



TECHNICAL REPORT

SIDS and Other Sleep-Related Infant Deaths: Expansion of Recommendations for a Safe Infant Sleeping Environment

TASK FORCE ON SUDDEN INFANT DEATH SYNDROME

KEY WORDS

SIDS, sudden infant death, infant mortality, sleep position, bed-sharing, tobacco, pacifier, immunization, bedding, sleep surface

ABBREVIATIONS

CPSC—Consumer Product Safety Commission

AAP—American Academy of Pediatrics

SIDS—sudden infant death syndrome

SUID—sudden unexpected infant death

ICD—*International Classification of Diseases*

ASSB—accidental suffocation and strangulation in bed

5-HT—5-hydroxytryptamine

OR—odds ratio

CI—confidence interval

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

www.pediatrics.org/cgi/doi/10.1542/peds.2011-2285

doi:10.1542/peds.2011-2285

All technical reports from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Copyright © 2011 by the American Academy of Pediatrics

abstract

FREE

Despite a major decrease in the incidence of sudden infant death syndrome (SIDS) since the American Academy of Pediatrics (AAP) released its recommendation in 1992 that infants be placed for sleep in a non-prone position, this decline has plateaued in recent years. Concurrently, other causes of sudden unexpected infant death occurring during sleep (sleep-related deaths), including suffocation, asphyxia, and entrapment, and ill-defined or unspecified causes of death have increased in incidence, particularly since the AAP published its last statement on SIDS in 2005. It has become increasingly important to address these other causes of sleep-related infant death. Many of the modifiable and nonmodifiable risk factors for SIDS and suffocation are strikingly similar. The AAP, therefore, is expanding its recommendations from being only SIDS-focused to focusing on a safe sleep environment that can reduce the risk of all sleep-related infant deaths including SIDS. The recommendations described in this report include supine positioning, use of a firm sleep surface, breastfeeding, room-sharing without bed-sharing, routine immunization, consideration of a pacifier, and avoidance of soft bedding, overheating, and exposure to tobacco smoke, alcohol, and illicit drugs. The rationale for these recommendations is discussed in detail in this technical report. The recommendations are published in the accompanying “Policy Statement—Sudden Infant Death Syndrome and Other Sleep-Related Infant Deaths: Expansion of Recommendations for a Safe Infant Sleeping Environment,” which is included in this issue (www.pediatrics.org/cgi/doi/10.1542/peds.2011-2220). *Pediatrics* 2011;128:e1341–e1367

METHODOLOGY

Literature searches using PubMed were conducted for each of the topics in this technical report and concentrated on articles published since 2005 (when the last policy statement¹ was published). In addition, to provide additional information regarding sleep-environment hazards, a white paper was solicited from the US Consumer Product Safety Commission (CPSC).² Strength of evidence for recommendations³ was determined by the task force members. Draft versions of the policy statement⁴ and technical report were submitted to relevant committees and sections of the American Academy of Pediatrics (AAP) for review and comment. After the appropriate revisions were made, a

final version was submitted to the AAP Executive Committee and Board of Directors for final approval.

SUDDEN INFANT DEATH SYNDROME AND SUDDEN UNEXPECTED INFANT DEATH: DEFINITIONS AND DIAGNOSTIC ISSUES

Sudden Infant Death Syndrome and Sudden Unexpected Infant Death

Sudden infant death syndrome (SIDS) is a cause assigned to infant deaths that cannot be explained after a thorough case investigation that includes a scene investigation, autopsy, and review of the clinical history.⁵ Sudden unexpected infant death (SUID), also known as sudden unexpected death in infancy (SUDI), is a term used to describe any sudden and unexpected death, whether explained or unexplained (including SIDS), that occurs during infancy. After case investigation, SUIDs can be attributed to suffocation, asphyxia, entrapment, infection, ingestions, metabolic diseases, and trauma (accidental or nonaccidental). The distinction between SIDS and other SUIDs, particularly those that occur during an observed or unobserved sleep period (sleep-related infant deaths), such as accidental suffocation, is challenging and cannot usually be determined by autopsy alone. Scene investigation and review of the clinical history are also required. A few deaths that are diagnosed as SIDS are found, after further specialized investigations, to be attributable to metabolic disorders or arrhythmia-associated cardiac channelopathies.

Although standardized guidelines for conducting thorough case investigations have been developed,⁶ these guidelines have not been uniformly adopted across the more than 2000 US medical examiner and coroner jurisdictions.⁷ Information from emergency responders, scene investigators, and

caregiver interviews can provide additional evidence to assist death certifiers (ie, medical examiners and coroners) in accurately determining the cause of death. However, death certifiers represent a diverse group with varying levels of skills and education as well as diagnostic preferences. Recently, much attention has been focused on reporting differences among death certifiers. At one extreme, some certifiers have abandoned using SIDS as a cause-of-death explanation.⁷ At the other extreme, some certifiers will not classify a death as suffocation in the absence of a pathologic marker of asphyxia at autopsy (ie, pathologic findings diagnostic of oronasal occlusion or chest compression⁸), even with strong evidence from the scene investigation that suggests a probable accidental suffocation.

US Trends in SIDS, Other SUIDs, and Postneonatal Mortality

To monitor trends in SIDS and other SUIDs nationally, the United States classifies diseases and injuries according to the *International Classification of Diseases* (ICD) diagnostic codes. This classification system is designed to promote national and international comparability in the assignment of cause-of-death determinations; however, this system might not provide the optimal precision in classification desired by clinicians and researchers. In the United States, the National Center for Health Statistics assigns a SIDS diagnostic code (ICD-10 R95) if the death is classified with terminology such as SIDS (including presumed, probable, or consistent with SIDS), sudden infant death, sudden unexplained death in infancy, sudden unexpected death in infancy, or sudden unexplained infant death on the certified death certificate. A death will be coded as “other ill-defined and unspecified causes of mortality” (ICD-10 R99) if the cause of the death is reported as

unknown or unspecified. A death is coded as “accidental suffocation and strangulation in bed” (ASSB) (ICD-10 W75) when the terms “asphyxia,” “asphyxiated,” “asphyxiation,” “strangled,” “strangulated,” “strangulation,” “suffocated,” or “suffocation” are reported, along with the terms “bed” or “crib.” This code also includes deaths while sleeping on couches and armchairs.

Although SIDS was defined somewhat loosely until the mid-1980s, there was minimal change in the incidence of SIDS in the United States until the early 1990s. In 1992, in response to epidemiologic reports from Europe and Australia, the AAP recommended that infants be placed for sleep in a nonprone position as a strategy for reducing the risk of SIDS.⁹ The “Back to Sleep” campaign was initiated in 1994 under the leadership of the National Institute of Child Health and Human Development as a joint effort of the Maternal and Child Health Bureau of the Health Resources and Services Administration, the AAP, the SIDS Alliance (now First Candle), and the Association of SIDS and Infant Mortality Programs.¹⁰ The Eunice Kennedy Shriver National Institute of Child Health and Human Development began conducting national surveys of infant care practices to evaluate the implementation of the AAP recommendation. Between 1992 and 2001, the SIDS rate declined, and the most dramatic declines occurred in the years immediately after the first nonprone recommendations, consistent with the steady increase in the prevalence of supine sleeping (Fig 1).¹¹ The US SIDS rate declined from 120 deaths per 100 000 live births in 1992 to 56 deaths per 100 000 live births in 2001, representing a decrease of 53% over 10 years. However, from 2001 to 2006 (the latest year from which data are available), the rate has remained constant (Fig 1). In 2006, 2327 infants

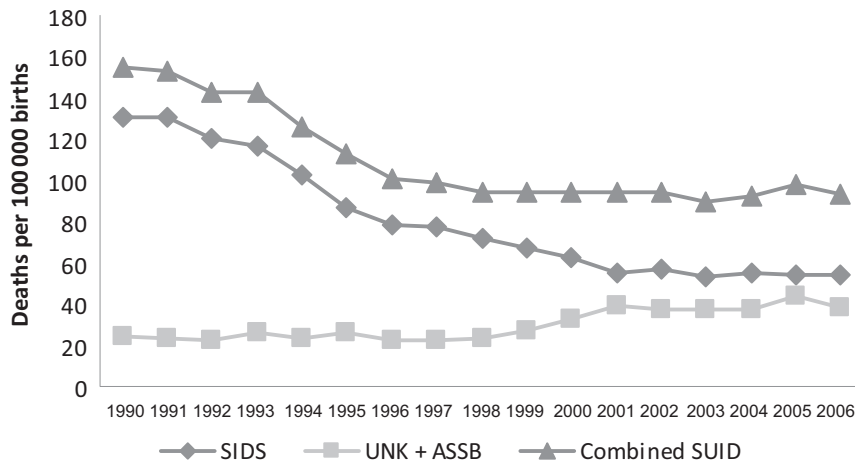


FIGURE 1

Trends in SIDS and other SUID mortality: United States 1990–2006. UNK indicates ill-defined or unspecified deaths.

died from SIDS. Although SIDS rates have declined by more than 50% since the early 1990s, SIDS remains the third-leading cause of infant mortality and the leading cause of postneonatal mortality (28 days to 1 year of age).

The all-cause postneonatal death rate has followed a trend similar to the SIDS rate: there was a 29% decline from 1992 to 2001 (from 314 to 231 per 100 000 live births). From 2001 until 2006, postneonatal mortality rates have also remained fairly unchanged (from 231 to 224 per 100 000 live births); the average decline is 3%.¹²

Several recent studies have revealed that some deaths previously classified as SIDS are now being classified as other causes of infant death (eg, accidental suffocation and other ill-defined or unspecified causes).^{13,14} Since 1999, much of the decline in SIDS rates might be explained by increasing rates of these other causes of SUID, particularly over the years 1999–2001.^{13,15} A notable change is in deaths attributable to ASSB. Between 1984 and 2004, ASSB infant mortality rates more than quadrupled, from 2.8 to 12.5 deaths per 100 000 live births,¹⁵ which represents 513 infant deaths attributed to ASSB in 2004 compared with 103 in 1984.

Sleep Position

The apparent leveling of the previously declining SIDS rate is occurring coincident with a slowing in the reduction of the prevalence of prone positioning. The prevalence of supine sleep positioning, as assessed from an ongoing national sampling, increased from 13% in 1992 to 72% in 2001. From 2001 until 2010, the prevalence of supine sleep positioning has been fairly stagnant (prevalence in 2010: 75%).¹¹

The 1998 and 2005 AAP policy statements and the Back to Sleep campaign not only addressed the importance of back sleeping but also provided recommendations for other infant care

practices that may reduce the risk of SIDS and other sleep-related infant deaths.^{1,9} Unfortunately, the ability to measure the prevalence of these other risk factors is limited by lack of data. Death certificates are useful for monitoring trends in SIDS mortality, but the circumstances and events that lead to death are not captured in vital statistics data.¹⁶ The Centers for Disease Control and Prevention recently began to pilot a SUID case registry that will provide supplemental surveillance information about the sleep environment at the time of death, infant health history, and the comprehensiveness of the death scene investigation and autopsy. These factors will better describe the circumstances surrounding SIDS and other sleep-related infant deaths and assist researchers in determining the similarities and differences between these deaths.

Racial and Ethnic Disparities

SIDS mortality rates, similar to other causes of infant mortality, have notable racial and ethnic disparities (Fig 2).¹⁷ Despite the decline in SIDS in all races and ethnicities, the rate of SIDS in non-Hispanic black (99 per 100 000 live births) and American Indian/Alaska Native (112 per 100 000 live births) infants was double that of non-Hispanic white infants (55 per 100 000)

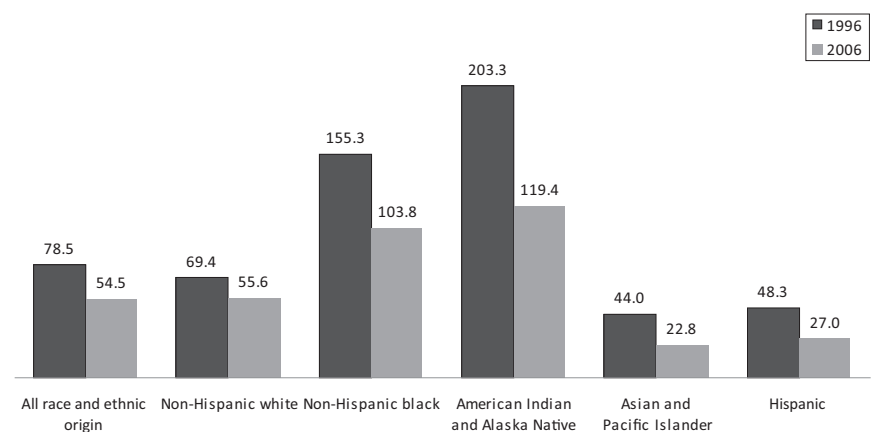


FIGURE 2

Comparison of US rates of SIDS according to maternal race and ethnic origin in 1996 and 2006.

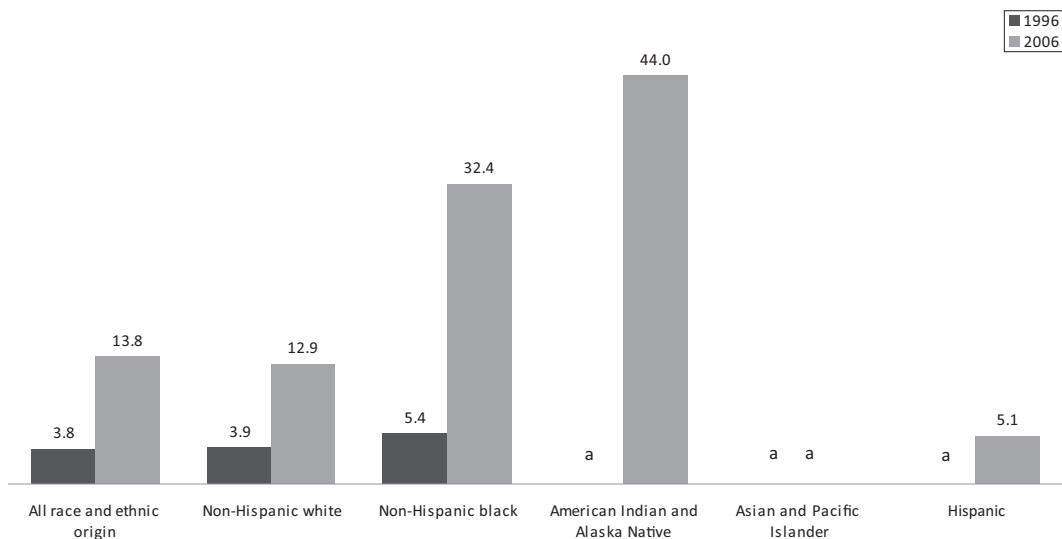


FIGURE 3 Comparison of US rates of death resulting from ASSB according to maternal race and ethnic origin in 1996 and 2006. ^a The figure does not meet standards of reliability or precision on the basis of fewer than 20 deaths in the numerator.

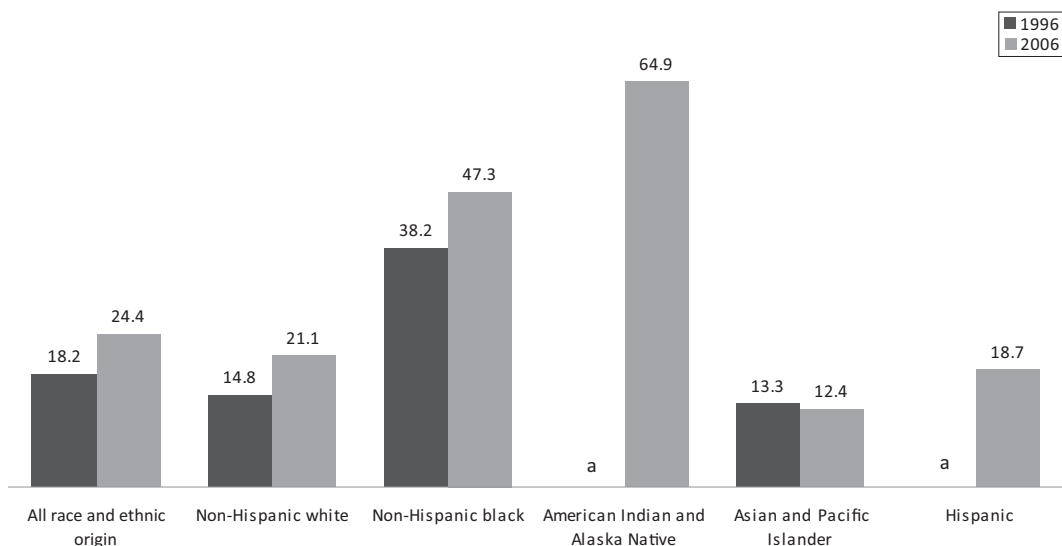


FIGURE 4 Comparison of US rates of cause ill-defined or unspecified death according to maternal race and ethnic origin in 1996 and 2006. ^a The figure does not meet standards of reliability or precision on the basis of fewer than 20 deaths in the numerator.

live births) in 2005 (Fig 2). SIDS rates for Asian/Pacific Islander and Hispanic infants were nearly half the rate for non-Hispanic white infants. Furthermore, similar racial and ethnic disparities have been seen with deaths attributed to both ASSB (Fig 3) and ill-defined or unspecified deaths (Fig 4). Differences in the prevalence of supine positioning and other sleep-environment conditions among ra-

cial and ethnic populations might contribute to these disparities.¹⁷ The prevalence of supine positioning in 2010 among white infants was 75%, compared with 53% among black infants (Fig 5). The prevalence of supine sleep positioning among Hispanic and Asian infants was 73% and 80%, respectively.¹¹ Parent-infant bed-sharing^{18–20} and use of soft bedding are also more common among black families

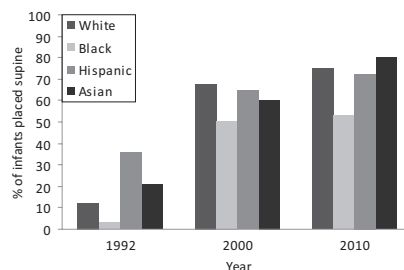


FIGURE 5 Prevalence of supine sleep positioning according to maternal race and ethnic origin, 1992–2010. Data source: National Infant Sleep Position Study.¹¹

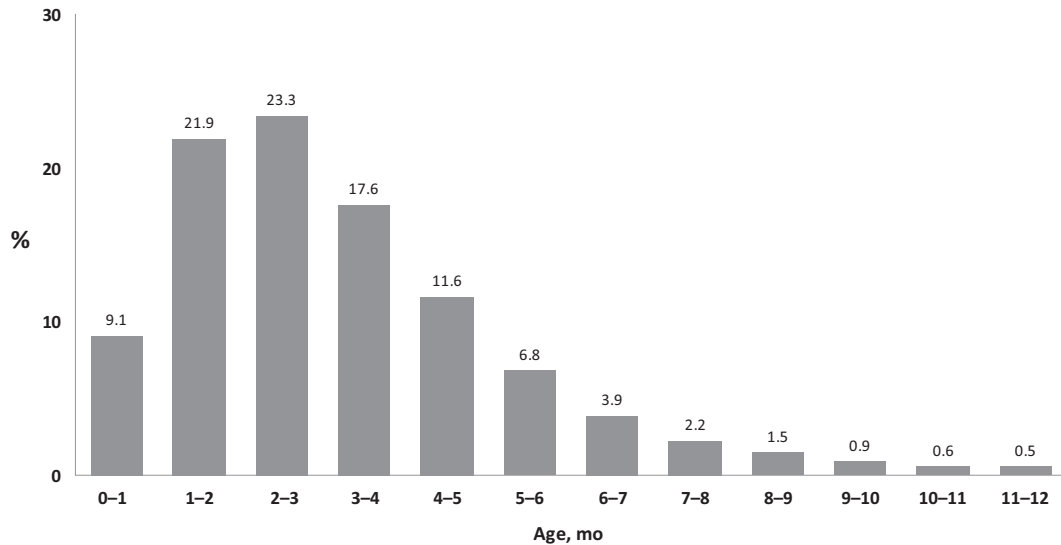


FIGURE 6
Percent distribution of SIDS deaths according to age at death: United States, 2004–2006.

