

AMERICAN ACADEMY OF PEDIATRICS

POLICY STATEMENT

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of All Children

Committee on Fetus and Newborn

Apnea, Sudden Infant Death Syndrome, and Home Monitoring

ABSTRACT. More than 25 years have elapsed since continuous cardiorespiratory monitoring at home was suggested to decrease the risk of sudden infant death syndrome (SIDS). In the ensuing interval, multiple studies have been unable to establish the alleged efficacy of its use. In this statement, the most recent research information concerning extreme limits for a prolonged course of apnea of prematurity is reviewed. Recommendations regarding the appropriate use of home cardiorespiratory monitoring after hospital discharge emphasize limiting use to specific clinical indications for a predetermined period, using only monitors equipped with an event recorder, and counseling parents that monitor use does not prevent sudden, unexpected death in all circumstances. The continued implementation of proven SIDS prevention measures is encouraged.

ABBREVIATIONS. SIDS, sudden infant death syndrome; ALTE, apparent life-threatening event.

INTRODUCTION

Significant new information has been forthcoming in recent decades on sudden infant death syndrome (SIDS) and apnea during early infancy.¹⁻⁸ This statement focuses on the epidemiologic aspects of SIDS, the lack of a proven association between episodic apnea and SIDS, strategies for prevention of SIDS, and appropriate use of home cardiorespiratory monitoring.

Apnea monitors were first introduced in the mid-1960s for the management of apnea of prematurity in hospital settings.⁹ Subsequently, cardiorespiratory monitoring has become widely used in the care of infants with a variety of acute and chronic disorders.

The hypothesis that apnea is the pathophysiologic precursor to SIDS was first proposed in 1972.¹⁰ Apnea documented by cardiorespiratory monitoring during prolonged hospitalizations was reported for 2 infants, both of whom were siblings of 3 infants who had died suddenly at home. Both siblings subsequently died unexpectedly after discharge from the hospital. More than 2 decades later, evidence of infanticide for all 5 infants in the original report became known. The apnea theory never has been proven despite extensive independent research in the several decades after that report.¹⁻⁵ Nevertheless,

the home cardiorespiratory monitoring industry, fueled by increasing demand from parents concerned about the risk of SIDS, rapidly developed products aimed at preventing SIDS.¹¹ Despite the absence of a scientific foundation or evidence of efficacy,^{12,13} home cardiorespiratory monitoring continues to be a common practice in this country.

The American Academy of Pediatrics Committee on Infant and Preschool Child in 1975 recommended that home monitoring to prevent SIDS should be limited to ongoing research studies.¹⁴ Subsequently, in the early 1980s a Task Force on Prolonged Infantile Apnea was formed to evaluate the evidence for the theory that apnea is a precursor to SIDS. It concluded in a 1985 statement that "a causal relationship between prolonged apnea and SIDS has not been established."¹⁵ The recommendations left the use of home cardiorespiratory monitoring in individual situations to physician judgment.

The costs of home monitoring are substantial. In 1999, 44% of 26 000 infants weighing 501 to 1500 g at birth and cared for in 325 neonatal units within the Vermont Oxford Network were discharged from the hospital on monitors.¹⁶ A conservative estimate of the annual cost of monitoring preterm infants weighing less than 1500 g in the United States is \$24 million, and this projection does not include physician fees, repeat pneumograms or sleep studies, other ancillary medical costs, or the costs of other populations of infants who are monitored. In this context, the question of efficacy of home monitoring becomes even more important.

DEFINITIONS

SIDS has been defined as "the sudden death of an infant under 1 year of age that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history."¹⁷

Apnea of infancy is defined as "an unexplained episode of cessation of breathing for 20 seconds or longer, or a shorter respiratory pause associated with bradycardia, cyanosis, pallor, and/or marked hypotonia." The term "apnea of infancy" generally refers to infants with gestational age of 37 weeks or more at the onset of apnea.¹⁸

Apnea of prematurity is defined as sudden cessation of breathing that lasts for at least 20 seconds or

is accompanied by bradycardia or oxygen desaturation (cyanosis) in an infant younger than 37 weeks' gestational age.⁹ It usually ceases by 37 weeks' postmenstrual age but may persist for several weeks beyond term, especially in infants born before 28 weeks' gestation.⁶⁻⁸ The most recent data indicate that extreme episodes usually cease at approximately 43 weeks' postconceptional age.^{*19}

