

SIDS Risk: It's More Than Just the Sleep Environment

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It is widely acknowledged that dramatic declines in the rate of sudden infant death syndrome (SIDS) in the United States resulted from recommendations that infants not be placed prone to sleep, first by the American Academy of Pediatrics in 1992,¹ followed by the national Back to Sleep campaign 2 years later.² Other recommendations, such as room-sharing without bed-sharing and avoidance of soft bedding, were introduced and reinforced over the next 2 decades and, with supine positioning, have been the mainstay of SIDS risk reduction guidelines.^{3,4} However, there has been much concern in recent years because the rate of SIDS (International Classification of Diseases, 10th Revision [ICD-10] R95) has plateaued and rates of other sleep-related infant deaths, such as accidental suffocation and strangulation in bed (ICD-10 W75) and ill-defined deaths (ICD-10 R99), have increased.² Indeed, the rate of postneonatal mortality, which encompasses all of these diagnoses, has not declined since the late 1990s.² The risk factors for these different categories of death are strikingly similar,⁵ and researchers have documented a diagnostic shift, ie, deaths that were previously classified as SIDS 2 to 3 decades ago are now being classified as suffocation, strangulation, asphyxia, and ill-defined deaths.⁶⁻⁹ The analysis of infant deaths from 1983 to 2012 by Goldstein et al,¹⁰ published in this month's issue of *Pediatrics*, reinforces that there has indeed been a diagnostic shift.

However, this diagnostic shift has led many to believe that SIDS is not a true

entity and that all of these sudden unexpected deaths are accidental deaths that can be entirely prevented. Thus, public health efforts have refocused on reducing environmental risks, such as prone and side sleep positioning, bed-sharing, and soft bedding use,^{3,4} and clearly those efforts will continue to be important. However, Goldstein et al remind us also that some proportion of these infants who die of sleep-related deaths have an intrinsic risk. Furthermore, they make the case that some of the reduction in SIDS rates can be attributable to reductions in intrinsic risk from improvements in care, such as the use of antenatal steroids that reduce respiratory distress of the newborn. Interestingly, despite the fact that infants born prematurely and with low birth weight are at higher risk of SIDS, increased survival of low birth weight infants past the neonatal period has not been accompanied by an increase in SIDS rates. In addition, reductions in intrinsic risk from general improvements in care may help to explain why SIDS rates began to decline even before the Back to Sleep campaign was launched.

SIDS is generally believed to occur when an intrinsically vulnerable infant during a critical period of development (with those <6 months of age at greatest risk) is exposed to an exogenous stressor, such as prone position or airway obstruction.¹¹ Thus, death may occur when a vulnerable infant is in a potentially asphyxiating sleep environment and does not arouse or otherwise respond appropriately when in such an environment. Kinney et al¹² and Paterson et al¹³ have

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Opinions expressed in these commentaries are those of the author and not necessarily those of the American Academy of Pediatrics or its Committees.

DOI: 10.1542/peds.2015-3665

Accepted for publication Oct 9, 2015

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

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

COMPANION PAPER: A companion to this article can be found online at www.pediatrics.org/cgi/doi/10.1542/peds.2015-2298.

To cite: Moon RY and Hauck FR. SIDS Risk: It's More Than Just the Sleep Environment. *Pediatrics*. 2016;137(1):e20153665

found pathologic evidence in SIDS victims of brainstem abnormalities in neurotransmitters that are responsible for arousal and other autonomic responses. This finding likely explains why some infants die when placed in an unsafe sleep environment, whereas most infants, when placed in that same environment, do not, and why some infants (who presumably have severe intrinsic vulnerabilities) die even when in a safe sleep environment.

Although we cannot yet identify which infants have an intrinsic risk, there are factors that influence this intrinsic risk. Infants who have been exposed in utero or postnatally to tobacco smoke have alterations in their arousal and autonomic activity,^{14–16} resulting in blunted arousal responses.^{17–24} Smoke exposure confers a much higher risk of SIDS in a dose-dependent manner.^{25–29} It has been estimated that one-third of SIDS deaths could be prevented if maternal smoking during pregnancy is eliminated.^{30,31} Prenatal exposure to drugs and alcohol may also increase intrinsic risk.^{32–36} Infants who are breastfed have decreased arousal thresholds^{37,38} (ie, they awake more easily and often), which may be one of the reasons that it provides strong protection against SIDS (adjusted odds ratio for ever breastfeeding: 0.55–0.64).^{39,40} Another may be improved immune function and decreased incidence of respiratory or gastrointestinal infections, both of which are associated with the occurrence of SIDS. Regular prenatal care, which also has been shown to decrease SIDS risk,^{41–44} may do so by decreasing both intrinsic and extrinsic risk.

We are very much in agreement that reductions in SIDS rates have resulted from some combination of reductions in extrinsic risk factors, changes in classification of cause of death for sudden unexpected infant deaths, changes in intrinsic risk

factors, and changes in factors that affect both intrinsic and extrinsic risk. If we are to further impact infant mortality rates and eliminate SIDS, focus on the sleep environment will continue to be important but will likely be insufficient. Public health efforts will need to also focus on decreasing intrinsic risk through the promotion of smoking cessation, elimination of in utero drug and alcohol exposure, and increasing rates of breastfeeding and access to high-quality prenatal care.

ABBREVIATIONS

ICD-10: International Classification of Diseases, 10th Revision
SIDS: sudden infant death syndrome

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Pediatrics 2016;137;

DOI: 10.1542/peds.2015-3665 originally published online December 2, 2015;

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