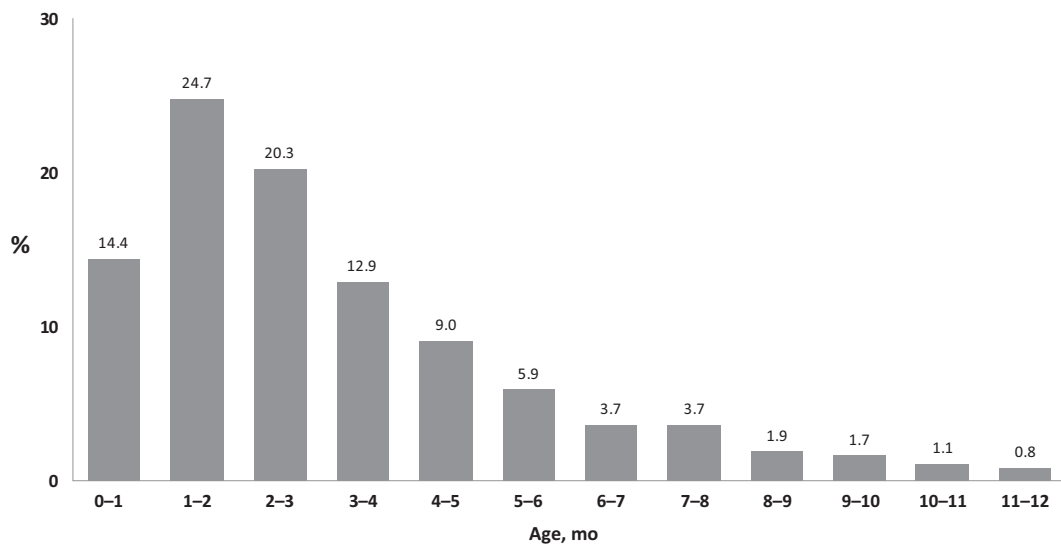


FIGURE 7
Percent distribution of deaths caused by ASSB according to age at death: United States, 2004–2006.

than among other racial/ethnic groups.^{21,22} Additional work in promoting appropriate infant sleep position and sleep-environment conditions is necessary to resume the previous rate of decline (observed during the 1990s) for SIDS and all-cause postneonatal mortality.

Age at Death

Ninety percent of SIDS cases occur before an infant reaches the age of 6 months. The rate of SIDS peaks be-

tween 1 and 4 months of age. Although SIDS was once considered a rare event during the first month of life, in 2004–2006, nearly 10% of cases coded as SIDS occurred during the first month. SIDS is uncommon after 8 months of age (Fig 6).¹⁴ A similar age distribution is seen for ASSB (Fig 7).

Seasonality of SIDS

A pattern in seasonality of SIDS is no longer apparent. SIDS deaths have historically been observed more fre-

quently in the colder months, and the fewest SIDS deaths occurred in the warmest months.²³ In 1992, SIDS rates had an average seasonal change of 16.3%, compared with only 7.6% in 1999,²⁴ which is consistent with reports from other countries.²⁵

PATHOPHYSIOLOGY AND GENETICS OF SIDS

A working model of SIDS pathogenesis includes a convergence of exogenous triggers or “stressors” (eg, prone

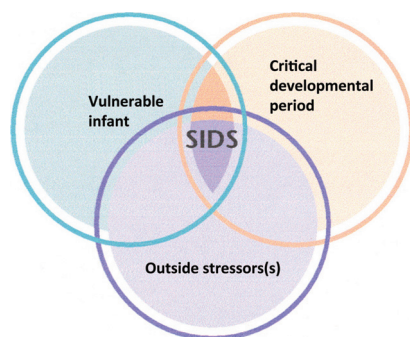


FIGURE 8
Triple-risk model for SIDS.²⁶

sleep position, overbundling, airway obstruction), a critical period of development, and dysfunctional and/or immature cardiorespiratory and/or arousal systems (intrinsic vulnerability) that lead to a failure of protective responses (see Fig 8).²⁶ Convergence of these factors ultimately results in a combination of progressive asphyxia, bradycardia, hypotension, metabolic acidosis, and ineffectual gasping, leading to death.²⁷ The mechanisms responsible for dysfunctional cardiorespiratory and/or arousal protective responses remain unclear but might be the result of in utero environmental conditions and/or genetically determined maldevelopment or delay in maturation. Infants who die from SIDS are more likely to be born at low birth weight or growth restricted, which suggests an adverse intrauterine environment. Other adverse in utero environmental conditions include exposure to nicotine or other components of cigarette smoke and alcohol.

Recent studies have explored how prenatal exposure to cigarette smoke may result in an increased risk of SIDS. In animal models, exposure to cigarette smoke or nicotine during fetal development alters the expression of the nicotinic acetylcholine receptor in areas of the brainstem important for autonomic function,²⁸ alters the neuronal excitability of neurons in the nucleus tractus solitarius (a brainstem region

important for sensory integration),²⁹ and alters fetal autonomic activity and medullary neurotransmitter receptors.³⁰ In human infants, there are strong associations between nicotinic acetylcholine receptor and serotonin receptors in the brainstem during development.³¹ Prenatal exposure to tobacco smoke attenuates recovery from hypoxia in preterm infants,³² decreases heart rate variability in preterm³³ and term³⁴ infants, and abolishes the normal relationship between heart rate and gestational age at birth.³⁵ Moreover, infants of smoking mothers exhibit impaired arousal patterns to trigeminal stimulation in proportion to urinary cotinine levels.³⁵ It is important to note also that prenatal exposure to tobacco smoke alters the normal programming of cardiovascular reflexes such that there is a greater-than-expected increase in blood pressure and heart rate in response to breathing 4% carbon dioxide or a 60° head-up tilt.³⁶ These changes in autonomic function, arousal, and cardiovascular reflexes might all increase an infant's vulnerability to SIDS. Brainstem abnormalities that involve the medullary serotonergic (5-hydroxytryptamine [5-HT]) system in up to 70% of infants who die from SIDS are the most robust and specific neuropathologic findings associated with SIDS and have been confirmed in several independent data sets and laboratories.^{37–40} This area of the brainstem plays a key role in coordinating many respiratory, arousal, and autonomic functions and, when dysfunctional, might prevent normal protective responses to stressors that commonly occur during sleep. Since the Task Force on Sudden Infant Death Syndrome report in 2005, more specific abnormalities have been described, including decreased 5-HT_{1A} receptor binding, a relative decrease in binding to the serotonin transporter, and in-

creased numbers of immature 5-HT neurons in regions of the brainstem that are important for autonomic function.⁴¹ These findings are not confined to nuclei containing 5-HT neurons but also include relevant projection sites. The most recent study report described in these same regions decreased tissue levels of 5-HT and tryptophan hydroxylase, the synthesizing enzyme for serotonin, and no evidence of excessive serotonin degradation as assessed by levels of 5-hydroxyindoleacetic acid (the main metabolite of serotonin) or ratios of 5-hydroxyindoleacetic acid to serotonin.³⁰ A recent article described a significant association between a decrease in medullary 5-HT_{1A} receptor immunoreactivity and specific SIDS risk factors, including tobacco smoking.⁴⁰ These data confirm results from earlier studies in humans^{39,41} and are also consistent with studies in piglets that revealed that postnatal exposure to nicotine decreases medullary 5-HT_{1A} receptor immunoreactivity.⁴² Animal studies have revealed that serotonergic neurons located in the medullary raphe and adjacent paragigantocellularis lateralis play important roles in many autonomic functions including the control of respiration, blood pressure, heart rate, thermoregulation, sleep and arousal, and upper airway patency. Engineered mice with decreased numbers of 5-HT neurons and rats or piglets with decreased activity secondary to 5-HT_{1A} autoreceptor stimulation have diminished ventilator responses to carbon dioxide, dysfunctional heat production and heat-loss mechanisms, and altered sleep architecture.⁴³ These studies linked SIDS risk factors with possible pathophysiology.

There is no evidence of a strong heritable contribution for SIDS. However, genetic alterations have been observed that may increase the vulnera-

bility to SIDS. Genetic variation can take the form of common base changes (polymorphisms) that alter gene function or rare base changes (mutations) that often have highly deleterious effects. Several categories of physiologic functions relevant to SIDS have been examined for altered genetic makeup. Genes related to the serotonin transporter, cardiac channelopathies, and the development of the autonomic nervous system are the subject of current investigation.⁴⁴ The serotonin transporter recovers serotonin from the extracellular space and largely serves to regulate overall serotonin neuronal activity. Results of a recent study support those in previous reports that polymorphisms in the promoter region that enhance the efficacy of the transporter (L) allele seem to be more prevalent in infants who die from SIDS compared with those reducing efficacy (S)⁴⁵; however, at least 1 study did not confirm this association.⁴⁶ It has also been reported that a polymorphism (12-repeat intron 2) of the promoter region of the serotonin transporter, which also enhances serotonin transporter efficiency, was increased in black infants who died from SIDS⁴⁴ but not in a Norwegian population.⁴⁵

It has been estimated that 5% to 10% of infants who die from SIDS have novel mutations in the cardiac sodium or potassium channel genes that result in long QT syndrome as well as in other genes that regulate channel function.⁴⁴ A recent report described important new molecular and functional evidence that implicates specific *SCN5A* (sodium channel gene) β subunits in SIDS pathogenesis.⁴⁷ The identification of polymorphisms in genes pertinent to the embryologic origin of the autonomic nervous system in SIDS cases also lends support to the hypothesis that a genetic predisposition contributes to the etiology of SIDS. There have

also been a number of reports of polymorphisms or mutations in genes that regulate inflammation,^{48,49} energy production,^{50–52} and hypoglycemia⁵³ in infants who died from SIDS, but these associations require more study to determine their importance.

ISSUES RELATED TO SLEEP POSITION

The Supine Sleep Position Is Recommended for Infants to Reduce the Risk of SIDS; Side Sleeping Is Not Safe and Is Not Advised

The prone or side sleep position can increase the risk of rebreathing expired gases, resulting in hypercapnia and hypoxia.^{54–57} The prone position also increases the risk of overheating by decreasing the rate of heat loss and increasing body temperature compared with infants sleeping supine.^{58,59} Recent evidence suggests that prone sleeping alters the autonomic control of the infant cardiovascular system during sleep, particularly at 2 to 3 months of age,⁶⁰ and can result in decreased cerebral oxygenation.⁶¹ The prone position places infants at high risk of SIDS (odds ratio [OR]: 2.3–13.1).^{62–66} However, recent studies have demonstrated that the SIDS risks associated with side and prone position are similar in magnitude (OR: 2.0 and 2.6, respectively)⁶³ and that the population-attributable risk reported for side sleep position is higher than that for prone position.^{65,67} Furthermore, the risk of SIDS is exceptionally high for infants who are placed on their side and found on their stomach (OR: 8.7).⁶³ The side sleep position is inherently unstable, and the probability of an infant rolling to the prone position from the side sleep position is significantly greater than rolling prone from the back.^{65,68} Infants who are unaccustomed to the prone position and are placed prone for sleep are also at

greater risk than those usually placed prone (adjusted OR: 8.7–45.4).^{63,69,70} Therefore, it is critically important that every caregiver use the supine sleep position for every sleep period.

Despite these recommendations, the prevalence of supine positioning has remained stagnant for the last decade.⁷¹ One of the most common reasons that parents and caregivers cite for not placing infants supine is fear of choking or aspiration in the supine position.^{72–80} Parents often misconstrue coughing or gagging, which is evidence of a normal protective gag reflex, for choking or aspiration. Multiple studies in different countries have not found an increased incidence of aspiration since the change to supine sleeping.^{81–83} There is often particular concern for aspiration when the infant has been diagnosed with gastroesophageal reflux. The AAP supports the recommendations of the North American Society for Pediatric Gastroenterology and Nutrition, which state that infants with gastroesophageal reflux should be placed for sleep in the supine position, with the rare exception of infants for whom the risk of death from gastroesophageal reflux is greater than the risk of SIDS⁸⁴—specifically, infants with upper airway disorders for whom airway protective mechanisms are impaired, which may include infants with anatomic abnormalities, such as type 3 or 4 laryngeal clefts, who have not undergone antireflux surgery. Elevating the head of the infant's crib while the infant is supine is not effective in reducing gastroesophageal reflux^{85,86}; in addition, this elevation can result in the infant sliding to the foot of the crib into a position that might compromise respiration and, therefore, is not recommended.

The other reason often cited by parents for not using the supine sleep position is the perception that the infant is uncomfortable or does not sleep

well.^{72–80} An infant who wakes frequently is normal and should not be perceived as a poor sleeper. Physiologic studies have found that infants are less likely to arouse when they are sleeping in the prone position.^{87–95} The ability to arouse from sleep is an important protective physiologic response to stressors during sleep,^{96–100} and the infant's ability to sleep for sustained periods might not be physiologically advantageous.

Preterm Infants Should Be Placed Supine as Soon as Possible

Infants born prematurely have an increased risk of SIDS,^{101,102} and the association between prone sleep position and SIDS among low birth weight infants is equal to, or perhaps even stronger than, the association among those born at term.⁶⁹ Therefore, preterm infants should be placed supine for sleep as soon as their clinical status has stabilized. The task force supports the recommendations of the AAP Committee on Fetus and Newborn, which state that hospitalized preterm infants should be placed in the supine position for sleep by 32 weeks' postmenstrual age to allow them to become accustomed to sleeping in that position before hospital discharge.¹⁰³ Unfortunately, preterm and very low birth weight infants continue to be more likely to be placed prone for sleep after hospital discharge.^{104,105} Preterm infants are placed prone initially to improve respiratory mechanics^{106,107}; although respiratory parameters are no different in the supine or prone positions in preterm infants who are close to discharge,¹⁰⁸ both infants and their caregivers likely become accustomed to using the prone position, which makes it more difficult to change. One study of NICU nurses found that only 50% of nurses place preterm infants supine during the transition to an open crib,

and more than 20% never place preterm infants supine or will only place them supine 1 to 2 days before discharge.¹⁰⁹ Moreover, very prematurely born infants studied before hospital discharge have longer sleep duration, fewer arousals from sleep, and increased central apneas while in the prone position.⁸⁸ The task force believes that neonatologists, neonatal nurses, and other health care professionals responsible for organizing the hospital discharge of infants from NICUs should be vigilant about endorsing SIDS risk-reduction recommendations from birth. They should model the recommendations as soon as the infant is medically stable and significantly before the infant's anticipated discharge. In addition, NICUs are encouraged to develop and implement policies to ensure that supine sleeping and other safe sleep practices are modeled for parents before discharge from the hospital.

Newborn Infants Should Be Placed Supine Within the First Few Hours After Birth

Practitioners who place infants on their sides after birth in newborn nurseries continue to be a concern. The practice likely occurs because nursery staff believe that newborn infants need to clear their airways of amniotic fluid and may be less likely to aspirate while on their sides. No evidence that such fluid will be cleared more readily while in the side position exists. Finally, and perhaps most importantly, if parents observe health care professionals placing infants in the side or prone position, they are likely to infer that supine positioning is not important¹¹⁰ and, therefore, might be more likely to copy this practice and use the side or prone position at home.^{77,80,111} The AAP recommends that infants be placed on their backs as

soon as they are ready to be placed in a bassinet.

Once an Infant Can Roll From the Supine to Prone and From the Prone to Supine Position, the Infant Can Be Allowed to Remain in the Sleep Position That He or She Assumes

Parents and caregivers are frequently concerned about the appropriate strategy for infants who have learned to roll over, which generally occurs at 4 to 6 months of age. As infants mature, it is more likely that they will roll. In 1 study, 6% and 12% of 16- to 23-week-old infants placed on their backs or sides, respectively, were found in the prone position; among infants aged 24 weeks or older, 14% of those placed on their backs and 18% of those placed on their sides were found in the prone position.¹¹² Repositioning the sleeping infant to the supine position can be disruptive and might discourage the use of supine position altogether. Although data to make specific recommendations as to when it is safe for infants to sleep in the prone position are lacking, the AAP recommends that these infants continue to be placed supine until 1 year of age. If the infant can roll from supine to prone and from prone to supine, the infant can then be allowed to remain in the sleep position that he or she assumes. To prevent suffocation or entrapment if the infant rolls, soft or loose bedding should continue to be removed from the infant's sleep environment. Some caregivers use such bedding to prevent an infant from rolling, but this bedding could cause suffocation and entrapment. Parents can be reassured by the information that the incidence of SIDS begins to decline after 4 months of age (Fig 6).

Supervised, Awake Tummy Time on a Daily Basis Can Promote Motor Development and Minimize the Risk of Positional Plagiocephaly

Positional plagiocephaly, or plagiocephaly without synostosis (PWS), can be associated with supine sleeping position (OR: 2.5).¹¹³ It is most likely to result if the infant's head position is not varied when placed for sleep, if the infant spends little or no time in awake, supervised tummy time, and if the infant is not held in the upright position when not sleeping.^{113–115} Children with developmental delay and/or neurologic injury have increased rates of PWS, although a causal relationship has not been demonstrated.^{113,116–119} In healthy normal children, the incidence of PWS decreases spontaneously from 20% at 8 months to 3% at 24 months of age.¹¹⁴ Although data to make specific recommendations as to how often and how long tummy time should be undertaken are lacking, supervised tummy time while the infant is awake is recommended on a daily basis. Tummy time should begin as early as possible to promote motor development, facilitate development of the upper body muscles, and minimize the risk of positional plagiocephaly. The AAP clinical report on positional skull deformities¹²⁰ provides additional detail on the prevention, diagnosis, and management of positional plagiocephaly.

SLEEP SURFACES

Infants Should Sleep in a Safety-Approved Crib, Portable Crib, Play Yard, or Bassinet

Cribs should meet safety standards of the CPSC, Juvenile Product Manufacturers Association, and the ASTM International (formerly the American Society for Testing and Materials), including those for slat spacing, snugly fitting and firm mattresses, and no drop sides.¹²¹ The AAP recommends the use of new cribs, because older

cribs might no longer meet current safety standards, might have missing parts, or might be incorrectly assembled. If an older crib is to be used, care must be taken to ensure that there have been no recalls on the crib model, that all of the hardware is intact, and that the assembly instructions are available.

