



eNeonatal Review

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PROGRAM INFORMATION

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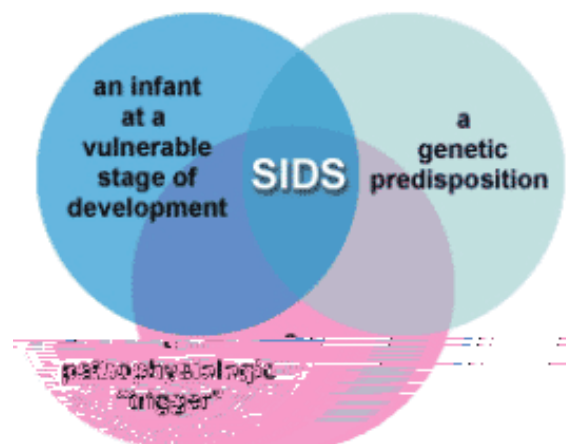
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Our focus this month is on the physiologic mechanisms of SIDS: Sudden Infant Death Syndrome. SIDS has been defined as the sudden unexpected death of an infant less than 1 year of age that remains unexplained after a thorough case investigation, including autopsy and forensic scene examination. After decades of research into the causal pathways of sudden infant death, one proposed model of disease is the "fatal triangle," where death occurs at the intersection of three susceptibilities:



This month's review will focus on the "trigger" arm of this model.

Numerous epidemiological studies have identified various factors associated with the occurrence of SIDS. Factors that are amenable to intervention include infant sleep position, smooth firm sleep surfaces, prevention of parental smoking (especially maternal), encouragement of breastfeeding, and prevention of hyperthermia. Public health interventions, such as the "Back to Sleep" Program, have focused on these factors and have been associated with a 40-50% reduction in the incidence of SIDS among many populations throughout the world.

Viral infections, impaired arousal from sleep, impaired cardiovascular control mechanisms, and genetic propensity are active areas of investigation in trying to identify mechanisms that may be the proximate cause of death; however, none as yet have been definitively linked.

The articles highlighted - published during the past few months - may help shed new light on potential SIDS death mechanisms.

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Edward E. Lawson, MD

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COMMENTARY

The Nordic Epidemiologic Study re-emphasizes the roles of sleep conditions, breast feeding, infections, and preventing exposure to cigarette smoke that have been the main foci regarding prevention of SIDS. As discussed in an editorial accompanying the study (see Reference 4 and link), the researchers demonstrated that a prone sleep position is more associated with day/evening deaths while night/morning deaths are more associated with infection and maternal smoking. The authors conclude that the epidemiological data from this study support the concept of multiple mechanisms for death in these infants. The papers we have chosen to emphasize demonstrate different potential mechanisms for these unexpected, and apparently sudden, infant deaths.

We have summarized several recent publications that emphasize the role of autoresuscitation in spontaneous recovery from apnea events preceding death. The mechanisms purporting to cause SIDS have been uncertain due to lack of accurate observations. The current articles support two mechanisms. One is the failure of infants to sustain gasping respiratory efforts that would result in recovery from loss of regular respiratory efforts. A second mechanism for onset of

apnea may be that due to sudden desaturations secondary to rebreathing. These mechanisms would be most involved in the deaths occurring during the daytime sleep states as described in the Nordic Epidemiologic study that are associated with prone sleep position.

Viral infections and their role in SIDS have been emphasized in a recent review by Martin Samuels (1). He cites numerous physiologic mechanisms that may incite sudden death including: chemosensory desensitization, onset of sudden hypoxemia, effects of hypoxemia on recovery from apnea, stimulation of laryngeal chemoreflex, co-infection with bacteria, and various mechanisms involving cytokines as intermediaries. The findings of Baasner et al uniquely identified a large subset of apparent SIDS victims with a presence of PCR positive material for parvovirus and enterovirus. Presence of viral DNA in myocardial tissue was demonstrated in one third of a series of patients dying of SIDS. Along with association with other respiratory illnesses, viral infections can be postulated to be associated with increase in circulating cytokines. The Kadhim paper indicates that IL-1 β may be present in brainstem nuclei associated with autonomic nervous system control. Using animal models, Olsson et al demonstrated direct evidence that IL-1 β depresses respiration and that survival during anoxia is reduced. These studies may be more relevant to the nighttime deaths that in the Nordic study were more likely associated with colds and smoking. Smoking can logically be assumed to have tissue inflammatory mechanisms as an intermediary (perhaps in addition to known effects of nicotine). Presumably, the mechanism by which increased brainstem cytokines affect the autonomic nervous system is inhibition of neurotransmission between the autonomic neurons that regulate the cardiorespiratory control systems.