An apparent life-threatening event (ALTE) is defined as "an episode that is frightening to the observer and is characterized by some combination of apnea (central or occasionally obstructive), color change (usually cyanotic or pallid but occasionally erythematous or plethoric), marked change in muscle tone (usually marked limpness), choking, or gagging."¹⁸

PREDICTION OF SIDS RISK

Peer-reviewed evidence indicates that apnea is not predictive of or a precursor to SIDS. To the contrary, the evidence indicates that there is no clear, unequivocal relationship between apnea and SIDS. The National Institute of Child Health and Human Development SIDS Cooperative Epidemiologic Study was a case-control study of 757 definite or probable cases of SIDS and 1514 control infants.²⁰ The investigators found no association between newborn apnea (apnea of prematurity) and SIDS and stated that the relationship with postneonatal apnea (apnea with onset after hospital discharge as defined in the study) was arguable.

The Collaborative Home Infant Monitoring Evaluation included 718 000 hours of documented monitoring of 1079 infants (infants with idiopathic ALTEs, siblings of infants who died of SIDS, symptomatic [having clinically apparent apnea/bradycardia episodes] and asymptomatic preterm infants weighing less than 1750 g at birth, and healthy term infants).¹⁹ Findings indicated that apnea and bradycardia at conventional alarm thresholds as well as extreme apnea and bradycardia occurred in all groups of infants. (The study protocol defined extreme apnea as episodes lasting longer than 30 seconds for all age infants and extreme bradycardia as heart rate of less than 60 beats per minute for infants of less than 44 weeks postmenstrual age and less than 50 beats per minute for those more than 44 weeks and lasting longer than 10 seconds.) Events exceeding the extreme threshold occurred in 10% of all infants and in 2.3% of healthy term infants. The only groups with an increased risk of such events compared with healthy term infants were the preterm infant groups, up to approximately 43 weeks' postmenstrual age. Infants being monitored for ALTEs had an increased risk of repeated extreme episodes, but the difference was statistically significant only for the preterm ALTE group. The risk of a recurrent extreme episode increased with each subsequent recurrence for all infants who had a single extreme episode. Apnea

and bradycardia occurred as independent events. This study documents that many infants experience apnea and bradycardia exceeding current conventional alarm thresholds and do not die. The mean postmenstrual age for SIDS occurrence is estimated to be 45.8 weeks for infants born at 24 to 28 weeks' gestation, compared with 52.3 weeks for term infants.²¹ Furthermore, apnea appears to resolve at a postnatal age before which most SIDS deaths occur. Events exceeding the extreme threshold diminished in occurrence at approximately 43 weeks' postmenstrual age. These data provide further evidence that apnea is not an immediate precursor to SIDS. The respiratory monitors used in the Collaborative Home Infant Monitoring Evaluation may have detected more episodes of apnea than current home monitors that do not detect obstructive apnea reliably. These results indicate that cardiorespiratory monitoring for apnea and bradycardia is not an effective tool to identify infants at great risk of SIDS. Pneumography, performed in a laboratory under controlled conditions, may identify infants who are having a prolonged course of apnea. However, such studies never have been shown to be predictive of SIDS.^{2,3,12}

The risk of sudden death in siblings of infants who died of SIDS is unclear. Efficacy of home cardiorespiratory monitoring to prevent SIDS in this group of infants is equally unproven. The rarity of a SIDS death and the more extreme rarity of a subsequent SIDS death of a sibling make it difficult to complete a definitive clinical trial to establish efficacy. Many studies that reported an increased risk for siblings were performed before the current understanding of the epidemiology evolved. The roles of infant sleep position and sleeping environment, smoking in the household,²²⁻²⁶ and death scene investigation to exclude infanticide are now recognized as significant factors in understanding the causation of SIDS.^{17,27} There is a body of evidence, although inconclusive, that suggests a genetic susceptibility to SIDS may exist,²⁸ although the risk of recurrence in siblings, if present, is most likely exceedingly low.