For some families, use of a crib might not be possible for financial reasons or space considerations. In addition, parents might be reluctant to place the infant in the crib because of concerns that the crib is too large for the infant or that “crib death” (ie, SIDS) only occurs in cribs. Alternate sleep surfaces, such as portable cribs/play yards and bassinets might be more acceptable for some families, because they are smaller and more portable. Local organizations throughout the United States can help to provide low-cost or free cribs or play yards. If a portable crib/play yard or bassinet is to be used, it should meet the following CPSC guidelines: (1) sturdy bottom and wide base; (2) smooth surfaces without protruding hardware; (3) legs with locks to prevent folding while in use; and (4) firm, snugly fitting mattress.¹²¹ In addition, other AAP guidelines for safe sleep, including supine positioning and avoidance of soft objects and loose bedding, should be followed. Mattresses should be firm and should maintain their shape even when the fitted sheet designated for that model is used, such that there are no gaps between the mattress and the side of the bassinet, playpen, portable crib, or play yard. Only mattresses designed for the specific product should be used. Pillows or cushions should not be used as substitutes for mattresses or in addition to a mattress. Any fabric on the sides or a canopy should be taut and firmly attached to the frame so as not to create a suffocation risk for the infant. Portable cribs, play yards, and

bassinets with vertical sides made of air-permeable material may be preferable to those with air-impermeable sides.¹²² Finally, parents and caregivers should adhere to the manufacturer's guidelines regarding maximum weight of infants using these products.^{122,123} If the product is a combination product (eg, crib/toddler bed), the manual should be consulted when the mode of use is changed.

There are no data regarding the safety of sleepers that attach to the side of an adult bed. However, there are potential safety concerns if the sleeper is not attached properly to the side of the adult bed or if the infant moves into the adult bed. Therefore, the task force cannot make a recommendation for or against the use of bedside sleepers. In addition, infants should not be placed for sleep on adult-sized beds because of the risk of entrapment and suffocation.¹²⁴ Portable bed rails (railings installed on the side of the bed that are intended to prevent a child from falling off of the bed) should not be used with infants because of the risk of entrapment and strangulation.¹²⁵

Car Seats and Other Sitting Devices Are not Recommended for Routine Sleep at Home or in the Hospital, Particularly for Young Infants

Some parents let their infants sleep in a car seat or other sitting device. Sitting devices include but are not restricted to car seats, strollers, swings, infant carriers, and infant slings. Parents and caregivers often use these devices, even when not traveling, because they are convenient. One study found that the average young infant spends 5.7 hours/day in a car seat or similar sitting device.¹²⁶ However, there are multiple concerns about using sitting devices as a usual infant sleep location. Placing an infant in such devices can potentiate gastro-

esophageal reflux¹²⁷ and positional plagiocephaly. Because they still have poor head control and often experience flexion of the head while in a sitting position, infants younger than 1 month in sitting devices might be at increased risk of upper airway obstruction and oxygen desaturation.^{128–132} In addition, there is increasing concern about injuries from falls resulting from car seats being placed on elevated surfaces.^{133–137} An analysis of CPSC data revealed 15 suffocation deaths between 1990 and 1997 resulting from car seats overturning after being placed on a bed, mattress, or couch.¹³⁶ The CPSC also warns about the suffocation hazard to infants, particularly those who are younger than 4 months, who are carried in infant sling carriers.¹³⁸ When infant slings are used for carrying, it is important to ensure that the infant's head is up and above the fabric, the face is visible, and that the nose and mouth are clear of obstructions. After nursing, the infant should be repositioned in the sling so that the head is up and is clear of fabric and the adult's body.

BED-SHARING

Room-Sharing Without Bed-Sharing Is Recommended

The terms “bed-sharing” and “cosleeping” are often used interchangeably, but they are not synonymous. Cosleeping is when parent and infant sleep in close proximity (on the same surface or different surfaces) so as to be able to see, hear, and/or touch each other.^{139,140} Cosleeping arrangements can include bed-sharing or sleeping in the same room in close proximity.^{140,141} Bed-sharing refers to a specific type of cosleeping when the infant is sleeping on the same surface with another person.¹⁴⁰ Because the term cosleeping can be misconstrued and does not precisely describe sleep arrangements,

the AAP recommends use of the terms “room-sharing” and “bed-sharing.”

The AAP recommends the arrangement of room-sharing without bed-sharing, or having the infant sleep in the parents' room but on a separate sleep surface (crib or similar surface) close to the parents' bed. There is evidence that this arrangement decreases the risk of SIDS by as much as 50%.^{64,66,142,143} and is safer than bed-sharing^{64,66,142,143} or solitary sleeping (when the infant is in a separate room).^{53,64} In addition, this arrangement is most likely to prevent suffocation, strangulation, and entrapment, which may occur when the infant is sleeping in the adult bed. Furthermore, room-sharing without bed-sharing allows close proximity to the infant, which facilitates feeding, comforting, and monitoring of the infant.

Parent-infant bed-sharing is common. In 1 national survey, 45% of parents responded that they had shared a bed with their infant (8 months of age or younger) at some point in the preceding 2 weeks.¹⁹ In some racial/ethnic groups, the rate of routine bed-sharing might be higher.^{18–20} There are often cultural and personal reasons why parents choose to bed-share, including convenience for feeding (breast-feeding or with formula) and bonding. In addition, many parents might believe that their own vigilance is the only way that they can keep their infant safe and that the close proximity of bed-sharing allows them to maintain vigilance, even while sleeping.¹⁴⁴ Some parents will use bed-sharing specifically as a safety strategy if the infant sleeps in the prone position^{21,144} or if there is concern about environmental dangers such as vermin and stray gunfire.¹⁴⁴

Parent-infant bed-sharing continues to be highly controversial. Although electrophysiologic and behavioral studies have offered a strong case for its effect

in facilitating breastfeeding^{145,146} and although many parents believe that they can maintain vigilance of the infant while they are asleep and bed-sharing,¹⁴⁴ epidemiologic studies have shown that bed-sharing can be hazardous under certain conditions.^{147–150} Bed-sharing might increase the risk of overheating,¹⁵¹ rebreathing¹⁵² or airway obstruction,¹⁵³ head covering,^{152,154–156} and exposure to tobacco smoke,¹⁵⁷ which are all risk factors for SIDS. A recent meta-analysis of 11 studies that investigated the association of bed-sharing and SIDS revealed a summary OR of 2.88 (95% confidence interval [CI]: 1.99–4.18) with bed-sharing.¹⁵⁸ Furthermore, bed-sharing in an adult bed not designed for infant safety exposes the infant to additional risks for accidental injury and death, such as suffocation, asphyxia, entrapment, falls, and strangulation.^{159,160} Infants, particularly those in the first 3 months of life and those born prematurely and/or with low birth weight, are at highest risk,¹⁶¹ possibly because immature motor skills and muscle strength make it difficult to escape potential threats.¹⁵⁸ In recent years, the concern among public health officials about bed-sharing has increased, because there have been increased reports of SUIDs occurring in high-risk sleep environments, particularly bed-sharing and/or sleeping on a couch or armchair.^{162–165}

There Is Insufficient Evidence to Recommend Any Bed-Sharing Situation in the Hospital or at Home as Safe; Devices Promoted to Make Bed-Sharing “Safe” Are Not Recommended

Epidemiologic studies have not found bed-sharing to be protective against SIDS and accidental suffocation for any subgroups of the population. It is acknowledged that there are some cultures for which bed-sharing is the norm and SIDS rates are low, but there

are other cultures for which bed-sharing is the norm and SIDS rates are high. In general, the bed-sharing practiced in cultures with low SIDS rates is often different from that in the United States and other Western countries (eg, with firm mats on the floor, separate mat for the infant, and/or absence of soft bedding). It is statistically much more difficult to demonstrate safety (ie, no risk) in small subgroups. Breastfeeding mothers who do not smoke and have not consumed alcohol or arousal-altering medications or drugs are 1 such subgroup. Furthermore, not all risks associated with bed-sharing (eg, parental fatigue) can be controlled. The task force, therefore, believes that there is insufficient evidence to recommend any bed-sharing situation in the hospital or at home as safe. In addition, there is no evidence that devices marketed to make bed-sharing “safe” (eg, in-bed cosleepers) reduce the risk of SIDS or suffocation or are safe. Such devices, therefore, are not recommended.

There Are Specific Circumstances in Which Bed-Sharing Is Particularly Hazardous, and It Should Be Stressed to Parents That They Avoid the Following Situations at All Times

The task force emphasizes that certain circumstances greatly increase the risk with bed-sharing. Bed-sharing is especially dangerous when 1 or both parents are smokers (OR: 2.3–17.7)^{64,65,158,166,167}; when the infant is younger than 3 months (OR: 4.7–10.4), regardless of parental smoking status^{64,66,143,158,168,169}; when the infant is placed on excessively soft surfaces such as waterbeds, sofas, and armchairs (OR: 5.1–66.9)^{62,64,65,143,169}; when soft bedding accessories such as pillows or blankets are used (OR: 2.8–4.1)^{62,170}; when there are multiple bed-sharers (OR: 5.4)⁶²; and when the parent has consumed alcohol (OR:

1.66)^{66,171} There is also a higher risk of SIDS when the infant is bed-sharing with someone who is not a parent (OR: 5.4).⁶²

A retrospective series of SIDS cases indicated that mean maternal body weight was higher for bed-sharing mothers than for non-bed-sharing mothers.¹⁷² The only case-control study to investigate the relationship between maternal body weight and bed-sharing did not find an increased risk of bed-sharing with increased maternal weight.¹⁷³

Infants May Be Brought Into the Bed for Feeding or Comforting but Should Be Returned to Their Own Crib or Bassinet When the Parent Is Ready to Return to Sleep

The risk of bed-sharing is higher the longer the duration of bed-sharing during the night.^{64,65,167,169} Returning the infant to the crib after bringing him or her into the bed for a short period of time is not associated with increased risk.^{65,169} Therefore, if the infant is brought into the bed for feeding, comforting, and bonding, the infant should be returned to the crib when the parent is ready for sleep. Because of the extremely high risk of SIDS, accidental suffocation, and entrapment on couches and armchairs,^{62,64,65,143,169} infants should not be fed on a couch or armchair when there is high risk that the parent may fall asleep.

It Is Prudent to Provide Separate Sleep Areas and Avoid Cobedding for Twins and Higher-Order Multiples in the Hospital and at Home

Cobedding of twins and other infants of multiple gestation is a frequent practice, both in the hospital setting and at home.¹⁷⁴ However, the benefits of cobedding twins and higher-order multiples have not been established.^{175–177} Twins and higher-order

multiples are often born prematurely and with low birth weight, so they are at increased risk of SIDS.^{101,102} Furthermore, there is increased potential for overheating and rebreathing while cobedding, and size discordance might increase the risk of accidental suffocation.¹⁷⁶ Most cobedded twins are placed on their sides rather than supine.¹⁷⁴ Finally, cobedding of twins and higher-order multiples in the hospital setting might encourage parents to continue this practice at home.¹⁷⁶ Because the evidence for the benefits of cobedding twins and higher-order multiples is not compelling and because of the increased risk of SIDS and suffocation, the AAP believes that it is prudent to provide separate sleep areas for these infants to decrease the risk of SIDS and accidental suffocation.

BEDDING

Pillows, Quilts, Comforters, Sheepskins, and Other Soft Surfaces Are Hazardous When Placed Under the Infant or Loose in the Sleep Environment

Bedding is used in infant sleep environments for comfort and safety.¹⁷⁸ Parents and caregivers who perceive that infants are uncomfortable on firm surfaces will often attempt to soften the surface with blankets and pillows. Parents and caregivers will also use pillows and blankets to create barriers to prevent the infant from falling off the sleep surface (usually an adult bed or couch) or to prevent injury if the infant hits the crib side. However, such soft bedding can increase the potential of suffocation and rebreathing.^{54,56,57,179–181} Pillows, quilts, comforters, sheepskins, and other soft surfaces are hazardous when placed under the infant^{62,147,182–187} or left loose in the infant’s sleep area^{62,65,184,185,188–191} and can increase SIDS risk up to fivefold independent of sleep position.^{62,147} Several reports have also described that

in many SIDS cases, the heads of the infants, including some infants who slept supine, were covered by loose bedding.^{65,186,187,191} It should be noted that the risk of SIDS increases 21-fold when the infant is placed prone with soft bedding.⁶² In addition, soft and loose bedding have both been associated with accidental suffocation deaths.¹⁴⁹ The CPSC has reported that the majority of sleep-related infant deaths in its database are attributable to suffocation involving pillows, quilts, and extra bedding.^{192,193} The AAP recommends that infants sleep on a firm surface without any soft or loose bedding. Pillows, quilts, and comforters should never be in the infant's sleep environment. Specifically, these items should not be placed loose near the infant, between the mattress and the sheet, or under the infant. Infant sleep clothing that is designed to keep the infant warm without the possible hazard of head covering or entrapment can be used in place of blankets; however, care must be taken to select appropriately sized clothing and to avoid overheating. If a blanket is used, it should be thin and tucked under the mattress so as to avoid head or face covering. These practices should also be modeled in hospital settings.

Wedges and Positioning Devices Are not Recommended

Wedges and positioning devices are often used by parents to maintain the infant in the side or supine position because of claims that these products reduce the risk for SIDS, suffocation, or gastroesophageal reflux. However, these products are frequently made with soft, compressible materials, which might increase the risk of suffocation. The CPSC has reports of deaths attributable to suffocation and entrapment associated with wedges and positioning devices. Most of these deaths occurred when infants were placed in the prone or side position with these

devices; other incidents have occurred when infants have slipped out of the restraints or rolled into a prone position while using the device.^{2,194} Because of the lack of evidence that they are effective against SIDS, suffocation, or gastroesophageal reflux and because there is potential for suffocation and entrapment, the AAP concurs with the CPSC and the US Food and Drug Administration in warning against the use of these products. If positioning devices are used in the hospital as part of physical therapy, they should be removed from the infant sleep area well before discharge from the hospital.

Bumper Pads and Similar Products Are not Recommended

Bumper pads and similar products that attach to crib slats or sides are frequently used with the thought of protecting infants from injury. Initially, bumper pads were developed to prevent head entrapment between crib slats.¹⁹⁵ However, newer crib standards that require crib slat spacing to be less than 2⁵/₈ inches have obviated the need for crib bumpers. In addition, infant deaths have occurred because of bumper pads. A recent report by Thach et al,¹⁹⁶ who used CPSC data, found that deaths attributed to bumper pads were from 3 mechanisms: (1) suffocation against soft, pillow-like bumper pads; (2) entrapment between the mattress or crib and firm bumper pads; and (3) strangulation from bumper pad ties. However, the CPSC believes that there were other confounding factors, such as the presence of pillows and/or blankets, that might have contributed to many of the deaths in this report.² Thach et al¹⁹⁶ also analyzed crib injuries that might have been prevented by bumper pad use and concluded that the use of bumper pads only prevents minor injuries. A more recent study of crib injuries that used data from the CPSC National Electronic Injury Surveillance System con-

cluded that the potential benefits of preventing minor injury with bumper pad use were far outweighed by the risk of serious injury such as suffocation or strangulation.¹⁹⁷ In addition, most bumper pads obscure infant and parent visibility, which might increase parental anxiety.¹⁹⁵ There are other products that attach to crib sides or crib slats that claim to protect infants from injury. However, there are no published data that support these claims. Because of the potential for suffocation, entrapment, and strangulation and lack of evidence to support that bumper pads or similar products that attach to crib slats or sides prevent injury in young infants, the AAP does not recommend their use.

PRENATAL AND POSTNATAL EXPOSURES (INCLUDING SMOKING AND ALCOHOL)

Pregnant Women Should Seek and Obtain Regular Prenatal Care

There is substantial epidemiologic evidence that links a lower risk of SIDS for infants whose mothers obtain regular prenatal care.^{198–200} Women should seek prenatal care early in the pregnancy and continue to obtain regular prenatal care during the entire pregnancy.

Smoking During Pregnancy, in the Pregnant Woman's Environment, and in the Infant's Environment Should Be Avoided

Maternal smoking during pregnancy is a major risk factor in almost every epidemiologic study of SIDS.^{201–204} Smoke in the infant's environment after birth is a separate major risk factor in a few studies,^{202,205} although separating this variable from maternal smoking before birth is problematic. Thirdhand smoke refers to residual contamination from tobacco smoke after the cigarette has been extinguished²⁰⁶; there is no research to date on the signifi-

cance of thirdhand smoke with regards to SIDS risk. Smoke exposure adversely affects infant arousal^{207–213}; in addition, smoke exposure increases risk of preterm birth and low birth weight, both of which are risk factors for SIDS. The effect of tobacco smoke exposure on SIDS risk is dose-dependent. Aside from sleep position, smoke exposure is the largest contributing risk factor for SIDS.¹⁴⁹ It is estimated that one-third of SIDS deaths could be prevented if all maternal smoking during pregnancy were eliminated.^{214,215} The AAP supports the elimination of all tobacco smoke exposure, both prenatally and environmentally.^{216,217}

Avoid Alcohol and Illicit Drug Use During Pregnancy and After the Infant's Birth

Several studies have specifically investigated the association of SIDS with prenatal and postnatal exposure to alcohol or illicit drug use, although substance abuse often involves more than 1 substance and it is difficult to separate these variables from each other and from smoking. However, 1 study of Northern Plains American Indians found that periconceptional maternal alcohol use (adjusted OR: 6.2 [95% CI: 1.6–23.3]) and maternal first-trimester binge drinking (adjusted OR: 8.2 [95% CI: 1.9–35.3])²¹⁸ were associated with increased SIDS risk independent of prenatal cigarette smoking exposure. Another study from Denmark, which was based on prospective data about maternal alcohol use, also found a significant relationship between maternal binge drinking and postneonatal infant mortality, including SIDS.²¹⁹

Postmortem studies of Northern Plains American Indian infants revealed that prenatal cigarette smoking was significantly associated with decreased serotonin receptor binding in the brainstem. In this study, the asso-

ciation of maternal alcohol drinking in the 3 months before or during pregnancy was of borderline significance on univariate analysis but was not significant when prenatal smoking and case-versus-control status were in the model.³⁹ However, this study had limited power for multivariate analysis because of its small sample size. One study found an association of SIDS with heavy alcohol consumption in the 2 days before the death.²²⁰ Although some studies have found a particularly strong association when alcohol consumption occurs in combination with bed-sharing,^{64–66,221} other studies have not found interaction between bed-sharing and alcohol to be significant.^{167,222}

Studies investigating the relationship of illicit drug use and SIDS have focused on specific drugs or illicit drug use in general. In utero exposure to opiates (primarily methadone and heroin) has been shown in retrospective studies to be associated with an increased risk of SIDS.^{223,224} With the exception of 1 study that did not show increased risk,²²⁵ population-based studies have generally shown an increased risk with in utero cocaine exposure.^{226–228} However, these studies did not control for confounding factors. A prospective cohort study found the SIDS rate to be significantly increased for infants exposed in utero to methadone (OR: 3.6 [95% CI: 2.5–5.1]), heroin (OR: 2.3 [95% CI: 1.3–4.0]), methadone and heroin (OR: 3.2 [95% CI: 1.2–8.6]), and cocaine (OR: 1.6 [95% CI: 1.2–2.2]), even after controlling for race/ethnicity, maternal age, parity, birth weight, year of birth, and maternal smoking.²²⁹ In addition, a meta-analysis of studies that investigated an association between in utero cocaine exposure and SIDS found an increased risk of SIDS to be associated with prenatal exposure to cocaine and illicit drugs in general.²³⁰

BREASTFEEDING

Breastfeeding Is Recommended

Earlier epidemiologic studies were not consistent in demonstrating a protective effect of breastfeeding on SIDS*[†]; some studies found a protective effect,^{67,239,240} and others did not.[†] Because many of the case-control studies demonstrated a protective effect of breastfeeding against SIDS in univariate analysis but not when confounding factors were taken into account,^{62,184,198,251,238} these results suggested that factors associated with breastfeeding, rather than breastfeeding itself, are protective. However, newer published reports support the protective role of breastfeeding on SIDS when taking into account potential confounding factors.^{243–245} Studies do not distinguish between nursing and expressed human milk. In the Agency for Healthcare Research and Quality's "Evidence Report on Breastfeeding in Developed Countries,"²⁴³ multiple outcomes, including SIDS, were examined. Six studies were included in the SIDS-breastfeeding meta-analysis, and in both unadjusted and adjusted analysis, ever breastfeeding was associated with a lower risk of SIDS (summary OR: 0.41 [95% CI: 0.28–0.58]; adjusted summary OR: 0.64 [95% CI: 0.51–0.81]). The German Study of Sudden Infant Death, the largest and most recent case-control study of SIDS, found that exclusive breastfeeding at 1 month of age halved the risk of SIDS (adjusted OR: 0.48 [95% CI: 0.28–0.82]). At all ages, control infants were breastfed at higher rates than SIDS victims, and the protective effect of partial or exclusive breastfeeding remained statistically significant after adjustment for confounders.²⁴⁴ A recent meta-analysis that included 18 case-control studies revealed an unadjusted summary OR for any breast-

*Refs 62, 65, 67, 184, 198, and 231–239.

