developed areas of the world to isolate newborn babies from their parents and its possible role in cot death. Dr. Odent, in a letter to the *Lancet*, recently expressed the view after visiting China in 1977, where the concept of cot death was virtually unknown among professionals, that "cot death is a disease of babies who spend their nights in an atmosphere of loneliness and that cot death is a disease of societies where the nuclear family has taken over" (Odent 1986).

Toke Hoppenbrouwers, Ph.D. and Joan Hodgman, M.D.
Department of Pediatrics, University of Southern California School of Medicine

. . . We have been working with another, physiological theory supported by more evidence, which has a better chance of being adequately tested (McGinty and Hoppenbrouwers 1983:375; Hoppenbrouwers 1983). This theory postulates that infants die because they fail to arouse. Wakefulness constitutes a strong stimulus to breathing and a failure to awaken from sleep may jeopardize adequate breathing and CNS oxygenation. Whenever breathing stops, CO_2 builds up. CO_2 is a very potent stimulus to arousal, operating via the brain stem upon the cortex. This mechanism seems intact in infants at risk (McCulloch et al. 1982:911; Ariagno et al. 1980:351; Van der Hal et al. 1985:848). However, an increased input to the cortex may be ineffective if the cortex is depressed. Alternatively, hypoxic hyperapnea may prevent a build-up of CO_2 (Tenney et al. 1971:81) and thus prevent arousal. Both of these scenarios postulate a relatively depressed cortex precipitating an arousal failure and death (Hoppenbrouwers and Hodgman 1986). These two theories in combination suggest intriguing and specific questions which new sophisticated research strategies can begin to answer. . . .

As indicated above, there are significant changes during the first three months of life, some representing a shift away from control by the brain stem toward control by higher centers, such as the cortex. Therefore, cortical depression is perhaps more critical at this time. Dr. McKenna acknowledges the presence of this shift, but he uses this to support a very different theory. He relies heavily on the simultaneous appearance of several functions such as breathing and auditory and vocal behavior as proof for their functional relationship. That seems very tenuous evidence, since during development a wealth of functions is bound to show temporal relationships without direct functional implications. . .

There is something very appealing about evolutionary theories. Our evolutionary theory is speculation at this time, as is Dr. McKenna's. His theoretical underpinning is less than convincing and the key evidence that infants' breathing can be affected by parental breathing is nonexistent. Even the indirect evidence that rocking can affect infant breathing is controversial (Saigal et al. 1986:384A).

Evolutionary theories, while interesting, may have only limited usefulness. They are not easy to test. Dr. McKenna's theory has another drawback: it has a component that blames the parents of the victim. That parents have somehow failed to do something to prevent SIDS is a cruel accusation to place at their feet without convincing evidence. Dr. McKenna has taken pains not to draw such a conclusion but his theory could easily be misused by the uninitiated. This is a persistent problem, as was evidenced by a current article in the *New England Journal of Medicine* (Bass et al. 1986:100). Ironically, the death-scene investigations described in that article implied overlying as a preventable cause in almost a quarter of the cases. Fortunately, as in this issue of *Medical Anthropology*, the editors of that journal provided a simultaneous opportunity for other scientists to comment (Thach 1986:126).

Melvin Konner, M.D., Ph.D., Professor of Biological Anthropology
Departments of Anthropology and Psychiatry, Emory University, Atlanta, GA 30322

James McKenna has fielded an ingenious hypothesis for the Sudden Infant Death Syndrome. Accepting the final common pathway of prolonged sleep apnea -- almost certainly a factor in many cases -- but without ignoring other possible contributing causes, he has reasoned that sleeping proximity between parent (usually mother) and infant may influence breathing rhythm in infants at risk. He goes on to analyze extensively the ways in which such an effect might be mediated, and in doing so reviews admirably much literature relevant to SIDS and infant breathing. His specific hypothesis of auditory stimulation and

stabilization of infant breathing rhythm during co-sleeping is worthy of further exploration.

Perhaps even more important than the specific mechanism, however, is the sleeping arrangement hypothesis. This speculation has been made independently by others (Konner and Super, 1987) and has implications broader than those pertaining directly to SIDS. Many with experience of higher primate infant care or infant care in nonindustrial societies have been impressed with the fact that immediate physical proximity, usually including continuous physical contact and night feeding, is the rule rather than the exception in those cases. Thus it is meaningful to propose that solitary sleeping in a separate crib or bed, especially in a separate room, may not be "natural" for human infants during the first year of life.