EFFICACY OF HOME CARDIORESPIRATORY MONITORING

Epidemiologic studies have failed to document any impact of home cardiorespiratory monitoring for apnea and/or bradycardia on the incidence of SIDS.^{29,30} There is no evidence that the presence of apnea and/or bradycardia identifies a group at increased risk of SIDS, that home cardiorespiratory monitoring can provide warning in time for intervention to prevent sudden death, or that intervention would be successful in preventing unexpected death. Given the lack of evidence that home cardiorespiratory monitoring has any impact on SIDS, prevention of SIDS is not an acceptable indication for home cardiorespiratory monitoring.

Evidence exists that preterm infants are at a greater risk of extreme apnea episodes than are term infants. This risk decreases with time, ceasing at approximately 43 weeks' postmenstrual age.¹⁹ There are no studies correlating long-term neurodevelopmental outcome with such episodes. Home cardiorespiratory

*As defined in various studies, gestational age at birth plus age in weeks from birth; more accurately designated as "postmenstrual age." References cited may have used different terms, but for the purposes of this statement, postmenstrual age is used hereafter.

spiratory monitoring after hospital discharge may be prescribed for some preterm infants with an unusually prolonged course of recurrent, extreme apnea (as defined previously). The physician, together with the parents, should consider the potential advantages and disadvantages of home cardiorespiratory monitoring.^{31–34} Current evidence suggests that if such monitoring is elected, it usually may be discontinued after 43 weeks' postmenstrual age, although extreme apnea may persist beyond that time in some infants. Using a monitor with event recording can be helpful in determining the appropriate time for discontinuance.

There are other groups of infants for whom use of a home cardiorespiratory monitor may be warranted, not because of an increased risk of SIDS, but because of other factors that increase the risk of sudden death. For example, home cardiorespiratory monitoring may be justified to allow rapid recognition of apnea, airway obstruction, respiratory failure, interruption of supplemental oxygen supply, or failure of mechanical respiratory support. Infants for whom these indications may apply include: 1) infants who have experienced an ALTE; 2) infants with tracheostomies or anatomic abnormalities that make them vulnerable to airway compromise; 3) infants with neurologic or metabolic disorders affecting respiratory control; and 4) infants with chronic lung disease (bronchopulmonary dysplasia), especially those requiring supplemental oxygen, continuous positive airway pressure, or mechanical ventilation. In these instances, home cardiorespiratory monitoring may allow the caregiver to respond more quickly and perhaps to decrease the duration of accompanying hypoxemia. However, such monitoring will not always prevent sudden death attributable to the triggering event or underlying condition. The parents of such infants should be counseled regarding the purpose of the home cardiorespiratory monitoring and realistic expectations of what it can and cannot contribute to an infant's well-being.

When cardiorespiratory monitoring in the hospital or home is prescribed, the physician should also establish a specific plan for periodic review and termination. Should monitoring beyond 43 weeks' postmenstrual age be recommended, clear documentation of the reasons for continuing monitoring is necessary.

If monitoring is to be used at home, parents and other caregivers must be trained in observation techniques, operation of the monitor, and infant cardiopulmonary resuscitation. Medical and technical support staff should always be available for direct or telephone consultation. Psychosocial assistance and respite personnel should also be available. A care plan including periodic reassessment of historical, physical, developmental, and laboratory data are essential. Long-term follow-up of neurodevelopmental status is advised.³⁵

Many monitors are available, and it is the physician's responsibility to prescribe equipment with specific capabilities. Cardiac and respiratory activity should be monitored simultaneously. Monitors capa-

ble of event recording for downloading and retrospective review for analysis of true versus false alarms should be used. None of the current monitors available for home use will detect obstructive apnea reliably.