†Refs 62, 184, 198, 231, 238, 241, and 242.

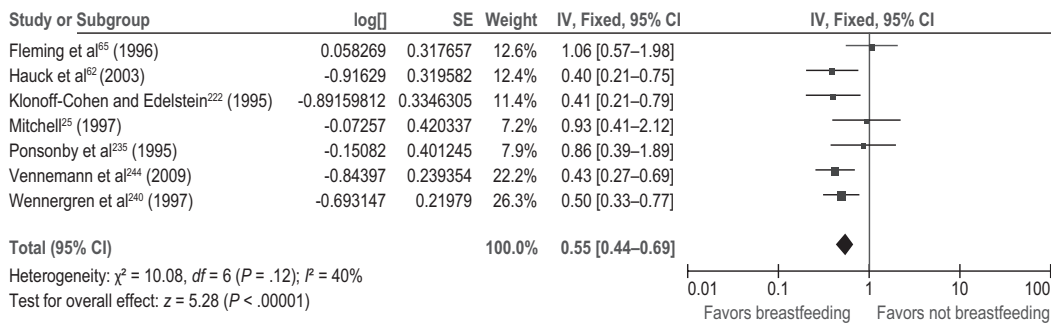


FIGURE 9

Multivariable analysis of any breastfeeding versus no breastfeeding. log[] indicates logarithm of the OR; weight, weighting that the study contributed to the meta-analysis (according to sample size); IV, fixed, 95% CI: fixed-effect OR with 95% CI.²⁴⁵

feeding of 0.40 (95% CI: 0.35–0.44). Seven of these studies provided adjusted ORs, and on the basis of these studies, the pooled adjusted OR remained statistically significant at 0.55 (95% CI: 0.44–0.69) (Fig 9).²⁴⁵ The protective effect of breastfeeding increased with exclusivity, with a univariable summary OR of 0.27 (95% CI: 0.24–0.31) for exclusive breastfeeding of any duration.²⁴⁵

Currently in the United States, 73% of mothers initiate breastfeeding, and 42% and 21% are still breastfeeding at 6 and 12 months, respectively.²⁴⁶ Non-Hispanic black mothers are least likely to initiate or to still be breastfeeding at 6 and 12 months (54%, 27%, and 12%, respectively), whereas Asian/Pacific Islander mothers initiate and continue breastfeeding more than other groups (81%, 52%, and 30%, respectively). Rates for initiating and continuing breastfeeding at 6 and 12 months for non-Hispanic white mothers are 74%, 43%, and 21%; rates for Hispanic mothers are 80%, 45%, and 24%; and rates for American Indian/Alaskan Native mothers are 70%, 37%, and 19%, respectively.

Physiologic sleep studies have found that breastfed infants are more easily aroused from sleep than their formula-fed counterparts.^{247,248} In addition, breastfeeding results in a decreased incidence of diarrhea, upper and lower respiratory infections, and

other infectious diseases²⁴⁹ that are associated with an increased vulnerability to SIDS and provides overall immune system benefits from maternal antibodies and micronutrients in human milk.^{250,251} Exclusive breastfeeding for 6 months has been found to be more protective against infectious diseases compared with exclusive breastfeeding to 4 months of age and partial breastfeeding thereafter.²⁴⁹

If a Breastfeeding Mother Brings the Infant Into the Adult Bed for Nursing, the Infant Should Be Returned to a Separate Sleep Surface When the Mother Is Ready for Sleep

Several organizations promote the practice of mother-infant bed-sharing (ie, sleeping in the same bed) as a way of facilitating breastfeeding.^{142,252,253} Breastfeeding is a common reason given by mothers for bed-sharing with their infants.²⁵⁴ Studies have found an association between bed-sharing and longer duration of breastfeeding, but their data cannot determine a temporal relationship (ie, it is not known whether bed-sharing promotes breastfeeding or if breastfeeding promotes bed-sharing, or if women who prefer 1 practice are also likely to prefer the other).²⁵⁵ Although bed-sharing may facilitate breastfeeding, it is not essential for successful breastfeeding.^{256,257} Furthermore, 1 case-control

study found that the risk of SIDS while bed-sharing was similar regardless of breastfeeding status, which indicates that the benefits of breastfeeding do not outweigh the increased risk associated with bed-sharing.²⁵⁸

PACIFIER USE

Consider Offering a Pacifier at Nap Time And Bedtime

Several studies^{62,66,167,251,259–262} have found a protective effect of pacifiers on the incidence of SIDS, particularly when used at the time of last sleep. Two meta-analyses revealed that pacifier use decreased the risk of SIDS by 50% to 60% (summary adjusted OR: 0.39 [95% CI: 0.31–0.50]²⁶⁵; summary unadjusted OR: 0.48 [95% CI: 0.43–0.54]²⁶⁴). Two later studies not included in these meta-analyses reported equivalent or even larger protective associations.^{265,266} The mechanism for this apparent strong protective effect is still unclear, but lowered arousal thresholds, favorable modification of autonomic control during sleep, and maintaining airway patency during sleep have been proposed.^{247,267–270} It is common for the pacifier to fall from the mouth soon after the infant falls asleep; even so, the protective effect persists throughout that sleep period.^{247,271} Two studies have shown that pacifier use is most protective when used for all sleep periods.^{169,266} However, these studies also

showed increased risk of SIDS when the pacifier was usually used but not used the last time the infant was placed for sleep; the significance of these findings is yet unclear.

Although some SIDS experts and policy-makers endorse pacifier use recommendations that are similar to those of the AAP,^{272,273} concerns about possible deleterious effects of pacifier use have prevented others from making a recommendation for pacifier use as a risk reduction strategy.²⁷⁴ Although several observational studies^{275–277} have found a correlation between pacifiers and reduced breastfeeding duration, the results of well-designed randomized clinical trials indicated that pacifiers do not seem to cause shortened breastfeeding duration for term and preterm infants.^{278,279} The authors of 1 study reported a small deleterious effect of early pacifier introduction (2–5 days after birth) on exclusive breastfeeding at 1 month of age and on overall breastfeeding duration (defined as any breastfeeding), but early pacifier use did not adversely affect exclusive breastfeeding duration. In addition, there was no effect on breastfeeding duration when the pacifier was introduced at 1 month of age.²⁸⁰ A more recent systematic review found that the highest level of evidence (ie, from clinical trials) does not support an adverse relationship between pacifier use and breastfeeding duration or exclusivity.²⁸¹ The association between shortened duration of breastfeeding and pacifier use in observational studies likely reflects a number of complex factors such as breastfeeding difficulties or intent to wean.²⁸¹ A large multicenter, randomized controlled trial of 1021 mothers who were highly motivated to breastfeed were assigned to 2 groups: mothers advised to offer a pacifier after 15 days and mothers advised not to offer a pacifier. At 3

months, there were no differences in breastfeeding rates between the 2 groups; 85.8% of infants in the offer-pacifier group were exclusively breastfed compared with 86.2% in the not-offered group.²⁸² The AAP policy statement on breastfeeding and the use of human milk includes a recommendation that pacifiers can be used during breastfeeding, but implementation should be delayed until breastfeeding is well established.²⁸³

Some dental malocclusions have been found more commonly among pacifier users than nonusers, but the differences generally disappeared after pacifier cessation.²⁸⁴ In its policy statement on oral habits, the American Academy of Pediatric Dentistry states that nonnutritive sucking behaviors (ie, fingers or pacifiers) are considered normal for infants and young children and that, in general, sucking habits in children to the age of 3 years are unlikely to cause any long-term problems.²⁸⁵ There is an approximate 1.2- to 2-fold increased risk of otitis media associated with pacifier use, particularly between 2 and 3 years of age.^{286,287} The incidence of otitis media is generally lower in the first year of life, especially the first 6 months, when the risk of SIDS is the highest.^{288–293} However, pacifier use, once established, may persist beyond 6 months, thus increasing the risk of otitis media. Gastrointestinal infections and oral colonization with *Candida* species were also found to be more common among pacifier users than nonusers.^{289–291}

The literature on infant digit-sucking and SIDS is extremely limited. Only 1 case-control study from the Netherlands has reported results.²⁶² This study did not find an association between usual digit-sucking (reported as “thumb-sucking”) and SIDS risk (OR: 1.38 [95% CI: 0.35–1.51]), but the wide CI suggests that there was insufficient

power to detect a significant association.

OVERHEATING, FANS, AND ROOM VENTILATION

Avoid Overheating and Head Covering in Infants

There is clear evidence that the risk of SIDS is associated with the amount of clothing or blankets on an infant and the room temperature.^{182,218,294,295} Infants who sleep in the prone position have a higher risk of overheating than do supine sleeping infants.¹⁸² It is unclear whether the relationship to overheating is an independent factor or merely a reflection of the increased risk of SIDS and suffocation with blankets and other potentially asphyxiating objects in the sleeping environment. Head covering during sleep is of particular concern. In a recent systematic review, the pooled mean prevalence of head covering among SIDS victims was 24.6% compared with 3.2% among control infants.¹⁵⁴ It is not known whether the risk associated with head covering is attributable to overheating, hypoxia, or rebreathing.

There has been some suggestion that room ventilation may be important. One study found that bedroom heating, compared with no bedroom heating, increases SIDS risk (OR: 4.5),²³⁵ and another study has also demonstrated a decreased risk of SIDS in a well-ventilated bedroom (windows and doors open) (OR: 0.4).²⁹⁶ In 1 study, the use of a fan seemed to reduce the risk of SIDS (adjusted OR: 0.28 [95% CI: 0.10–0.77]).²⁹⁷ However, because of the possibility of recall bias, the small sample size of controls using fans ($n = 36$), a lack of detail about the location and types of fans used, and the weak link to a mechanism, this study's results should be interpreted with caution. On the basis of available data, the task force cannot make a recommendation on the use

of a fan as a SIDS risk-reduction strategy.

SWADDLING

Although Swaddling May Be Used as a Strategy to Calm the Infant and Encourage Use of Supine Position, There Is Not Enough Evidence to Recommend It as a Strategy for Reducing the Risk of SIDS

Many cultures and newborn nurseries have traditionally used swaddling, or wrapping the infant in a light blanket, as a strategy to soothe infants and, in some cases, encourage sleep in the supine position. Swaddling, when done correctly, can be an effective technique to help calm infants and promote sleep.²⁹⁸ Some have argued that swaddling can alter certain risk factors for SIDS, thus reducing the risk of SIDS. For instance, it has been suggested that the physical restraint associated with swaddling may prevent infants placed supine from rolling to the prone position.²⁹⁹ One study's results suggested a decrease in SIDS rate with swaddling if the infant was supine,¹⁸² but it was notable that there was an increased risk of SIDS if the infant was swaddled and placed in the prone position.¹⁸² Although a recent study found a 31-fold increase in SIDS risk with swaddling, the analysis was not stratified according to sleep position.¹⁷¹ Although it may be more likely that parents will initially place a swaddled infant supine, this protective effect may be offset by the 12-fold increased risk of SIDS if the infant is either placed or rolls to the prone position when swaddled.^{182,300} Moreover, there is no evidence that swaddling reduces bed-sharing or use of unsafe sleep surfaces, promotes breastfeeding, or reduces maternal cigarette smoking.

There is some evidence that swaddling might cause detrimental physiologic

consequences. For example, it can cause an increase in respiratory rate,³⁰¹ and tight swaddling can reduce the infant's functional residual lung capacity.^{299,302,303} Tight swaddling can also exacerbate hip dysplasia if the hips are kept in extension and adduction.^{304–307} This is particularly important, because some have advocated that the calming effects of swaddling are related to the "tightness" of the swaddling. In contrast, "loose" or incorrectly applied swaddling could result in head covering and, in some cases, strangulation if the blankets become loose in the bed. Swaddling may also possibly increase the risk of overheating in some situations, especially when the head is covered or the infant has an infection.^{308,309} However, a recent study found no increase in abdominal skin temperature when infants were swaddled in a light cotton blanket from the shoulders down.³⁰²

Impaired arousal has often been postulated as a mechanism that contributes to SIDS, and several studies have investigated the relationship between swaddling, arousal, and sleep patterns in infants. Physiologic studies have demonstrated that, in general, swaddling decreases startling,³⁰¹ increases sleep duration, and decreases spontaneous awakenings.³¹⁰ Swaddling also decreases arousability (ie, increases cortical arousal thresholds) to a nasal pulsatile air-jet stimulus, especially in infants who are easily arousable when not swaddled but less so in infants who have high arousal thresholds when not swaddled.³⁰¹ One study found decreased arousability in infants at 3 months of age who were not usually swaddled and then were swaddled but found no effect on arousability in routinely swaddled infants.³⁰¹ In contrast, another group of investigators showed decreased arousal thresholds³¹⁰ and increases in autonomic (subcortical) responses³¹¹ to an auditory stimulus

when swaddled. Thus, although swaddling clearly promotes sleep and decreases the number of awakenings, the effects on arousability to an external stimulus remain unclear. There is accumulating evidence, however, that there are only minimal effects of routine swaddling on arousal. In addition, there have been no studies investigating the effects of swaddling on arousal to more relevant stimuli such as hypoxia or hypercapnia.

In summary, it is recognized that swaddling is one of many child care practices that can be used to calm infants and promote sleep. However, there is insufficient evidence to recommend routine swaddling as a strategy for reducing the incidence of SIDS. Moreover, as many have advocated, swaddling must be correctly applied to avoid possible hazards such as hip dysplasia, head covering, and strangulation. It is important to note that swaddling does not reduce the necessity to follow recommended safe sleep practices.

IMMUNIZATIONS AND SIDS

Infants Should Be Immunized in Accordance With Recommendations of the AAP and Centers for Disease Control and Prevention

The incidence of SIDS peaks at a time when infants are receiving numerous immunizations. Case reports of a cluster of deaths shortly after immunization with diphtheria-tetanus-pertussis in the late 1970s created concern of a possible causal relationship between vaccinations and SIDS.^{312–315} Case-control studies were performed to evaluate this temporal association. Four of the 6 studies found no relationship between diphtheria-tetanus-pertussis vaccination and subsequent SIDS,^{316–319} and results of the other 2 studies suggested a temporal relationship but only in specific subgroup anal-

ysis.^{320,321} In 2003, the Institute of Medicine of the National Academy of Sciences reviewed available data and concluded that “[t]he evidence favors rejection of a causal relationship between exposure to multiple vaccinations and SIDS.”³²² Additional subsequent large population case-control trials consistently have found vaccines to be protective against SIDS^{323–325}; however, confounding factors (social, maternal, birth, and infant medical history) might account for this protective effect.³²⁶ It also has been theorized that the decreased SIDS rate immediately after vaccination was attributable to infants being healthier at time of immunization, or “the healthy vaccinee effect.”³²⁷ Recent illness would both place infants at higher risk of SIDS and make them more likely to have immunizations deferred.³²⁸

Recent studies have attempted to control for confounding by social, maternal, birth, and infant medical history.^{323,325,328} In a meta-analysis, Vennemann et al³²⁸ found a multivariate summary OR for immunizations and SIDS to be 0.54 (95% CI: 0.39–0.76), which indicates that the risk of SIDS is halved by immunization. The evidence continues to show no causal relationship between immunizations and SIDS and suggests that vaccination may have a protective effect against SIDS.

HOME MONITORS, SIDS, AND APPARENT LIFE-THREATENING EVENTS

There Is no Evidence That Apparent Life-Threatening Events Are Precursors to SIDS, and Infant Home Monitors Should Not Be Used as a Strategy for Preventing SIDS

For many years it was believed that apparent life-threatening events were the predecessors of SIDS, and home apnea monitors were used as a strat-

egy for preventing SIDS.³²⁹ However, there is no evidence that home monitors are effective for this purpose.^{330–333} The task force concurs with the AAP Committee on Fetus and Newborn, which has recommended that infant home monitoring not be used as a strategy to prevent SIDS, although it can be useful for some infants who have had an apparent life-threatening event.³³⁴

POTENTIAL TOXICANTS AND SIDS

There Is no Evidence Linking Various Toxicants to SIDS

Many theories link various toxicants and SIDS. Currently, no studies have substantiated a causal relationship between metals, such as silver, cadmium, cobalt, lead, or mercury, and SIDS.^{335–337} Although an ecological study found correlation of the maximal recorded nitrate levels of drinking water with local SIDS rates in Sweden,³³⁸ no case-control study has demonstrated a relationship between nitrates in drinking water and SIDS. Furthermore, an expert group in the United Kingdom analyzed data pertaining to a hypothesis that SIDS is related to toxic gases, such as antimony, phosphorus, or arsenic, being released from mattresses^{339,340} and found the toxic-gas hypothesis to be unsubstantiated.³⁴¹ Finally, 2 case-control studies found that wrapping mattresses in plastic to reduce toxic gas emission did not protect against SIDS.^{191,342}

HEARING SCREENS

Newborn Hearing Screens Should Not Be Used as a Screening Test for SIDS

A single, small, retrospective case-control study examined the use of newborn transient evoked otoacoustic emission hearing screening tests as a tool for identifying infants at subsequent risk of SIDS.³⁴³ Infants who sub-

sequently died from SIDS did not fail their hearing tests but, compared with controls, showed a decreased signal-to-noise ratio score in the right ear only (at frequencies of 2000, 3000, and 4000 Hz). Methodologic concerns have been raised about the validity of the study methods used in this study,^{344,345} and these results have not been substantiated by others. A larger but non-peer-reviewed report of hearing screening data in Michigan revealed no relationship between hearing screening test results and SIDS cases.³⁴⁶ Until additional data are available, hearing screening should not be considered as a valid screening tool for determining which infants might be at subsequent risk of SIDS. Furthermore, an increased risk of SIDS should not be inferred from an abnormal hearing screen result.