Marjorie Elias and her colleagues (Elias et al., 1986) have shown that infants in Boston whose mothers sleep with them and feed them during the night (in contrast to a matched control group) have night waking throughout the first year of the sort that is usually associated with the first few weeks of life. This finding is equally characteristic of the !Kung of Botswana and the Kipsigis of Kenya (Konner and Supper, 1987) among other preindustrial societies. Such findings raise the possibility that sleeping through the night, like solitary sleeping, may be unnatural for human infants.

Other possible consequences of solitary sleep range over a broad spectrum of physiological and psychological variables that may be unrelated to SIDS but nevertheless important. The anthropological hypotheses of the consequences of sleeping proximity proposed by McKenna and others deserve much closer research attention.

Jeffrey T. Laitman, Ph.D.
Department of Anatomy, Mount Sinai School of Medicine, City University of New York

Few killers strike with the unexpectedness and cruelty to both victim and family as does Sudden Infant Death Syndrome. Despite the obvious toll SIDS has taken, and the considerable advances that have been made in related areas of basic and clinical research, the key to understanding SIDS continues to elude us. In an attempt to shed light on this problem, McKenna has combined an impressive synthesis of existing studies on SIDS with suggestions on how an anthropological perspective may offer new insight into the problem. I have found his approach to be both innovative and of great potential value.

By viewing the forest rather than each individual tree, McKenna has caused us to focus upon some major developmental and evolutionary

patterns of our own species whose appreciation is crucial to a basic understanding of SIDS. One such feature is the unique nature of aspects of both our development and our evolutionary history. His observations on the uniqueness of certain features of the human upper respiratory trace, my own area of specific interest, are of particular importance. As our studies have shown, the human upper respiratory tract differs in a number of important ways from that of other mammals (see Laitman and Crelin 1976; Laitman, Crelin and Conlogue 1977; Laitman 1983). For example, the larynx of most mammals, ranging from aquatic forms such as whales and dolphins to terrestrial species such as our closest relatives, the monkeys and apes, is positioned very high in the neck. This high position allows the larynx to literally "lock" into the nasopharynx, providing a direct airtube from the nasal cavity to the trachea and lungs. Due to this configuration, liquid, or in some species even solid material, can flow around the larynx to the esophagus while an animal is breathing. This "two-tube" system, with one separate route for respiration and one for deglutition, is a basic mammalian characteristic.

The change from the (human) infant pattern of the upper respiratory tract to the adult configuration, a change from the basic mammalian pattern to a completely new condition, is indeed unique among mammals. While the exact timing of the transition has yet to be determined conclusively, the transition itself is an undeniable occurrence. To our knowledge no other mammal undergoes such a radical developmental transformation in the anatomy of its upper respiratory tract. McKenna's suggestion to investigate these unique aspects of our developmental anatomy and related neurophysiology in order to provide clues as to why we alone exhibit SIDS is, I feel, particularly well founded. Further, his discussion of the difficulties in producing successful animal models for SIDS in light of a number of our peculiarly human characteristics is also well taken.

As research on the underlying mechanisms for SIDS progresses, it is becoming clear that no single, simple explanation for this syndrome will emerge. While many of us have sought to explain the role that individual pieces of the puzzle may play, the larger picture has remained out of reach. Contributions such as McKenna's, which go beyond isolated paths of inquiry to assault the larger picture, will, I feel, play an increasingly important role. McKenna's own hypotheses on parent-infant respiratory synchronies and their possible relationships to SIDS are well made and deserve further examination. Yet the greatest value of this work has been the demonstration that fresh insight into SIDS can be gained by using an anthropological perspective to examine the cross-disciplinary data. McKenna has made his point. I hope others follow his admirable lead.

Lewis P. Lipsitt, Ph.D. and Barbara Burns, Ph.D.
Department of Psychology, Brown University

We are intrigued by McKenna's eclectic scholarship which has led him to such an interesting proposal about the natural history of the sudden infant death "syndrome." McKenna's review, interpretation of existing data, and hypothesis concerning SIDS provide researchers with a rather nontraditional framework within which to view this tragic and frustrating phenomenon. Of special interest to us is the fact that McKenna's proposal is entirely compatible with, and may be viewed as a specific example of, Lipsitt's more general hypothesis (Lipsitt 1978, 1983; Burns and Lipsitt 1986) that sudden death in babies often is the consequence of a *behavioral failure* resulting from a confluence of physiological (most likely, nervous system) insufficiencies dating from birth or before, and environmental hazards. . . .

