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PN23 - Sudden infant death: physiology
(348)

991104-012
Apnea of prematurity and risk for sudden infant death syndrome. Freed GE, Meny RG (1999), Pediatrics vol 103, no 2, August 1999, pp 297-298
Commentary on the possible role of apnoea of prematurity as a risk factor for sudden infant death. The effectiveness of home monitoring of at risk infants is discussed. (8 references) (KL)

990911-008
Not time to put cot death to bed. Limerick S (1999), BMJ vol 319, no 7211, 11 September 1999, pp 698-700
Second part of a two part debate, the author argues the case for keeping the term 'sudden infant death' for unexplained deaths, despite recent speculation that infant homicides may play a greater part in such figures than previously thought. (24 references) (JAL)

990911-007
Time to put 'cot death' to bed?. Green MA (1999), BMJ vol 319, no 7211, 11 September 1999, pp 697-698
First part of a two part debate, the author argues that the terms 'cot death' or 'sudden infant death' can be too readily applied in cases where pathologists are unable to identify an actual cause. The author believes that many such deaths should be investigated with a greater level of suspicion, and that the cause of death should be recorded by the coroner as 'not ascertained' in far more instances than at present. (14 References) (JAL)

990902-001
Caffeine and alcohol as risk factors for sudden infant death syndrome. Alm B, Wennergren G, Norvenius G, et al (1999), Archives of Disease in Childhood vol 81, no 2, August 1999, pp 107-111
Objective: To assess whether alcohol and caffeine are independent risk factors for sudden infant death syndrome (SIDS). Materials and methods: Analyses based on data from the Nordic epidemiological SIDS study, a case control study in which all parents of SIDS victims in the Nordic countries from 1 September 1992 to 31 August 1995 were invited to participate with parents of four controls, matched for sex and age at death. Odds ratios (ORs) were calculated by conditional logistic regression analysis. Results: The crude ORs for caffeine consumption > 800mg/24 hours both during and after pregnancy were significantly raised: 3.9 (95% confidence interval (CI), 1.9 to 8.1) and 3.1 (95% CI, 1.5 to 6.3), respectively. However, after adjustment for maternal smoking in 1st trimester, maternal age, education and parity, no significant effect of caffeine during or after pregnancy remained. For maternal and paternal alcohol use, no significant risk increase was found after adjusting for social variables, except for heavy postnatal intake of alcohol by the mother, where the risk was significantly increased. Conclusions: Caffeine during or after pregnancy was not found to be an independent risk factor for SIDS after adjustment for maternal age, education, parity, and smoking during pregnancy. Heavy postnatal but not prenatal intake of alcohol by the mother increased the risk. (32 references) (Author)

990712-011
Sudden infant death syndrome among twins. Malloy MH, Freeman DH (1999), Archives of Pediatrics and Adolescent Medicine

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vol 153, no 7, July 1999, pp 736-740

Background: Sudden infant death syndrome (SIDS) is a major contributor to infant mortality. Previous studies have suggested that infants born of twin pregnancies are at greater risk for SIDS and that a twin who survives after a co-twin dies is at increased risk for SIDS. Objective: To attempt to confirm the increased risk of SIDS among and within twin pairs through the use of US vital statistics data. Methods: We analyzed data from the US-linked birth and infant death certificate tapes for the years 1987 through 1991 to determine the risk of SIDS in twin births compared with singleton births and to describe the characteristics of twin pairs in who SIDS occurred. The analysis was limited to live births with weights of 500 g or more and gestational ages of 24 weeks or more. We used an algorithm to match co-twins (infants within a twin pair) to measure sex and birth weight concordancy; to identify twin pairs, in which one or both twins died of SIDS; and to examine, when both twins died, whether they died on the same day. Results: There were 23 464 singleton SIDS deaths and 1056 twin SIDS deaths during the 5-year period. The crude relative risk for SIDS among twins compared with singleton births was 2.06 (95% confidence interval, 1.94-2.19). The adjusted relative risk independent of birth weight and sociodemographic variables was 1.13 (95% confidence interval, 0.97-1.31). We successfully matched the co-twins of 172 029 twin pregnancies. Of these, 767 were twin pregnancies in which one or both twins died of SIDS. Among the 767 twin pregnancies in which one or both twins experienced SIDS, there were only 7 in which both twins died of SIDS rate ratio, 4.0 per 100 000 twin pregnancies). In only 1 of these 7 did both twins die on the same day (rate ratio, 0.58 per 100 000 twin pregnancies). The relative risk for a second twin dying of SIDS was 8.17 (90% confidence interval, 1.18-56.67). Conclusions: Independent of birth weight, twins do not appear to be at greater risk for SIDS compared with singleton births. In addition, the occurrence of both twins dying of SIDS in uncommon, and the occurrence of both twins dying on the same day is extremely uncommon. (29 references) (Author)

990702-029

Interactions of infectious symptoms and modifiable risk factors in sudden infant death syndrome. The Nordic Epidemiological SIDS study. Helweg-Larsen K, Lundemose JB, Oyen N, and others (1999), Acta Paediatrica vol 88, no 5, May 1999, pp 521-527

The aim of the study was to investigate the effect of infection on sudden infant death syndrome (SIDS) and to analyse whether modifiable risk factors of SIDS, prone sleeping, covered head and smoking act as effect modifiers. In a consecutive multicentre case-control study of SIDS in Denmark, Norway and Sweden, questionnaires on potential risk factors for SIDS were completed by parents of SIDS victims, and for at least two controls matched for gender, age and place of birth. All SIDS cases were verified by an autopsy. The study comprised 244 SIDS cases and 869 controls, analysed by conditional logistic regression. Significantly more cases than controls presenting symptoms of infectious diseases during the last week and/or last day were treated with antibiotics and had been seen by a physician. The finding is consistent with the hypothesis of an infectious mechanism in SIDS induced by local microorganism growth and toxin or cytokine production, and also adds further support to a possible association between infection and SIDS by loss of protective mechanisms, such as arousal. The risk of SIDS among infants with the combined presence of infectious symptoms and either of the other modifiable risk factors, prone sleeping, head covered or parental smoking, was far greater than the sum of each individual factor. These risk factors thus modify the dangerousness of infection in infancy. (32 references) (Author)

990601-015*

Exploring infant health: a review commissioned by the Foundation for the Study of Infant Deaths. Conroy S, Smith M (1999), London: Foundation for the Study of Infant Deaths 1999. 178p

Comprehensive review of causes and risks of sudden infant death in the United Kingdom. The first section explores parenting in relation to socially disadvantaged variables, including infant feeding, infant sleeping, use of infant health services, parenting knowledge, beliefs and sources, maternal health, and interventions in parenting. Part 2 covers the impact of social and environmental factors in parental smoking including: social patterns of smoking; concurrent lifestyle and environment; and factors relating to onset, maintenance, cessation and relapse in smoking. Part 3 covers general issues, research design and methods of data collection. (KL)

990505-050

Maternal placental abnormality and the risk of sudden infant death syndrome. Li DK, Wi S (1999), American Journal of Epidemiology vol 149, no 7, 1 April 1999, pp 608-611

To determine whether placental abnormality (placental abruption or placental previa) during pregnancy predisposes an infant to a high risk of sudden infant death syndrome (SIDS), the authors conducted a population-based case-control study using 1989-1991 California linked birth and death certificate data. They identified 2,107 SIDS cases, 96% of whom were diagnosed through autopsy. Ten controls were randomly selected for each case from the same linked birth-death certificate data, matched to the case on year of birth. About 1.4% of mothers of cases and 0.7% of mothers of controls had either placental abruption or placenta previa during the index pregnancy. After adjustment for potential confounders, placental abnormality during pregnancy was associated with a twofold increase in the risk of SIDS in offspring (odds ratio = 2.1, 95% confidence interval 1.3-3.1). The individual effects of placental abruption and placenta previa on the risk of SIDS did not differ significantly. An impaired fetal development due to placental abnormality may predispose an infant to a high risk of SIDS. (25 references) (Author)

990313-004

Home apnea monitoring and sudden infant death syndrome. Malloy MH, Hoffman HJ (1996), Preventive Medicine vol 25, no 6, 1996, pp 645-649

Objective: To estimate the U.S. national prevalence of apnea monitor use by birth weight classification and to examine the relationship between the use of apnea monitors and the occurrence of sudden infant death syndrome (SIDS). Design and setting: Data obtained from the 1988 National Maternal and Infant Health Survey (NMIHS) were used. Prevalence estimates of apnea monitor use were obtained by weighting survey data, and the relationship between monitor use and SIDS was accomplished by a case-control analysis using SIDS deaths and live controls obtained from the NMIHS. Outcome measure: Weighted estimates of the prevalence of apnea monitor use and odds ratios for the odds of use of an apnea monitor among SIDS victims compared with the odds of use of an apnea monitor among living controls. Results: The national prevalence estimates for home apnea monitor use among birth weight strata of 500 to 1,499 g, 1,500 to 2,499 g, and 2,500 g or more for blacks were 19.9, 2.6, and 1.1% compared with 44.0, 8.8, and 1.2% for non-blacks. The only significant association between the use of apnea monitors and SIDS was for black 500- to 1,499-g infants. The adjusted odds ratio for SIDS among monitored black 500- to 1,499-g infants vs unmonitored infants was 3.93 (1.09, 14.17). Conclusions: This analysis suggests a marked difference in reported monitor use between U.S. black and non-black infants. In addition, black very low birth weight infants at highest risk for SIDS appear to be preferentially selected for monitoring. The protective effect of home apnea monitoring in this survey population is unclear. (13 references) (Author)

990213-034

Short QTc interval as an important factor in sudden infant death syndrome. Davies DP (1999), Archives of Disease in Childhood vol 80, no 2, February 1999, pp 105-106

The rate of sudden infant death was drastically reduced when parents were advised to sleep their babies on their back. However, in 1997, 403 babies died for no apparent reason, although most of these deaths occurred in families where the mother smoked or there was exposure to tobacco smoke after birth. Much research is now going on into the prolongation of the QT interval which has been shown to be a risk factor for SIDS. Further research is needed into the significance of the QT interval, particularly where it is associated with other stress factors. (19 references) (VDD)

990210-018

Prenatal brain damage, placental pathology and SIDS. Laurini RN (1995), In: Johnson P editor. Impact of antenatal and postnatal environmental factors on infant outcome. Proceedings of the Third Congress of the European Society for the Study and Prevention of Infant Deaths, Oxford, August 1993. Oxford: John Radcliffe Hospital, Maternal Infant Healthcare Research Centre 1995, pp 49-52

Sudden infant death (SIDS) remains a multifactorial phenomenon were the intrauterine environment plays a major role in a significant number of cases. In particular events leading to intrauterine growth deviations and prenatal hypoxic-ischaemic encephalopathy lesions define a subset of cases with subtle changes of chronic hypoxia that increases the risk of SIDS. Detail assessment of postmortem findings in embryonic and fetal deaths including the placenta give further support to this concept. Therefore it is proposed that gliosis and other brain lesions are secondary to prenatal events and the disturbances in state (i.e. respiratory control) a result rather than a cause of gliosis. Although these findings fail to explain this phenomenon, it does provide data program prevention. (16 references) (Author)

990115-018

Has this man discovered the cause of cot death?. Hubbard B (1999), Natural Parent January/February 1999, pp 33-35

Dr Anthony Parish has a theory that sudden infant death (SIDS) is the result of a sudden heart attack caused by a form of chemical poisoning usually following use of pain-killing drugs during labour. A baby's immature immune system could also be damaged by cigarette smoke or carbon monoxide from cars, although these have less of an impact. This poisoning can be triggered when the baby experiences some kind of stress such as overheating. The findings of his research have not been accepted among organisations investigating causes of sudden infant death. Other possible causes of SIDS, such as vaccinations, prescribed drugs, fetal problems, and bedding are discussed. (KL)

990113-009

Unexplained deaths in infancy. (1999), Lancet vol 353, no 9148, 16 January 1999, p 161

Commentary on the recently published article (1) in which the author questions whether a significant proportion of deaths in infancy classified as sudden infant deaths are in fact deaths as a result of child abuse. 1. Meadows R. Unnatural sudden infant death. Archives of Disease in Childhood, vol 80, 1999, pp 7-14. (KL)

990113-005

Some 'cot deaths' are child abuse. White C (1999), BMJ vol 318, no 7177, 16 January 1999, p 147

Commentary on the recently published article (1) which questioned whether some cases classed as sudden infant death were in fact cases of fatal child abuse. 1. Meadows R. Unnatural sudden infant death. Archives of Disease in Childhood, vol 80, 1999, pp 7-14. (KL)

981203-047

Apnea of prematurity and risk for SIDS. Hodgman JE (1998), Pediatrics vol 102, no 4, part 1, October 1998, pp 969-971

Although the premature infant is at increased risk of sudden infant death syndrome (SIDS) there is no evidence that cardiorespiratory recordings at or near the time of discharge are effective in identifying the infant who is more likely to die. (9 references) (MS)

980807-036

Pertussis epidemic and sudden infant death syndrome. Jan MMS, Halperin S (1998), Clinical Pediatrics vol 37, no 7, July 1998, pp 449-452

Report of a study undertaken in Nova Scotia, Canada to investigate the relationship between whooping cough inaccurately diagnosed or missed and increased incidence of sudden infant death (SIDS). A total of 405 cases of whooping cough were examined during a five year period, and 64 cases of sudden infant death were identified in the same period. No associated increase in the number of SIDS cases was found in an epidemic year of whooping cough. (9 references) (KL)

980409-043

Infectious agents and SIDS: analysis of risk factors and preventive measures. Blackwell CC, Weir DM, Busuttil A (1997), Journal of Sudden Infant Death Syndrome and Infant Mortality vol 2, no 2, June 1997, pp 61-76

Epidemiological factors associated with sudden infant death syndrome (SIDS) closely resemble those for respiratory tract infections. We suggested some cases of SIDS are due to pathophysiological responses elicited by combinations of microbial products and/or cigarette smoke during a developmental stage when infants' endocrine responses are less able to 'damp down' the effects of inflammatory mediators. In this review we assess results of epidemiological and laboratory studies on interactions between developmental and environmental risk factors that could affect (a) mucosal colonization of infants by potentially pathogenic bacteria and viruses and (b) infants' inflammatory responses to infectious agents. These include: age range affected; prone sleeping position; minor respiratory infection; overheating; maternal smoking; bottle feeding; delayed immunization/lack of immunization. Recent findings help explain how risk factors might make infants more vulnerable to infection and inflammatory responses and partly explain the effectiveness of current recommendations for reducing the risk of SIDS. (84 references) (Author)