EDUCATIONAL INTERVENTIONS

Educational and Intervention Campaigns Are Often Effective in Altering Practice

Intervention campaigns for SIDS have been extremely effective, especially with regard to avoidance of prone positioning.³⁴⁷ Furthermore, there is evidence that primary care-based educational interventions, particularly those that address caregiver concerns and misconceptions about safe sleep recommendations, can be effective in altering practice. For instance, addressing concerns about infant comfort, choking, and aspiration while the infant is sleeping prone is helpful.^{348,349} Similar interventions for improving behavior of medical and nursing staff and child care providers have shown that these professionals have similar concerns about the supine sleep position.^{350–353} Primary care providers should be encouraged to develop quality improvement initiatives to improve

adherence with safe sleep recommendations among their patients.

MEDIA MESSAGES

Media and Manufacturers Should Follow Safe Sleep Guidelines in Their Messaging and Advertising

A recent study found that, in magazines targeted toward childbearing women, more than one-third of pictures of sleeping infants and two-thirds of pictures of infant sleep environments portrayed unsafe sleep positions and sleep environments.³⁵⁴ Media exposures (including movie, television, magazines, newspapers, and Web sites), manufacturer advertisements, and store displays affect individual behavior by influencing beliefs and attitudes. Frequent exposure to health-related media messages can affect individual health decisions,^{355,356}

and media messages have been quite influential in decisions regarding sleep position.^{77,80} Media and advertising messages contrary to safe sleep recommendations may create misinformation about safe sleep practices. Safe sleep messages should be reviewed, revised, and reissued at least every 5 years to address the next generation of new parents and products on the market.

RECOMMENDATIONS

The AAP's recommendations for a safe infant sleeping environment to reduce the risk of both SIDS and other sleep-related infant deaths are specified in the accompanying policy statement.⁴

LEAD AUTHOR

Rachel Y. Moon, MD

TASK FORCE ON SUDDEN INFANT DEATH SYNDROME, 2010–2011

Rachel Y. Moon, MD, Chairperson

Robert A. Darnall, MD
Michael H. Goodstein, MD
Fern R. Hauck, MD, MS

CONSULTANTS

Marian Willinger, PhD – Eunice Kennedy Shriver National Institute for Child Health and Human Development
Carrie K. Shapiro-Mendoza, PhD, MPH – Centers for Disease Control and Prevention

STAFF

James Couto, MA

ACKNOWLEDGMENTS

The task force acknowledges the contributions provided by others to the collection and interpretation of data examined in preparation of this report. The task force is particularly grateful for the report submitted by Dr Suad Wanna-Nakamura (CPSC) and for the assistance of Sarah McKinnon, PhD, MPH, and Cristina Rodríguez-Hart, MPH, with the statistics and graphs.

REFERENCES

1. Kattwinkel J, Hauck FR, Keenan ME, Malloy MH, Moon RY; American Academy of Pediatrics, Task Force on Sudden Infant Death Syndrome. The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. *Pediatrics*. 2005;116(5):1245–1255
2. Wanna-Nakamura S. *White Paper: Unsafe Sleep Settings—Hazards Associated With the Infant Sleep Environment and Unsafe Practices Used by Caregivers: A CPSC Staff Perspective*. Bethesda, MD: US Consumer Product Safety Commission; 2010
3. US Preventive Services Task Force. Grade definitions. Available at: www.uspreventiveservicestaskforce.org/uspstf/grades.htm. Accessed March 22, 2011
4. Moon RY; American Academy of Pediatrics, Task Force on Sudden Infant Death Syndrome. Policy statement: SIDS and other sleep-related infant deaths: expansion of recommendations for a safe infant sleeping environment. *Pediatrics*. 2011;128(5):1030–1039
5. Willinger M, James LS, Catz C. Defining the sudden infant death syndrome (SIDS): deliberations of an expert panel convened by the National Institute of Child Health and Human Development. *Pediatr Pathol*. 1991; 11(5):677–684
6. Centers for Disease Control and Prevention. Sudden unexplained infant death investigation reporting form (SUIDIRF). Available at: www.cdc.gov/SIDS/SUIDIRF.htm. Accessed September 20, 2011
7. Camperlengo LT, Shapiro-Mendoza CK, Kim SY. Sudden infant death syndrome: diagnostic practices and investigative policies, 2004. *Am J Forensic Med Pathol*. 2011; In press
8. Krous HF, Chadwick AE, Haas EA, Stanley C. Pulmonary intra-alveolar hemorrhage in SIDS and suffocation. *J Forensic Leg Med*. 2007;14(8):461–470
9. Kattwinkel J, Brooks J, Myerberg D; American Academy of Pediatrics, Task Force on Infant Positioning and SIDS. Positioning and SIDS [published correction appears in *Pediatrics*. 1992;90(2 pt 1):264]. *Pediatrics*. 1992;89(6 pt 1):1120–1126
10. Eunice Kennedy Shriver National Institute of Child Health and Human Development/ National Institutes of Health. Back to Sleep public education campaign. Available at: www.nichd.nih.gov/sids/sids.cfm. Accessed July 7, 2010
11. National Infant Sleep Position Study [home page]. Available at: http://dccwww.bumc.bu.edu/ChimeNisp/Main_Nisp.asp. Accessed April 5, 2011
12. US Department of Health and Human Services, Centers of Disease Control and Prevention, National Center for Health Statistics, Office of Analysis and Epidemiology, Division of Vital Statistics. Compressed mortality data: underlying cause-of-death—mortality for 1979–1998 with ICD 9 codes; and Mortality for 1999–2007 with ICD 10 codes. Available at: <http://wonder.cdc.gov/mortSQL.html>. Accessed July 8, 2010
13. Malloy MH, MacDorman M. Changes in the classification of sudden unexpected infant deaths: United States, 1992–2001. *Pediatrics*. 2005;115(5):1247–1253
14. Shapiro-Mendoza CK, Tomashek KM, Anderson RN, Wingo J. Recent national trends in sudden, unexpected infant deaths: more evidence supporting a change in classification or reporting. *Am J Epidemiol*. 2006;163(8):762–769
15. Shapiro-Mendoza CK, Kimball M, Tomashek KM, Anderson RN, Blanding S. US infant mortality trends attributable to accidental suffocation and strangulation in bed from 1984 through 2004: are rates increasing? *Pediatrics*. 2009;123(2):533
16. Shapiro-Mendoza CK, Kim SY, Chu SY, Kahn

- E, Anderson RN. Using death certificates to characterize sudden infant death syndrome (SIDS): opportunities and limitations. *J Pediatr*. 2010;156(1):38–43
17. Hauck FR, Moore CM, Herman SM, et al. The contribution of prone sleeping position to the racial disparity in sudden infant death syndrome: the Chicago Infant Mortality Study. *Pediatrics*. 2002;110(4):772–780
 18. Lahr MB, Rosenberg KD, Lapidus JA. Maternal-infant bedsharing: risk factors for bedsharing in a population-based survey of new mothers and implications for SIDS risk reduction. *Matern Child Health J*. 2007;11(3):277–286
 19. Willinger M, Ko CW, Hoffman HJ, Kessler RC, Corwin MJ. Trends in infant bed sharing in the United States, 1993–2000: the National Infant Sleep Position study. *Arch Pediatr Adolesc Med*. 2003;157(1):43–49
 20. Fu LY, Colson ER, Corwin MJ, Moon RY. Infant sleep location: associated maternal and infant characteristics with sudden infant death syndrome prevention recommendations. *J Pediatr*. 2008;153(4):503–508
 21. Flick L, White DK, Vemulapalli C, Stulac BB, Kemp JS. Sleep position and the use of soft bedding during bed sharing among African American infants at increased risk for sudden infant death syndrome. *J Pediatr*. 2001;138(3):338–343
 22. Rasinski KA, Kuby A, Bzdusek SA, Silvestri JM, Weese-Mayer DE. Effect of a sudden infant death syndrome risk reduction education program on risk factor compliance and information sources in primarily black urban communities. *Pediatrics*. 2003;111(4 pt 1). Available at: www.pediatrics.org/cgi/content/full/111/4/e347
 23. Osmond C, Murphy M. Seasonality in the sudden infant death syndrome. *Paediatr Perinat Epidemiol*. 1988;2(4):337–345
 24. Malloy MH, Freeman DH. Age at death, season, and day of death as indicators of the effect of the back to sleep program on sudden infant death syndrome in the United States, 1992–1999. *Arch Pediatr Adolesc Med*. 2004;158(4):359–365
 25. Mitchell EA. The changing epidemiology of SIDS following the national risk reduction campaigns. *Pediatr Pulmonol Suppl*. 1997;(16):117–119
 26. Filiano JJ, Kinney HC. A perspective on neuropathologic findings in victims of the sudden infant death syndrome: the triple-risk model. *Biol Neonate*. 1994;65(3–4):194–197
 27. Kinney HC. Brainstem mechanisms underlying the sudden infant death syndrome: evidence from human pathologic studies. *Dev Psychobiol*. 2009;51(3):223–233
 28. Browne CJ, Sharma N, Waters KA, Machaalani R. The effects of nicotine on the alpha-7 and beta-2 nicotinic acetylcholine receptor subunits in the developing piglet brainstem. *Int J Dev Neurosci*. 2010;28(1):1–7
 29. Sekizawa S, Joad JP, Pinkerton KE, Bonham AC. Secondhand smoke exposure alters K⁺ channel function and intrinsic cell excitability in a subset of second-order airway neurons in the nucleus tractus solitarius of young guinea pigs. *Eur J Neurosci*. 2010;31(4):673–684
 30. Duncan JR, Paterson DS, Hoffman JM, et al. Brainstem serotonergic deficiency in sudden infant death syndrome. *JAMA*. 2010;303(5):430–437
 31. Duncan JR, Paterson DS, Kinney HC. The development of nicotinic receptors in the human medulla oblongata: interrelationship with the serotonergic system. *Auton Neurosci*. 2008;144(1–2):61–75
 32. Schneider J, Mitchell I, Singhal N, Kirk V, Hasan SU. Prenatal cigarette smoke exposure attenuates recovery from hypoxemic challenge in preterm infants. *Am J Respir Crit Care Med*. 2008;178(5):520–526
 33. Thiriez G, Bouhaddi M, Mourot L, et al. Heart rate variability in preterm infants and maternal smoking during pregnancy. *Clin Auton Res*. 2009;19(3):149–156
 34. Fifer WP, Fingers ST, Youngman M, Gomez-Gribben E, Myers MM. Effects of alcohol and smoking during pregnancy on infant autonomic control. *Dev Psychobiol*. 2009;51(3):234–242
 35. Richardson HL, Walker AM, Horne RS. Maternal smoking impairs arousal patterns in sleeping infants. *Sleep*. 2009;32(4):515–521
 36. Cohen G, Vella S, Jeffery H, Lagercrantz H, Katz-Salamon M. Cardiovascular stress hyperreactivity in babies of smokers and in babies born preterm. *Circulation*. 2008;118(18):1848–1853
 37. Panigrahy A, Filiano J, Sleeper LA, et al. Decreased serotonergic receptor binding in rhombic lip-derived regions of the medulla oblongata in the sudden infant death syndrome. *J Neuropathol Exp Neurol*. 2000;59(5):377–384
 38. Ozawa Y, Takashima S. Developmental neurotransmitter pathology in the brainstem of sudden infant death syndrome: a review and sleep position. *Forensic Sci Int*. 2002;130(suppl):S53–S59
 39. Kinney HC, Randall LL, Sleeper LA, et al. Serotonergic brainstem abnormalities in Northern Plains Indians with the sudden infant death syndrome. *J Neuropathol Exp Neurol*. 2003;62(11):1178–1191
 40. Machaalani R, Say M, Waters KA. Serotonergic receptor 1A in the sudden infant death syndrome brainstem medulla and associations with clinical risk factors. *Acta Neuropathol (Berl)*. 2009;117(3):257–265
 41. Paterson DS, Trachtenberg FL, Thompson EG, et al. Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. *JAMA*. 2006;296(17):2124–2132
 42. Say M, Machaalani R, Waters KA. Changes in serotonergic receptors 1A and 2A in the piglet brainstem after intermittent hypercapnic hypoxia (IHH) and nicotine. *Brain Res*. 2007;1152:17–26
 43. Kinney HC, Richerson GB, Dymecki SM, Darnall RA, Nattie EE. The brainstem and serotonin in the sudden infant death syndrome. *Annu Rev Pathol*. 2009;4:517–550
 44. Weese-Mayer DE, Ackerman MJ, Marazita ML, Berry-Kravis EM. Sudden infant death syndrome: review of implicated genetic factors. *Am J Med Genet A*. 2007;143A(8):771–788
 45. Opdal SH, Vege A, Rognum TO. Serotonin transporter gene variation in sudden infant death syndrome. *Acta Paediatr*. 2008;97(7):861–865
 46. Paterson DS, Rivera KD, Broadbelt KG, et al. Lack of association of the serotonin transporter polymorphism with the sudden infant death syndrome in the San Diego dataset. *Pediatr Res*. 2010;68(5):409–413
 47. Tan BH, Pundi KN, Van Norstrand DW, et al. Sudden infant death syndrome-associated mutations in the sodium channel beta subunits. *Heart Rhythm*. 2010;7(6):771–778
 48. Ferrante L, Opdal SH, Vege A, Rognum T. Cytokine gene polymorphisms and sudden infant death syndrome. *Acta Paediatr*. 2010;99(3):384–388
 49. Ferrante L, Opdal SH, Vege A, Rognum TO. IL-1 gene cluster polymorphisms and sudden infant death syndrome. *Hum Immunol*. 2010;71(4):402–406
 50. Opdal SH, Rognum TO, Vege A, Stave AK, Dupuy BM, Egeland T. Increased number of substitutions in the D-loop of mitochondrial DNA in the sudden infant death syndrome. *Acta Paediatr*. 1998;87(10):1039–1044
 51. Opdal SH, Rognum TO, Torgersen H, Vege A. Mitochondrial DNA point mutations detected in four cases of sudden infant death syndrome. *Acta Paediatr*. 1999;88(9):957–960
 52. Santorelli FM, Schlessel JS, Slonim AE, Di-

- Mauro S. Novel mutation in the mitochondrial DNA tRNA glycine gene associated with sudden unexpected death. *Pediatr Neurol.* 1996;15(2):145–149
53. Forsyth L, Hume R, Howatson A, Busuttill A, Burchell A. Identification of novel polymorphisms in the glucokinase and glucose-6-phosphatase genes in infants who died suddenly and unexpectedly. *J Mol Med.* 2005;83(8):610–618
 54. Kanetake J, Aoki Y, Funayama M. Evaluation of rebreathing potential on bedding for infant use. *Pediatr Int.* 2003;45(3):284–289
 55. Kemp JS, Thach BT. Quantifying the potential of infant bedding to limit CO₂ dispersal and factors affecting rebreathing in bedding. *J Appl Physiol.* 1995;78(2):740–745
 56. Kemp JS, Livne M, White DK, Arfken CL. Softness and potential to cause rebreathing: differences in bedding used by infants at high and low risk for sudden infant death syndrome. *J Pediatr.* 1998;132(2):234–239
 57. Patel AL, Harris K, Thach BT. Inspired CO₂ and O₂ in sleeping infants rebreathing from bedding: relevance for sudden infant death syndrome. *J Appl Physiol.* 2001;91(6):2537–2545
 58. Tuffnell CS, Petersen SA, Wailoo MP. Prone sleeping infants have a reduced ability to lose heat. *Early Hum Dev.* 1995;43(2):109–116
 59. Ammari A, Schulze KF, Ohira-Kist K, et al. Effects of body position on thermal, cardiorespiratory and metabolic activity in low birth weight infants. *Early Hum Dev.* 2009;85(8):497–501
 60. Yiallourou SR, Walker AM, Horne RS. Prone sleeping impairs circulatory control during sleep in healthy term infants: implications for SIDS. *Sleep.* 2008;31(8):1139–1146
 61. Wong FY, Witcombe NB, Yiallourou SR, et al. Cerebral oxygenation is depressed during sleep in healthy term infants when they sleep prone. *Pediatrics.* 2011;127(3). Available at: www.pediatrics.org/cgi/content/full/127/3/e558
 62. Hauck FR, Herman SM, Donovan M, et al. Sleep environment and the risk of sudden infant death syndrome in an urban population: the Chicago Infant Mortality Study. *Pediatrics.* 2003;111(5 pt 2):1207–1214
 63. Li DK, Petitti DB, Willinger M, et al. Infant sleeping position and the risk of sudden infant death syndrome in California, 1997–2000. *Am J Epidemiol.* 2003;157(5):446–455
 64. Blair PS, Fleming PJ, Smith IJ, et al. Babies sleeping with parents: case-control study of factors influencing the risk of the sudden infant death syndrome. CESDI SUDI Research Group. *BMJ.* 1999;319(7223):1457–1462
 65. Fleming PJ, Blair PS, Bacon C, et al. Environment of infants during sleep and risk of the sudden infant death syndrome: results of 1993–5 case-control study for confidential inquiry into stillbirths and deaths in infancy. Confidential Enquiry Into Stillbirths and Deaths Regional Coordinators and Researchers. *BMJ.* 1996;313(7051):191–195
 66. Carpenter RG, Irgens LM, Blair PS, et al. Sudden unexplained infant death in 20 regions in Europe: case control study. *Lancet.* 2004;363(9404):185–191
 67. Mitchell EA, Tuohy PG, Brunt JM, et al. Risk factors for sudden infant death syndrome following the prevention campaign in New Zealand: a prospective study. *Pediatrics.* 1997;100(5):835–840
 68. Waters KA, Gonzalez A, Jean C, Morielle A, Brouillette RT. Face-straight-down and face-near-straight-down positions in healthy, prone-sleeping infants. *J Pediatr.* 1996;128(5 pt 1):616–625
 69. Oyen N, Markestad T, Skjaerven R, et al. Combined effects of sleeping position and prenatal risk factors in sudden infant death syndrome: the Nordic Epidemiological SIDS Study. *Pediatrics.* 1997;100(4):613–621
 70. Mitchell EA, Thach BT, Thompson JMD, Williams S. Changing infants' sleep position increases risk of sudden infant death syndrome. *Arch Pediatr Adolesc Med.* 1999;153(11):1136–1141
 71. National Infant Sleep Position survey database. Available at: http://dccwww.bumc.bu.edu/ChimeNisp/NISP_Data.asp. Accessed September 7, 2010
 72. Oden R, Joyner BL, Ajao TI, Moon R. Factors influencing African American mothers' decisions about sleep position: a qualitative study. *J Natl Med Assoc.* 2010;102(10):870–872, 875–880
 73. Colson ER, McCabe LK, Fox K, et al. Barriers to following the Back-to-Sleep recommendations: insights from focus groups with inner-city caregivers. *Ambul Pediatr.* 2005;5(6):349–354
 74. Mosley JM, Stokes SD, Ulmer A. Infant sleep position: discerning knowledge from practice. *Am J Health Behav.* 2007;31(6):573–582
 75. Moon RY, Omon R. Determinants of infant sleep position in an urban population. *Clin Pediatr (Phila).* 2002;41(8):569–573
 76. Ottolini MC, Davis BE, Patel K, Sachs HC, Gershon NB, Moon RY. Prone infant sleeping despite the "Back to Sleep" campaign. *Arch Pediatr Adolesc Med.* 1999;153(5):512–517
 77. Willinger M, Ko CW, Hoffman HJ, Kessler RC, Corwin MJ. Factors associated with caregivers' choice of infant sleep position, 1994–1998: the National Infant Sleep Position Study. *JAMA.* 2000;283(16):2135–2142
 78. Moon RY, Bilitier WM. Infant sleep position policies in licensed child care centers after Back to Sleep campaign. *Pediatrics.* 2000;106(3):576–580
 79. Moon RY, Weese-Mayer DE, Silvestri JM. Nighttime child care: inadequate sudden infant death syndrome risk factor knowledge, practice, and policies. *Pediatrics.* 2003;111(4 pt 1):795–799
 80. Von Kohorn I, Corwin MJ, Rybin DV, Heeren TC, Lister G, Colson ER. Influence of prior advice and beliefs of mothers on infant sleep position. *Arch Pediatr Adolesc Med.* 2010;164(4):363–369
 81. Byard RW, Beal S. Gastric aspiration and sleeping position in infancy and early childhood. *J Paediatr Child Health.* 2000;36(4):403–405
 82. Malloy MH. Trends in postneonatal aspiration deaths and reclassification of sudden infant death syndrome: impact of the "Back to Sleep" program. *Pediatrics.* 2002;109(4):661–665
 83. Tablizo MA, Jacinto P, Parsley D, Chen ML, Ramanathan R, Keens TG. Supine sleeping position does not cause clinical aspiration in neonates in hospital newborn nurseries. *Arch Pediatr Adolesc Med.* 2007;161(5):507–510
 84. Vandenplas Y, Rudolph CD, Di Lorenzo C, et al. Pediatric gastroesophageal reflux clinical practice guidelines: joint recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN). *J Pediatr Gastroenterol Nutr.* 2009;49(4):498–547
 85. Meyers WF, Herbst JJ. Effectiveness of positioning therapy for gastroesophageal reflux. *Pediatrics.* 1982;69(6):768–772
 86. Tobin JM, McCloud P, Cameron DJ. Posture and gastro-oesophageal reflux: a case for left lateral positioning. *Arch Dis Child.* 1997;76(3):254–258
 87. Kahn A, Groswasser J, Sottiaux M, Rebuffat E, Franco P, Dramaix M. Prone or supine body position and sleep characteristics in infants. *Pediatrics.* 1993;91(6):1112–1115