The Zeitgeist of SIDS research itself may be undergoing a transition of fundamental importance at this time. As hypotheses which regard both physiological dispositions and early environmental factors (including learning experiences) become more prevalent in our thinking about developing systems of self-regulation, researchers will surely examine closely a host of alternative factors relating experiential factors to SIDS. Researchers will examine an entire range of experience which may be important to consider when modeling the etiology of SIDS. By considering more carefully the interaction of constitutional and experiential facets of the life histories of infants, a more comprehensive albeit more complex understanding of the SIDS phenomenon may be achieved. Surely it is of scientific import that we get our understanding of SIDS right; that it is also of immense humane concern goes without saying. McKenna's contribution to this advance is commendable.

Evelyn B. Thoman, Ph.D.
Department of Biobehavioral Sciences, University of Connecticut

Since the human species is unique in its relatively recent separation of parent and infant during sleep, McKenna's general hypothesis is a very reasonable and exciting one, namely that evolution designed parents and infants so that the process of physiological entrainment, which occurs during the prenatal period, continues after the infant's birth. I will describe a very recent study of ours that is highly relevant to this notion. On the other hand, the more specific hypothesis, that social sleeping should protect vulnerable infants from SIDS, is neither tenable nor testable, and the consequences of applying such a notion may be as serious as SIDS itself. . . .

An inherent part of the problem is that it would not be feasible, perhaps not even possible, for research purposes to randomly assign parents and

their infants to social or isolated sleeping. For example, some parents would undoubtedly object to cosleeping out of fear of "overlying." This may not be an unreasonable concern in the absence of breastfeeding, which has typically accompanied cosleeping. A mother's full breasts serve as a major physical constraint on her rolling over, thus preventing her from overlying her infant. McKenna dismisses parents' concerns with overlying as a "remote possibility." SIDS is also a remote possibility: 2 (or fewer) instances in 1000 infants. There are no statistics on overlying where cosleeping is generally practiced. In cosleeping circumstances, it has always been impossible to discriminate between the occurrence of SIDS and overlying. . . .

This brings me to consideration of the more specific hypothesis, that social sleeping may reduce SIDS. The strategies of science involve hypothesis testing based on evidence from previous hypothesis confirmation. Until and unless clear confirmation of the fundamental, more general hypothesis, discussed above, is obtained, any postulation based on that hypothesis is clearly in the realm of speculation. Thus, proposing the specific hypothesis, and efforts at testing such a hypothesis, does not derive from a logical model-building strategy. . . .

The crosscultural data that are cited in support of the idea of social sleeping as a protection against SIDS are not convincing. The differences in rates between countries with relatively high and relatively low rates are so great as to leave little variance that might possibly be accounted for by the mode of sleeping. . .

Clearly, however, there is a serious need for research concerning environmental factors that play a role in SIDS. This is a most difficult problem because we do not yet know what it is about the baby that must be prevented, or modified -- other than the final tragic event. And the event is an extremely rare one in any population. Thus, to investigate this phenomenon and the role of the social environment in regulating physiological rhythms, animal models offer the greatest hope for serious progress (see McGinty and Harper 1974; Baker and McGinty 1977; Bowes et al. 1980).

The major argument against animal models is the "uniqueness" hypothesis about humans. Thus, McKenna's scholarly review of the literature delineates uniqueness in the anatomy and developmental neurophysiology for language in humans. However, the data do not warrant the conclusion that *SIDS* is unique to humans. It should be noted that handedness and brain laterality were also considered at one time to be unique to humans, and specific to the development of language. It is now known that neither of these is a uniquely human characteristic (see review by Denenberg 1981). Given the commonalities in brain structures and function and the presence of homologous behaviors across species, it seems premature to dismiss the possibility of an animal model for SIDS, even among lower

mammalian species. Clearly an animal model is most appropriate for investigating the more general hypothesis of the role of parental entrainment postnatally.

IX. My Response: Conflict and Congruence (as published)

. . . The most serious criticisms of this SIDS research model and perspective are put forth by three of six commentators most closely associated with SIDS research. They raise pertinent paradigmatic, interpretive, and methodological issues and in several instances discuss the possible implications of my working hypothesis for parents of SIDS victims and raise for further discussion the issue of parent-infant cosleeping. I would like, therefore, to respond in detail to their criticisms and concerns and in so doing, and wherever possible, to make use of the insights provided by other commentators. . . .