RECOMMENDATIONS

1. Home cardiorespiratory monitoring should not be prescribed to prevent SIDS.
2. Home cardiorespiratory monitoring may be warranted for premature infants who are at high risk of recurrent episodes of apnea, bradycardia, and hypoxemia after hospital discharge. The use of home cardiorespiratory monitoring in this population should be limited to approximately 43 weeks' postmenstrual age or after the cessation of extreme episodes, whichever comes last.
3. Home cardiorespiratory monitoring may be warranted for infants who are technology dependent (tracheostomy, continuous positive airway pressure), have unstable airways, have rare medical conditions affecting regulation of breathing, or have symptomatic chronic lung disease.
4. If home cardiorespiratory monitoring is prescribed, the monitor should be equipped with an event recorder.
5. Parents should be advised that home cardiorespiratory monitoring has not been proven to prevent sudden unexpected deaths in infants.
6. Pediatricians should continue to promote proven practices that decrease the risk of SIDS—supine sleep position, safe sleeping environments, and elimination of prenatal and postnatal exposure to tobacco smoke.

COMMITTEE ON FETUS AND NEWBORN, 2002–2003

Lillian R. Blackmon, MD, Chairperson
 Daniel G. Batton, MD
 Edward F. Bell, MD
 William A. Engle, MD
 William P. Kanto, Jr, MD
 Gilbert I. Martin, MD
 Warren N. Rosenfeld, MD
 Ann R. Stark, MD

*James A. Lemons, MD, Past Committee Chairperson

LIAISONS

Keith J. Barrington, MD
 Canadian Paediatric Society
 Jenny Ecord, MS, RNC, NNP, PNP
 American Nurses Association
 Association of Women's Health, Obstetric, and Neonatal Nurses
 National Association of Neonatal Nurses
 Laura E. Riley, MD
 American College of Obstetricians and Gynecologists
 Kay M. Tomashek, MD
 Centers for Disease Control and Prevention
 Linda L. Wright, MD
 National Institutes of Health