980312-028

Closing in on SIDS. (1998), AWHONN Lifelines vol 2, no 1, February 1998, p 15

Brief discussion of recent evidence on risk factors for sudden infant death, including research into malfunctions in the arcuate nucleus of the infant brain. (KL)

971120-029

Sudden infant death syndrome: a hypothesis. David CM (1997), Medical Hypotheses vol 49, no 1, July 1997, pp 61-67

A study of the strikingly low incidence of sudden infant death syndrome in Eastern countries revealed significant differences in infant handling thought to have an etiological bearing; therefore this writer suggested that adoption of certain Eastern methods of nursing may reduce the incidence of sudden infant death syndrome. A dramatic fall in incidence has resulted from implementing one of the suggestions made by the writer in 1983, namely the abandonment of the prone position, after initial opposition. The present hypothesis sets out to give a scientific explanation for this fall, and is a unified hypothesis explaining certain puzzling and disparate features of sudden infant death syndrome such as the remarkable winter incidence, age incidence, and the occurrence of sudden infant death syndrome during sleep, and is based on a postulated disturbance in thermoregulatory function (a unique hypothermia). Recommendations are made for evolving a test for sudden infant death syndrome-proneness and a possible method of treatment of a fatality within a short time frame. (Author)

971111-020

Sudden infant death syndrome: a proposed discovery. Parish AR (1997), Medical Hypotheses vol 49, no 2, August 1997, pp 177-179

Sudden cardiac death is a leading cause of fatality in the industrially developed world. Sudden infant death syndrome, has not hitherto been regarded as the same disease. However, the 55% reduction in the recorded rate of death from sudden infant death syndrome following the removal of stress-related problems caused by babies sleeping on their stomachs and overheating from tight and heavy clothing has, I propose, revealed that the babies may be dying from a similar stress-related cause, which can be prevented. (Author)

970502-002

Maternal selenium levels and sudden infant death syndrome (SIDS). McGlashan ND, Cook SJ, Melrose W, and others (1996), Australian and New Zealand Journal of Medicine vol 26, no 5, October 1996, pp 677-682

Background: The possibility is tested that low anti-oxidant status and/or low levels of selenium (Se) might predispose to Sudden Infant Death Syndrome (SIDS). Aim: This study was undertaken to collect evidence on the Se status of pregnant and non-pregnant women and newborn babies and to establish whether babies who later died of cot death had significantly divergent levels of blood Se at birth. Methodology: Aliquots of blood were collected from all newly pregnant mothers in Tasmania and from the cords of all newborn babies. These were analysed for Se and glutathione

peroxidase (GPx) content and compared by season and with non-pregnant, age standardised blood donors in three areas of Tasmania and three mainland Australian States. Results: Cot death babies' cordbloods were not significantly different in Se or GPx-status, in this small sample, from those of other babies, nor was a seasonal variation in these parameters demonstrated among 390 randomly selected Tasmanian mothers. Mothers-to-be showed a decrease in enzyme levels during pregnancy and Tasmanian blood donors had significantly lower levels than donors from other States. Conclusion: While no evidence can safely be drawn about a relationship between Se or GPx-status and SIDS, this study provides base level measures for populations showing that Tasmanian residents have low levels of these anti-oxidants. (Author)

960814-084

Symptoms, sweating and reactivity of infants who die of SIDS compared with community controls. Taylor BJ, Williams SM, Mitchell EA, and others (1996), Journal of Paediatrics and Child Health vol 32, no 4, August 1996, pp 316-322

Objective: To describe the symptoms of illness reported by the parents of infants who have died of sudden infant death syndrome (SIDS) compared with those reported by community controls. Methodology: A nationwide case-control study involving regions of New Zealand with 78% of all births between 1987 and 1990. Home interviews were completed with parents of 393 (81% of total) infants who died from SIDS in the post neonatal age group, and 1592 (88.4% of total) controls who were a representative sample of all hospital births in the study region. Results: Symptoms of infection were common in both cases and controls, but were not significantly different. Infants dying of SIDS, however, were likely to have symptoms suggestive of more severe illness in the 2 days before death (odds ratio [OR] = 3.02, 95% confidence interval [CI] 1.69-5.38). After adjusting for potential confounding this was still statistically significant (adjusted OR 2.36, 95% CI 1.14-4.90). Also, babies dying of SIDS were more likely to have been less reactive to their environment in the 2 weeks before death compared with the controls (univariate OR 0.88, 95% CI 0.55-1.39, adjusted OR 0.55, 95% CI 0.29-0.88). 'Drenching' sweats at least weekly were reported for 15.6% of case infants compared with 5.9% of control infants (adjusted OR 2.12, 95% CI 1.53-3.39). Forty per cent of these infants had this symptom in the first 4 weeks of life when it was also associated with a significantly raised risk of SIDS. Apnoea lasting more than 20s was reported for 13.2% of case infants compared with 5.3% of control infants (adjusted OR 1.93, 95% CI 1.17-3.17). Similarly, 71.8% of case infants' faces were reported to never turn red while awake compared to 49.8% of control infants (adjusted OR 2.98, 95% CI 2.19-4.07). Conclusions: Only a small number (6.4%) of babies who die of SIDS have symptoms of serious illness in the 2 days before death. There is support for the hypothesis that there is a group of babies dying of SIDS who have subtle abnormalities in autonomic control or arousal ability. (Author)

960812-028

SIDS is not always truly sudden. Meny RG, Jesurun CA, Lin CH, and others (1996), Journal of Sudden Infant Death Syndrome and Infant Mortality vol 1, no 1, March 1996, pp 51-54

The authors have identified two SIDS deaths which were not truly sudden. (SJH)

960812-025

A probability model for the age distribution of SIDS. Mage DT (1996), Journal of Sudden Infant Death Syndrome and Infant Mortality vol 1, no 1, March 1996, pp 13-31

Universal epidemiological characteristics of sudden infant death syndrome (SIDS) are a lognormal-type age distribution, and a 50% risk of male SIDS that suggests a possible X-linkage. A combined set of 19,755 SIDS ages from 15 global data sets is modelled by a four-parameter lognormal distribution, with a mode at 63 days and median at 94 days, bounded between birth and 41.2 months of life. This model can be interpreted as the probability that a genetically susceptible infant has four independent risk factors occurring simultaneously. The four risk factors (A,B,C,D) are interpreted as follows: (A) a constant rate of risk for a severe apneic episode by a susceptible infant, with (B) a risk rising in probability from 0 at birth, to a peak at 3.1 months, and then falling back to 0 at 41.2 months, eg intrinsic infant anemia, with (C) a risk rising in probability from 0 at birth to 1 at age 41.2 months eg from an extrinsic source of infection, with (D) a risk decreasing in probability from 1 at birth toward 0 at 41.2 months, eg an intrinsic deficit in neurological development. The cause of SIDS is hypothesized to be prolonged apnea during a status of

cerebral hypoxia. The four risks can be related to almost all epidemiological factors associated with SIDS, eg chronic hypoxia, with higher infection rates found in SIDS over 3 months, and higher deficits in neurological development found in SIDS below 3 months. (Author)

960701-022

Sudden infant death syndrome: orthostatic intolerance and disordered antigravity postural mechanisms. Reid GM, Tervit H (1996), Medical Hypotheses vol 46, no 2, February 1996, p 162

The authors speculate that, in SIDS, the pressure gradients and blood flow responses to the brain are underdeveloped. They postulate that the prolonged bed-rest disorders evident in human studies on the effects of microgravity are also encountered in neonates lacking the physiological adjustments of postural mechanisms which NASA investigators describe as orthostatic intolerance. (Author)

960701-015

Deficient heat shock protein expression: a potential mechanism for the sudden infant death syndrome. Gozal D (1996), Medical Hypotheses vol 46, no 1, January 1996, pp 52-54

Induction of heat shock proteins follows a metabolic stress and protects from subsequent stresses. Stressors proposed for the sudden infant death syndrome include infection, environmentally induced hyperthermia and hypoxia. Failure to express heat shock proteins to such stressful conditions may lead to reduced tolerance, and enhance inappropriate physiologic responses and vulnerability which ultimately may lead to infant death. (Author)

960613-122

Risk factors for sudden infant death syndrome: further change in 1992-3. Hiley CMH, Morley CJ (1996), BMJ vol 312, no 7043, 1 June 1996, pp 1397-1398

Update of information (1) on changes in risk factors for sudden infant death syndrome in three East Anglian health districts first published in 1992. 1. Hiley CMH, Morley CJ. BMJ, vol 309, 1994, pp 703-704.

960608-019*

Sudden infant death syndrome: an update. Chantler S (1996), Hospital Update vol 22, no 6, June 1996, pp 212, 214-216

Sudden infant death is a distressing and mysterious syndrome. Dr Chantler reviews what is known about the condition and describes recent discoveries. (Author)

960605-030

Dysfunctional development of the diaphragm in SIDS and the prone sleeping position. Jones RE (1996), Clinical Pediatrics vol 35, no 3, March 1996, pp 173-174

Discussion of the aetiological significance of dysfunctional development of the diaphragm and its relationship to rapid eye movement (REM) sleep, another factor that has been observed in SIDS. (KL)

960605-009

Cytokines may give insight into mechanisms of death in sudden infant death syndrome. Sayers NM, Drucker DB, Grencis RK (1995), Medical Hypotheses vol 45, 1995, pp 369-374

There are a number of postulated causes of sudden infant death syndrome, including bacterial toxins, defects in thermoregulation and hypersensitivity. This paper formulates the hypothesis that analysis of cytokine profiles in suspected sudden infant death syndrome victims may give an insight into mechanisms of death. These cytokine profiles may also help to identify specific causes of sudden infant death syndrome or indicate that different causes act in concert in individual cases. (Author)

960419-014

Respiratory disease and sudden infant death syndrome. Anderson R, Britton J, Esmail A, and others (1995), In: Botting B editor. The health of our children: decennial supplement. London: HMSO 1995, pp 113-134

Discussion of trends in respiration disorders and sudden infant death in England and Wales 1958 to 19990. (75 references) (KL)

960415-001

Child virus is blamed for 50% of cot deaths. Rogers L (1996), Sunday Times 7 April 1996, p 3

A research team at Royal Liverpool children's hospital led by Tony Hart believe that a respiratory virus caught by all small children could be the cause of half the cot deaths in Britain. A further 750 newborn babies are killed by the virus every year. The research is funded by the Foundation for the Study of Infant Deaths. (KL)

960328-052

The timing of SIDS deaths in premature infants in an urban population. Lipsky CL, Gibson E, Cullen JA, and others (1995), Clinical Pediatrics vol 34, no 8, August 1995, pp 410-414

Previous reports have demonstrated that premature infants are at greatly increased risk for sudden infant death syndrome (SIDS). Although only 9% of infants are born at less than 36 weeks' gestation, 20% of SIDS victims are former premature infants. The objective of this study was to characterize the time course of SIDS in premature infants and to determine why SIDS occurs at such a high rate in this patient population. A database of all cases of SIDS in Philadelphia from 1987 through 1991 was used to establish the time course for SIDS deaths in term and preterm infants. Gestational age was established by Dubowitz exam. To evaluate distinctly different age groups, infants from 32-36 weeks were excluded from analysis. Age at death and postconceptional age of death were compared for both groups. Data are described in weeks (mean +- SEM), and analyzed using unpaired t-test and log-rank test to compare survival rate between term and preterm infants. A significant difference ($P < 0.01$) was noted in age at death of term versus preterm infants. No difference was found in postconceptional age of death. The survival rates were also different ($P < 0.001$). Preterm infants showed a much wider distribution in age of death from SIDS. The term infants followed the classic SIDS curve. By 32 weeks' postnatal age, 95% of all SIDS had taken place in the term group, but only 75% in the preterm group. The age at death for SIDS differs in the preterm infant. These data reinforce the concept of prolonged vulnerability of preterm infants to SIDS. Survival of greater numbers of premature infants makes it increasingly important to focus efforts for SIDS prevention in this group for a longer period of time. (Author)

960129-055

Autopsies of sudden infant death syndrome - classification and epidemiology. Hatton F, Bouvier-Colle MH, Barois A, and others (1995), Acta Paediatrica vol 84, no 12, December 1995, pp 1366-1371

An enquiry into sudden infant death syndrome (SIDS) in 1987 furnished us with detailed epidemiological data for 281 cases that underwent a thorough post-mortem examination. This analysis uses these data to evaluate the role the autopsy plays in explaining sudden death. The cases were classified into three diagnostic groups: explained causes of death (group 1), unexplained deaths with anomalies (group 2), and no anomaly (group 3). These 281 cases show the three essential features that characterize SIDS: over-representation of males, increased deaths during the second and third months of life, and increased deaths during winter. The autopsy examination revealed that many of these deaths had a medical explanation. Almost half were assigned to group 1. At the time of autopsy, no precise pathology could be diagnosed for 147 deaths; of these, 140 showed histological anomalies. There were only seven sudden deaths for which no abnormal sign was evident at the autopsy. These results are compared with those of similar studies and discussed in connection with three factors: the initial selection of cases, the nature and degree of the investigations, and the possible interpretations of the symptoms uncovered. (Author)

960107-009

Lethal synergy between toxins of staphylococci and enterobacteria: implications for sudden infant death syndrome.