88. Bhat RY, Hannam S, Pressler R, Rafferty GF, Peacock JL, Greenough A. Effect of prone and supine position on sleep, apneas, and arousal in preterm infants. *Pediatrics*. 2006;118(1):101–107
89. Ariagno RL, van Liempt S, Mirmiran M. Fewer spontaneous arousals during prone sleep in preterm infants at 1 and 3 months corrected age. *J Perinatol*. 2006;26(5):306–312
90. Franco P, Groswasser J, Sottiaux M, Broadfield E, Kahn A. Decreased cardiac responses to auditory stimulation during prone sleep. *Pediatrics*. 1996;97(2):174–178
91. Galland BC, Reeves G, Taylor BJ, Bolton DP. Sleep position, autonomic function, and arousal. *Arch Dis Child Fetal Neonatal Ed*. 1998;78(3):F189–F194
92. Galland BC, Hayman RM, Taylor BJ, Bolton DP, Sayers RM, Williams SM. Factors affecting heart rate variability and heart rate responses to tilting in infants aged 1 and 3 months. *Pediatr Res*. 2000;48(3):360–368
93. Horne RS, Ferens D, Watts AM, et al. The prone sleeping position impairs arousability in term infants. *J Pediatr*. 2001;138(6):811–816
94. Horne RS, Bandopadhyay P, Vitkovic J, Cranage SM, Adamson TM. Effects of age and sleeping position on arousal from sleep in preterm infants. *Sleep*. 2002;25(7):746–750
95. Kato I, Scaillet S, Groswasser J, et al. Spontaneous arousability in prone and supine position in healthy infants. *Sleep*. 2006;29(6):785–790
96. Phillipson EA, Sullivan CC. Arousal: the forgotten response to respiratory stimuli. *Am Rev Respir Dis*. 1978;118(5):807–809
97. Kahn A, Groswasser J, Rebuffat E, et al. Sleep and cardiorespiratory characteristics of infant victims of sudden death: a prospective case-control study. *Sleep*. 1992;15(4):287–292
98. Schechtman VL, Harper RM, Wilson AJ, Southall DP. Sleep state organization in normal infants and victims of the sudden infant death syndrome. *Pediatrics*. 1992;89(5 pt 1):865–870
99. Harper RM. State-related physiological changes and risk for the sudden infant death syndrome. *Aust Paediatr J*. 1986;22(suppl 1):55–58
100. Kato I, Franco P, Groswasser J, et al. Incomplete arousal processes in infants who were victims of sudden death. *Am J Respir Crit Care Med*. 2003;168(11):1298–1303
101. Malloy MH, Hoffman HJ. Prematurity, sudden infant death syndrome, and age of death. *Pediatrics*. 1995;96(3 pt 1):464–471
102. Sowter B, Doyle LW, Morley CJ, Altmann A, Halliday J. Is sudden infant death syndrome still more common in very low birthweight infants in the 1990s? *Med J Aust*. 1999;171(8):411–413
103. American Academy of Pediatric, Committee on Fetus and Newborn. Hospital discharge of the high-risk neonate. *Pediatrics*. 2008;122(5):1119–1126
104. Vernacchio L, Corwin MJ, Lesko SM, et al. Sleep position of low birth weight infants. *Pediatrics*. 2003;111(3):633–640
105. Pollack HA, Frohna JG. Infant sleep placement after the Back to Sleep campaign. *Pediatrics*. 2002;109(4):608–614
106. Baird TM, Paton JB, Fisher DE. Improved oxygenation with prone positioning in neonates: stability of increased transcutaneous P_{O_2} . *J Perinatol*. 1991;11(4):315–317
107. Martin RJ, DiFiore JM, Korenke CB, Randal H, Miller MJ, Brooks LJ. Vulnerability of respiratory control in healthy preterm infants placed supine. *J Pediatr*. 1995;127(4):609–614
108. Levy J, Habib RH, Liptsen E, et al. Prone versus supine positioning in the well preterm infant: Effects on work of breathing and breathing patterns. *Pediatr Pulmonol*. 2006;41(8):754–758
109. Grazel R, Phalen AG, Polomano RC. Implementation of the American Academy of Pediatrics recommendations to reduce sudden infant death syndrome risk in neonatal intensive care units: an evaluation of nursing knowledge and practice. *Adv Neonatal Care*. 2010;10(6):332–342
110. Moon RY, Oden RP, Joyner BL, Ajao TI. Qualitative analysis of beliefs and perceptions about sudden infant death syndrome (SIDS) among African-American mothers: implications for safe sleep recommendations. *J Pediatr*. 2010;157(1):92–97.e2
111. Brenner R, Simons-Morton BG, Bhaskar B, et al. Prevalence and predictors of the prone sleep position among inner-city infants. *JAMA*. 1998;280(4):341–346
112. Willinger M, Hoffman HJ, Wu KT, et al. Factors associated with the transition to nonprone sleep positions of infants in the United States: the National Infant Sleep Position Study. *JAMA*. 1998;280(4):329–335
113. Hutchison BL, Thompson JM, Mitchell EA. Determinants of nonsynostotic plagiocephaly: a case-control study. *Pediatrics*. 2003;112(4). Available at: www.pediatrics.org/cgi/content/full/112/4/e316
114. Hutchison BL, Hutchison LA, Thompson JM, Mitchell EA. Plagiocephaly and brachycephaly in the first two years of life: a prospective cohort study. *Pediatrics*. 2004;114(4):970–980
115. van Vlimmeren LA, van der Graaf Y, Boere-Boonekamp MM, L'Hoir MP, Helders PJ, Engelbert RH. Risk factors for deformational plagiocephaly at birth and at 7 weeks of age: a prospective cohort study. *Pediatrics*. 2007;119(2). Available at: www.pediatrics.org/cgi/content/full/119/2/e408
116. Miller RI, Clarren SK. Long-term developmental outcomes in patients with deformational plagiocephaly. *Pediatrics*. 2000;105(2). Available at: www.pediatrics.org/cgi/content/full/105/2/e26
117. Panchal J, Amirshaybani H, Gurwitsch R, et al. Neurodevelopment in children with single-suture craniosynostosis and plagiocephaly without synostosis. *Plast Reconstr Surg*. 2001;108(6):1492–1498; discussion 1499–1500
118. Balan P, Kushnerenko E, Sahlin P, Huotilainen M, Naatanen R, Hukki J. Auditory ER: Ps reveal brain dysfunction in infants with plagiocephaly. *J Craniofac Surg*. 2002;13(4):520–525; discussion 526
119. Chaddock WM, Kast J, Donahue DJ. The enigma of lambdoid positional molding. *Pediatr Neurosurg*. 1997;26(6):304–311
120. Laughlin J, Luerssen TG, Dias MS; American Academy of Pediatrics, Committee on Practice and Ambulatory Medicine, Section on Neurological Surgery. Clinical report—prevention and management of positional skull deformities in infants. *Pediatrics*. 2011;128(6). In press
121. US Consumer Product Safety Commission. Crib Safety Tips: Use Your Crib Safely. Washington, DC: US Consumer Product Safety Commission. CPSC document 5030
122. Pike J, Moon RY. Bassinet use and sudden unexpected death in infancy. *J Pediatr*. 2008;153(4):509–512
123. Jackson A, Moon RY. An analysis of deaths in portable cribs and playpens: what can be learned? *Clin Pediatr (Phila)*. 2008;47(3):261–266
124. Nakamura S, Wind M, Danello MA. Review of hazards associated with children placed in adult beds. *Arch Pediatr Adolesc Med*. 1999;153(10):1019–1023
125. US Consumer Product Safety Commission. Staff Briefing Package, Draft Proposed Rule for Portable Bed Rails (CPSIA Section 104). Bethesda, MD: US Consumer Product Safety Commission; 2011
126. Callahan CW, Sisler C. Use of seating devices in infants too young to sit. *Arch Pediatr Adolesc Med*. 1997;151(3):233–235
127. Orenstein SR, Whittington PF, Orenstein DM.

- The infant seat as treatment for gastroesophageal reflux. *N Engl J Med*. 1983;309(13):760–763
128. Bass JL, Bull M. Oxygen desaturation in term infants in car safety seats. *Pediatrics*. 2002;110(2 pt 1):401–402
 129. Cerar LK, Scirica CV, Stucin Gantar I, Osredkar D, Neubauer D, Kinane TB. A comparison of respiratory patterns in healthy term infants placed in car safety seats and beds. *Pediatrics*. 2009;124(3). Available at: www.pediatrics.org/cgi/content/full/124/3/e3396
 130. Côté A, Bairam A, Deschenes M, Hatzakis G. Sudden infant deaths in sitting devices. *Arch Dis Child*. 2008;93(5):384–389
 131. Merchant JR, Worwa C, Porter S, Coleman JM, deRegnier RA. Respiratory instability of term and near-term healthy newborn infants in car safety seats. *Pediatrics*. 2001;108(3):647–652
 132. Willett LD, Leuschen MP, Nelson LS, Nelson RM Jr. Risk of hypoventilation in premature infants in car seats. *J Pediatr*. 1986;109(2):245–248
 133. Desapriya EB, Joshi P, Subzwari S, Nolan M. Infant injuries from child restraint safety seat misuse at British Columbia Children's Hospital. *Pediatr Int*. 2008;50(5):674–678
 134. Graham CJ, Kittredge D, Stuemky JH. Injuries associated with child safety seat misuse. *Pediatr Emerg Care*. 1992;8(6):351–353
 135. Parikh SN, Wilson L. Hazardous use of car seats outside the car in the United States, 2003–2007. *Pediatrics*. 2010;126(2):352–357
 136. Pollack-Nelson C. Fall and suffocation injuries associated with in-home use of car seats and baby carriers. *Pediatr Emerg Care*. 2000;16(2):77–79
 137. Wickham T, Abrahamson E. Head injuries in infants: the risks of bouncy chairs and car seats. *Arch Dis Child*. 2002;86(3):168–169
 138. US Consumer Product Safety Commission. *Infant Deaths Prompt CPSC Warning About Sling Carriers for Babies*. Washington, DC: US Consumer Product Safety Commission; 2010
 139. McKenna JJ, Thoman EB, Anders TF, Sadeh A, Schechtman VL, Glotzbach SF. Infant-parent co-sleeping in an evolutionary perspective: implications for understanding infant sleep development and the sudden infant death syndrome. *Sleep*. 1993;16(3):263–282
 140. McKenna JJ, Ball HL, Gettler LT. Mother-infant cosleeping, breastfeeding and sudden infant death syndrome: what biological anthropology has discovered about normal infant sleep and pediatric sleep medicine. *Am J Phys Anthropol*. 2007;(suppl 45):133–161
 141. McKenna J. *Sleeping With Your Baby: A Parent's Guide to Cosleeping*. Washington, DC: Platyus Media, LLC; 2007
 142. Mitchell EA, Thompson JMD. Co-sleeping increases the risk of SIDS, but sleeping in the parents' bedroom lowers it. In: Rognum TO, ed. *Sudden Infant Death Syndrome: New Trends in the Nineties*. Oslo, Norway: Scandinavian University Press; 1995:266–269
 143. Tappin D, Ecob R, Brooke H. Bedsharing, roomsharing, and sudden infant death syndrome in Scotland: a case control study. *J Pediatr*. 2005;147(1):32–37
 144. Joyner BL, Oden R, Ajao TI, Moon R. Where should my baby sleep? A qualitative study of African-American infant sleep location decisions. *J Natl Med Assoc*. 2010;102(10):881–889
 145. Mosko S, Richard C, McKenna J. Infant arousals during mother-infant bed sharing: implications for infant sleep and sudden infant death syndrome research. *Pediatrics*. 1997;100(5):841–849
 146. McKenna JJ, Mosko S, Richard CA. Bed-sharing promotes breastfeeding. *Pediatrics*. 1997;100(2 pt 1):214–219
 147. Scheers NJ, Dayton CM, Kemp JS. Sudden infant death with external airways covered: case-comparison study of 206 deaths in the United States. *Arch Pediatr Adolesc Med*. 1998;152(6):540–547
 148. Unger B, Kemp JS, Wilkins D, et al. Racial disparity and modifiable risk factors among infants dying suddenly and unexpectedly. *Pediatrics*. 2003;111(2). Available at: www.pediatrics.org/cgi/content/full/111/2/e127
 149. Kemp JS, Unger B, Wilkins D, et al. Unsafe sleep practices and an analysis of bed-sharing among infants dying suddenly and unexpectedly: results of a four-year, population-based, death-scene investigation study of sudden infant death syndrome and related deaths. *Pediatrics*. 2000;106(3). Available at: www.pediatrics.org/cgi/content/full/106/3/e41
 150. Drago DA, Dannenberg AL. Infant mechanical suffocation deaths in the United States, 1980–1997. *Pediatrics*. 1999;103(5). Available at: www.pediatrics.org/cgi/content/full/103/5/e59
 151. Baddock SA, Galland BC, Beckers MG, et al. Bed-sharing and the infant's thermal environment in the home setting. *Arch Dis Child*. 2004;89(12):1111–1116
 152. Baddock SA, Galland BC, Bolton DP, Williams SM, Taylor BJ. Differences in infant and parent behaviors during routine bed sharing compared with cot sleeping in the home setting. *Pediatrics*. 2006;117(5):1599–1607
 153. McIntosh CG, Tonkin SL, Gunn AJ. What is the mechanism of sudden infant deaths associated with co-sleeping? *N Z Med J*. 2009;122(1307):69–75
 154. Blair PS, Mitchell EA, Heckstall-Smith EM, Fleming PJ. Head covering: a major modifiable risk factor for sudden infant death syndrome—a systematic review. *Arch Dis Child*. 2008;93(9):778–783
 155. Baddock SA, Galland BC, Taylor BJ, Bolton DP. Sleep arrangements and behavior of bed-sharing families in the home setting. *Pediatrics*. 2007;119(1). Available at: www.pediatrics.org/cgi/content/full/119/1/e200
 156. Ball H. Airway covering during bed-sharing. *Child Care Health Dev*. 2009;35(5):728–737
 157. American Academy of Pediatrics, Task Force on Infant Sleep Position and Sudden Infant Death Syndrome. Changing concepts of sudden infant death syndrome: implications for infant sleeping environment and sleep position. *Pediatrics*. 2000;105(3 pt 1):650–656
 158. Vennemann M, Hense HW, Bajanowski T, et al. Bed sharing and the risk of SIDS: can we resolve the debate? *J Pediatr*. 2011; In press
 159. Ostfeld BM, Perl H, Esposito L, et al. Sleep environment, positional, lifestyle, and demographic characteristics associated with bed sharing in sudden infant death syndrome cases: a population-based study. *Pediatrics*. 2006;118(5):2051–2059
 160. Scheers NJ, Rutherford GW, Kemp JS. Where should infants sleep? A comparison of risk for suffocation of infants sleeping in cribs, adult beds, and other sleeping locations. *Pediatrics*. 2003;112(4):883–889
 161. Blair P, Ward Platt MP, Smith IJ, Fleming PJ. Sudden infant death syndrome and sleeping position in pre-term and low birthweight infants: an opportunity for targeted intervention. *Arch Dis Child*. 2006;91(2):101–106
 162. Lin RG. Infant deaths prompt warning. *Los Angeles Times*. April 24, 2008. Available at: <http://articles.latimes.com/2008/apr/24/local/me-deaths24>. Accessed September 6, 2011
 163. Cambria N. Officials issue warning to prevent infant death. *St Louis Post-Dispatch*. January 24, 2009. Available at: www.stltoday.com/news/article_738ffce-5343-534c-bf89-0582e440adf5.html. Accessed September 20, 2011