STAFF

Jim Couto, MA

*Lead author

REFERENCES

1. Franks CI, Watson JB, Brown BH, Foster EF. Respiratory patterns and risk of sudden unexpected death in infancy. *Arch Dis Child*. 1980;55:595–599
2. Southall DP, Richards JM, Rhoden KJ, et al. Prolonged apnea and cardiac arrhythmias in infants discharged from neonatal intensive care units: failure to predict an increased risk for sudden infant death syndrome. *Pediatrics*. 1982;70:844–851
3. Rosen CL, Frost JD Jr, Harrison GM. Infant apnea: polygraphic studies and follow-up monitoring. *Pediatrics*. 1983;71:731–736
4. Southall DP, Richards JM, Stebbens V, et al. Cardiorespiratory function in 16 full-term infants with sudden infant death syndrome. *Pediatrics*. 1986;78:787–796
5. Schwartz PJ, Southall DP, Valdes-Dapena M. The sudden infant death syndrome: cardiac and respiratory mechanisms and interventions. Proceedings. May 24–27, 1987, Como, Italy. *Ann N Y Acad Sci*. 1988;533:1–474
6. Henderson-Smart DJ. The effect of gestational age on the incidence and duration of recurrent apnoea in newborn babies. *Aust Paediatr J*. 1981;17:273–276
7. Eichenwald EC, Aina A, Stark AR. Apnea frequently persists beyond term gestation in infants delivered at 24 to 28 weeks. *Pediatrics*. 1997;100:354–359
8. Darnall RA, Kattwinkel J, Nattie C, Robinson M. Margin of safety for discharge after apnea in preterm infants. *Pediatrics*. 1997;100:795–801
9. Daily WJ, Klaus M, Meyer HB. Apnea in premature infants: monitoring, incidence, heart rate changes, and an effect of environmental temperature. *Pediatrics*. 1969;43:510–518
10. Steinschneider A. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics*. 1972;50:646–654
11. Bergman AB, Beckwith JB, Ray CG. The apnea monitor business. *Pediatrics*. 1975;56:1–3
12. 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;50:147–153
13. Hodgman JE, Hoppenbrouwers T. Home monitoring for the sudden infant death syndrome: the case against. *Ann N Y Acad Sci*. 1988;533:164–175
14. American Academy of Pediatrics, Committee on Infant and Preschool Child. Home monitoring for sudden infant death. *Pediatrics*. 1975;55:144–145
15. American Academy of Pediatrics, Task Force on Prolonged Infantile Apnea. Prolonged infantile apnea: 1985. *Pediatrics*. 1985;76:129–131
16. Vermont Oxford Network. 1999 database summary. Available at: <http://www.vtoxford.org>. Accessed May 10, 2002
17. 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;11:677–684
18. National Institutes of Health, Consensus Development Conference on Infantile Apnea and Home Monitoring, Sept 29 to Oct 1, 1986. *Pediatrics*. 1987;79:292–299
19. 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 N Y Acad Sci*. 1988;533:13–30
20. 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;285:2199–2207
21. Malloy MH, Hoffman HJ. Prematurity, sudden infant death syndrome, and age of death. *Pediatrics*. 1995;96:464–471
22. 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;105:650–656
23. Kemp JS, Unger B, Wilkins D, et al. Unsafe sleep practices and an analysis of bedsharing 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
24. Thogmartin JR, Siebert CF Jr, Pellan WA. Sleep position and bedsharing in sudden infant deaths: an examination of autopsy findings. *J Pediatr*. 2001;138:212–217
25. Skadberg BT, Morild I, Markestad T. Abandoning prone sleeping: effect on the risk of sudden infant death syndrome. *J Pediatr*. 1998;132:340–343
26. Carleton JN, Donoghue AM, Porter WK. Mechanical model testing of rebreathing potential in infant bedding materials. *Arch Dis Child*. 1998;78:323–328
27. American Academy of Pediatrics Committee on Child Abuse and Neglect. Distinguishing sudden infant death syndrome from child abuse fatalities. *Pediatrics*. 2001;107:437–441
28. Hunt CE. Sudden infant death syndrome and other causes of infant mortality: diagnosis, mechanisms, and risk for recurrence in siblings. *Am J Respir Crit Care Med*. 2001;164:346–357
29. MacKay M, Abreu e Silva FA, MacFadyen UM, Williams A, Simpson H. Home monitoring for central apnoea. *Arch Dis Child*. 1984;59:136–142
30. Ward SL, Keens TG, Chan LS, et al. Sudden infant death syndrome in infants evaluated by apnea programs in California. *Pediatrics*. 1986;77:451–458
31. Leonard BJ, Scott SA, Sootsman J. A home-monitoring program for parents of premature infants: a comparative study of the psychological effects. *J Dev Behav Pediatr*. 1989;10:92–97
32. Phipps S, Drotar D, Joseph C, Geiss C, Doershuk C. Psychological impact of home apnea monitoring: temporal effects, family resources, and maternal coping style. *J Dev Behav Pediatr*. 1989;10:7–12
33. Cote A, Hum C, Brouillette RT, Themens M. Frequency and timing of recurrent events in infants using home cardiorespiratory monitors. *J Pediatr*. 1998;132:783–789
34. Abendroth D, Moser DK, Dracup K, Doering LV. Do apnea monitors decrease emotional distress in parents of infants at high risk for cardiopulmonary arrest? *J Pediatr Health Care*. 1999;13:50–57
35. American Academy of Pediatrics, Committee on Fetus and Newborn. Hospital discharge of the high-risk neonate—proposed guidelines. *Pediatrics*. 1998;102:411–417

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

Apnea, Sudden Infant Death Syndrome, and Home Monitoring

Committee on Fetus and Newborn

Pediatrics 2003;111;914

DOI: 10.1542/peds.111.4.914

Updated Information & Services

including high resolution figures, can be found at:
<http://pediatrics.aappublications.org/content/111/4/914>

References

This article cites 33 articles, 19 of which you can access for free at:
<http://pediatrics.aappublications.org/content/111/4/914#BIBL>

Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):
Committee on Fetus & Newborn
http://www.aappublications.org/cgi/collection/committee_on_fetus_newborn
Fetus/Newborn Infant
http://www.aappublications.org/cgi/collection/fetus:newborn_infant_sub

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
<http://www.aappublications.org/site/misc/Permissions.xhtml>

Reprints

Information about ordering reprints can be found online:
<http://www.aappublications.org/site/misc/reprints.xhtml>

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Apnea, Sudden Infant Death Syndrome, and Home Monitoring

Committee on Fetus and Newborn

Pediatrics 2003;111;914

DOI: 10.1542/peds.111.4.914

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/111/4/914>

Pediatrics is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. Pediatrics is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2003 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 1073-0397.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