Hoppenbrouwers and Hodgman's Alternative Explanation

. . . In order to understand, as Drs. Hoppenbrouwers and Hodgman seek to do, the bases of postnatal adaptive failure, is it not just as important and logical, in fact, to their own argument to consider carefully the effect that deviations from the evolutionarily expected environments might have -- microenvironments within which infant vulnerability, i.e., "vestigial" births, originally could have been favorably selected? The relationship between cortical depression, failure to arouse, and hypoxia leading to death could be made more clear, I think, by considering the coevolving postnatal environments that act to minimize the number of adaptive failures. If it is theoretically justifiable to use evolutionarily based characteristics to help understand infant vulnerability in the first place, it seems equally justifiable and indeed necessary to consider the evolved developmental setting in which this vulnerability is offset or counteracted. After all, if wakefulness constitutes a strong stimulus to breathing and failure to awaken from sleep may jeopardize breathing -- as Drs. Hoppenbrouwers and Hodgman point out, why is it not important to understand what socioenvironmental conditions foster wakefulness, i.e., the effects, for example, of parent-infant cosleeping?

On Parent-Infant Cosleeping and Suffocation from a Systems View

. . . Dr. Thoman suggests that the issue of parent-infant cosleeping should be discussed from a systems approach -- one that considers the interacting constellations of family circumstances, including parental attitudes, knowledge, psychological and even physiological statuses -- a position I fully support. But this, I believe, is clearly a separable set of issues, no less important than, yet different from the question of whether under normal circumstances of high parental responsibility some or all infants benefit physiologically from nocturnal parent-infant contact, or whether the sensory cues to which infants have access in

evolutionarily stable environments can be expected to aid them in overriding breathing control errors.

I do not, moreover, mean to dismiss altogether the possibility that the number of infants possibly saved from SIDS by cosleeping may be offset by the special new dangers infants face in cosleeping in urban environments wherein drug-insensitized sleeping companions, parental obesity, or soft mattresses create increased chances of suffocation (see Bass et al. 1986). This may be a real possibility, and so careful discussion of whether, or if, and under what circumstances parent-infant cosleeping is encouraged should be done on a case-by-case basis; but still, if we are attempting to understand SIDS dynamics and its proximate unfolding pathogenesis, then the significance of an evolutionarily based adaptation to cosleeping and the physiological regulatory effects that cosleeping potentially offers must at some point be recognized. . . .

On Animal Models of SIDS and Human Uniqueness

I did not mean to suggest that because SIDS may be a species-specific malady that the search for an animal model of SIDS should not continue. Rather my point is that it is critical to consider the ways in which unique human developmental characteristics coalesce with evolutionarily novel environmental circumstances to create crib death risk. We should be prepared, however, for the possibility that an animal model will not be found.

Nonhuman primates offer excellent subjects to study because of the high degree of cortically based vocalizing and breathing control, coupled with the slow development of infants who, like humans, are physiologically affected by parental contact. It may be that diving sea mammals (whales, dolphins, and seals) are also particularly appropriate to study because of their unique breathing control system, which is partly reflexive and partly under voluntary control -- a pattern reminiscent of our own.

Dr. Laitman's experimental and comparative studies of the mammalian laryngeal tract, including his studies of human infants, further confirm the uniqueness of the human being with respect to developmental changes related to our communication that in the first six months of life create increased respiratory system instability. He stresses that no other mammal undergoes "such a radical transformation" in the anatomy of the respiratory tract. His studies show that it is not until 1-1/2 to 2 years of life that a true descent of the larynx begins; but interestingly, and I suppose predictably, at the same time as voluntary breathing in the form of speech breathing is being mastered, the infant begins to exhibit oral tidal respiration, which possibly contributes to the instability of respiratory behavior during this time period.

The Possibility of SIDS Prevention: The "Blame Factor"

Only parents of SIDS victims can ever really appreciate what it means to lose an infant to this syndrome. Parental feelings of guilt are common since

no physical, structural, or anatomical "cause" can be pinpointed to provide a context within which parents can begin to come to grips with, or sort out, what has happened to them upon the death of their infant. Hence, health professionals and SIDS researchers alike are exceedingly sensitized to the need for counseling, to help ameliorate feelings of guilt or blame. Drs. Hoppenbrouwers and Hodgman worry about whether this research model and hypothesis might cause parents of SIDS victims to blame themselves further; they suggest therefore that this model has a serious "drawback" to it.

Without a doubt this is an issue around which much thought has been and should be given. As I have been careful to point out, this model does not deal with causes of SIDS, but stresses factors that may increase or decrease the ease by which primary defects find expression in the form of SIDS. The heterogeneity of the epidemiological data on SIDS suggests that the environmental components I outlined are suggested to be relevant only to certain subclasses of SIDS victims or future victims. Nobody who interprets my model correctly can assume that any particular SIDS victims could have been saved, or that infants necessarily are automatically protected by parental contact in the form of cosleeping.