Sayers NM, Drucker DB, Morris JA, and others (1995), Journal of Clinical Pathology vol 48, 1995, pp 929-932

Aim: To test the hypothesis that lethal synergy occurs between toxin preparations of nasopharyngeal staphylococci and enterobacteria from sudden infant death syndrome (SIDS) victims and matched healthy infants. Methods: SIDS and matched healthy babies were studied if both staphylococcal and enterobacterial strains were isolated from the nasopharynx. The lethality of toxin preparations from each bacterial isolate (separately and combined) was assessed over a range of dilutions using the chick embryo assay system. Results: Staphylococci and enterobacteria were isolated together from the nasopharynx of seven SIDS babies but from only one normal healthy infant. Enterobacterial toxins were lethal at high dilutions. Staphylococcal toxins were less toxic. Simultaneous testing in the chick assay of staphylococcal and enterobacterial toxins, from each baby, at non-lethal concentrations enhanced lethality levels by 177 to 1011% compared with lethality expected by an additive effect alone. Conclusions: Synergy occurs between the toxins of nasopharyngeal staphylococci and enterobacteria. This combination of strains is more likely to occur in the nasopharynx of SIDS victims than that of healthy infants. (Author)

951211-038

Immunisation and the sudden infant death syndrome. Mitchell EA, Stewart AW, Clements M, et al (1995), Archives of Disease in Childhood vol 73, no 6, December 1995, pp 498-501

Aims: To examine the relation between immunisation and the risk of sudden infant death syndrome (SIDS). Methods: A large nationwide case-control study. Parental held records were used to measure immunisation status. Results: Infants were at increased risk of SIDS if they had not received the 6 week, 3 month, and 5 month immunisations. After controlling for potential confounding variables, including those which measured health care use and infant illness, the relative risk of SIDS for infants not being immunised at 6 weeks was 2.1 (95% confidence interval = 1.2, 3.5). Four per cent of cases died within four days of immunisation and 7.6% of control infants had been immunised within four days of the nominated date. There was a reduced chance of SIDS in the four days immediately following immunisation (OR = 0.5; 95% CI = 0.2 to 0.9). Conclusions: Immunisation does not increase the risk of SIDS and may even lower the risk. (Author)

951129-009

Cot death update: the current state of knowledge regarding the risk factors for cot death. Silvester J (1995), Journal of Neonatal Nursing vol 1, no 4, July 1995, pp 17-19

The sudden and unexpected death of an apparently healthy baby for no obvious reason is a tragedy for the family concerned and an enigma for the healthcare professionals involved. The 'Reduce the Risk' campaign has considerably decreased the incidence of cot death, but many questions remain unanswered. Further research is required to elucidate all the risk factors concerned and reduce still further the unexplained infant deaths which occur each year. (Author)

951111-024

The relationship between intra uterine growth retardation (IUGR) and the risk for SIDS. van Velzen D (1995), Foundation for the Study of Infant Deaths (FSID) News no 51, September 1995, pp 7-8

Poor growth of a baby in the womb may result in intra uterine growth retardation (IUGR), a condition where a baby born at term is smaller than it should be. It may be present in low birth weight babies but also in apparently normal weight babies. The FSID-funded Liverpool group has investigated important organs for evidence of deficient development which indicates IUGR. These include the kidney, the lungs, the brain, and the muscle of the diaphragm and the nerve that innervates it (the 'phrenic' nerve). The research has shown organ deficiencies to be present in both low and apparently normal birth weight babies that died of sudden infant death syndrome and may explain why some babies are more vulnerable. (Author)

951101-014

Vaccination and SIDS: information from the South Australian SIDS database. Byard RW, Mackenzie J, Beal SM (1995),

Medical Journal of Australia vol 163, no 8, 16 October 1995, pp 443-444

Statistics from the Sudden Infant Death (SIDS) Database in South Australia is used to refute claims of a link between vaccination for diptheria-tetanus-pertussis is a risk factor for sudden infant death. (KL)

951031-079

Antenatal and intrapartum factors associated with Sudden Infant Death Syndrome in the New Zealand Cot Death Study. Stewart AJ, Williams SM, Mitchell EA, and others (1995), Journal of Paediatrics and Child Health vol 31, no 5, October 1995, pp 473-478

Objective: To describe the relationship between antenatal and intrapartum factors and sudden infant death syndrome (SIDS). Methodology: The New Zealand Cot death Study was a 3 year case-control study, with 485 infants who died from SIDS in the postneonatal period and 1800 randomly selected control infants. Data were obtained from obstetric records, parental interview and community nursing records. Results: This study confirms many of the antenatal and intrapartum risk factors for SIDS noted in studies from both the southern and northern hemispheres. After controlling for potential confounders, such as occupational group and marital status, significant inverse effects were noted for interpregnancy interval, birthweight and gestation. Other factors that retained status, significant inverse effects were noted for interpregnancy interval, birthweight and gestation. Other factors that retained a significantly increased risk of SIDS were: increasing parity, bacteriological evidence of urinary tract infection (UTI) (adjusted odds ratio 1.73, 95% CI 1.10-2.73); smoking antenatally (AdjOR 2.14 5% CI 1.61-2.84); less than six antenatal checks attended (ADjOR 3.23, 95% CI 1.70-6.02). No interaction was observed between maternal haemoglobin and antenatal smoking. Interactions were tested for and not found between antenatal smoking and three antenatal risk factors (UTI, short second stage of labour and number of antenatal appointments). The only significant interaction between these three factors and three modifiable postnatal risk factors (prone sleeping, bed sharing and bottle feeding) was between bed sharing and fewer antenatal appointments. The risk of SIDS associated with bed sharing was greater among those whose mothers had fewer antenatal appointments. Conclusions: Although many of the previously identified antenatal and intrapartum risk factors for SIDS are confirmed, the risks of SIDS associated with obstetric factors are in general considerably lower than the risks associated with the four modifiable postnatal risk factors. (Author)

951031-072

Higher rates of SIDS persist in low income groups. Ford RPK, Nelson KP (1995), Journal of Paediatrics and Child Health vol 31, no 5, October 1995, pp 408-411

Objective: To examine how changes in the rates of sudden infant death syndrome (SIDS) have varied in different income groups during a 25-year-period. Methodology: Census data were obtained for five census periods (1971, 1976, 1981, 1986 and 1991) on the number of SIDS cases and inevitable deaths. Census area units (CAU) were ranked according to the average income earned by adults over the age of 15 years for each census year. The CAU were then divided into three equal income groups: low, middle and high. Results: The rates of SIDS differed significantly between the three income groups for the 1991 census period with the low income SIDS rate being 4.6/1000 births compared to 1.2/1000 live births for the higher income groups (Chi-squared = 18.3, P<0.0001). There was no association between rates of inevitable deaths and income groups. Conclusion: Currently, low income groups have three times the rate of SIDS compared to those higher income groups. The reason for this is probably because the disadvantaged groups carry n overall higher burden of risk factors for SIDS. This must be kept in mind as further SIDS educational programmes are developed and implemented. (Author)

951031-063

Pathology review of sudden and unexpected death in Aboriginal and non-Aboriginal infants. Alessandri LM, Read AW, Dawes VP, and others (1995), Paediatric and Perinatal Epidemiology vol 9, no 4, October 1995, pp 406-419

Summary: Previous research showed that the sudden infant death syndrome (SIDS) rate for Aboriginal infants significantly increased during the 1980s in Western Australia (WA) and raised the possibility of a diagnostic transfer of Aboriginal infant deaths from our causes to SIDS over this period. Here, therefore we review the pathology of SIDS and other sudden and unexpected deaths in infancy (SUDI) for Aboriginal and non-Aboriginal infants in WA between

1980 and 1988. The aim was to investigate whether there had been differences in the diagnosis and/or classification of SIDS according to whether the infants were Aboriginal or non-Aboriginal. The study population comprised: (1) all Aboriginal cases of SIDS and other SUDI between 1980 and 1988, and (2) corresponding random samples of non-Aboriginal cases. A two-stage process was employed for the review. First, histology slides were reviewed for each case where the aboriginality of the infant was Aboriginal and the original cause of death were unknown to the pathologists. Second, all paper records (i.e. death scene investigations, laboratory tests and medical reports) except for the original cause of death information were reviewed by the pathologists. The results showed that there was excellent agreement between the final review diagnosis and the original diagnosis for both Aboriginal and non-Aboriginal SUDI. Thus, there was no evidence for a diagnostic shift among Aboriginal infant deaths and the review supported the observed increase in the SIDS rate for Aboriginal infants. (Author)

951031-003

Prematurity, sudden infant death syndrome, and age of death. Malloy MH, Hoffman HJ (1995), Pediatrics vol 96, no 3, part 1, September 1995, pp 464-471

Objective: To determine if preterm infants are at greater risk for sudden infant death syndrome (SIDS) than term infants and to determine if the postconceptional age of SIDS deaths varies by gestational age a birth. Methods: A cohort analysis was conducted using data from the 1987 United states' Birth Cohort Linked Birth/Infant Death Certificate tapes. SIDS was defined as the death of any infant who was > 24 weeks gestation at birth; weighed > 500 g at birth; was assigned an International Classification of Diseases-9th Revision (ICD-9) underlying cause of death of 7980; and had an autopsy. Results: The overall SIDS rate using our definition was 1.20 deaths/1000 live births. The SIDS rates by gestational age categories of 24 to 28 weeks, 29 to 32 weeks, 33 to 36 weeks, and 37 or more weeks were 3.52, 3.01, 2.27, and 1.06 deaths/1000 live births, respectively. Because of misclassification of gestational age among the most pre-term infants, a restricted analysis was conducted on SIDS victims whose gestational ages fell within cutoff values derived from a methodology that excluded gestational age assessments assumed to be invalid. This subgroup analysis showed a mean (standard deviates) postconceptional age of death for SIDS for infants of 24 to 28 weeks, 29 to 32 weeks, and 33 to 36 weeks gestation to be 45.8 (8.3), 47.3 (8.6), and 48.0 (8.3) weeks, respectively, compared with 52.3 (8.5) weeks for term infants (ANOVA P = .0001). Conclusions: We infer from this analysis that preterm infants are at higher risk for SIDS than term infants, and that the postconceptional age of peak vulnerability for SIDS may differ by 4 to 6 weeks between preterm and term infants. (Author).

951009-117

Newborn acoustic cry characteristics of infants subsequently dying of sudden infant death syndrome. Corwin MJ, Lester BM, Sepkoski C, and others (1995), Pediatrics vol 96, no 1, part 1, July 1995, pp 73-77

Objective: To test the hypothesis that the occurrence of a neonatal cry exhibiting a high first formant is a risk factor for sudden infant death syndrome (SIDS) and to evaluate the association between SIDS and other acoustic cry variables. Method: We recorded cries and obtained medical and demographic data for 21 880 apparently healthy term newborns. Two cries were recorded between days 2 and 7 of life, after a painful stimulus at the time of routine blood drawing. Acoustic variables were measured with an automated computer-based analysis system. Twelve infants died of SIDS. Age at death ranged from 19 days to 6.5 months. Autopsies were performed in all cases. At least one cry was analyzed for all 12 infants who died of SIDS and 20 167 infants without SIDS. Two cries were analyzed for 9 infants who died of SIDS and 14 235 infants without SIDS. Results: Newborns whose first cries exhibited a high first formant were more likely to die of SIDS than infants whose first cries did not have this characteristic (relative risk, 3.5; 95% confidence interval [CI], 1.1 to 12). The relative risk for SIDS increased to 8.8 (95% CI, 2.2 to 35) for newborns whose second cries showed that this characteristic persisted. Newborns with the combination of both a high first formant and a high number of mode changes on both of two cries had a relative risk of 32 (95% CI, 8.7 to 120). Conclusions: We have shown an association between alterations in neonatal cry acoustics and SIDS. Cry analysis represents a potentially important research tool that, when studied in relation to other physiologic measures, may lead to an improved understanding of SIDS. (Author)

950922-049

Infant dreaming and fetal memory: a possible explanation of sudden infant death syndrome. Christos GA (1995), Medical Hypotheses vol 44, no 4, April 1995, pp 243-250

During rapid-eye-movement sleep, when we dream, the brain is thought to be processing stored memory. The memory of a newborn infant is dominated by its fetal experience, and the infant is likely to dream about its life in the womb. Research with lucid (or conscious) dreaming has shown that dream images are supported by the corresponding body actions, using those muscles which remain active during rapid-eye-movement sleep. We suggest that sudden infant death syndrome or cot death may be a result of an infant dreaming about its life (or memory) as a fetus. In the course of that dream, since a fetus does not breathe (in the usual sense) the infant may cease to breathe and may die. This simple hypothesis is consistent with all of the known facts about sudden infant death syndrome (pathological and epidemiological), such as the age at death curve (the observed exponential decay and possibly the peak at 2-3 months), the higher risk with the prone sleeping position (but not excluding the supine position), and the observed climatic variation (seasonal and regional) in the incidence of sudden infant death syndrome. Many of these well-established facts have no other known explanation and other theories can generally only account for a few of the known facts about sudden infant death syndrome. Our hypothesis is also supported by recent findings that, as a group, sudden infant death syndrome infants have a higher proportion of rapid-eye-movement sleep and also that they have an average higher heart rate (corresponding to possible fetal dreams) but only during rapid-eye-movement sleep. The infant dreaming hypothesis also offers an explanation of why all of the chemoreflexes, and other protective mechanisms, that would normally awake the infant, may fail simultaneously. On the basis of our theory, we make suggestions as to how the infant dreaming hypothesis can be tested further and how the risk of sudden infant death syndrome may be reduced. (Author)

950921-085

Aldolase B A149P mutation and hereditary fructose intolerance are not associated with sudden infant death syndrome. Aarskog NK, Ogreid D (1995), Acta Paediatrica vol 84, no 8, August 1995, pp 947-948

No abstract available.