164. Department of Human Services. *DHS and Health Department Launch New Campaign Warning to Parents* [press release]. Philadelphia, PA: Department of Human Services; 2007. Available at: www.phila.gov/dhs/news_sleepingSafely.html. Accessed September 6, 2011
165. Brewington K. Don't share the bed with infant, parents told. *Baltimore Sun*. January 26, 2009
166. Arnestad M, Andersen M, Vege A, Rognum TO. Changes in the epidemiological pattern of sudden infant death syndrome in south-east Norway, 1984–1998: implications for future prevention and research. *Arch Dis Child*. 2001;85(2):108–115
167. Scragg R, Mitchell EA, Taylor BJ, et al. Bed sharing, smoking, and alcohol in the sudden infant death syndrome. New Zealand Cot Death Study Group. *BMJ*. 1993;307(6915):1312–1318
168. McGarvey C, McDonnell M, Hamilton K, O'Regan M, Matthews T. An 8 year study of risk factors for SIDS: bed-sharing vs. non bed-sharing. *Arch Dis Child*. 2006;91(4):318–323
169. McGarvey C, McDonnell M, Chong A, O'Regan M, Matthews T. Factors relating to the infant's last sleep environment in sudden infant death syndrome in the Republic of Ireland. *Arch Dis Child*. 2003;88(12):1058–1064
170. Fu LY, Moon R, Hauck FR. Bed sharing among black infants and sudden infant death syndrome: interactions with other known risk factors. *Acad Pediatr*. 2010;10(6):376–382
171. Blair PS, Sidebotham P, Evason-Coombe C, Edmonds M, Heckstall-Smith EM, Fleming P. Hazardous cosleeping environments and risk factors amenable to change: case-control study of SIDS in south west England. *BMJ*. 2009;339:b3666
172. Carroll-Pankhurst C, Mortimer EAJ. Sudden infant death syndrome, bedsharing, parental weight, and age at death. *Pediatrics*. 2001;107(3):530–536
173. Mitchell E, Thompson J. Who cosleeps? Does high maternal body weight and duvet use increase the risk of sudden infant death syndrome when bed sharing? *Paediatr Child Health*. 2006;11(suppl):14A–15A
174. Hutchison BL, Stewart AW, Mitchell EA. The prevalence of cobedding and SIDS-related child care practices in twins. *Eur J Pediatr*. 2010;169(12):1477–1485
175. Hayward K. Cobedding of twins: a natural extension of the socialization process? *MCN Am J Matern Child Nurs*. 2003;28(4):260–263
176. Tomashek KM, Wallman C; American Academy of Pediatrics, Committee on Fetus and Newborn. Cobedding twins and higher-order multiples in a hospital setting [published correction appears in *Pediatrics*. 2008;121(1):227]. *Pediatrics*. 2007;120(6):1359–1366
177. National Association of Neonatal Nurses Board of Directors. NANN position statement 3045: cobedding of twins or higher-order multiples. *Adv Neonatal Care*. 2009;9(6):307–313
178. Moon RY, Oden RP, Joyner BL, Ajao TI. Reasons that African-American parents use soft bedding and soft sleep surfaces for their infants [abstract]. *J Paediatr Child Health*. 2010;16(suppl 3):43
179. Chiodini BA, Thach BT. Impaired ventilation in infants sleeping facedown: potential significance for sudden infant death syndrome. *J Pediatr*. 1993;123(5):686–692
180. Kemp JS, Nelson VE, Thach BT. Physical properties of bedding that may increase risk of sudden infant death syndrome in prone-sleeping infants. *Pediatr Res*. 1994;36(1 pt 1):7–11
181. Sakai J, Kanetake J, Takahashi S, Kanawaku Y, Funayama M. Gas dispersal potential of bedding as a cause for sudden infant death. *Forensic Sci Int*. 2008;180(2–3):93–97
182. Ponsonby AL, Dwyer T, Gibbons LE, Cochrane JA, Wang YG. Factors potentiating the risk of sudden infant death syndrome associated with the prone position. *N Engl J Med*. 1993;329(6):377–382
183. Mitchell EA, Thompson JMD, Ford RPK, Taylor BJ. Sheepskin bedding and the sudden infant death syndrome. New Zealand Cot Death Study Group. *J Pediatr*. 1998;133(5):701–704
184. Brooke H, Gibson A, Tappin D, Brown H. Case-control study of sudden infant death syndrome in Scotland, 1992–5. *BMJ*. 1997;314(7093):1516–1520
185. Kemp JS, Kowalski RM, Burch PM, Graham MA, Thach BT. Unintentional suffocation by rebreathing: a death scene and physiologic investigation of a possible cause of sudden infant death. *J Pediatr*. 1993;122(6):874–880
186. Ponsonby AL, Dwyer T, Couper D, Cochrane J. Association between use of a quilt and sudden infant death syndrome: case-control study. *BMJ*. 1998;316(7126):195–196
187. Mitchell EA, Scragg L, Clements M. Soft cot mattresses and the sudden infant death syndrome. *N Z Med J*. 1996;109(1023):206–207
188. L'Hoir MP, Engelberts AC, van Well GTJ, et al. Risk and preventive factors for cot death in the Netherlands, a low-incidence country. *Eur J Pediatr*. 1998;157(8):681–688
189. Markestad T, Skadberg B, Hordvik E, Morrild I, Irgens L. Sleeping position and sudden infant death syndrome (SIDS): effect of an intervention programme to avoid prone sleeping. *Acta Paediatr*. 1995;84(4):375–378
190. Beal SM, Byard RW. Accidental death or sudden infant death syndrome? *J Paediatr Child Health*. 1995;31(4):269–271
191. Wilson CA, Taylor BJ, Laing RM, Williams SM, Mitchell EA. Clothing and bedding and its relevance to sudden infant death syndrome: further results from the New Zealand Cot Death Study. *J Paediatr Child Health*. 1994;30(6):506–512
192. Chowdhury RT. *Nursery Product-Related Injuries and Deaths Among Children Under Age Five*. Washington, DC: US Consumer Product Safety Commission; 2009
193. Chowdhury RT. *Nursery Product-Related Injuries and Deaths Among Children Under Age Five*. Washington, DC: US Consumer Product Safety Commission; 2010
194. US Food and Drug Administration. CPSC and FDA warn against using infant sleep positioners because of suffocation risk: initial communication. Available at: www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm227301.htm. Accessed September 6, 2011
195. Moon RY. "And things that go bump in the night": nothing to fear? *J Pediatr*. 2007;151(3):237–238
196. Thach BT, Rutherford GW, Harris K. Deaths and injuries attributed to infant crib bumper pads. *J Pediatr*. 2007;151(3):271–274
197. Yeh ES, Rochette LM, McKenzie LB, Smith GA. Injuries associated with cribs, playpens, and bassinets among young children in the US, 1990–2008. *Pediatrics*. 2011;127(3):479–486
198. Kraus JF, Greenland S, Bulterys M. Risk factors for sudden infant death syndrome in the US Collaborative Perinatal Project. *Int J Epidemiol*. 1989;18(1):113–120
199. Paris C, Remler R, Daling JR. Risk factors for sudden infant death syndrome: changes associated with sleep position recommendations. *J Pediatr*. 2001;139(6):771–777
200. Stewart A, Williams S, Mitchell E, Taylor BJ, Ford R, Allen EM. Antenatal and intrapartum factors associated with sudden infant death syndrome in the New Zealand Cot Death Study. *J Paediatr Child Health*. 1995;31(5):473–478

201. MacDorman MF, Cnattingius S, Hoffman HJ, Kramer MS, Haglund B. Sudden infant death syndrome and smoking in the United States and Sweden. *Am J Epidemiol*. 1997; 146(3):249–257
202. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*. 1992;90(6):905–908
203. Malloy MH, Kleinman JC, Land GH, Schramm WF. The association of maternal smoking with age and cause of infant death. *Am J Epidemiol*. 1988;128(1):46–55
204. Haglund B, Cnattingius S. Cigarette smoking as a risk factor for sudden infant death syndrome: a population-based study. *Am J Public Health*. 1990;80(1):29–32
205. Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. *Pediatrics*. 1993;91(5):893–896
206. Winickoff JP, Friebely J, Tanski SE, et al. Beliefs about the health effects of “third-hand” smoke and home smoking bans. *Pediatrics*. 2009;123(1). Available at: www.pediatrics.org/cgi/content/full/123/1/e74
207. Tirosh E, Libon D, Bader D. The effect of maternal smoking during pregnancy on sleep respiratory and arousal patterns in neonates. *J Perinatol*. 1996;16(6):435–438
208. Franco P, Groswasser J, Hassid S, Lanquart JP, Scaillet S, Kahn A. Prenatal exposure to cigarette smoking is associated with a decrease in arousal in infants. *J Pediatr*. 1999;135(1):34–38
209. Horne RS, Ferens D, Watts AM, et al. Effects of maternal tobacco smoking, sleeping position, and sleep state on arousal in healthy term infants. *Arch Dis Child Fetal Neonatal Ed*. 2002;87(2):F100–F105
210. Sawnani H, Jackson T, Murphy T, Beckerman R, Simakajornboon N. The effect of maternal smoking on respiratory and arousal patterns in preterm infants during sleep. *Am J Respir Crit Care Med*. 2004; 169(6):733–738
211. Lewis KW, Bosque EM. Deficient hypoxia awakening response in infants of smoking mothers: possible relationship to sudden infant death syndrome. *J Pediatr*. 1995; 127(5):691–699
212. Chang AB, Wilson SJ, Masters IB, et al. Altered arousal response in infants exposed to cigarette smoke. *Arch Dis Child*. 2003; 88(1):30–33
213. Parsiow PM, Cranage SM, Adamson TM, Harding R, Horne RS. Arousal and ventilatory responses to hypoxia in sleeping infants: effects of maternal smoking. *Respir Physiol Neurobiol*. 2004;140(1):77–87
214. Mitchell EA, Milerad J. Smoking and the sudden infant death syndrome. *Rev Environ Health*. 2006;21(2):81–103
215. Dietz PM, England LJ, Shapiro-Mendoza CK, Tong VT, Farr SL, Callaghan WM. Infant morbidity and mortality attributable to prenatal smoking in the U.S. *Am J Prev Med*. 2010;39(1):45–52
216. American Academy of Pediatrics, Committee on Environmental Health; Committee on Substance Abuse; Committee on Adolescence; Committee on Native American Child. Policy statement—tobacco use: a pediatric disease [published correction appears in *Pediatrics*. 2010;125(4):861]. *Pediatrics*. 2009;124(5):1474–1487
217. Best D; Committee on Environmental Health; Committee on Native American Child Health; Committee on Adolescence. Technical report—secondhand and prenatal tobacco smoke exposure. *Pediatrics*. 2009;124(5). Available at: www.pediatrics.org/cgi/content/full/124/5/e1017
218. Iyasu S, Randall LL, Welty TK, et al. Risk factors for sudden infant death syndrome among Northern Plains Indians. *JAMA*. 2002;288(21):2717–2723
219. Strandberg-Larsen K, Gronboek M, Andersen AM, Andersen PK, Olsen J. Alcohol drinking pattern during pregnancy and risk of infant mortality. *Epidemiology*. 2009;20(6):884–891
220. Alm B, Wennergren G, Norvenius G, et al. Caffeine and alcohol as risk factors for sudden infant death syndrome. Nordic Epidemiological SIDS Study. *Arch Dis Child*. 1999;81(2):107–111
221. James C, Klenka H, Manning D. Sudden infant death syndrome: bed sharing with mothers who smoke. *Arch Dis Child*. 2003; 88(2):112–113
222. Klonoff-Cohen H, Edelstein SL. Bed sharing and the sudden infant death syndrome. *BMJ*. 1995;311(7015):1269–1272
223. Rajegowda BK, Kandall SR, Falciglia H. Sudden unexpected death in infants of narcotic-dependent mothers. *Early Hum Dev*. 1978;2(3):219–225
224. Chavez CJ, Ostrea EM Jr, Stryker JC, Smialek Z. Sudden infant death syndrome among infants of drug-dependent mothers. *J Pediatr*. 1979;95(3):407–409
225. Bauchner H, Zuckerman B, McClain M, Frank D, Fried LE, Kayne H. Risk of sudden infant death syndrome among infants with in utero exposure to cocaine. *J Pediatr*. 1988;113(5):831–834
226. Durand DJ, Espinoza AM, Nickerson BG. Association between prenatal cocaine exposure and sudden infant death syndrome. *J Pediatr*. 1990;117(6):909–911
227. Ward SL, Bautista D, Chan L, et al. Sudden infant death syndrome in infants of substance-abusing mothers. *J Pediatr*. 1990;117(6):876–881
228. Rosen TS, Johnson HL. Drug-addicted mothers, their infants, and SIDS. *Ann NY Acad Sci*. 1988;533:89–95
229. Kandall SR, Gaines J, Habel L, Davidson G, Jessop D. Relationship of maternal substance abuse to subsequent sudden infant death syndrome in offspring. *J Pediatr*. 1993;123(1):120–126
230. Fares I, McCulloch KM, Raju TN. Intrauterine cocaine exposure and the risk for sudden infant death syndrome: a meta-analysis. *J Perinatol*. 1997;17(3):179–182
231. Tappin D, Brooke H, Ecob R, Gibson A. Used infant mattresses and sudden infant death syndrome in Scotland: case-control study. *BMJ*. 2002;325(7371):1007–1012
232. Ford RP, Taylor BJ, Mitchell EA, et al. Breastfeeding and the risk of sudden infant death syndrome. *Int J Epidemiol*. 1993; 22(5):885–890
233. Hoffman HJ, Damus K, Hillman L, Krongrad E. Risk factors for SIDS: results of the National Institute of Child Health and Human Development SIDS Cooperative Epidemiological Study. *Ann NY Acad Sci*. 1988;533: 13–30
234. Henderson-Smart DJ, Ponsonby AL, Murphy E. Reducing the risk of sudden infant death syndrome: a review of the scientific literature. *J Paediatr Child Health*. 1998; 34(3):213–219
235. Ponsonby AL, Dwyer T, Kasl SV, Cochrane JA. The Tasmanian SIDS Case-Control Study: univariable and multivariable risk factor analysis. *Paediatr Perinat Epidemiol*. 1995;9(3):256–272
236. Alm B, Wennergren G, Norvenius SG, et al. Breast feeding and the sudden infant death syndrome in Scandinavia, 1992–95. *Arch Dis Child*. 2002;86(6):400–402
237. McVea KLSP, Turner PD, Peppler DK. The role of breastfeeding in sudden infant death syndrome. *J Hum Lact*. 2000;16(1): 13–20
238. Gilbert RE, Wighfield RE, Fleming PJ, Berry PJ, Rudd PT. Bottle feeding and the sudden infant death syndrome. *BMJ*. 1995; 310(6972):88–90
239. l’Hoir MP, Engelberts AC, van Well GTJ, et al. Case-control study of current validity of previously described risk factors for SIDS in the Netherlands. *Arch Dis Child*. 1998; 79(5):386–393
240. Wennergren G, Alm B, Oyen N, et al. The decline in the incidence of SIDS in Scandinavia and its relation to risk-intervention

- campaigns. Nordic Epidemiological SIDS Study. *Acta Paediatr*. 1997;86(9):963–968
241. Biering-Sørensen F, Jørgensen T, Hilden J. Sudden infant death in Copenhagen 1956–1971: I. Infant feeding. *Acta Paediatr Scand*. 1978;67(2):129–137
242. Watson E, Gardner A, Carpenter RG. An epidemiological and sociological study of unexpected death in infancy in nine areas of southern England. I: Epidemiology. *Med Sci Law*. 1981;21(2):78–88
243. Ip S, Chung M, Raman G, Trikalinos TA, Lau J. A summary of the Agency for Healthcare Research and Quality's evidence report on breastfeeding in developed countries. *Breastfeed Med*. 2009;4(suppl 1):S17–S30
244. Vennemann MM, Bajanowski T, Brinkmann B, et al; GeSID Study Group. Does breastfeeding reduce the risk of sudden infant death syndrome? *Pediatrics*. 2009;123. Available at: www.pediatrics.org/cgi/content/full/123/3/e406
245. Hauck FR, Thompson J, Tanabe KO, Moon RY, Vennemann M. Breastfeeding and reduced risk of sudden infant death syndrome: a meta-analysis. *Pediatrics*. 2011;128(1):103–110
246. Centers for Disease Control and Prevention. Racial and ethnic differences in breastfeeding initiation and duration, by state: National Immunization Survey, United States, 2004–2008. *MMWR Morb Mortal Wkly Rep*. 2010;59(11):327–334
247. Franco P, Scaillet S, Wermenbol V, Valente F, Groswasser J, Kahn A. The influence of a pacifier on infants' arousals from sleep. *J Pediatr*. 2000;136(6):775–779
248. Horne RS, Parslow PM, Ferens D, Watts AM, Adamson TM. Comparison of evoked arousability in breast and formula fed infants. *Arch Dis Child*. 2004;89(1):22–25
249. Duijts L, Jaddoe VW, Hofman A, Moll HA. Prolonged and exclusive breastfeeding reduces the risk of infectious diseases in infancy. *Pediatrics*. 2010;126(1). Available at: www.pediatrics.org/cgi/content/full/126/1/e18
250. Heinig MJ. Host defense benefits of breastfeeding for the infant: effect of breastfeeding duration and exclusivity. *Pediatr Clin North Am*. 2001;48(1):105–123, ix
251. Kramer MS, Guo T, Platt RW, et al. Infant growth and health outcomes associated with 3 compared with 6 mo of exclusive breastfeeding. *Am J Clin Nutr*. 2003;78(2):291–295
252. Academy of Breastfeeding Medicine Protocol Committee. ABM clinical protocol #6: guideline on co-sleeping and breastfeeding. Revision, March 2008. *Breastfeed Med*. 2008;3(1):38–43
253. McKenna J. Sleeping with your baby. *New Beginnings (La Leche League International)*. 2009;26(1):4–9
254. Hauck FR, Signore C, Fein SB, Raju TN. Infant sleeping arrangements and practices during the first year of life. *Pediatrics*. 2008;122(suppl 2):S113–S120
255. Horsley T, Clifford T, Barrowman N, et al. Benefits and harms associated with the practice of bed sharing: a systematic review. *Arch Pediatr Adolesc Med*. 2007;161(3):237–245
256. Hauck FR, Signore C, Fein SB, Raju TN. Infant feeding practices and mother-infant bed sharing: is there an association? Results from the Infant Feeding Practices Study II [abstract]. Presented at: American Public Health Association annual meeting; October 25, 2007; Washington, DC
257. Vogel A, Hutchison BL, Mitchell EA. Factors associated with the duration of breastfeeding. *Acta Paediatr*. 1999;88(12):1320–1326
258. Ruys JH, de Jonge GA, Brand R, Engelberts AC, Semmekrot BA. Bed-sharing in the first four months of life: a risk factor for sudden infant death. *Acta Paediatr*. 2007;96(10):1399–1403
259. Arnestad M, Andersen M, Rognum TO. Is the use of dummy or carry-cot of importance for sudden infant death? *Eur J Pediatr*. 1997;156(12):968–970
260. Mitchell EA, Taylor BJ, Ford RPK, et al. Dummies and the sudden infant death syndrome. *Arch Dis Child*. 1993;68(4):501–504
261. Fleming PJ, Blair PS, Pollard K, et al. Pacifier use and sudden infant death syndrome: results from the CESDI/SUDI case control study. CESDI SUDI Research Team. *Arch Dis Child*. 1999;81(2):112–116
262. L'Hoir MP, Engleberts AC, van Well GTJ, et al. Dummy use, thumb sucking, mouth breathing and cot death. *Eur J Pediatr*. 1999;158(11):896–901
263. Hauck FR, Omojokun OO, Siadaty MS. Do pacifiers reduce the risk of sudden infant death syndrome? A meta-analysis. *Pediatrics*. 2005;116(5). Available at: www.pediatrics.org/cgi/content/full/116/5/e716
264. Mitchell EA, Blair PS, L'Hoir MP. Should pacifiers be recommended to prevent SIDS? *Pediatrics*. 2006;117(5):1755–1758
265. Li DK, Willinger M, Petitti DB, Odouli R, Liu L, Hoffman HJ. Use of a dummy (pacifier) during sleep and risk of sudden infant death syndrome (SIDS): population based case-control study. *BMJ*. 2006;332(7532):18–22
266. Vennemann MM, Bajanowski T, Brinkmann B, Jorch G, Sauerland C, Mitchell EA; GeSID Study Group. Sleep environment risk factors for sudden infant death syndrome: the German Sudden Infant Death Syndrome Study. *Pediatrics*. 2009;123(4):1162–1170
267. Kahn A, Sawaguchi T, Sawaguchi A, et al. Sudden infant deaths: from epidemiology to physiology. *Forensic Sci Int*. 2002;130(suppl):S8–S20
268. Franco P, Chabanski S, Scaillet S, Groswasser J, Kahn A. Pacifier use modifies infant's cardiac autonomic controls during sleep. *Early Hum Dev*. 2004;77(1–2):99–108
269. Tonkin SL, Lui D, McIntosh CG, Rowley S, Knight DB, Gunn AJ. Effect of pacifier use on mandibular position in preterm infants. *Acta Paediatr*. 2007;96(10):1433–1436
270. Horne RS, Witcombe NB, Yiallourou SR, Richardson HL. Sudden infant death syndrome: implications of altered physiological control during sleep. *Curr Pediatr Rev*. 2010;6(1):30–38
271. Weiss P, Kerbl R. The relatively short duration that a child retains a pacifier in the mouth during sleep: implications for sudden infant death syndrome. *Eur J Pediatr*. 2001;160(1):60–70
272. Foundation for the Study and Prevention of Infant Mortality (Cot Death Foundation). Safe sleeping for your baby. Available at: www.wiegedood.nl/files/download_vs_engels.pdf. Accessed September 21, 2011
273. Foundation for the Study of Infant Deaths. Factfile 2: research background to the Reduce the Risk of Cot Death advice by the Foundation for the Study of Infant Deaths. Foundation for the Study of Infant Deaths. Available at: <http://fsid.org.uk/Document.Doc?id=42>. Accessed September 21, 2011
274. SIDS and Kids. National Scientific Advisory Group. *Information Statement: Pacifier/Dummy Use*. Melbourne, Australia: National SIDS Council of Australia; 2009. Available at: www.sidsandkids.org/wp-content/uploads/Pacifier_Dummy_Use.pdf. Accessed September 11, 2011
275. Aarts C, Hörnell A, Kylberg E, Hofvander Y, Gebre-Medhin M. Breastfeeding patterns in relation to thumb sucking and pacifier use. *Pediatrics*. 1999;104(4). Available at: www.pediatrics.org/cgi/content/full/104/4/e50
276. Benis MM. Are pacifiers associated with early weaning from breastfeeding? *Adv Neonatal Care*. 2002;2(5):259–266
277. Scott JA, Binns CW, Oddy WH, Graham KI. Predictors of breastfeeding duration: evidence from a cohort study. *Pediatrics*.