But the issue is actually much larger and more complex than this. The belief underlying all SIDS research is that even though we do not understand SIDS now, nor can we prevent it now, aggressive research will continue to delineate the range of its organic and environmental bases to that eventually it can be prevented altogether, or its distribution reduced. The question of prevention and how to handle descriptions of SIDS and explanations to parents of SIDS victims, in the face of the fact that none are available, has led to serious and bitter disagreements within the SIDS research community itself (see Guntheroth's 1982 discussion, for example). Aspects of this "drawback factor" are certainly not new, or applicable to my theory alone. In fact, it can be argued that in an important way the level of sensitivity required to help parents of SIDS victims cope with their loss has influenced SIDS research protocols and the general sociopolitical climate within which SIDS researchers operate (see Guntheroth 1982). A tension exists between the claims that SIDS cannot be prevented to ease the plight of grieving parents on one hand, and the critical belief in the need for ongoing work that eventually might eliminate SIDS or lower its frequency on the other.

Because my research emphasis breaks from traditional ones insofar as it brings specifically into focus factors over which parents assert some control and which, better yet, they can understand, this perspective lends itself to immediate controversy. Seemingly immune from the kind of controversy this model generates are, first, research that focuses more on constitutional factors such as those proposed by pathologists, neurophysiologists, respiratory physiologists, and sleep researchers -- factors outside both the control and understanding of parents -- and, second, theories that are more consistent with the traditional SIDS hypotheses which assume that constitutional factors occur independently of interacting environmental components and suggest the compelling primacy of internal controlling mechanisms reducible to a closed as compared with an open

interactional system, or one in which variables are seen to occur in mutual interaction (von Bertalanffy 1969).

It may be that the "closed system approach" is an appropriate research strategy and yet, from my reading of the epidemiological data, and from my knowledge of the evolution of human infant development and the clinical data, I believe that this approach is unlikely to yield results that can explain *all* SIDS deaths and that the interactional model is just as appropriate to employ. The difference between my hypotheses and the more traditional ones, then, is not simply a case of one and not the other practicing logical model building. Rather the difference reflects different points in the historical development of a theory and how strongly each of the paradigms is able to accept each other's basic underlying assumptions -- around which the model can be built, tested, and then modified.

One simple truth is that investigators could never *not* examine legitimate questions that emerge from diverse ongoing research. Perhaps it is ultimately healthy that investigators from different fields looking at the same problem differ as to what point in time, and under what evidential circumstances, research indicates that a change in research assumptions is necessary. Fortunately, it will be the validity of the proposed change itself rather than the particular way any investigator argues the change that will determine its usefulness, assuming, of course, that at least one person tests the model.

. . . From my own vantage point, the work described by Drs. Thoman, Hoppenbrouwers, and Hodgman is not in conflict with the evolutionary perspective, but rather represents different levels at which explanations can begin. For example, Drs. Hoppenbrouwers and Hodgman delimit SIDS explanatory components to proximate physiological mechanisms presumed to be independent of external influences, i.e., the failure to arouse due to cortical depression; Dr. Thoman's perspective based on her breathing teddy bear studies examines the effects of external breathing cues on human prematures and establishes that both internal and external factors are somehow linked. My own model provides an evolutionary based rationale, context, and set of mechanisms that explain why, in some cases at least, reference to external sensory cues is important, as well as the species-specific neurological adaptations for speech breathing and language that help explain why human infants are especially vulnerable to this syndrome in the first place.

I am very appreciative of this dialogue and realize that simply because I speak last does not mean that I have the final word. My sincere hope is that this paper will stimulate ongoing discussion and, most critically, some SIDS research that seriously incorporates an evolutionary conceptualization of the human infant.

X. Conclusions/Student Study Questions

Disagreements between scientists are common and certainly the reactions of other scientists, both positive and negative, can be instructive. Ultimately, conflict is necessary for reserch to proceed. Rarely, if at all, does any one person propose *the* right answer. Sometimes, many times, the right answer emerges little by little by way of the contributions of thousands of scientists. Not all the issues can be resolved, either. But this is part of science, too. Answers do not come in neat packages, nor are they always, if ever, completely satisfying, or what initially we thought they would be.

