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SUDDEN INFANT DEATH SYNDROME

—by Robert M. Reece

Profound emotions are aroused when an infant dies. When an infant who was previously healthy and apparently normal dies unexpectedly and suddenly, the resulting anguish sweeps over a family in a tidal wave of grief, helplessness, doubt, suspicion, guilt, and anger. The inevitable and invariable question asked is, "Why did my baby die?" Answers are ambiguous, speculative, and shrouded in our ignorance as to the etiology of this age-old condition known variously as crib death, cot death, and sudden infant death. In this country and increasingly around the world, this phenomenon is becoming known as sudden infant death syndrome, or SIDS.

The inevitable and invariable question asked is, "Why did my baby die?" Answers are ambiguous, speculative, and shrouded in our ignorance as to the etiology of this age-old condition.

The sudden death of an infant is the most devastating of life events for young parents who, along with other family members and friends, have expectations, hopes, and dreams for the infant and his future. The typical scenario involves a previously apparently healthy infant who is put to bed after having been fed his usual diet of formula or breast milk. The child's parents, on retiring, look in on the baby and find him to be fine. In the morning, one of the parents goes to the baby's bed to get him up for a feeding and finds the baby immobile, unresponsive, often mottled in color, sometimes rigid and cold. In panic, the parent attempts to resuscitate the baby and summons help from the emergency responders in their locale. The rescue squad arrives, attempts resuscitation and then transports the baby to the hospital where the baby is ultimately pronounced dead. In some locales, support groups are available to help the parents through the initial shock and sometimes through long-term grieving. Also, depending on location and jurisdiction, a postmortem examination is conducted to ascertain the cause of death and a review is made of the death scene, medical history, and other factors surrounding the death.

The grieving reaction of parents and other family members runs the gamut from quiet resignation to uncontrolled emotional outbursts of grief, anger, and denial. Many SIDS parents blame themselves initially for doing something, or not doing something, that they feel could have contributed to the death their infant. Using parents' self-blame to raise the suspicion that they were perpetrators of infanticide is unjustified and unfair, since guilt is the norm in the excruciating first hours and days of the realization of the death.

The definition of SIDS, promulgated by the National Institutes of Child Health and Human Development in 1989, is "the sudden death of an infant under one year of age which remains unexplained after the performance of a complete postmortem investigation, including an autopsy, an examination of the scene of death and review of the case history." Another term heard in this field is Sudden Unexpected Death (SUD). This is a generic term describing an event but not suggesting a diagnostic category.

The NIH Consensus Statement on Terminol-

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ogy (1987) suggests the following terms and definitions:

Apnea. The cessation of respiratory airflow.

Pathologic apnea. A respiratory pause is abnormal if it is 20 seconds or longer or associated with: cyanosis; abrupt, marked pallor or hypotonia; or bradycardia (slowing of the heart).

Periodic breathing. A breathing pattern in which there are three or more respiratory pauses of greater than three seconds' duration with less than 20 seconds of respiration between pauses. Periodic breathing can be a normal event.

Apnea of prematurity. Periodic breathing with pathologic apnea in a premature infant. Apnea of prematurity usually ceases by 37 weeks post-conceptional age but occasionally persists for several weeks past term (40 weeks).

Apparent life-threatening event (ALTE). An episode that is frightening to the observer and is characterized by some combination of apnea, color change (usually cyanotic or pallid but occasionally erythematous (red) or plethoric (flushed), marked change in muscle tone (usually limpness), choking or gagging. In some cases the observer fears that the infant has died. Earlier terminology such as "aborted crib death" or "near-miss SIDS" should be abandoned.

Several so-called risk factors have been shown to be overrepresented in groups of future SIDS victims, but they are by no means invariable.

Apnea of infancy. An unexplained episode of cessation of breathing for 20 seconds or longer or a shorter respiratory pause associated with bradycardia, cyanosis, pallor and/or marked hypotonia. Apnea of infancy should be reserved for those infants for whom no cause of an ALTE can be identified.

Sudden Infant Death Syndrome is a recognized cause of death all over the world. It is, as yet, neither preventable nor predictable. It has been called a cause of death that is exclusionary of readily recognizable reasons for the death, but it has clinical, epidemiological, and postmortem characteristics that are typical and diagnostic.

Current statistics about the incidence of SIDS in the United States must be considered estimates. Over the past several years the annual rate of death from SIDS has remained relatively constant in the 5,000-6,000 range, representing a 1.2 to 1.5 per 1,000 live birth incidence. Incidence figures for other parts of the world vary from a low of 0.036 per 1,000 live births in Hong Kong (Davies, 1985) to 6.3 per 1,000 in Tasmania (Newman, 1986). Kraus and Bultreys, in a careful review of SIDS and socioeconomic status (SES), conclude that the evidence suggests a consistent inverse relationship between SIDS and SES, but suggest the possibility that SES acts as a confounder, effect modifier, or intermediate variable (Kraus and Bultreys, 1991).