The current state of knowledge regarding potential mechanisms has not led to a strategy for SIDS prevention, other than avoidance of known conditions supported by epidemiology studies. Nevertheless, much effort has been expended utilizing home apnea monitors in situations that are seemingly high risk for sudden unexpected infant deaths. Use of home monitors in these patients has not been demonstrated to effectively prevent infant deaths, though anecdotal experiences have demonstrated apparently successful infant resuscitations in situations that apparently would have lead to death. The recent American Academy of Pediatric Policy statement regarding home monitoring reviews these data and concludes that appropriate use of home monitoring has these characteristics: 1) a limited, predetermined use period, 2) use of monitors with event recorders, and 3) parent counseling that monitors do not prevent sudden death. In specific, the AAP Policy Statement does not support use of home monitoring simply for prematurely born infants having experienced prior apnea during their hospital stay. Recent papers indicate that use of home monitoring does not shorten hospital stay and that wide variations exist between NICUs on prescribing this therapy.

CIRCADIAN PATTERN

Daltveit AK, Irgens LM, Oyen N, et al. Circadian variations in sudden infant death syndrome: Association with maternal smoking, sleeping position and infections. The Nordic Epidemiological SIDS study. *Acta Paediatrica* 2003; 92:1007-1013.

Nordic investigators reported a circadian pattern of sudden infant death associated with several possible triggers, including maternal smoking, infant respiratory infection, and infant sleep position.

Investigators from Denmark, Norway, and Sweden conducted a questionnaire-based case-control study of the circadian variation in SIDS and associated risk factors. Two hundred and forty four SIDS victims and 869 control infants were recruited between September 1992 and August 1995. The questionnaire contained 272 items including information on pregnancy, infant care practices, and sociodemographic conditions. The main outcome variable was the hour found dead.

A significant circadian pattern was observed among 242 SIDS cases with known time found dead, with a peak incidence from 8:00 a.m. to 8:59 a.m., and a smaller peak from 2:00 p.m. to 2:59 p.m. Most cases were found dead between 6:00 a.m. and 11:59 a.m. (n=141, 58%). The proportion of infants found dead between 6:00 a.m. and 11:59 a.m. (night/morning SIDS) was lowest among infants of non-smoking mothers (39%) and highest among mothers who smoked more than 20 cigarettes per day (91%). The proportion of night/morning SIDS was also highest among infants of mothers with low education, infants with a reported cold, and infants sleeping side/supine. Among infants sleeping prone, there was also a circadian pattern, with prone

sleeping increasing the risk for both night/morning SIDS (odds ratio=11.0) as well as day/evening SIDS (odds ratio=21.6), compared with supine sleepers.

A methodological problem with this study is that the exact time of death is unknown, and therefore the study relies on the hour found dead. The bias is most likely to be largest in the morning, when caregivers wake from a night's sleep. However, when time of death is classified into 12 midnight-11:59 a.m. and 12 noon-11:59 p.m., the time of death for the majority of SIDS cases in this study (167 out of 242), based on both the time found dead and the time of last interaction with the infant before death, can be ascertained to have occurred during the night and morning hours.

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O₂ DESATURATION

Patel AL, Paluszynska D, Harris KA, Thach BT. Occurrence and mechanisms of sudden oxygen desaturation in infants who sleep face down. *Pediatrics* 2003; 111:e328-e332.

From the Washington University School of Medicine, Bradley Thach and his group reported an association between oxygen desaturation in the blood and both rebreathing and central apnea in infants who sleep in the prone position.

In this study, investigators studied the respiratory tracings and videotapes of 56 healthy 1- to 6-month-old infants who were sleeping face down and rebreathing on soft bedding in a sleep laboratory. Four of the infants were born prematurely, and were studied at a post-conception age of 292-377 days. An additional two infants were siblings of SIDS cases. The goal was to determine the frequency of failure to arouse and escape from the facedown position during periods of rapid desaturation in healthy infants.

The investigators compared the frequency of desaturation during rebreathing and nonbreathing periods, respiratory frequency and apnea occurrence before desaturation and nonbreathing control episodes, and minute ventilation changes during the desaturation episodes. Desaturation was defined as a decrease of at least 3% SaO₂, dropping to a level at or below 93% SaO₂ over 20 to 30 seconds. Rebreathing was defined as an elevation in inspired CO₂ level of at least 1%.