This perspective does not suggest that we return to the days when researchers assumed that all infants were passive protoplasmic blobs unable to engage with their environments -- unable to do anything but lie there. Clearly, what we have learned about infant abilities during the last thirty years has earned them the "amazing newborn" description so frequently used in infancy literature, video tapes, and film. But I do think that being *prepared to adapt* to a caregiver and being *already adapted*, are two different things; and that in our rush to recognize the infant as a competent individual, autonomous and independent of others, infant researchers have inadvertently confused one for the other. Cross-cultural, cross-species, and evolutionary data suggest that in our enthusiasm to "Americanize" infants by giving them as much autonomy as we can at the earliest possible age, we have pushed too far the notion of the infant's natural physiological independence from its caregiver. Sleep is just one behavior for which researchers, reflecting their own values, nonconscious ideologies, and expectations, have favored research strategies which assume the correctness of an accelerated developmental trajectory of infant sleep wherein prolonged and uninterrupted periods of consolidated sleep early in the first year of life are deemed safe, natural, and desirable.

But millions of years of evolution have shaped and designed the infant's developmental needs in conjunction with co-evolving parental caregiving behaviors. While versatile and flexible, the human infant's developmental systems are not infinitely malleable nor are all infants the same as to their own individual constitutional versatility. Moreover, infant developmental patterns and the timing of development or maturation cannot change as quickly as do patterns of caregiving, including sleeping arrangements, which change with prevailing cultural needs and values. The extent to which parents and infants physiologically influence each other while co-sleeping is a good reminder of some of these evolutionary realities. The clinical significance of this recent deviation is as yet unknown.

Study Questions:

1. What is SIDS? Why is it so difficult to study?

2. What are some of the known SIDS risk factors?

3. Why might Afro-American infants be at greater risk of dying from SIDS?

4. What is the reason for discussing monkeys and apes in the context of possible environmental factors involved in SIDS?

5. Bipedalism and SIDS? What is the connection to the research strategy advocated here?

6. Why are American infants encourage to sleep alone? Who makes these kinds of recommendations? Where does the ideology come from?

7. What specific theories emerging from SIDS research on respiratory behavior are most relevant to the hypothesis proposed here?

8. What is the connection between ideology and scientific research related to SIDS, as argued here?

9. Other than the ones mentioned, can you think of any other possible benefits of parent-infant co-sleeping?

10. Suppose that co-sleeping may help some infants to resist successfully a potential SIDS fatality. Would this finding justify a general recommendation, say by the Academy of American Pediatricians, that all parents sleep with their infants? What complicating factors might make this recommendation difficult?

11. To prove the hypothesis here, why couldn't we just use the cross-cultural data on SIDS rates?

12. I assert here that SIDS researchers should begin their research with a different conceptualization of the biological and social nature of the infant than they currently do. Why?

13. Hoppenbrouwers and Hodgman respond to my ideas as published in 1986 by stating that their position and mine are vastly different. I do not accept that; in fact, in my response I point out that the two perspectives are complementary and supportive of one another. What do you think? Why is it, do you suppose, that they feel the two approaches are so vastly different?

14. How would you respond to the suggestion that I should not publish this hypothesis since it is likely to make some SIDS parents feel that if they had only slept with their infants they would be alive? Is this what I'm saying?

15. Briefly describe the preliminary results of our laboratory studies. Do they prove anything about SIDS? Why did we conduct these studies if they cannot directly prove our hypothesis that co-sleeping should lower SIDS rates?

References

Anderson, J. R.
1984 Ethology and Ecology of Sleep in Monkeys and Apes. *In Advances in the Study of Behavior*, Vol. 14 J. S. Rosenblatt, C. Beer, M. C. Busnel, and P. J. Slater, eds. Pp. 166-229. Orlando: Academic Press.

Aries, Philippe
1962 *Centuries of Childhood.* New York: Vintage Books.

Baba, N., J. Quattrochi, C. Reiner, W. Adrion, P. T. McBride, and A.J. Yates
1983 Possible Role of the Brain Stem in Sudden Infant Death Syndrome. Journal of the American Medical Association 249:2789-2791.

Beal, S.M.
1983 Some Epidemiological Factors about Sudden Infant Death Syndrome (SIDS) in South Australia. *In Sudden Infant Death Syndrome.* J. T. Tildon, L. M. Roeder, and A. Steinschneider, eds. Pp. 15-28. New York: Academic Press.

Becker, P. T., and E. B. Thoman
1983 Organization of Sleeping and Waking States in Infants: Consistency Across Contexts. Physiology and Behavior 31:405-410.

Campbell, B.
1986 *Humankind Emerging.* New York: Scott Foresman.

Chiswick, M. L.
1985 Book Review of *Sudden Infant Death*, by Jean Golding, S. Limerick, and Aiden Macfarlane. Lancet (May 25):1193.