950921-050

Accidental death or sudden infant death syndrome?. Beal SM, Byard RW (1995), Journal of Paediatrics and Child Health vol 31, no 4, August 1995, pp 269-271

Objective: To describe the reasons why it is difficult to decide whether to attribute some infant deaths to accidents or to SIDS. Methodology: To extract from infant deaths data in South Australia those where the cause of death is debatable. Results: The risks associated with rocking cradles, bed sharing, bedclothes, couch sleeping, unsafe cots or beds and the prone position are presented. Conclusions: Uniform worldwide death scene investigations for all infant deaths should help identify unsafe sleeping conditions for infants. (Author)

950713-001

The Tasmanian SIDS case-control study: univariable and multivariable risk factor analysis. Ponsonby A-L, Dwyer T, Kasl SV, and others (1995), Paediatric and Perinatal Epidemiology Vol 9, no 3, July 1995, pp 256-272

Summary: A population-based retrospective case-control study has been conducted in Tasmania since October 1988. Study measurements pertained to the scene of death of last sleep, as well as a verbal questionnaire on relevant exposures. From 1 October 1988 to 1 October 1991, 62 cases of sudden infant death syndrome (SIDS) occurred. Case response rate for retrospective interviews was 94% (58/62). The initial control response rate was 84% (101/121). After stratification for maternal age and birthweight, there was no increase in risk associated with the usual side position (odds ratio [OR] 1.05 [0.27, 5.02], compared with the supine position (OR 1.00, reference). The prone position was associated with increased risk [OR 5.70(1.67, 25.58)], relative to the supine position. In the final multivariable model, predictors of SIDS in this study were usual prone position (P<0.001), maternal smoking (P=0.008), a family history of asthma (P= 0.0045) and bedroom heating during last sleep (P=0.039). Protective factors were maternal age over 25

years (P=0.013) and more than one child health clinic attendance (p=0.003). The results provide further support for current health education activities which aim to inform parents of modifiable risk factors for SIDS, including the prone sleeping position, thermal stress and infant exposure to tobacco smoke. (Author)

950711-066

A reexamination of the risk factors for the sudden infant death syndrome. Taylor JA, Sanderson M (1995), Journal of Pediatrics vol 126, no 6, June 1995, pp 887-891

Objective: To determine which risk factors are specific for the sudden infant death syndrome (SIDS) rather than characteristics of postneonatal deaths in general. Study population: The live births and infant death cohorts of the 1988 National Maternal and Infant Health Survey. Methods: Information on live births, deaths from SIDS, and postneonatal deaths from other causes was abstracted from the National Maternal and Infant Health Survey. To account for oversampling of certain populations, the data were weighted to reflect national counts. Risk factors were defined as black race, birth weight less than 1500 gm, birth weight less than 2500 gm, gestational age at birth less than 37 weeks, 5-minute Apgar score less than 7, male gender, more than two previous pregnancies, maternal age less than 20 years, maternal education level less than 12 years, multiple births, and maternal smoking during pregnancy. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to compare the SIDS with the live births cohort, infants who died of other causes with the live births cohort, and SIDS with non-SIDS deaths. The population-attributable risk percentage was computed for risk factors independently associated with SIDS when compared with other postneonatal deaths. Results: For all characteristics other than a 5-minute Apgar score less than 7, the ORs comparing infants who died of SIDS with the live births cohort were significantly greater than 1.0. Similarly, ORs comparing infants who died of other causes with the live births cohort were also greater than 1.0, except for male gender. When the two infant death cohorts were compared, only maternal smoking during pregnancy and low maternal education level were significantly more common among the SIDS group. After we controlled for cigarette smoking, the adjusted OR for low maternal education level was not significantly greater than 1.0. However, even after control for low maternal education level, prenatal exposure to tobacco was significantly more common among the SIDS group than in infants dying of other causes (OR = 1.97; 95% CI, 1.59 to 2.45). On the basis of an adjusted OR of 2.92 when the SIDS group was compared with the live births cohort, the population-attributable risk percentage for maternal smoking as a risk factor for SIDS was 30%. Conclusion: Among characteristics generally thought to be risk factors, only maternal smoking during pregnancy was independently associated with SIDS. Data from this nationally representative sample indicate that if women refrained from smoking while pregnant, up to 30% of SIDS might be prevented. (Author)

950605-038

Fetal behaviour and the sudden infant death syndrome (SIDS). Smoleniec J, James D (1995), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 72, no 3, May 1995, pp F168-F171

To examine whether differences in sleep maturation could be identified before birth, behavioural studies were carried out in 28 fetuses. Studies were possible in all 28 fetuses at 28 weeks, but only in 26 fetuses at 36 weeks (two fetuses delivered before 36 weeks). The risk of sudden infant death syndrome (SIDS) was determined using the Oxford SIDS scoring system. The fetuses at greater risk of SIDS had coincidence of behavioural characteristics for a significantly lower percentage of the time than those at low risk. This difference reached significance (p ó 0.05) only at 36 weeks. (Author)

950511-094

Sudden infant death syndrome and subsequent siblings. Hunt CE (1995), Pediatrics vol 95, no 3, March 1995, pp 430-432

There is an increased risk of sudden infant death (SIDS) among subsequent siblings of SIDS victims. Recurrence rates, risk factors and the cost-effectiveness of home monitoring are discussed. (KL)

950417-006

The sleep of reason. (1995), Guardian (Weekend) 1 April 1995, pp 24-30

Seven years ago Barry Richardson came up with what remains the most persuasive explanation of cot death. It makes far more sense than the latest theory - that the syndrome is caused by smoking. So why is his work ignored or condemned? (Author)

950308-028

Sudden infant death syndrome: effect of breast and formula feeding on frontal cortex and brainstem lipid composition. Byard RW, Makrides M, Need M, and others (1995), Journal of Paediatrics and Child Health vol 31, no 1, February 1995, pp 14-16

Methodology: Docosahexaenoic acid levels were measured by gas chromatography in samples of frontal lobe and brainstem taken from 28 and 26 infants, respectively, who had died of sudden infant death syndrome (SIDS). Results: Significantly higher levels of docosahexaenoic acid were present in the frontal lobe tissues derived from the 13 breast fed infants (age range = 3.3-36.3 weeks; mean 15.9 +- 11.3 weeks) compared to the 15 formula fed infants (age range = 6.9-47.7 weeks; mean 19.3 +- 10.6 weeks); mean (+- s.d.) levels were 8.5 +- 1.1% and 7.6 +- 0.8% of total fatty acids (P = 0.019). There was, however, no significant difference in brainstem docosahexaenoic acid levels between breast and formula fed infants. Conclusions: Given these variable findings, further investigation of the relationship between dietary fatty acid intake and cerebral lipid levels may help to clarify whether different modes of feeding have a role in the pathogenesis of SIDS. (Author)

950201-067

Changes in deep body temperature before a cot death. Jackson JA, Petersen SA, McKeever PA, and others (1995), Archives of Disease in Childhood vol 72, no 1, January 1995, p 97

No abstract available.

950117-009

Bottle feeding and the sudden infant death syndrome. Gilbert RE, Wigfield RE, Fleming PJ, and others (1995), BMJ vol 310, no 6972, 14 January 1995, pp 88-90

Objective: To determine whether the risk of the sudden infant death syndrome is increased in bottle fed babies. Design: Population based case-control study matching for age and time. Subjects: All babies aged 1 week to 1 year dying of sudden infant death syndrome during November 1987 to April 1989 or February 1990 to June 1991 and two live controls. Setting: Avon and North Somerset. Main outcome measures: Breast or bottle feeding, sleeping position, maternal smoking, parental employment, and length of gestation. Results: Compared with being fully breast fed, the crude odds ratio for sudden infant death in fully bottle fed babies was 3.1 and for mixed breast and bottle fed babies 1.5. These odds ratios fell to 1.8 (95% confidence interval 0.7 to 4.8) and 1.2 (0.5 to 2.7) respectively after maternal smoking, parental employment, preterm gestation, and sleeping position had been adjusted for. Sleeping position partly masked the effect of being bottle fed on sudden infant death as breast fed babies were more likely to have slept prone than bottle fed babies. Conclusions: Bottle feeding is not a significant independent risk factor for the sudden infant death syndrome. Patterns of maternal smoking, preterm gestation, and parental employment status account for most of the apparent association with bottle feeding. (Author)

940925-021

Infant care practices and the investigation of physiological mechanisms. Johnson P (1994), Early Human Development vol 38, no 3, 15 September 1994, pp 165-179

It is strange that some aspects of infant care have been strongly promoted by modern medicine while others have been neglected. Thus prone sleeping which has been strongly promoted is now related to an increase in SIDS, whereas the promotion of breast feeding in developed countries has been less successful. Unfortunately there has not been sufficient physiological investigation of many infant care practices and some of the proposed mechanisms for SIDS and prone sleeping have not been substantiated. Thus further work is needed on hypercapnia, hypothermia and periodic breathing and respiratory control. Studying infants alone may leave out important physiological mechanisms

such as the effect on body warmth when the infant is close to the mother. More investigation is needed of antenatal factors related to SIDS and it is critically important that physiological investigation should not look for single mechanisms but be concerned with the interaction of many physiological factors. (Author)

940331-049

Well health care and the sudden infant death syndrome. Ford RPK, Mitchell EA, Taylor BJ (1994), Journal of Paediatrics and Child Health vol 30, no 2, April 1994, pp 140-143

The aim of this study was to examine whether poor attendance at routine antenatal and postnatal `well child' health services was associated with a higher risk of sudden infant death syndrome (SIDS, or cot death). A nationwide case-control study of SIDS in New Zealand enrolled 485 postneonatal deaths due to SIDS and 1800 control infants who were selected randomly. The risk for SIDS was found to be higher for infants whose mothers attended their first antenatal check later than 3 months into the pregnancy, made fewer antenatal visits, and did not go to antenatal education classes. However, this increased risk was largely explained by high parity, maternal smoking, the mother not being married, mother being <20 years old at the birth of her first child, and delivery during the winter months. Infants not attending a 6 week postnatal check had an almost three-fold increased risk of SIDS compared with those who did attend (odds ratio [OR] 2.86; 95% confidence interval [CI] 1.93, 4.24). Similarly, infants not attending well child clinics were at increased risk of SIDS (OR 2.75; CI 2.09, 3.62). These differences persisted when adjusted for likely confounders. This study demonstrates that infants who miss child health nurse clinics are those most at risk for SIDS and are those who warrant increased surveillance. (Author)

930918-040

Ethnic differences in infant-rearing practices and their possible relationship to the incidence of sudden infant death syndrome (SIDS). Farooqi S, Perry IJ, Beevers DG (1993), Paediatric and Perinatal Epidemiology vol 7, no 3, July 1993, pp 245-252

The aetiology of sudden infant death syndrome (SIDS) is still uncertain, although associations with overheating and the prone sleeping position have been reported. In the UK, the incidence of SIDS is considerably lower in infants of Asian origin, but as yet no explanation for this has been suggested. We have studied a group of 202 white and 172 Asian multiparous mothers attending an antenatal clinic to compare the sleeping position and home environment of infants in each ethnic group. We found that significantly more white infants (31%) than Asians (11%) were placed in the prone position at night and that 94% of Asian infants slept in their parents' bedroom, compared with 61% of whites. These observations demonstrate marked differences in the infant rearing practices favoured by Asians and whites and lend support to the concept that the prone position and separate bedrooms may be contributors to the development of sudden infant death. (Author)

900905-011

Ethnic differences in incidence of sudden infant death syndrome in Birmingham. Kyle D, Sunderland R, Stonehouse M, and others (1990), Archives of Disease in Childhood vol 65, no 8, August 1990, pp 830-833

Among the 45 204 live births in Birmingham in the three calendar years 1981-3, there were 218 postneonatal deaths, giving a postneonatal mortality rate of 4.82 per 1000 live births. Postneonatal mortality rates were 4.22 for whites, 5.91 for Asians (relative risk 1.26, 95% confidence interval (CI) 1.04 to 1.53) and 8.20 for Afro-Caribbeans (relative risk (relative risk 1.78, 95% CI 1.25 to 2.55). Among Asians malformations were common (3.36) and sudden infant death syndrome rare (1.18), in contrast to Afro-Caribbeans among whom the rates were 0.66 and 5.25, respectively. Logistic regression analysis demonstrated a significantly lower risk of sudden infant death syndrome (SIDS) in Asians and significantly raised risks of SIDS in very low birthweight babies and those with unemployed parent(s). Ethnic differences persisted after controlling for maternal age, social class, and birth weight. Studies of sociocultural differences in child rearing practices are needed and may uncover important aetiological factors of sudden infant death syndrome. (Author)

2024-03455

Preterm infants experience a nadir in cerebral oxygenation during sleep three months after hospital discharge. Yee AK, Shetty M, Siriwardhana LS, et al (2024), Acta Paediatrica 20 February 2024, online
Full URL: https://doi.org/10.1111/apa.17166

Aim
Preterm infants are at increased risk of Sudden Infant Death Syndrome (SIDS) and frequently experience short central apnoeas which can occur in isolation or a repetitive pattern (periodic breathing). We investigated the relationship between central apnoeas experienced before and over the 6 months after hospital discharge and cerebral oxygenation.

Methods
Preterm infants born between 28 and 32 weeks gestational age (GA) were studied during supine daytime sleep at 32–36 weeks post menstrual age (PMA) (n = 40), 36–40 weeks PMA (n = 27), 3-months corrected age (CA) (n = 20) and 6-months CA (n = 26). Cerebral tissue oxygenation (TOI), peripheral oxygenation (SpO2) and heart rate were recorded continuously. The percentage total sleep time (%TST) spent having central apnoeas at each study and cerebral fractional oxygen extraction (SpO2-TOI/SpO2) were calculated.

Results
%TST spent with central apnoeas decreased with increasing age in both active sleep (AS) and quiet sleep (QS). TOI tended to be lower and cerebral fractional oxygen extraction higher at 3 months compared to the other studies and this reached statistical significance compared to 32–36 weeks in QS.

Conclusion
The nadir in cerebral tissue oxygenation at 3 months of age coincides with the peak risk period for SIDS and this may contribute to increased risk in these infants. (Author)

2023-11268

Understanding the immune profile of sudden infant death syndrome – proteomic perspectives. Ferrante L, Opdal SH, Byard RW (2024), Acta Paediatrica vol 113, no 2, February 2024, pp 249-255
Full URL: https://doi.org/10.1111/apa.16988

Aim
The aim of this study was to investigate a panel of immune proteins in cases of sudden infant death syndrome (SIDS). It was hypothesised that, in at least a subset of SIDS, a dysregulated immune response may be a contributing factor leading to death.

Methods
The subjects included 46 SIDS cases and 41 controls autopsied at the Department of Forensic Sciences, Norway. The causes of death in the controls were accidents/trauma. Samples of cerebrospinal fluid (CSF) were analysed quantitatively by Proximity Extension Assay (PEA).