A total of 25 desaturation episodes occurred during the experiment, affecting 11 of the 56 study infants (20%). Spells did not occur in the SIDS siblings or the former preterm infants. Desaturation occurred more frequently during rebreathing than during nonbreathing periods. Respiratory frequency was not significantly different during the 10 seconds before desaturation than nonbreathing control periods. Eleven of the 25 desaturation episodes were preceded by 1 or more short apneas, of which all but one were central apneas. Average minute ventilation decreased during the desaturation episodes, and in 9 of the 17 desaturation episodes that could be evaluated, minute ventilation decreased by an average of 33% during the episode. Five desaturation events were preceded by a spontaneous shift in head position, which increased airway contact with the bedding during the 10 seconds before desaturation. Most episodes of desaturation resolved when the infant turned or lifted his/her head. However, 2 infants had desaturation below 85% resulting in intervention by the examiner.

The authors found that sudden small desaturations while rebreathing are not uncommon. These desaturations are not necessarily clinically relevant, but they could lead to potentially serious larger desaturations in infants who fail to escape or arouse. In infants who sleep in the prone position, desaturation occurred more often while they were rebreathing than not rebreathing. The authors concluded that in the low oxygen environment produced by the infant's face being covered by bedding, normal respiratory patterns might produce a rapid desaturation. When this is coupled with an arousal activity that is ineffective in gaining access to fresh air, such desaturation can be potentially lead to death.

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GENETIC/ACQUIRED AUTORESUSCITATION DEFECT

Sridhar R, Thach BT, Kelly DH, Henslee JA. Characterization of successful and failed autoresuscitation in human infants, including those dying of SIDS. *Pediatric Pulmonology* 2003; 36:113-122.

Investigators at Washington University and the Southwest SIDS research institute proposed that a genetic or an acquired defect in the autoresuscitation mechanism may be lethal for some infants when faced with hypoxia.

Using cardiorespiratory recordings of 24 infants who died suddenly while being monitored at home, the authors investigated physiologic mechanisms relevant to autoresuscitation from hypoxic apnea in infants dying suddenly and unexpectedly. Their aims were to determine if hypoxic gasping occurs prior to death in moribund infants, to determine if recovery from bradycardia is associated with gasping, and to determine if SIDS infants differ from cases with other diagnoses. The infants were aged 0.8 to 21 months, and the cardiorespiratory tracings were obtained from home monitoring companies (any patient identifiers were removed from the record). Of 41 recordings available, 17 were eliminated because resuscitative efforts created artifacts that obscured interpretation. Of the remaining 24 infant tracings, 10 belonged to infants born prematurely, who were being monitored for apnea. The others were being monitored for other reasons, including 4 who were siblings of SIDS cases, and 1 with a history of an acute life-threatening event. To serve as controls, 15 healthy infants were recruited for impedance pneumography in the sleep laboratory. The tracings of both the case infants and the control infants were analyzed for several parameters, including hypoxic gasp inspiratory and expiratory duration, gasp frequency, gasping duration, presence of partial or complete autoresuscitation, and double gasps or other unusual features.

The duration of hypoxic gasping, and the number of gasps, were highly variable, and no clear associations were seen. The case infants did, however, have a significantly shorter inspiratory time and longer expiratory time during their gasps. Double gasps occurred in 11 subjects, and almost always occurred at the onset of gasping. Infants with evidence of partial autoresuscitation had a sustained increase in heart rate during gasping, followed by continued gasping. Infants with evidence of complete autoresuscitation had a sustained increase in heart rate during gasping, followed by a return to a eupneic respiratory pattern.

Among the 5 SIDS cases, 4 infants had a double or triple gasp pattern in their tracings, compared to 1 out of 7 non-SIDS infants with these patterns ($p=0.04$). The SIDS infants also had longer average gasp duration than the other infants (8.4 minutes versus 6.5 minutes, respectively), but this difference was not statistically significant.

The authors concluded that hypoxic gasping is common in infants who are dying or are in danger of dying. This gasping can produce partial or complete autoresuscitation by introducing sufficient oxygen into the alveoli. Both groups had repeated gasps, but there were significantly more double and triple gasps in the SIDS infants, suggesting abnormal regulation of gasping in SIDS infants compared to controls. The SIDS infants in this study had no evidence of complete autoresuscitation, and only 1 of 5 infants had partial autoresuscitation. This was significantly different from non-SIDS infants, the majority of who showed some form of autoresuscitation. The authors speculated that SIDS infants may have a genetic or acquired defect in the autoresuscitation mechanism.

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VIRAL MYOCARDITIS

Baasner A, Dettmeyer R, Graebe M, Rissland J, Madea B. PCR-based diagnosis of enterovirus and parvovirus B19 in paraffin-embedded heart tissue of children with suspected sudden infant death syndrome. *Laboratory Investigation* 2003; 83:1451-1455.