The peak incidence of SIDS is between two and four months of age. Occasional deaths occur during the first month of life, and the deaths decrease in number after the third month of life. Ninety percent of all cases of SIDS occur by six months of age. It is more common in males, occurs more frequently in the winter months, and is seen more frequently in multiple births (twins and triplets). Recently, sleeping position has been found to influence rates of death due to SIDS, and evidence is mounting that the prone position is seen more often in babies dying of SIDS (Engelberts and DeJonge, 1990; Southall and Samuels, 1992; Wigfield et al., 1992).

Several so-called risk factors have been shown to be overrepresented in groups of future SIDS victims, but they are by no means invariable. These include maternal cigarette smoking during pregnancy, prematurity and low birth weight, and younger age of mothers. More future SIDS babies have had thrush, pneumonia, and illnesses requiring hospitalization. More have histories of episodes of rapid respiration, rapid heart rate, cyanotic spells, or vomiting during the newborn period. Autopsy findings in SIDS babies show increased blood-forming activity in the liver, and gliosis (reaction to insult) in the brainstem, but these findings have not been universal. Despite the occurrence of these factors, it is still not possible for future SIDS victims to be identified prior to the terminal event. Another potential risk factor considered in earlier speculation about etiology was that of familial propensity to SIDS. Was there a genetic or at least a familial tendency in SIDS? Several studies have examined the possibility of recurrent SIDS within families. The best studies have concluded that it is highly unlikely to have a familial recurrence of SIDS and there is no demonstrable genetic etiology.

Theories of etiology

Research into the causes of this medical mystery has involved investigation of practically every organ system of the body. Because the terminal event is a cessation of breathing and heart activity, it was only natural that these two organ systems were the focus of most serious attention in the 1970s and 1980s. Steinschneider (1972) began the quest into a respiratory etiology when he reported on two patients with prolonged apnea and cyanotic spells who subsequently died, presumably of SIDS. This report and others gave rise to the "apnea hypothesis," which stated that prolonged and recurrent apneic episodes, even when not observed, were the precursors to SIDS. If infants with these spells could be identified by pneumograms (breathing recordings) and equipped with home monitoring devices that would signal when abnormal respiratory or cardiac events occurred, then the parents could stimulate and/or resuscitate the baby and prevent death. This approach was tried in several centers for several years, but it failed change the rate of SIDS, and despite careful monitoring, there are anecdotal

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reports of infants dying while on home monitors. The belief in the apnea hypothesis began to diminish when Southall and colleagues demonstrated in a prospective study of over 9,000 infants that none of the 29 future SIDS victims had had abnormalities prior to death either clinically or by pneumogram/electrocardiogram. The current consensus is that the overlap between SIDS and apnea is at most 5% because most infants dying of SIDS have had no previous apneic spells as manifested either clinically or by pneumogram (Southall et al., 1983).

Another theory, upper airway obstruction leading to prolonged apnea, also has adherents. This theory claims that pharyngeal obstruction, caused by backward falling of the tongue, reflex laryngospasm due to gastroesophageal reflux, pharyngeal collapse during sleep, or neck flexion leads to obstruction of the airway. This in turn leads to carbon dioxide accumulation in the bloodstream. In the older child and adult, carbon dioxide stimulates the respiratory center of the brainstem to increase respiratory effort. The respiratory center in infants is relatively unresponsive to carbon dioxide levels, and this, so the theory goes, is what leads to fatal apnea. Supporters of this concept cite the presence of intrathoracic petechiae (blood spots) in the thoracic viscera seen in the postmortem examination of SIDS victims as highly suggestive of a terminal event involving high intrathoracic pressures, a condition compatible with obstruction. Although sleep-related upper airway obstruction can occasionally be responsible for cyanotic episodes, the parents of SIDS victims do not report these as part of their experience at the time of death and it would seem reasonable to expect clinical findings suggesting this.

Lethal cardiac arrhythmias (irregularities of the heartbeat) were postulated as a silent cause of SIDS, based on data showing electrocardiographic abnormalities in some future SIDS victims. Currently there is no consensus on the importance of cardiac etiologies in most SIDS cases.

All of the countries that experienced a rapid decline in prone sleeping also had reductions of approximately 50% in their SIDS rates.

The influence of toxic agents on the developing fetus has received increasing scrutiny since Naeye et al. (1976) first noted that 59% of mothers of SIDS victims smoked during pregnancy. Haglund and Cnattingius (1990) found that maternal smoking doubled the risk of SIDS in a study of nearly 280,000 infants. There is no doubt that there is an increased risk, to the extent that the National Institute for Children's Health and Human Development showed that maternal cigarette smoking during pregnancy carried the single strongest statistical association with future SIDS victims.

Substance abuse during pregnancy has also been shown to increase significantly the risk of SIDS. Infants born to mothers who use drugs during pregnancy are said to have a 5 to 10 times increased risk of dying of SIDS (Chasnoff et al., 1985; Davidson-Ward et al., 1990; Durand, Espinoza, and Nickerson, 1990). Before single agent causation is established, attention needs to be paid to confounding variables such as SES, prematurity, race, and crowded living conditions, as well as the timing and the dosage of the drugs and the presence of numerous other risk factors such as polydrug use and concomitant cigarette smoking.