Results
Initial results revealed that normalised protein expression differed in 35 proteins. For the purposes of this report five proteins that are involved in immune system were selected for analysis: IFNLR1 ($p = 0.003$), IL10 ($p = 0.007$), IRAK4 ($p < 0.001$) and IL6 ($p = 0.035$); all had lower protein concentrations in SIDS cases compared to controls except for CD28 ($p = 0.024$) which had higher protein concentrations in SIDS cases.

Conclusion
The results confirm previous studies indicating that a dysregulation of the immune system may be a predisposing factor for SIDS. The results may indicate that these aberrant protein concentrations could lead to an inadequate response to immune triggers and uncontrolled defence mechanisms towards the common cold or other non-fatal infections. (Author)

2023-11137

Racial and Ethnic Disparities in Sudden Unexpected Infant Death Among US Infants Born Preterm. Hwang SS, Bourque SL, Hannan KE, et al (2023), The Journal of Pediatrics Vol 260, September 2023, 113498

Objective

To investigate among US infants born at <37 weeks gestation (a) racial and ethnic disparities in sudden unexpected infant death (SUID) and (b) state variation in SUID rates and non-Hispanic Black (NHB)-non-Hispanic White (NHW) SUID disparity ratio.

Methods

In this retrospective cohort analysis of linked birth and death certificates from 50 states from 2005 to 2014, SUID was defined by the following International Classification of Diseases, 9th or 10th edition, codes listed on death certificates: (7980, R95 or Recode 135; ASSB: E913, W75 or Recode 146; Unknown: 7999 R99 or Recode 134). Multivariable models were used to assess the independent association between maternal race and ethnicity and SUID, adjusting for several maternal and infant characteristics. The NHB-NHW SUID disparity ratios were calculated for each state.

Results

Among 4 086 504 preterm infants born during the study period, 8096 infants (0.2% or 2.0 per 1000 live births) experienced SUID. State variation in SUID ranged from the lowest rate of 0.82 per 1000 live births in Vermont to the highest rate of 3.87 per 1000 live births in Mississippi. Unadjusted SUID rates across racial and ethnic groups varied from 0.69 (Asian/Pacific Islander) to 3.51 (NHB) per 1000 live births. In the adjusted analysis, compared with NHW infants, NHB and Alaska Native/American Indian preterm infants had greater odds of SUID (aOR, 1.5;[95% CI, 1.42-1.59] and aOR, 1.44 [95% CI, 1.21-1.72]) with varying magnitude of SUID rates and NHB-NHW disparities across states.

Conclusions

Significant racial and ethnic disparities in SUID among preterm infants exist with variation across US states. Additional research to identify the drivers of these disparities within and across states is needed. (Author)

2023-09448

Midwives' experiences of safer infant sleep discussions at a southwest London hospital: a work-based learning project. Jolly L, Gregory J (2023), MIDIRS Midwifery Digest vol 33, no 3, September 2023, pp 278-284

Sudden infant death syndrome (SIDS) is defined as 'the sudden unexpected death of an infant under one year of age, with onset of the fatal episode apparently occurring during sleep, which remains unexplained after thorough investigation' (Willinger et al 1991). The aim of this work-based learning (WBL) project was to review how the maternity workforce at a southwest London trust undertakes Safer Infant Sleep Discussions (SISD). Women's and midwives' experiences of SISD were explored to identify barriers and facilitators, alongside a review of interventions to assist midwives with SISD. Analysis of the results enabled quality improvement and practice-based recommendations with a reflection on the learning process. (Author)

2023-06929

The San Diego SIDS definition—20 Years on. Byard RW, Tan L (2023), Acta Paediatrica vol 112, no 7, July 2023, pp 1389-1391

Aim

As it is now 20 years since the San Diego definition of sudden infant death syndrome (SIDS) was proposed, it is timely to examine the impact of this consensus statement.

Results

Concerns at the time were expressed that 'death scene' had been replaced by circumstances of death and so it may have been more useful to have a more inclusive statement of 'death scene, including circumstances of death'. The category of unclassified sudden infant deaths (USID) that was proposed has not been widely adopted. More disturbing, however, is the increasing failure to use either the San Diego or earlier definitions in published research,

with recent studies showing that almost two-thirds of peer-reviewed SIDS publications (2019–2021) did not quote or reference internationally accepted definitions. This is a decrease of 33% from the 68% of papers that correctly used SIDS definitions in 2011. The definition is therefore not being uniformly applied and in addition, diagnostic shift is occurring, with more pathologists favouring 'undetermined' over a designation of SIDS.

Conclusions

Given these developments, how can we correctly interpret conclusions relating to SIDS research, and can we accurately monitor trends in SIDS mortality? The authors would suggest that unfortunately, at present we cannot with any precision. (Author)

2023-04506

Polymorphisms of the hypothalamic–pituitary–adrenal axis may lead to an inadequate response to stress and contribute to sudden infant death syndrome. Uzuntas E, Schürmann P, Rothämel T, et al (2023), Acta Paediatrica vol 112, no 7, July 2023, pp 1478-1484

Full URL: https://doi.org/10.1111/apa.16772

Aim

Impaired resilience to stress may be a factor in sudden infant death syndrome (SIDS). However, no comprehensive studies have been performed on polymorphisms that are relevant to the hypothalamic–pituitary–adrenal (HPA) axis, which regulates the stress hormone cortisol.

Methods

We analysed 22 relevant single nucleotide polymorphisms (SNPs) in 206 anonymised SIDS cases who died at a mean of 131 days (range: 5–343) and 256 adult controls who were recruited from paternity testing cases. Additional stratified analyses were performed for sex, age and season of death. Both the cases and the controls were Caucasian.

Results

Variants for rs2235543 (HSD11B1) and rs3779250 (CRHR2) were associated with SIDS in the overall analysis, and borderline for rs2446432 (CRH), at least before corrections for multiple testing. A combination of these three variants was observed in 52.9% of SIDS cases but only 43.0% of controls ($p = 0.039$). Five or more variants showed an association in the subgroups.

Conclusion

Our findings suggest that the HPA axis influences SIDS and supports the hypothesis that an inadequate stress response may add to the risk. The associated variants for rs2235543, rs3779250 and rs2446432 appeared to decrease the cortisol concentration and impair an appropriate stress response. (Author)

2023-02859

Changing diagnostic patterns in cases of sudden and unexpected natural death in infants and young children: 1994–2018. Byard RW, Tan L (2023), Acta Paediatrica vol 112, no 6, June 2023, pp 1236-1239

Full URL: https://doi.org/10.1111/apa.16669

Aim

To determine whether there has been a change in the incidence and type of conditions causing sudden and unexpected natural death in infants and young children in recent years.

Methods

A search was undertaken of pathology records at Forensic Science SA in Adelaide, Australia for all cases of sudden and unexpected natural death in children aged less than 10 years at the time of death over two time periods: 1994–1998 and 2014–2018.

Results

Overall, 136 cases were identified consisting of 81 boys and 55 girls (M:F = 16:11; age range 0–9 years). No difference was shown in the numbers of sudden unexplained deaths in infants and young children between the two time periods (80 vs. 56; p = 0.18). A trend was shown for a prominent decrease in SIDS cases (55 vs. 12) with an increase in undetermined cases, <1 year (5 vs. 18). However, when the two categories were combined there was no statistical difference between the two periods (60/80 vs. 30/56) (p = 0.26), although a decrease in numbers had occurred.

Conclusion

Analysis of numbers of fatalities reported from medicolegal institutes should be undertaken with an awareness of the potential effect of diagnostic shift. (Author)

2023-01966

Sudden unexpected death in infancy: current practices in virological investigations and documentation in the French registry. Martin-Perceval L, Scherdel P, Jarry B, et al (2023), The Journal of Pediatrics vol 257, June 2023, 113324

Objective

To describe pre-COVID-19 pandemic current practices in virological investigations, including type, frequency of samplings, and documented viruses, in sudden unexpected death in infancy (SUDI) and to compare results according to the cause of death.

Study design

Between May 2015 and December 2019, infants under 2 years of age included in the French SUDI registry were classified in one of four groups by causes of death according to the Goldstein et al. classification: unexplained (SIDS), infectious, explained but noninfectious, and undetermined. Sampling sites and viruses detected were described, then SIDS and explained deaths (control group) were compared.

Results

Among 639 infants, 3.6% died from an established viral infection. From 23 sampling sites and 2,238 samples, 19 virus species were detected. Overall, 43.3% of infants carried a virus, with no significant difference between SIDS infants and the control group (p=0.06). We found wide variations in frequencies of samples by site (550 for nasopharynx to one for saliva). The highest positivity rate was from the nasopharynx (195/2,238; 8.7%). Rhinovirus was the predominant virus detected (135/504, 26.8%), mostly in SIDS (83/254, 32.7%). We found no significant difference between positivity rates and distribution of viruses between the SIDS and control groups. At-autopsy virological analysis never contributed to determining the cause of death.

Conclusion

Current practices in virological investigations in SUDI are heterogeneous, with wide variability despite published guidelines. Investigations should be limited to the most relevant sites, and systematic at-autopsy sampling should be reconsidered. We found no association between virus detection and SIDS. (Author)

2023-01801

Protecting Infants from Sudden Unexpected Infant Death: Guidelines for Interventions during the Perinatal Period from the French National College of Midwives. Weiss S (2022), Journal of Midwifery & Women's Health vol 67, suppl 1, December 2022, pp 83-92

Full URL: https://doi.org/10.1111/jmwh.13430

With 300–400 annual deaths in France, sudden unexpected infant death (SUID) is the leading cause of mortality in France among infants from the end of their first through their 12th month of life. These clinical practice guidelines aim to identify strategies for (future) parents to prevent avoidable SUIDs. They are based on a narrative literature review and an analysis of the existing reports and guidelines available on the topic in 2019–2020. In summary, it is recommended that parents ensure that their infants sleep on their back on a firm, empty surface in a sufficiently ventilated environment, share the parental bedroom, and be breastfed and vaccinated. All of these actions create protective factors against SUID. Conversely, parents should know that several factors increase the risk of SUID: unsafe sleep, maternal smoking, passive smoking after birth, exposure to alcohol or other psychoactive substances, and

excessive heat. (Author)

2023-00742

Epidemiology of sudden infant death syndrome in Mexico, 2005–2020. Martinez-Valdez L, Richardson V, Bautista-Marquez A, et al (2022), 08 December 2022, Online

Full URL: https://doi.org/10.3389/fped.2022.1001089

Background: Sudden Infant Death Syndrome (SIDS) constitutes one of the main causes of mortality in children under one year of age in developed countries; it's frequency to varies geographically. In Mexico the real incidence of SIDS is not known.

Methods: National databases of deaths in children under one year of age, from 2005 to 2020, were analyzed, due to Sudden Unexpected Infant Death (SUID) [SIDS (R95), accidental suffocation in a sleeping environment (W75), and other ill-defined and unspecified causes of mortality (R99), according to the International Classification of Diseases, tenth revision (ICD 10)]. Mortality rates per year of occurrence due to SUID and their subcategories were calculated. Simple frequencies of SIDS were obtained per year and month of occurrence, state of residence, age, place of death, and access to social security services.

Results: In the study period 473,545 infant deaths occurred; 7,714 (1.62%) deaths were due to SUID; of these, 6,489 (84%) were due to SIDS, which is among the 10 leading causes of infant death in Mexico. The average mortality rate for SUID was 22.4/100,000 live births, for SIDS was 18.8/100,000 live births. Mortality rates within the states were variable, ranging from 2.4/100,000 to 105.1/100,000 live births. In 81% of SIDS records there was no autopsy; 38% of deaths due to SIDS occurred in infants under one month of age, up to 87% of deaths occurred in families without social security services or it was unknown, and 76.2% of deaths occurred at home. Deaths were more frequent during the last months of autumn and during winter.

Conclusion: In Mexico there is an underregistry of SIDS as cause of death, along with other SUID categories. Health workers need to be trained to improve diagnosis and data registration, including the practice of autopsies; additionally, it is necessary to implement a public health campaign. (Author)

2023-00453

Sudden unexpected infant death risk profiles in the first month of life. Hegyi T, Ostfeld BM (2022), Journal of Maternal-Fetal and Neonatal Medicine 04 October 2022, Online

Full URL: https://doi.org/10.1080/14767058.2022.2128662

Background

Limited improvement in current SUID rates requires further identification of its characteristics, including age-specific risk patterns.

Objective

Compare SUID risk factors in the first week versus the remainder in the first month of life.

Design/methods

We compared maternal and infant data from New Jersey databases for SUID from 2000 to 2015 in infants ≥ 34 weeks GA in the two groups.

Results

In the period studied, 123 died in the first 27 days, 24 before seven. Deaths in the first week had a higher percentage of mothers with post-High School education (OR 3.50, CI: 1.38–8.87) and a primary Cesarean section delivery (OR 4.0, CI: 1.39–11.49), and a smaller percentage with inadequate prenatal care (OR 0.36, CI: 0.14, 0.94). A smaller percentage of first-week deaths had mothers who smoked during pregnancy or identified as Black, non-Hispanic, but these

findings did not reach significance ($p < .08$ and $p < .09$, respectively).

Conclusions

SUID in the first week and the first month of life is rare. However, despite a limited sample size, data suggest that even within the first month of life, there are differences in risk patterns for SUID based on age at death. Age-specific profiles may lead to new hypotheses regarding causality and more refined risk-reduction guidelines and warrant further study. (Author)

2022-11025

Sudden and unexpected deaths in infancy and childhood. National Child Mortality Database Programme thematic report. Williams T, Sleap V, Pease A, et al for the National Child Mortality Database (NCMD) Programme (2022), December 2022. 64 pages

Full URL: https://www.hqip.org.uk/wp-content/uploads/2022/12/Ref-321-SUDIC-Thematic-report_FINAL.pdf

This report covers the two-year period from 2019 to 2021 and is unique in two ways. It is the first national report to have investigated all unexpected deaths of infants and children – not just those that remained unexplained. It is also the first national review of the "multi-agency investigation process" into unexpected deaths.

The report found that, of all infant and child deaths occurring between April 2019 and March 2021 in England, 30% occurred suddenly and unexpectedly, and of these 64% had no immediately apparent cause.