Darwish, H. Z., and D. D. McMillan
1983 Apnea in the Newborn. *In Topics in Neonatal Neurology.* R. Stralton, ed. Orlando: Grune and Stratton.

Davies, D. P.
1985 Cot Death in Hong Kong: A Rare Problem? *The Lancet*, Dec. 2(8468):1346-1349.

Einspieler, C., J. Widder, A. Holzer, and T. Kenner
1988 The Predictive Value of Behavioral Risk Factors for Sudden Infant Death. Early Human Development 18:101-109.

Elias, M. F., N. Nicholson, C. Bora, and J. Johnston
1986 Sleep/Wake Patterns of Breast-Fed Infants in the First Two Years of Life. Pediatrics 77(3):322-329.

Elias, M. F., N. Nicholson, and M. Konner
1987 Two Subcultures of Maternal Care in the United States. *In Current Perspectives in Primate Social Dynamics.* D. Taub and F. King, eds. Pp. 31-36. New York: Van Nostrand Reinhold.

Fardig, J. A.
1980 A Comparison of Skin to Skin Contact and Radiant Heaters in Promoting Neonatal Thermoregulation. Journal of Nurse-Midwifery 25:19-28.

Field, T. et al.
1986 Tactile/Kinesthetic Stimulation Effects on Preterm Neonates. Pediatrics 77:654-658.

Flandrin, Jean-Louis
1979 Families in Former Times: Kinship, Household and Sexuality. Cambridge: Cambridge University Press.

Fleagle, J.
1988 Primate Evolution. New York: Academic Press.

Foley, Robert
1987 *Another Unique Species: Patterns in Evolutionary Ecology.* London: Longman Company.

Froggatt, P.
1983 The Epidemiology of SIDS: Problems, Progress, and Prospects. *In Sudden Infant Death Syndrome.* J. T. Tildon, L. M. Roeder, and A. Steinschneider, eds. Pp. 85-88. New York: Academic Press.

Guntheroth, W.
1990 *Sudden Infant Death Syndrome.* New York: Futura Press.

Haddad, G. G., and R. B. Mellins
1983 Cardiorespiratory Aspects of SIDS: An Overview. *In Sudden Infant Death Syndrome.* J. T. Tildon, L. M. Roeder, and A. Steinschneider, eds. Pp. 357-374. New York: Academic Press.

Harper, R. M., B. Leake, H. Hoffman, D. O. Walter, T. Hoppenbrouwers, J. Hodgman, and M. B. Sterman
1981 Periodicity of Sleep States Is Altered in Infants at Risk for the SIDS. Science 213:1030-1032.

Hillman, L.
1984 Maternal and Newborn Medical Factors. Paper presented at the International Symposium on Sudden Infant Death Syndrome, 17th Annual Intra-Science Symposium, February 22-24, Santa Monica, California.

Hoffman, H.
1984 Adverse Reproductive Factors. Paper presented at the International Symposium on Sudden Infant Death Syndrome, 17th Annual Intra-Science Symposium, February 22-24, Santa Monica, California.

Hoffman, H. J., K. Damus, L. Hillman, and E. Krongrad
1988 Risk Factors for SIDS: Results of the National Institute of Child Health and Human Development SIDS Cooperative Epidemiological Study. *Sudden Infant Death Syndrome: Cardiac and Respiratory Mechanisms and Interventions.* P. Schwartz, D. Southall, and M. Valdes-Dapena, eds. Annals of the New York Academy of Science 533:13-30.

Jolly, A.
1986 *Evolution of Primate Behavior.* New York: MacMillan.

Kagan, J.
1984 *The Nature of the Child.* New York: Basic Books.

Kahlia, M. P.
1981 Anatomical Organization of Central Respiratory Nerves. Annual Review of Physiology 43:105-120.

Kellum, Barbara A.
1974 Infanticide in England in the Later Middle Ages. History of Childhood Quarterly: The Journal of Psychohistory 1:367-388.

Konner, M. J., and C. Worthman
1980 Nursing Frequency, Gonadal Function and Birth-spacing among Kung Hunters and Gatherers. Science 207:788-791.

Kraus, J. F.
1984 Demographic and Socioeconomic Factors NICHD Cooperative Epidemiological Study of SIDS Risk Factors. Paper presented at the International Symposium on Sudden Infant Death Syndrome, 17th Annual Intra-Science Symposium, February 22-24, Santa Monica, California.

Kuhn, S.
1969 *The Structure of Scientific Revolutions*. Chicago: University of Chicago Press.

Lancaster, J. B., and C. S. Lancaster
1982 Parental Investment: The Hominid Adaptation. In How Humans Adapt: A Biocultural Odyssey. D. Ortner, ed. Washington, D.C.: Smithsonian Institution Press.