German investigators proposed that viral myocarditis may be the trigger for sudden infant death.

Because myocarditis has previously been associated with SIDS, the researchers used polymerase chain reaction (PCR) to analyze myocardial tissue samples from 60 cases of SIDS and 36 cases of sudden death in children up to 10 years of age. The myocardial tissue was evaluated for enteroviruses, and parvovirus B19, two common causes of myocarditis in infancy. During post-mortem examination, each case had tissue sampling from two regions of the heart (the interventricular septum and posterior wall of the left ventricle). In addition, sampling of liver and splenic tissue occurred concurrently, and these samples were also tested for presence of enterovirus or parvovirus B19.

Enteroviruses were detected in 14 of 60 cases of suspected SIDS, and parvovirus B19 was found in 8 of the 60 cases. In contrast, no enterovirus was detected in samples from the control group, and parvovirus B19 was found in 3 of 36 controls (all above 1 year of age). Despite the detection of myocardial viral infection in 22 of the 60 SIDS cases, the tissue samples for only one patient fulfilled the Dallas criteria for myocarditis. Also of note, none of the liver or spleen tissue samples was positive for enterovirus or parvovirus.

The authors comment on the possibility that false-negative PCR assays may have limited the results of this study. The possibility of false-positive PCR assays was addressed by using immunohistochemical detection of enterovirus capsid protein VP1, which confirmed the diagnosis of enterovirus in 7 of the 8 cases. It is also possible that other viruses may be implicated in triggering sudden infant death by myocarditis, such as adenovirus or Epstein-Barr virus. In conclusion, the authors speculated that the myocardial pathology associated with SIDS might have resulted from an immune response triggered by a virus no longer present in the myocardium at the time of death.

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NEURONAL INHIBITION

Kadhim H, Kahn A, and Sébire G. Distinct cytokine profile in SIDS brain: A common denominator in a multifactorial syndrome? *Neurology* 2003; 61:1256-1259.

Belgian investigators demonstrated presence of high concentrations of interleukin immunoreactivity in the arcuate and dorsal vagal nuclei of SIDS victims

Interleukin 1 β (IL-1 β) was found in the brainstem arcuate and dorsal vagal nuclei in most tested SIDS victims, but only in a minority (<20%) of non-matched control infants.

Proinflammatory cytokines may exert neuromodulatory effects resulting in neuronal inhibition. The mechanisms of interaction of the cytokines with neurotransmitters in the brainstem is not known but presumed to be inhibition of neurotransmission. The region specific findings of this study correlate well with the known deficiency of acetylcholine receptor binding found in the arcuate nucleus of SIDS victims described by Kinney and her coworkers (2). Further, presence of cytokines may be associated with viral mediators of infection and may become central due to local production or by axonal transport from peripheral infected tissues.

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ACTIVATED IMMUNE SYSTEM

Olsson A, Kayhan G, Lagercrantz H, Herlenius E. IL-1 β depresses respiration and anoxic survival via a prostaglandin-dependent pathway in neonatal rats. *Pediatric Research* 2003; 54:326-331.

Researchers at the Karolinska Institute in Sweden reported that an activated immune system may be the predisposing condition for sudden death in a neonatal rat model.

Because infection, hypoxia, and apnea have each been implicated in the pathogenesis of SIDS, the pro-inflammatory cytokine IL-1 β may serve as the critical link between them. The investigators proposed that IL-1 β might affect brainstem respiratory control via a prostaglandin-dependent

mechanism. In the rat model, they studied the effects of IL-1 β , lipopolysaccharide (LPS), and indomethacin on respiratory activity during normoxia and anoxia.

Seven-day-old rats received an initial injection of NaCl or indomethacin, followed by a second injection at 30 minutes of NaCl, LPS, or IL-1 β . After the second injection, the animal was placed in a plethysmograph chamber and exposed to room air. The inlet gas was then changed to 100% nitrogen, and respiratory activity was recorded during anoxia. One minute after initiation of secondary apnea, 100% oxygen was administered into the chamber. Measurements included mean respiratory frequency, tidal volume and minute ventilation. In addition, the duration of hyperpnea, primary apnea, gasping, secondary apnea, and time required to autoresuscitate were also noted.

During normoxia, animals treated with IL-1 β exhibited a significantly lower respiratory frequency than control animals or animals treated only with indomethacin. A lower respiratory frequency during normoxia was correlated to a longer gasping duration, a depressed gasping frequency, and reduced survival. Indomethacin reversed the depressive effects of IL-1 β and LPS on respiratory frequency, but did not stimulate respiration above basal levels in control animals.