Other key findings relating to sudden and unexpected infant deaths (under 1 year) include:

- 70% were aged between 28 and 364 days, and 57% were male
- Infant death rates were higher in urban areas and the most deprived neighbourhoods
- For sudden and unexpected infant deaths that occurred during 2020 and had been fully reviewed, 52% were classified as unexplained (ie Sudden Infant Death Syndrome) and 48% went on to be explained by other causes eg metabolic or cardiac conditions.

(Publisher)

2022-10915

Mitochondrial DNA content: a new potential biomarker for Sudden Infant Death Syndrome. Danusso R, Alfonsi G, Ferrero S, et al (2022), Pediatric Research vol 92, no 5, November 2022, pp 1282-1287

Background

Sudden Infant Death Syndrome (SIDS) occurs in apparently healthy infants and is unpredictable and unexplained despite thorough investigations and enormous research efforts. The hypothesis tested in this case–control study concerns mitochondrial involvement in SIDS occurrence.

Methods

Mitochondrial DNA content (MtDNAcn) was measured in 24 SIDS cerebral cortex samples and 18 controls using real-time PCR.

Results

The median (interquartile range) mtDNAcn in SIDS and controls was 2578 (2224–3838) and 1452 (724–2517) copies per nuclear DNA, respectively ($P = 0.0001$). MtDNAcn values were higher in SIDS victims born to non-smoking parents ($n = 7$) 4984 (2832–6908) compared to the controls ($n = 5$) 2020 (478–2386) ($P = 0.006$). Increased levels of mtDNAcn have been observed in the SIDS cases with mild defects in nuclei not essential for life compared to those found in SIDS cases with severe alterations of respiratory function ($P = 0.034$) 3571 (2568–5053) ($n = 14$) 2356 (1909–3132) ($n = 8$), respectively.

Conclusions

Our study revealed for the first time higher mtDNAcn in the cerebral cortex of the SIDS cases than the controls, indicating metabolic alterations. MtDNAcn plays an important role in compensatory mechanisms against environmental factors affecting human health. Despite the small sample size, mtDNA may prove to be a potential forensic biomarker for autopsied SIDS victims for gaining new insights into the etiology of SIDS.

Impact
Mitochondrial DNA content evaluated in cerebral cortex samples is higher in SIDS victims than controls.

These results represent a novel line of investigation for the etiology of SIDS and could have a significant role in the compensatory mechanism due to environmental factors affecting human health.

These findings suggest that the mitochondria are involved in SIDS: mtDNA content may represent a biomarker of this syndrome. (Author)

2022-10897

Genetic variants in eleven central and peripheral chemoreceptor genes in sudden infant death syndrome. Neubauer J, Forst AL, Warth R, et al (2022), Pediatric Research vol 92, no 4, October 2022, pp 1026-1033
Full URL: https://doi.org/10.1038/s41390-021-01899-4

Background
Sudden infant death syndrome (SIDS) is still one of the leading causes of postnatal infant death in developed countries. The occurrence of SIDS is described by a multifactorial etiology that involves the respiratory control system including chemoreception. It is still unclear whether genetic variants in genes involved in respiratory chemoreception might play a role in SIDS.

Methods
The exome data of 155 SIDS cases were screened for variants within 11 genes described in chemoreception. Pathogenicity of variants was assigned based on the assessment of variant types and in silico protein predictions according to the current recommendations of the American College of Medical Genetics and Genomics.

Results
Potential pathogenic variants in genes encoding proteins involved in respiratory chemoreception could be identified in 5 (3%) SIDS cases. Two of the variants (R137S/A188S) were found in the KNCJ16 gene, which encodes for the potassium channel Kir5.1, presumably involved in central chemoreception. Electrophysiologic analysis of these KCNJ16 variants revealed a loss-of-function for the R137S variant but no obvious impairment for the A188S variant.

Conclusions
Genetic variants in genes involved in respiratory chemoreception may be a risk factor in a fraction of SIDS cases and may thereby contribute to the multifactorial etiology of SIDS.

Impact
What is the key message of your article?

Gene variants encoding proteins involved in respiratory chemoreception may play a role in a minority of SIDS cases.

What does it add to the existing literature?

Although impaired respiratory chemoreception has been suggested as an important risk factor for SIDS, genetic variants in single genes seem to play a minor role.

What is the impact?

This study supports previous findings, which indicate that genetic variants in single genes involved in respiratory control do not have a dominant role in SIDS. (Author)

2022-10879

Sudden infant death syndrome revisited: serotonin transporter gene, polymorphisms and promoter methylation.
Pfisterer N, Meyer-Bockenkamp F, Qu D, et al (2022), Pediatric Research vol 92, no 3, September 2022, pp 694-699
Full URL: https://doi.org/10.1038/s41390-021-01773-3

Background
Based on findings in the brain stems of SIDS victims, the serotonin transporter (5-HTT) gene has been discussed to be associated with SIDS.

Methods
In the largest study to date, we investigated the promoter length (5-HTTLPR) and intron 2 VNTR polymorphisms in 274 cases and 264 controls and the Ile425Val polymorphism in 65 cases and 64 controls. Moreover, the methylation of the internal promoter region was investigated in 35 cases and 14 controls.

Results
For 5-HTTLPR, we observed a trend towards an association of allele L (58.8% vs. 53.4%) with SIDS and significant results were observed after stratifying for age, season at death, and prone position. Nevertheless, when pooling all published data, a significant association of allele L with SIDS is confirmed (p: 0.001). For the intron 2 VNTR polymorphism, no significant differences were observed. After pooling, a significant accumulation of the rare allele 9 was observed in SIDS (2.1% vs. 0.6%; p: 0.018). For the Ile425Val polymorphism, no differences were observed.

Conclusion
We conclude that genetic variation at this gene might be of some importance in SIDS. Epigenetic analysis of the internal promoter, however, revealed no influence on the relative risk to succumb to SIDS.

Impact
This is the largest study published up to now on 5-HTT gene polymorphisms and SIDS.

Polymorphisms in the 5-HTT gene appear to contribute (although to a small degree) to the risk to die from SIDS.

There is no evidence that a methylation of the promoter region is of impact for the etiology of SIDS. (Author)

2022-10664

Infant Mortality [written answer]. House of Commons (2022), Hansard Written question 85056, 11 November 2022
Full URL: https://questions-statements.parliament.uk/written-questions/detail/2022-11-11/85056

Maria Caulfield responds to a written question from Carla Lockhart to the Secretary of State for Health and Social Care, regarding how many sudden infant deaths occurred in the UK in each of the last three years. (JSM)

2022-09963

Pacifiers and the reduced risk of sudden infant death syndrome. Smith RW, Colpitts M (2020), Paediatrics & Child Health vol 25, no 4, June 2020, pp 205-206

Extract
For the current issue of the Journal, we asked Dr. Ryan Smith and his collegue Dr. Melanie Colpitts to comment on and put into context the recent Cochrane Review on the use of infant pacifiers for reduction in risk of sudden infant death syndrome (SIDS).

BACKGROUND
Sudden infant death syndrome (SIDS) has been most recently defined as the sudden unexpected death of an infant less than 1 year of age, with onset of the fatal episode apparently occurring during sleep that remains unexplained after a thorough investigation, including the performance of a complete autopsy and a review of the circumstances of death and clinical history. Despite the success of several prevention campaigns, SIDS remains a leading cause of infant mortality. In 1994, a 'triple risk model' for SIDS was proposed that described SIDS as an event that results from the intersection of three factors: a vulnerable infant; a critical development period in homeostatic control (age related); and an exogenous stressor. The association between pacifier (dummy) use and reduced incidence of SIDS has been shown in demi logical studies since the early 1990s. Pacifier use, given its low cost, might be a cost-effective intervention for SIDS prevention if it is confirmed effective in randomized controlled trials. (Author)

2022-08543

Nottingham maternity review to examine 10 years of baby cases. Cowley V, Lowbridge C (2022), BBC News 13 September 2022

Full URL: https://www.bbc.co.uk/news/uk-england-nottinghamshire-62888582?at_medium=RSS&at_campaign=KARANGA

A review examining failings by Nottingham's maternity services will consider cases going back at least 10 years, it has been announced. (Author)

2022-06306

Sudden Unexpected Death in Infancy [SUDI]: What the clinician, pathologist, coroner and researchers want to know. Fitzgerald DA, Jeffery H, Arbuckle S, et al (2022), Paediatric Respiratory Reviews vol 41, March 2022, pp 14-20

The loss of an apparently healthy infant is confronting for any family, puzzling for a clinician and challenging for the pathologist charged with the task of demonstrating a cause for death. The term "cot death" evolved to "sudden infant death syndrome" [SIDS] and now "sudden unexpected death in infancy [SUDI]" as the epidemiology and pathology of infant death changed. Community interventions were successful in changing sleep practices for young babies. The current research focus is on understanding genetic predispositions to unexpected death in early childhood. Whilst much has been achieved in reducing the infant mortality rate from SUDI by between 50%, and 80% in some countries, over the last 30 years, there remain challenges for improving rates of accurate diagnosis and reaching out to more vulnerable families with clearly modifiable risk factors for SUDI. These challenges directly involve the clinician through taking a systematic and detailed history and better standardised death scene evaluations with specifically accredited assessors. Better knowledge regarding circumstances of SUDI cases will help Coroners and researchers provide answers for grieving families now, and in the future contribute to further reductions in the rate of SUDI in communities across the world. (Author)

2022-04425

Only Halfway There with Sudden Infant Death Syndrome. Goldstein RD, Kinney HC, Guttmacher AE (2022), The New England Journal of Medicine vol 386, no 20, 19 May 2022, pp 1873-1875

Medicine promotes a logically inconsistent message regarding SIDS: authoritative statements imply that SIDS is a consequence of unsafe sleep in biologically normal children, but there is vague acknowledgment that important biologic factors are involved in these deaths. (Author)

2022-03989

Butyrylcholinesterase is a potential biomarker for Sudden Infant Death Syndrome. Harrington CT, Al Hafid N, Waters KA (2022), EBioMedicine vol 80, June 2022, 104041

Full URL: https://doi.org/10.1016/j.ebiom.2022.104041

Background
Autonomic dysfunction has been implicated in the pathophysiology of the Sudden Infant Death Syndrome (SIDS). Butyrylcholinesterase (BChE) is an enzyme of the cholinergic system, a major branch of the autonomic system, and

may provide a measure of autonomic (dys)function. This study was undertaken to evaluate BChE activity in infants and young children who had died from Sudden Infant Death or Sudden Unexpected Death.

Methods
In this case-control study we measured BChE activity and total protein in the eluate of 5µL spots punched from the dried blood spots taken at birth as part of the newborn screening program. Results for each of 67 sudden unexpected deaths classified by the coroner (aged 1 week-104 weeks) = Cases, were compared to 10 date of birth - and gender-matched surviving controls (Controls), with five cases reclassified to meet criteria for SIDS, including the criterion of age 3 weeks to 1 year.

Findings
Conditional logistic regression showed that in groups where cases were reported as “SIDS death” there was strong evidence that lower BChE specific activity (BChEsa) was associated with death (OR=0·73 per U/mg, 95% CI 0·60-0·89, P=0·0014), whereas in groups with a “Non-SIDS death” as the case there was no evidence of a linear association between BChEsa and death (OR=1·001 per U/mg, 95% CI 0·89-1·13, P=0·99).

Interpretation
BChEsa, measured in dried blood spots taken 2-3 days after birth, was lower in babies who subsequently died of SIDS compared to surviving controls and other Non-SIDS deaths. We conclude that a previously unidentified cholinergic deficit, identifiable by abnormal -BChEsa, is present at birth in SIDS babies and represents a measurable, specific vulnerability prior to their death.

Funding
All funding provided by a crowd funding campaign https://www.mycause.com.au/p/184401/damiens-legacy. (Author)

2022-02182

Is there a common denominator for Brief Resolved Unexplained Events, Sudden Infant Death Syndrome, and alleged Shaken Baby Syndrome?. Lynøe N, Eriksson A (2020), Medical Hypotheses vol 144, November 2020, 109939
Full URL: https://doi.org/10.1016/j.mehy.2020.109939

We propose the medical hypothesis that a common denominator may be the precursor for Brief Resolved Unexplained Events (BRUE), cases of Sudden Infant Death Syndrome (SIDS) as well as to cases of alleged Shaken Baby Syndrome (SBS) without external signs of trauma. Although previous studies have emphasized differences, we have focused on the overarching common denominators of the three conditions in terms of mechanism theories.

In consequence, fatal cases with subdural hemorrhage (SDH) classified as SBS could be classified as high risk BRUE with SDH. Fatal cases without SDH could be classified as SIDS. Non-fatal cases with SDH and retinal hemorrhages (RHs), currently classified as SBS, could be classified as BRUE with SDH and RHs, leaving a fourth group of BRUE without SDH and RHs.

While both the BRUE and the SIDS diagnoses have been refined and developed, alleged abusive head trauma (AHT) cases with and without external signs of trauma have been indiscriminately combined. This is analogous to indiscriminately grouping together, e.g., headache due to a brain tumor or headache after head trauma. Alleged AHT cases with external signs of trauma and high velocity impact might be explained by the traditional AHT mechanism theories, whereas the one-third of all alleged AHT cases without external signs of trauma could be explained by the hypoxia cascade theory and/or other non-shaking theories. (Author)

2022-02157

How might non nutritional sucking protect from sudden infant death syndrome. Abed BZ, Oneto S, Abreu AR (2020), Medical Hypotheses vol 143, October 2020, 109868

Epidemiology has identified an association between the use of pacifiers and protection from sudden infant death syndrome (SIDS). The use of pacifiers for SIDS prevention fails to gain adoption partly because there is no widely accepted physiologic mechanism to explain the epidemiologic association. Additionally, the scientific literature available on pacifier use focuses largely on the probable adverse effects. We hypothesize that pacifier use and all other forms of non-nutritional sucking (specifically digit sucking, also known as thumb sucking) is a life saving defense mechanism meant to splint open and stabilize the collapsible portion of the upper airway in infants. The main objective of this review article is to propose a mechanism to explain how pacifiers might help prevent SIDS. If the medical community accepts this mechanism, it can help promote pacifier use by the public and potentially reduce the incidence of SIDS. (Author)

2022-02138

A possible cause of Sudden Infant Death Syndrome. Blix AS (2019), Medical Hypotheses vol 136, March 2020, 109520

It is suggested that an orienting response to loud sound causes apnea, which, in already asphyxic infants, triggers a maximal secondary chemoreceptor response, with massive vagal stimulation of the heart, which causes heart arrest. (Author)

2022-00404

State-level trends in sudden unexpected infant death and immunization in the United States: an ecological study.