Lee, N. Y., Y. F. Chan, D. P. Davies, E. Lau, and D. C. P. Yip
1989 Sudden Infant Death Syndrome in Hong Kong: Confirmation of Low Incidence. British Medical Journal 298:721.

Lozoff, B., A. W. Wolf, and N. S. Davis
1984 Co-sleeping in Urban Families with Young Children in the United States. Pediatrics 74:171-182.

McCrary, Susan
1988 An Analysis of Factors Which Influence Co-Family Sleeping Senior thesis. Pomona College, Claremont, CA.

McKenna, J.
1986 An Anthropological Perspective on the Sudden Infant Death Syndrome (SIDS): The Role of Parental Breathing Cues and Speech Breathing Adaptations. Medical Anthropology 10(1). Special issue with commentaries.

McKenna, J., and S. Mosko
1990 Evolution and the Sudden Infant Death Syndrome (SIDS) Part III: Infant Arousal and Parent-Infant Co-sleeping. Human Nature 1:291-330.

McKenna, J., S. Mosko, C. Dungy, J. McAninch
1990 Sleep and Arousal Patterns of Co-sleeping Human Mother-Infant Pairs: A Preliminary Physiological Study with Implications for the Study of SIDS. American Journal of Physical Anthropology. Vol. 83, pp. 331-347.

Mitchell, R. A., and A. J. Berger
1981 Neural Regulation of Respiration. In Regulation of Breathing (Part 1). T. F. Hornbein, ed. Pp. 540-620. New York: Marcel Dekker, Inc.

Naeye, R. L.
1973 Pulmonary Arterial Abnormalities in Sudden Infant Death Syndrome. New England Journal of Medicine 289:1167-1170.

Norvenius, G.
1984 The Contribution of SIDS to Infant Mortality Trends in Sweden. Paper presented at the International Symposium on Sudden Infant Death Syndrome, 17th Annual Intra-Science Symposium, February 22-24, Santa Monica, California.

Peterson, D.
1983 Epidemiology of the Sudden Infant Death Syndrome: Problems, Progress, Prospects--A Review. In *Sudden Infant Death Syndrome.* J. T. Tildon, L. M. Roeder, and A. Steinschneider, eds. Pp. 89-98. New York: Academic Press.

Plum, F., and J. Leigh
1981 Abnormalities of Central Mechanisms. In Lung Biology in Health and Disease, Vol. 17. Regulation of Breathing, Part 1. C. Lenfant, E. Semes, and T. Hornbein, eds. Pp. 987-1067. New York: Marcel Dekker.

Quattrochi, J. J., N. Baba, L. Liss, and W. Adrion
1980 Sudden Infant Death Syndrome (SIDS): A Preliminary Study of Reticular Dendritic Spines in Infants with SIDS. Brain Research 181:245-249.

Ryerson, Alice
1961 Medical Advice on Child Rearing, 1550-1900. Harvard Educational Review 31:302-323.

Schwartz, P. J.
1983 Autonomic Nervous System, Ventricular Fibrillation, and SIDS. In Sudden Infant Death Syndrome. J. T. Tildon, L. M. Roeder, and A. Steinschneider, eds. Pp. 319-340. New York: Academic Press.

Shand, Nancy
1985 Culture's Influence in Japanese and American Maternal Role Perception and Confidence. Psychiatry 48:52-67.

Shostak, Marjorie
1981 Nisa: Story of a !Kung Woman. New York: Cambridge University Press.

Stanton, A. N.
1984 Overheating and Cot Death. The Lancet (Nov.):1199-1201.

Steinschneider, A.
1972 Prolonged Apnea and the Sudden Infant Death Syndrome: Clinical and Laboratory Observations. Pediatrics 50:646-654.

Stone, Lawrence
1977 The Family, Sex and Marriage in England, 1500-1800. New York: Harper & Row.

Valdes-Dapena, M. A.
1978 Sudden Infant Death Syndrome, 1970-1975. Washington, D.C.: U.S. Department of Health, Education, and Welfare. Publication no. 78-5255.

1980a Sudden Infant Death Syndrome: A Review of the Medical Literature, 1974-1979. Pediatrics 66(4):567-614.

1980b Sudden Infant Death Syndrome. Washington, D.C.: U.S. Department of Health, Education, and Welfare. Publication no. 80-5255.

Whoolley, P. V.
1945 Mechanical Suffocation during Infancy: Relation to Total Problem of Sudden Death. Journal of Pediatrics 26:572.