- 2006;117(4). Available at: www.pediatrics.org/cgi/content/full/117/4/e646
278. Kramer MS, Barr RG, Dagenais S, et al. Pacifier use, early weaning, and cry/fuss behavior. *JAMA*. 2001;286(3):322–326
 279. Collins CT, Ryan P, Crowther CA, McPhee AJ, Paterson S, Hiller JE. Effect of bottles, cups, and dummies on breast feeding in preterm infants: a randomised controlled trial. *BMJ*. 2004;329(7459):193–198
 280. Howard CR, Howard FM, Lanphear B, et al. Randomized clinical trial of pacifier use and bottle-feeding or cupfeeding and their effect on breastfeeding. *Pediatrics*. 2003; 111(3):511–518
 281. O'Connor NR, Tanabe KO, Siadaty MS, Hauck FR. Pacifiers and breastfeeding: a systematic review. *Arch Pediatr Adolesc Med*. 2009;163(4):378–382
 282. Jenik AG, Vain NE, Gorestein AN, Jacobi NE; Pacifier and Breastfeeding Trial Group. Does the recommendation to use a pacifier influence the prevalence of breastfeeding? *J Pediatr*. 2009;155(3): 350–354.e1
 283. Gartner LM, Morton J, Lawrence RA, et al; American Academy of Pediatrics, Section on Breastfeeding. Breastfeeding and the use of human milk. *Pediatrics*. 2005; 115(2):496–506
 284. Larsson E. The effect of dummy-sucking on the occlusion: a review. *Eur J Orthod*. 1986; 8(2):127–130
 285. American Academy of Pediatric Dentistry, Council on Clinical Affairs. Policy on oral habits. Available at: www.aapd.org/media/Policies_Guidelines/P_OralHabits.pdf. Accessed April 15, 2011
 286. Niemelä M, Uhari M, Möttönen M. A pacifier increases the risk of recurrent acute otitis media in children in day care centers. *Pediatrics*. 1995;96(5 pt 1):884–888
 287. Niemelä M, Pihakari O, Pokka T, Uhari M. Pacifier as a risk factor for acute otitis media: a randomized, controlled trial of parental counseling. *Pediatrics*. 2000; 106(3):483–488
 288. Jackson JM, Mourino AP. Pacifier use and otitis media in infants twelve months of age or younger. *Pediatr Dent*. 1999;21(4): 255–260
 289. Daly KA, Giebink GS. Clinical epidemiology of otitis media. *Pediatr Infect Dis J*. 2000; 19(5 suppl):S31–S36
 290. Darwazeh AM, al-Bashir A. Oral candidal flora in healthy infants. *J Oral Pathol Med*. 1995;24(8):361–364
 291. North K, Fleming PJ, Golding J. Pacifier use and morbidity in the first six months of life. *Pediatrics*. 1999;103(3). Available at: www.pediatrics.org/cgi/content/full/103/3/e34
 292. Niemelä M, Uhari M, Hannuksela A. Pacifiers and dental structure as risk factors for otitis media. *Int J Pediatr Otorhinolaryngol*. 1994;29(2):121–127
 293. Uhari M, Mantysaari K, Niemelä M. A meta-analytic review of the risk factors for acute otitis media. *Clin Infect Dis*. 1996;22(6): 1079–1083
 294. Fleming P, Gilbert R, Azaz Y, et al. Interaction between bedding and sleeping position in the sudden infant death syndrome: a population based case-control study. *BMJ*. 1990;301(6743):85–89
 295. Ponsonby AL, Dwyer T, Gibbons LE, Cochrane JA, Jones ME, McCall MJ. Thermal environment and sudden infant death syndrome: case-control study. *BMJ*. 1992; 304(6822):277–282
 296. McGlashan ND. Sudden infant deaths in Tasmania, 1980–1986: a seven year prospective study. *Soc Sci Med*. 1989;29(8): 1015–1026
 297. Coleman-Phox K, Odouli R, Li DK. Use of a fan during sleep and the risk of sudden infant death syndrome. *Arch Pediatr Adolesc Med*. 2008;162(10):963–968
 298. Chisholm JS. Swaddling, cradleboards and the development of children. *Early Hum Dev*. 1978;2(3):255–275
 299. Gerard CM, Harris KA, Thach BT. Physiologic studies on swaddling: an ancient child care practice, which may promote the supine position for infant sleep. *J Pediatr*. 2002;141(3):398–403
 300. van Sleuwen BE, Engelberts AC, Boere-Boonekamp MM, Kuis W, Schulpen TW, L'Hoir MP. Swaddling: a systematic review. *Pediatrics*. 2007;120(4). Available at: www.pediatrics.org/cgi/content/full/120/4/e1097
 301. Richardson HL, Walker AM, Horne RS. Influence of swaddling experience on spontaneous arousal patterns and autonomic control in sleeping infants. *J Pediatr*. 2010; 157(1):85–91
 302. Richardson HL, Walker AM, Horne RS. Minimizing the risks of sudden infant death syndrome: to swaddle or not to swaddle? *J Pediatr*. 2009;155(4):475–481
 303. Narangerel G, Pollock J, Manaseki-Holland S, Henderson J. The effects of swaddling on oxygen saturation and respiratory rate of healthy infants in Mongolia. *Acta Paediatr*. 2007;96(2):261–265
 304. Kutlu A, Memik R, Mutlu M, Kutlu R, Arslan A. Congenital dislocation of the hip and its relation to swaddling used in Turkey. *J Pediatr Orthop*. 1992;12(5):598–602
 305. Chaarani MW, Al Mahmeid MS, Salman AM. Developmental dysplasia of the hip before and after increasing community awareness of the harmful effects of swaddling. *Qatar Med J*. 2002;11(1):40–43
 306. Yamamuro T, Ishida K. Recent advances in the prevention, early diagnosis, and treatment of congenital dislocation of the hip in Japan. *Clin Orthop Relat Res*. 1984;(184): 34–40
 307. Coleman SS. Congenital dysplasia of the hip in the Navajo infant. *Clin Orthop Relat Res*. 1968;56:179–193
 308. Tronick EZ, Thomas RB, Daltabuit M. The Quechua manta pouch: a caretaking practice for buffering the Peruvian infant against the multiple stressors of high altitude. *Child Dev*. 1994;65(4):1005–1013
 309. Manaseki S. Mongolia: a health system in transition. *BMJ*. 1993;307(6919):1609–1611
 310. Franco P, Seret N, Van Hees JN, Scaillet S, Groswasser J, Kahn A. Influence of swaddling on sleep and arousal characteristics of healthy infants. *Pediatrics*. 2005;115(5): 1307–1311
 311. Franco P, Scaillet S, Groswasser J, Kahn A. Increased cardiac autonomic responses to auditory challenges in swaddled infants. *Sleep*. 2004;27(8):1527–1532
 312. Hutcheson R. DTP vaccination and sudden infant deaths: Tennessee. *MMWR Morb Mortal Wkly Rep*. 1979;28:131–132
 313. Hutcheson R. Follow-up on DTP vaccination and sudden infant deaths: Tennessee. *MMWR*. 1979;28:134–135
 314. Bernier RH, Frank JA Jr, Dondero TJ Jr, Turner P. Diphtheria-tetanus toxoids-pertussis vaccination and sudden infant deaths in Tennessee. *J Pediatr*. 1982; 101(3):419–421
 315. Baraff LJ, Ablon WJ, Weiss RC. Possible temporal association between diphtheria-tetanus toxoid-pertussis vaccination and sudden infant death syndrome. *Pediatr Infect Dis*. 1983;2(1):7–11
 316. Griffin MR, Ray WA, Livengood JR, Schaffner W. Risk of sudden infant death syndrome after immunization with the diphtheria-tetanus-pertussis vaccine. *N Engl J Med*. 1988;319(10):618–623
 317. Hoffman JJ, Hunter J, Damus K, et al. Diphtheria-tetanus-pertussis immunization and sudden infant death: results of the National Institute of Child Health and Human Development Cooperative Epidemiological Study of Sudden Infant Death Syndrome Risk Factors. *Pediatrics*. 1987; 79(4):598–611
 318. Taylor EM, Emergy JL. Immunization and cot deaths. *Lancet*. 1982;2(8300):721
 319. Flahault A, Messiah A, Jouglu E, Bouvet E,

- Perin J, Hatton F. Sudden infant death syndrome and diphtheria/tetanus toxoid/pertussis/poliomyelitis immunisation. *Lancet*. 1988;1(8585):582–583
320. Walker AM, Jick H, Perera DR, Thompson RS, Knauss TA. Diphtheria-tetanus-pertussis immunization and sudden infant death syndrome. *Am J Public Health*. 1987;77(8):945–951
321. Jonville-Bera AP, Autret E, Laugier J. Sudden infant death syndrome and diphtheria-tetanus-pertussis-poliomyelitis vaccination status. *Fundam Clin Pharmacol*. 1995;9(3):263–270
322. Immunization Safety Review Committee. In: Stratton K, Almario DA, Wizemann TM, McCormick MC, eds. *Immunization Safety Review: Vaccinations and Sudden Unexpected Death in Infancy*. Washington, DC: National Academies Press; 2003
323. Mitchell EA, Stewart AW, Clements M, Ford RPK. Immunisation and the sudden infant death syndrome. New Zealand Cot Death Study Group. *Arch Dis Child*. 1995;73(6):498–501
324. Jonville-Béra AP, Autret-Leca E, Barbeillon F, Paris-Llado J; French Reference Centers for SIDS. Sudden unexpected death in infants under 3 months of age and vaccination status: a case-control study. *Br J Clin Pharmacol*. 2001;51(3):271–276
325. Fleming PJ, Blair PS, Platt MW, Tripp J, Smith IJ, Golding J. The UK accelerated immunisation programme and sudden unexpected death in infancy: case-control study. *BMJ*. 2001;322(7290):822
326. Fine PEM, Chen RT. Confounding in studies of adverse reactions to vaccines. *Am J Epidemiol*. 1992;136(2):121–135
327. Virtanen M, Peltola H, Paunio M, Heinonen OP. Day-to-day reactogenicity and the healthy vaccinee effect of measles-mumps-rubella vaccination. *Pediatrics*. 2000;106(5). Available at: www.pediatrics.org/cgi/content/full/106/5/e62
328. Vennemann MM, Höffgen M, Bajanowski T, Hense HW, Mitchell EA. Do immunisations reduce the risk for SIDS? A meta-analysis. *Vaccine*. 2007;25(26):4875–4879
329. Steinschneider A. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics*. 1972;50(4):646–654
330. Hodgman JE, Hoppenbrouwers T. Home monitoring for the sudden infant death syndrome: the case against. *Ann NY Acad Sci*. 1988;533:164–175
331. Ward SL, Keens TG, Chan LS, et al. Sudden infant death syndrome in infants evaluated by apnea programs in California. *Pediatrics*. 1986;77(4):451–458
332. Monod N, Plouin P, Sternberg B, et al. Are polygraphic and cardiopneumographic respiratory patterns useful tools for predicting the risk for sudden infant death syndrome? A 10-year study. *Biol Neonate*. 1986;50(3):147–153
333. Ramanathan R, Corwin MJ, Hunt CE, et al. Cardiorespiratory events recorded on home monitors: comparison of healthy infants with those at increased risk for SIDS. *JAMA*. 2001;285(17):2199–2207
334. American Academy of Pediatrics, Committee on Fetus and Newborn. Apnea, sudden infant death syndrome, and home monitoring. *Pediatrics*. 2003;111(4 pt 1):914–917
335. Patriarca M, Lyon TD, Delves HT, Howatson AG, Fell GS. Determination of low concentrations of potentially toxic elements in human liver from newborns and infants. *Analyst*. 1999;124(9):1337–1343
336. Kleemann WJ, Weller JP, Wolf M, Troger HD, Bluthgen A, Heeschen W. Heavy metals, chlorinated pesticides and polychlorinated biphenyls in sudden infant death syndrome (SIDS). *Int J Legal Med*. 1991;104(2):71–75
337. Erickson MM, Poklis A, Gantner GE, Dickinson AW, Hillman LS. Tissue mineral levels in victims of sudden infant death syndrome I. Toxic metals: lead and cadmium. *Pediatr Res*. 1983;17(10):779–784
338. George M, Wiklund L, Aastrup M, et al. Incidence and geographical distribution of sudden infant death syndrome in relation to content of nitrate in drinking water and groundwater levels. *Eur J Clin Invest*. 2001;31(12):1083–1094
339. Richardson BA. Sudden infant death syndrome: a possible primary cause. *J Forensic Sci Soc*. 1994;34(3):199–204
340. Sprott TJ. Cot death: cause and prevention—experiences in New Zealand 1995–2004. *J Nutr Environ Med*. 2004;14(3):221–232
341. Department of Health. *Expert Group to Investigate Cot Death Theories*. London, United Kingdom: Her Majesty's Stationary Office; 1998. Available at: http://sids-network.org/experts/expert_group_to_investigate_cot_.htm. Accessed September 6, 2011
342. Blair P, Fleming P, Bensley D, Smith I, Bacon C, Taylor E. Plastic mattresses and sudden infant death syndrome. *Lancet*. 1995;345(8951):720
343. Rubens DD, Vohr BR, Tucker R, O'Neil C A, Chung W. Newborn oto-acoustic emission hearing screening tests: preliminary evidence for a marker of susceptibility to SIDS. *Early Hum Dev*. 2008;84(4):225–229
344. Hamill T, Lim G. Otoacoustic emissions does not currently have ability to detect SIDS. *Early Hum Dev*. 2008;84(6):373
345. Krous HF, Byard RW. Newborn hearing screens and SIDS. *Early Hum Dev*. 2008;84(6):371
346. Farquhar LJ, Jennings P. Newborn hearing screen results for infants that died of SIDS in Michigan 2004–2006. *Early Hum Dev*. 2008;84(10):699
347. Hauck FR, Tanabe KO. SIDS. *Clin Evid (Online)*. 2009; pii: 0315
348. Moon RY, Oden RP, Grady KC. Back to Sleep: educational intervention with Women, Infants, and Children program clients. *Pediatrics*. 2004;113(3 pt 1):542–547
349. Colson ER, Joslin SC. Changing nursery practice gets inner-city infants in the supine position for sleep. *Arch Pediatr Adolesc Med*. 2002;156(7):717–720
350. Moon RY, Calabrese T, Aird L. Reducing the risk of sudden infant death syndrome in child care and changing provider practices: lessons learned from a demonstration project. *Pediatrics*. 2008;122(4):788–798
351. Moon RY, Oden RP. Back to sleep: can we influence child care providers? *Pediatrics*. 2003;112(4):878–882
352. Lerner H, McClain M, Vance JC. SIDS education in nursing and medical schools in the United States. *J Nurs Educ*. 2002;41(8):353–356
353. Price SK, Gardner P, Hillman L, Schenk K, Warren C. Changing hospital newborn nursery practice: results from a statewide “Back to Sleep” nurses training program. *Matern Child Health J*. 2008;12(3):363–371
354. Joyner BL, Gill-Bailey C, Moon RY. Infant sleep environments depicted in magazines targeted to women of childbearing age. *Pediatrics*. 2009;124(3). Available at: www.pediatrics.org/cgi/content/full/124/3/e416
355. Yanovitzky I, Blitz CL. Effect of media coverage and physician advice on utilization of breast cancer screening by women 40 years and older. *J Health Commun*. 2000;5(2):117–134
356. Marketing Evolution. *Measuring Media Effectiveness: Comparing Media Contribution Throughout the Purchase Funnel*. New York, NY: Magazine Publishers of America; 2006. Available at: www.magazine.org/content/Files/MEFullStudy2006.pdf. Accessed September 6, 2011