Müller-Nordhorn J, Neumann K, Keil T, et al (2021), BMC Pediatrics vol 21, no 274, 11 June 2021

Full URL: https://doi.org/10.1186/s12887-021-02733-w

Background

Sudden unexpected infant death (SUID) continues to be a major contributor to infant mortality in the United States. The objective was to analyze time trends in SUID and their association with immunization coverage.

Methods

The number of deaths and live births per year and per state (1992–2015) was obtained from the Centers for Disease Control and Prevention (CDC). We calculated infant mortality rates (i.e., deaths below one year of age) per 1000 live births for SUID. We obtained data on immunization in children aged 19–35 months with three doses or more of diphtheria-tetanus-pertussis (3+ DTP), polio (3+ Polio), and Haemophilus influenzae type b (3+ Hib) as well as four doses or more of DTP (4+ DTP) from the National Immunization Survey, and data on infant sleep position from the Pregnancy Risk Assessment Monitoring System (PRAMS) Study. Data on poverty and race were derived from the Current Population and American Community Surveys of the U.S. Census Bureau. We calculated mean SUID mortality rates with 95% confidence interval (CI) as well as the annual percentage change using breakpoint analysis. We used Poisson regression with random effects to examine the dependence of SUID rates on immunization coverage, adjusting for sleep position and poverty (1996–2015). In a second model, we additionally adjusted for race (2000–2015).

Results

Overall, SUID mortality decreased in the United States. The mean annual percent change was − 9.6 (95% CI = − 10.5, − 8.6) between 1992 and 1996, and − 0.3 (95% CI = − 0.4, − 0.1) from 1996 onwards. The adjusted rate ratios for SUID mortality were 0.91 (95% CI = 0.80, 1.03) per 10% increase for 3+ DTP, 0.88 (95% CI = 0.83, 0.95) for 4+ DTP, 1.00 (95% CI = 0.90, 1.10) for 3+ polio, and 0.95 (95% CI = 0.89, 1.02) for 3+ Hib. After additionally adjusting for race, the rate ratios were 0.76 (95% CI = 0.67, 0.85) for 3+ DTP, 0.83 (95% CI = 0.78, 0.89) for 4+ DTP, 0.81 (95% CI = 0.73, 0.90) for 3+ polio, and 0.94 (95% CI = 0.88, 1.00) for 3+ Hib.

Conclusions

SUID mortality is decreasing, and inversely related to immunization coverage. However, since 1996, the decline has slowed down. (Author)

2021-13037

Association between auditory system pathology and sudden infant death syndrome (SIDS): a systematic review. Dahl K, Andersen M, Henriksen TB, et al (2021), BMJ Open vol 11, no 12, December 2021, e055318

Full URL: http://dx.doi.org/10.1136/bmjopen-2021-055318

Objective A theory has emerged, suggesting that abnormalities in the auditory system may be associated with sudden infant death syndrome (SIDS). However, current clinical evidence has never been systematically reviewed.

Design A systematic review was conducted according to the guideline of Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

Data sources PubMed, Embase and Web of Science were systematically searched through 7 September 2020.

Eligibility criteria for selecting studies Only human studies with a reference group were included. Studies were eligible for inclusion if they examined infants exposed to otoacoustic emissions (OAEs), auditory brainstem response (ABR) or had autopsies with brainstem histology of the auditory system. SIDS was the primary outcome, while the secondary outcome was near-miss sudden infant death syndrome episodes.

Data extraction and synthesis Two independent reviewers extracted data and assessed risk of bias, and the quality of evidence. Due to high heterogeneity, a narrative synthesis was conducted. Risk of bias and quality of evidence was assessed using the Newcastle-Ottawa Scale and Grading of Recommendations Assessment, Development and Evaluation.

Results Twelve case–control studies were included. Seven studies on OAEs or ABR had a high degree of inconsistency. Contrarily, four out of five studies reporting on brainstem histology found that auditory brainstem abnormalities were more prevalent in SIDS cases than in controls. However, the quality of evidence across all studies was very low.

Conclusion This systematic review found no clear association between auditory system pathology and SIDS. The higher prevalence of histological abnormalities in the auditory system of SIDS may indicate an association. However, further studies of higher quality and larger study populations are needed to determine whether these findings are valid.

PROSPERO registration number CRD42020208045. (Author)

2021-12345

Sudden infant death syndrome prevention. Jullien S (2021), BMC Pediatrics vol 21, no 320, 8 September 2021

Full URL: https://doi.org/10.1186/s12887-021-02536-z

We looked at existing recommendations and supporting evidence for successful strategies to prevent the sudden infant death syndrome (SIDS).

We conducted a literature search up to the 14th of December 2020 by using key terms and manual search in selected sources. We summarized the recommendations and the strength of the recommendation when and as reported by the authors. We summarized the main findings of systematic reviews with the certainty of the evidence as reported.

Current evidence supports statistical associations between risk factors and SIDS, but there is globally limited evidence by controlled studies assessing the effect of the social promotion strategies to prevent SIDS through knowledge, attitude and practices, due to obvious ethical reasons. A dramatic decline in SIDS incidence has been observed in many countries after the introduction of “Back to Sleep” campaigns for prevention of SIDS. All infants should be placed to sleep in a safe environment including supine position, a firm surface, no soft objects and loose bedding, no head covering, no overheating, and room-sharing without bed-sharing. Breastfeeding on demand and the use of pacifier during sleep time protect against SIDS and should be recommended. Parents should be advised against the use of tobacco, alcohol and illicit drugs during gestation and after birth. (Author)

2021-09253

Sudden Unexplained Infant Deaths - New Study Findings Related to Day of Life. Beal JA (2020), MCN - American Journal of Maternal/Child Nursing vol 45, no 3, May/June 2020, p 185

New data on classification and risk factors for sudden unexplained infant deaths based on age of infants at death have been published. Our pediatric nursing expert, Dr. Beal, explains the study findings and how they can help nurses promote safe infant sleep. (Author)

2021-06981

Sleep-related risk and worrying behaviours: a retrospective review of a tertiary centre's experience. Vigo A, Noce S, Costagliola G, et al (2019), European Journal of Pediatrics vol 178, no 12, pp 1841-1847

This retrospective study aims at helping physicians select babies considered at risk for fatal events during sleep. It does so by describing the clinical features and outcome of worrying infants' behaviour during sleep, with the activation of an emergency medical service and/or emergency department, subsequently referred to the Centre for Paediatric Sleep Medicine and sudden infant death syndrome, Regina Margherita Children's Hospital, Turin, Italy. We analysed the medical records of infants < 12 months whose parents reported they had worrying behaviour during sleep in the period 1 January 2009– 31 December 2015. Regional guidelines suggest performing anamnesis and capillary blood gas analysis in case of apparent life-threatening events. There were 33 males, average age 55 ± 54.37 days. On arrival at the emergency medical service/emergency department 97 % infants were asymptomatic; 61 % patients had a capillary blood gas analysis as suggested by the regional guidelines. A clear acid-base disorder was observed in two infants, asymptomatic at medical evaluation, that had assumed an unsafe sleeping position. Two patients presented recurrence of the episode at 3 months.

Conclusions: Most worrying infant behaviour during sleep can be related to paraphysiological phenomena; capillary blood gas analysis and anamnesis are pivotal to identify the cases at risk of fatal events. (Author)

2021-03266

Sudden Unexpected Postnatal Collapse Resulting in Newborn Death in the United States. Anderson TM, Ferres JML, Ramirez JM, et al (2021), MCN - American Journal of Maternal/Child Nursing vol 46, no 3, May/June 2021, pp 130-136

Full URL: https://doi.org/10.1097/NMC.0000000000000711

Background:

The sudden collapse of an apparently healthy newborn, or sudden unexpected postnatal collapse (SUPC) is fatal in about half of cases. Epidemiological characteristics of sudden unexpected infant death (SUID) in the first week of life differ from those in the postperinatal age group (7-365 days).

Aim:

To describe the characteristics of SUPC resulting in neonatal death.

Methods:

We analyzed the Centers for Disease Control and Prevention Birth Cohort Linked Birth/Infant Death Data Set (2003-2013: 41,125,233 births and 37,624 SUIDs). SUPC was defined as infants born ≥35 weeks gestational age, with a 5-minute Apgar score of ≥7, who died suddenly and unexpectedly in the first week of life.

Results:

Of the 37,624 deaths categorized as SUID during the study period, 616 met the SUPC criteria (1.5/100,000 live births). Eleven percent occurred on the first day of life and nearly three quarters occurred during postnatal days 3-6. SUPC deaths differed statistically from SUID deaths occurring 7-364 days of age, in particular for sex, marital status, and live birth order.

Implications:
These data support the need for adequate nurse staffing during the immediate recovery period and for the entire postpartum stay as well as nurse rounding for new mothers in the hospital setting. (Author) = [Erratum: MCN - American Journal of Maternal/Child Nursing, vol 46, no 3, May/June 2021, p 236].

20201221-36*

No association to sudden infant death syndrome detected by targeted amplicon sequencing of 24 genes. Ferrante L, Opdal SH, Nygaard V (2020), Acta Paediatrica vol 109, no 12, December 2020

Aim
The aim was to identify genetic variants associated with sudden infant death syndrome (SIDS) that can cause disease or introduce vulnerability. Genes reported in a previous SIDS study to have altered messenger ribonucleic acid (mRNA) expression in SIDS were investigated.
Methods
Samples from 81 SIDS (56 male/28 female) with a median age of 4 months (range 0.75-9 months) were analysed using Illumina TruSeq custom amplicon for 24 selected genes. Samples were collected from autopsy at Oslo university hospital from children whom died suddenly and unexpectedly from 1988 to 2006. The controls were the germline variation database, Norgene (no description of cases available).
Results
After filtering for rare variants, there were a total of 38 variants in the 81 SIDS cases and 462 variants in the 789 controls. After the filtration and curation steps, we found 36 rare variants. The overall occurrence of rare variants for all the SIDS samples was lower than for the Norgene population.
Conclusion
There was no association between rare variants in the included genes and SIDS. Although not statistically significant, two of the SIDS cases had a rare variant in the MyD88 gene: rs746651350, rs200424253. (Author)

20200604-89*

Factors associated with age of death in sudden unexpected infant death. Allen K, Anderson TM, Chajewska U, et al (2021), Acta Paediatrica vol 110, no 1, January 2021, pp 174-183

Aim
This study aimed to systematically analyse the pregnancy, birth and demographic-related factors associated with age of death in sudden unexpected infant death (SUID).
Methods
Data were analysed from the Centers for Disease Control and Prevention's Cohort Linked Birth/Infant Death data set (2011-2013; 11 737 930 live births). SUID was defined as deaths from sudden infant death syndrome, ill-defined causes, or accidental suffocation and strangulation in bed. There were 9668 SUID cases (7-364 days; gestation >28 weeks; 0.82/1000 live births). The odds of death at different ages were compared to determine which variables significantly affect the SUID age of death.
Results
Forty-three features indicated a significant change in age of death with two main patterns: (a) younger chronologic age at death was associated with maternal smoking and factors associated with lower socio-economic status, and (b) older age was associated with low birthweight, prematurity and admission to the neonatal intensive care unit. However, when age was corrected for gestation, these factors were associated with younger age.
Conclusion
Factors that varied with age of death are well-documented risk factors for SUID. The majority of these risk factors were associated with younger age at death after allowing for gestational age at birth. (Author)

20200417-51*

Prevalence of risk factors for sudden infant death among Indigenous and non-Indigenous people in Australia.
Shipstone RA, Young J, Kearney L, et al (2020), Acta Paediatrica vol 109, no 12, December 2020, pp 2614-2626

Aim
To examine differences in the prevalence of risk factors for sudden unexpected death in infancy (SUDI) between Aboriginal and Torres Strait Islander and non-Indigenous infants.
Methods
A retrospective cohort study of SUDI in Queensland during 2010-2014 examined exposure to SUDI risk factors, to identify factors accounting for higher SUDI mortality among Indigenous infants. A multistage algorithm was applied to linked data to determine Indigenous status.
Results
There were 228 SUDI, of which Indigenous infants comprised 26.8%. The Indigenous SUDI rate was 2.13/1000 live births compared to 0.72/1000 for non-Indigenous. The disparity between Indigenous and non-Indigenous SUDI was accounted for by surface sharing (OR = 2.93 95% CI = 1.41, 6.07), smoking (OR = 2.49, 95% CI = 1.13, 5.52), and a combination of background antenatal and sociodemographic factors (inadequate antenatal care [OR = 6.93, 95% CI = 2.20, 21.86], young maternal age at first birth [OR = 4.02, 95% CI = 1.49, 10.80] and outer regional [OR = 3.03, 95% CI = 1.37, 6.72] and remote locations [OR = 11.31, 95% CI = 3.47, 36.83]).
Conclusion
Culturally responsive prevention efforts, including wrap-around maternity care and strategies that reduce maternal smoking and promote safer yet culturally acceptable ways of surface sharing, may reduce Indigenous SUDI mortality. (Author)

20200113-7*

Distinct Populations of Sudden Unexpected Infant Death Based on Age. Ferres JML, Anderson TM, Johnston R, et al (2020), Pediatrics vol 145, no 1, January 2020, e20191637
Full URL: https://doi.org/10.1542/peds.2019-1637

OBJECTIVES: In most recent studies, authors combine all cases of sudden infant death syndrome, other deaths from ill-defined or unknown causes, and accidental suffocation and strangulation in bed as a single population to analyze sudden unexpected infant death (SUID). Our aim with this study is to determine if there are statistically different subcategories of SUID that are based on the age of death of an infant.
METHODS: In this retrospective, cross-sectional analysis, we analyzed the Centers for Disease Control and Prevention Birth Cohort Linked Birth/Infant Death Data Set (2003-2013: 41 125 233 births and 37 624 SUIDs). Logistic regression models were developed to identify subpopulations of SUID cases by age of death, and we subsequently analyzed the effects of a set of covariates on each group.
RESULTS: Two groups were identified: sudden unexpected early neonatal deaths (SUENDs; days 0-6) and postperinatal SUIDs (days 7-364). These groups significantly differed in the distributions of assigned International Classification of Diseases, 10th Revision code, live birth order, marital status, age of mother, birth weight, and gestational length compared to postperinatal SUIDs (days 7-364). Maternal smoking during pregnancy was not a significant risk factor for deaths that occurred in the first 48 hours.
CONCLUSIONS: SUEND should be considered as a discrete entity from postperinatal SUID in future studies. These data could help improve the epidemiological understanding of SUEND and SUID and provide clues to a mechanistic understanding underlying the causes of death. (Author)