During anoxia, IL-1 β significantly lowered the total gasping frequency compared with control animals. Indomethacin did not prevent this depression in gasping. IL-1 β , LPS, and indomethacin had no effect on the duration of hyperpnea, primary apnea, or gasping. Both IL-1 β and LPS worsened recovery after anoxia. Survival was significantly lower in animals given IL-1 β than in control animals and animals treated with indomethacin. A similar result occurred in animals that received LPS. Pretreatment with indomethacin significantly improved the outcome of animals subsequently given IL-1 β and LPS.

The authors concluded that peripheral IL-1 β depresses respiratory frequency and worsens recovery from anoxic challenge in neonatal rats, and that these effects are attenuated by pretreatment with indomethacin. They proposed that an activated immune system might alter central respiration and autoresuscitation via a cytokine-induced, prostaglandin-mediated pathway.

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AAP POLICY STATEMENT

The American Academy of Pediatrics Committee on Fetus and Newborn. Apnea, sudden infant death syndrome, and home monitoring. *Pediatrics* 2003; 111:914-917.

The American Academy of Pediatrics policy statement discourages the use of home cardiorespiratory monitors for the prevention of SIDS, and encourages parents to use other methods to decrease their infant's risk for SIDS

There is no evidence that the presence of apnea and/or bradycardia identifies a group at increased risk for SIDS, that home cardiorespiratory monitoring can provide warning in time for intervention to prevent sudden death, or that intervention would be successful in preventing unexpected death. Given the lack of evidence that home monitoring has any impact on SIDS, prevention of SIDS is not an acceptable indication for home cardiorespiratory monitoring.

Home cardiorespiratory monitoring after hospital discharge may be prescribed for some preterm infants with an unusual prolonged course of recurrent, extreme apnea. The physician, together with the parents, should consider the potential advantages and disadvantages of home cardiorespiratory monitoring.

There are other groups of infants for whom use of a home cardiorespiratory monitor may be warranted, not because of an increased risk of SIDS, but because of other factors that increase the risk of sudden death. These include: Infants who have experienced acute life-threatening events, infants with airway abnormalities or tracheotomies, infants with neurological or metabolic disorders affecting respiratory control, and infants with chronic lung disease receiving home oxygen therapy or other respiratory support*. In these instances, home cardiorespiratory monitoring may allow the caregiver to respond more quickly to decrease the duration of

accompanying hypoxemia. However, such monitoring will not always prevent sudden death attributable to the triggering event or underlying condition.

Pediatricians should continue to promote proven practices that decrease the risk of SIDS: a supine sleep position, safe sleeping environments, and elimination of prenatal and postnatal exposure to tobacco smoke.

** or perhaps CNS damage due to repeated hypoxia - EEL*

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Samuels M. Viruses and sudden infant death. Paediatric Respiratory Reviews 2003; 4: 178-183.



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Kinney HC, Filiano JJ, Sleeper LA, et al. Decreased muscarinic receptor binding in the arcuate nucleus in sudden infant death syndrome. Science 1995; 269:1446-1450.



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Froen JF, Akre H, Stray-Pedersen B, Saugstad OD. Adverse effects of nicotine and interleukin-1beta on autoresuscitation after apnea in piglets: implications for the sudden infant death syndrome. Pediatrics 2000;105:E52.



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Mitchell EA and Williams SM. Does circadian variation in risk factors for sudden infant death syndrome (SIDS) suggest there are two (or more) SIDS subtypes? Acta Paediatrica 2003; 92: 991-3. Commentary accompanying paper by Daltveit et al.



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Holdsworth MT, Fichtl RE, Behta M, Raisch DW, Mendez-Rico E, Adams A, Greifer M, Bostwick S, Greenwald BM. Incidence and Impact of Adverse Drug Events in Pediatric Inpatients. Arch Pediatr Adolesc Med. 2003 Jan;157:60-65.



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- Evaluate the recent research presented to develop an understanding of the possible mechanisms of death in SIDS patients.
- Use the information presented herein as a basis to counsel parents regarding SIDS monitoring.
- Understand that home monitoring is not recommended as a blanket policy for prevention of SIDS.

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- Dr. Nogee has indicated a financial relationship of grant/research support with Forest Laboratories and has received an honorarium from Forest Laboratories.
- Dr. Lawson has indicated a financial relationship of grant/research support from the NIH. He also receives financial/material support from Nature Publishing Group as the Editor of the Journal of Perinatology.

All other faculty have indicated that they have not received financial support for consultation, research, or evaluation, nor have financial interests relevant to this e-Newsletter.

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