Overheating and hyperthermia have been thought to be responsible for "febrile apnea." One study (Posonby et al., 1992), using a case-control method, examined 41 SIDS victims by measuring thermal conditions at the death scene and at the scene of last sleep for control infants. A questionnaire was also completed by the parents in both groups. In this study, the SIDS group had more excess thermal insulation for their given room temperatures than the matched controls.

Several retrospective studies have implicated the prone sleeping position as a contributor to SIDS (Engelberts and DeJonge, 1990; Southall and Samuels, 1992; Wigfield et al., 1992). These studies have prompted the American Academy of Pediatrics to issue a Policy Statement advising parents to place their babies in the supine (face up) position for sleep. The National Institutes of Health reported recently (Willinger, Hoffman, and Hartford, 1994) on their review of the current data regarding sleeping position and SIDS. The trends in SIDS rates from 1980 through 1992 in Australia, Britain, New Zealand, the Netherlands, Norway, Sweden and the United States were evaluated. All of the countries that experienced a rapid decline in prone sleeping also had reductions of approximately 50% in their SIDS rates. The major behavioral change in all targeted populations was in sleep position, with no significant changes being observed in the proportion of parents who smoked cigarettes or who breastfed.

If the SIDS rate does diminish over the next several years, it will be a challenge to determine whether it is due to altered sleeping position, decreased rates of prematurity, a reduction in cigarette smoking during pregnancy, changing patterns of substance abuse, an improvement in socioeconomic status, or other unknown factors.

The brainstem hypothesis postulates that SIDS occurs because of a defect in the respiratory and/or cardiovascular centers of the brainstem. Maturational factors are thought to play a role and this is consistent with the age distribution in SIDS. Kinney et al. (1991) found delayed myelination (the "insulation" layer of the nerves) in a group of SIDS infants in 25 of 62 white matter sites examined. They theorized that insults to these areas began prenatally and continued postnatally, and that delayed myelination most likely reflects a chronic underlying disorder that shares a common anteced-

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ent with sudden death, but is not directly responsible for it. Several possibilities are suggested: chronic hypoxemia; maternal cigarette smoking; nutritional deficiency; or inborn errors of metabolism inhibiting myelin formation. This theory has much appeal since it unifies the epidemiological observations, the individual and collective clinical and medical histories of SIDS cases, and the postmortem findings into an evolving sequence of events leading to a final common pathway for death.

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ACCIDENTAL INJURIES

—by Kenneth W. Feldman

It is important for pediatricians treating abused and neglected children to be knowledgeable in unintentional as well as intentional injuries. Understanding patterns of normal childhood injury helps us recognize the exceptional patterns that result from abuse. Knowledge about unintentional injuries also provides an indirect means of understanding the forces and mechanisms of intentional injuries. Without them, we are dependent on extrapolation from animal and biomechanical studies alone. For example, studies of head injuries resulting from childhood falls and bicycle accidents have obvious implications for understanding abusive head injury thresholds. Although both accident scenarios begin with a linear deceleration, these initial forces often impart rotational decelerations on the brain similar to those causing injury in shaken and/or beaten infants. Unintentional injuries also interface with issues of child neglect. Where does the boundary between "acts of God" and caretaker negligence lie?

Understanding patterns of normal childhood injury helps us recognize the exceptional patterns that result from abuse.

Accidents are currently the leading cause of death between ages one and 24 years, although the death rate of 27.2 per 100,000 in 1989 is 29% less than that of 1975 (Hoekelman, 1992).

Motor vehicle accidents

Motor vehicle injuries continue to be the leading cause of accidental death in childhood, accounting for two-thirds of these deaths (Hoekelman, 1992). From 1975 through 1987, the death rates in auto accidents for children up through age 14 years declined slowly but steadily. It is encouraging that the rate for infants to four-year-olds declined from 4.5 to 3.7 per 100,000, associated with an increase in auto restraint use to 80% (Agran et al., 1990). However, rates in older adolescents have been more erratic, rising from a low in 1983 to 33 per 100,000 in 1987. In 1987, 31% of older adolescents involved in fatal crashes had elevated blood alcohol levels. At the same time, only 25% of older teens used seat belts. Injury rates for adolescents are 75 times the fatality rates.

A number of injury prevention strategies have been considered or implemented. Adolescent risk might be modified by raising the age at which a person may receive a driver's license or drink legally. Night driving curfews or license restrictions and lower blood alcohol laws for teens have been considered. Passenger protection has been addressed by uniform restraint laws and passive passenger protections such as air bags and automatic seat belts. Ignition lockouts could be devised to prevent starting the engine if the driver had detectable breath alcohol or was unable to complete a rapid dexterity task. Roadway design to minimize traffic conflict may also reduce injuries.

In addition to motor vehicle occupants, pedestrians and bicyclists are injured in motor vehicle accidents. An estimated 50,000 child pedestrians are injured and 1,800 die annually (Rivara, 1990). They accounted for 15% of unintentional fatal

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