20190725-63*

Sudden infant death syndrome (SIDS) and the routine otoacoustic emission infant hearing screening test: an epidemiological retrospective case-control study. Blair PS, Rubens D, Pease A, et al (2019), BMJ Open vol 9, no 7, July 2019, e030026

Objectives To investigate whether decreased otoacoustic emission (OAE) signal recordings in the right ear are associated with an increased risk of sudden infant death syndrome (SIDS) and to monitor any temporal changes in risk factors.
Design Retrospective case-control study.
Setting Telephone interviews with families recruited in England between July 2016 and October 2017 who

experienced the unexpected death of a child <4 years old since 2008 and control families recruited from maternity wards in Bristol and Birmingham.
Participants We recruited 91 (89%) of the 102 bereaved families who made initial contact, 64 deaths were under 1 year (sudden unexpected death in infancy) of which 60 remained unexplained (SIDS). Of the 220 control families, 194 (88%) follow-up interviews were conducted. We had analysable hearing data for 24 SIDS infants (40%) and 98 controls (51%).
Results OAE signals were marginally increased rather than decreased among SIDS infants for the right ear, especially at lower frequencies, but not significantly so. The strongest predictors of SIDS were bed-sharing in hazardous (infant sleeping next to a carer who smoked, drank alcohol or slept on a sofa) circumstances (35% vs 3% controls, p<0.0001), infants found prone (33% vs 3% controls, p<0.0001) and infants whose health in the final week was 'not good' (53% vs 9% controls, p<0.0001). The prevalence of maternal smoking during pregnancy among both SIDS mothers (20%) and controls (10%) was much lower than previous studies.
Conclusions Hearing data were difficult to obtain; larger numbers would be needed to determine if asymmetrical differences between the right and left ear were a marker for SIDS. A national prospective registry for monitoring and a renewed campaign to a new generation of parents needs to be considered underlining the initial message to place infants on their backs for sleep and the more recent message to avoid bed-sharing in hazardous circumstances. (Author)

20190613-69*

Sudden unexpected infant death characteristics in the French region of West Provence-Alpes-Côte d'Azur. Tuchtan L, Delteil C, Levrat F, et al (2019), Paediatrics and International Child Health vol 39, no 2, 2019, pp 104-110

Background: Although the incidence of sudden unexpected infant death (SUID) has decreased since the 'Back to Sleep' campaign in English-speaking countries and other preventive campaigns, the circumstances of such deaths remain unclear.
Aim: To analyse infant deaths recorded at the referral centre for sudden infant death of the West Provence-Alpes-Côte d'Azur region of France (West PACA) and the forensic medicine department of Marseille University Hospital.
Methods: Information on all SUID cases from 2000 to 2017 was extracted from the referral centre for sudden infant deaths in West PACA and the forensic medicine department of Marseille.
Results: The study included 130 infants over the 17 years with a very similar distribution. There was a marked male preponderance, with 61.6% of boys whatever the age at death (sex ratio 1.6). Half of the deaths occurred in the first 6 months of life and the majority (61%) of infants died during autumn and winter. Nearly one-third (33.2%) had presented with minor infections and 21% had been seen by a doctor or had been admitted to hospital. Most deaths (86.4%) occurred during sleep (night or day). Nearly half of the infants (47.7%) were discovered in a prone position. A large majority of parents (90.7%) agreed to a post-mortem examination. Only 6.2% of deaths led to legal proceedings. Nearly 16.9% remained unexplained after compiling all the data included in the protocol and 9.2% remained unexplained because of incomplete investigation, including refusal of post-mortem examination. Abuse was involved in 2.3% of cases.
Conclusions: Asymptomatic infectious conditions were associated with a high proportion of SUID cases. Non-supine sleep positions were still practised. There is a need to increase SUID prevention campaigns. (40 references) (Author)

20190612-50*

Sudden Infant Death Syndrome: A Global Public Health Issue and Nursing's Response. Pretorius K, Rew L Comprehensive Child and Adolescent Nursing vol 42, no 2, pp 151-60

Sudden unexplained death in infancy, including sudden infant death syndrome (SIDS), is a global public health challenge. Despite public health campaigns and efforts, SIDS remains the leading cause of postneonatal mortality in many developed countries. In this article, we review SIDS, describe nursing's unique professional position in addressing this problem, and explore how the principles of social justice can inform nursing's response. Motivated by nursing's ethical and moral obligations, the profession is called to take an active role in educating others regarding this phenomenon, to participate in research, and to develop or advocate for policy that aims to reduce the incidence of

SIDS on an international scale. (Author)

20190125-2*

Child death review. Statutory and operational guidance (England). HM Government (2018), London: Cabinet Office October 2018. 71 pages

Full URL: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/773431/Child_death_review_statutory_and_operational_guidance_England.pdf

This guidance sets out key features of what a good child death review process should look like. This process combines best practice with statutory requirements that must be followed. The purpose of setting out key features of a robust child death review process in one document is to ensure that the outputs from reviews are standardised as far as possible and of a uniform quality. This will enable effective thematic learning from reviews, i.e. a local review may be able to identify specific learning but trends analysis at a national level may identify modifiable factors that could be altered to prevent future deaths. This requires a degree of standardisation that this document aims to outline; however, clinical commissioning groups (CCGs) and local authorities (the child death review partners) are able to make arrangements for child death reviews as they see fit in order to meet the statutory requirements under the Children Act 2004 (the Act). The process set out in this document runs from the moment of a child's death to the completion of the review by the Child Death Overview Panel (CDOP) or any equivalent arrangements put in place by child death review partners. This includes the immediate actions that should be taken after a child's death; the local review of a child's death by those who interacted with the child during life, and with the investigation after the child's death; through to the final stage of the child death review process which is the statutory review arranged by child death review partners. The process is designed to capture the expertise and thoughts of all individuals who have interacted with the case in order to identify changes that could save the lives of children. (Author)

20190103-89*

Polymorphisms in the myeloid differentiation primary response 88 pathway do not explain low expression levels in sudden infant death syndrome. Dybdrodt Bjørnvall C, Opdal SH, Rognum TO, et al (2019), Acta Paediatrica vol 108, no 7, July 2019, pp 1262-1266

Aim

The aim of this study was to investigate if a range of known rare and common genetic variants in the Toll-like receptor 4 (TLR4)/myeloid differentiation primary response 88 (MyD88) pathway were present or overrepresented in sudden infant death syndrome (SIDS) compared to controls.

Methods

Genetic variations in the genes encoding TLR4, MyD88 and Interleukin-1 receptor-associated kinase 4 were analysed. The subjects investigated included 158 SIDS cases with a median age of 15.25 weeks (2-47 weeks), 80 cases of infectious death with a median age of 24.9 weeks (0-285 weeks) and 199 adult controls with a median age of 50 years (11-86 years). The cases were collected in the years 1988-2017, and the autopsies were performed at the Department of Forensic Sciences at Oslo University Hospital, Oslo, Norway.

Results

The results showed that none of the genetic variants selected from the MyD88 pathway were associated with neither SIDS nor infectious death. Most of the rare genetic variants were homozygote for the common allele in all groups, while the rest revealed allelic variation.

Conclusion

The genetic variations investigated in this study did not appear to be involved in the pathogenesis of SIDS.

(24 references) (Author)

20181022-23*

The Sudden Infant Death Syndrome mechanism of death may be a non-septic hyper-dynamic shock. Gabbay U, Carmi D, Birk E, et al (2019), Medical Hypotheses vol 122, January 2019, pp 35-40

Background

Sudden Infant Death Syndrome (SIDS) mechanisms of death remains obscured. SIDS' Triple Risk Model assumed coexistence of individual subtle vulnerability, critical developmental period and stressors. Prone sleeping is a major risk factor but provide no clues regarding the mechanism of death. The leading assumed mechanisms of death are either an acute respiratory crisis or arrhythmias but neither one is supported with evidence, hence both are eventually speculations. Postmortem findings do exist but are inconclusive to identify the mechanism of death.
What does the proposed hypothesis based on?
1. The stressors (suggested by the triple risk model) share a unified compensatory physiological response of decrease in systemic vascular resistant (SVR) to facilitate a compensatory increase in cardiac output (CO). 2. The cardiovascular/cardiorespiratory control of the vulnerable infant during a critical developmental period may be impaired. 3. A severe decrease in SVR is associated with hyper-dynamic state, high output failure and distributive shock.
The hypothesis
Infant who is exposed to one or more stressors responds normally by decrease in SVR which increases CO. In normal circumstances once the needs are met both SVR and CO are stabilized on a new steady state. The incompetent cardiovascular control of the vulnerable infant fails to stabilize SVR which decreases in an uncontrolled manner. Accordingly CO increases above the needs to hyper-dynamic state, high output heart failure and hyper-dynamic shock.
Conclusions
The proposed hypothesis provides an appropriate alternative to either respiratory crises or arrhythmia though both speculations cannot be entirely excluded. (71 references) (Author)

20180917-10*

Ethnic variation in unexplained deaths in infancy, including sudden infant death syndrome (SIDS), England and Wales 2006-2012: national birth cohort study using routine data. Hroll ME, Quigley MA, Kurinczuk JJ, et al (2018), Journal of Epidemiology and Community Health vol 72, no 10, October 2018, pp 911-918
Full URL: https://jech.bmj.com/content/72/10/911

Background Unexplained deaths in infancy comprise 'sudden infant death syndrome' (SIDS) and deaths without ascertained cause. They are typically sleep-related, perhaps triggered by unsafe sleep environments. Preterm birth may increase risk, and varies with ethnicity. We aimed to compare ethnic-specific rates of unexplained infant death, explore sociodemographic explanations for ethnic variation, and examine the role of preterm birth.
Methods We analysed routine data for 4.6 million live singleton births in England and Wales 2006-2012, including seven non-White ethnic groups ranging in size from 29 313 (Mixed Black-African-White) to 180 265 (Pakistani). We calculated rates, birth-year-adjusted ORs, and effects of further adjustments on the $\chi 2$ for ethnic variation.
Results There were 1559 unexplained infant deaths. Crude rates per 1000 live singleton births were as follows: 0.1-0.2 for Indian, Bangladeshi, Pakistani, White Non-British, Black African; 0.4 for White British; 0.6-0.7 for Mixed Black-African-White, Mixed Black-Caribbean-White, Black Caribbean. Birth-year-adjusted ORs relative to White British ranged from 0.38 (95% CI 0.24 to 0.60) for Indian babies to 1.73 (1.21 to 2.47) for Black Caribbean ($\chi 2(10\ df)=113.6$, $p<0.0005$). Combined adjustment for parents' marital/registration status and mother's country of birth (UK/non-UK) attenuated the ethnic variation. Adjustments for gestational age at birth, maternal age and area deprivation made little difference.
Conclusion Substantial ethnic disparity in risk of unexplained infant death exists in England and Wales. Apparently not attributable to preterm birth or area deprivation, this may reflect cultural differences in infant care. Further research into infant-care practices in low-risk ethnic groups might enable more effective prevention of such deaths in the general population. (Author)

20180913-90*

Neuropathology of Early Sudden Infant Death Syndrome-Hypoplasia of the Pontine Kolliker-Fuse Nucleus: A Possible Marker of Unexpected Collapse during Skin-to-Skin Care. Lavezzi AM, Ferrero S, Paradiso B, et al (2019), American Journal of Perinatology vol 36, no 5, April 2019, pp 460-471

Objective
To find a possible pathogenetic mechanism of the early sudden infant death occurring in newborns during the skin-to-skin care (SSC), through the examination of neuronal centers regulating the vital activities.
Study Design
This is an in-depth examination of the brain stem in 22 healthy term newborns, suddenly died in the first hour of life without the identification of a cause at autopsy (early sudden infant death syndrome [eSIDS]), 12 of them concomitantly with SSC, and 10 with age-matched controls died of known pathology.
Results
Developmental alterations of neuronal structures of the brain stem were highlighted in 19 of the 22 eSIDS, but not in control. The hypoplasia of the pontine Kölliker-Fuse nucleus (KFN), an important respiratory center, was diagnosed at the histological examination, validated by morphometric quantifications, in 11 of the 12 eSIDS while they were placed on the mother's chest and in 2 of the 10 SSC unrelated neonatal deaths.
Conclusion
The delayed development of the KFN could represent a specific finding of eSIDS occurring during SSC. Therefore, it is necessary to point out that the SSC represents a further risk factor that must be added to others already known for sudden infant death syndrome. Then this practice needs appropriate monitoring strategies of the infant's conditions. (67 references) (Author)

20180416-24*

Dysfunction of NaV1.4, a skeletal muscle voltage-gated sodium channel, in sudden infant death syndrome: a case-control study. Männikkö R, Wong L, Tester DJ, et al (2018), The Lancet vol 391, no 10129, 14 April 2018, pp 1483-1492
Full URL: http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(18)30021-7/fulltext
Background
Sudden infant death syndrome (SIDS) is the leading cause of post-neonatal infant death in high-income countries. Central respiratory system dysfunction seems to contribute to these deaths. Excitation that drives contraction of skeletal respiratory muscles is controlled by the sodium channel NaV1.4, which is encoded by the gene SCN4A. Variants in NaV1.4 that directly alter skeletal muscle excitability can cause myotonia, periodic paralysis, congenital myopathy, and myasthenic syndrome. SCN4A variants have also been found in infants with life-threatening apnoea and laryngospasm. We therefore hypothesised that rare, functionally disruptive SCN4A variants might be over-represented in infants who died from SIDS.
Methods
We did a case-control study, including two consecutive cohorts that included 278 SIDS cases of European ancestry and 729 ethnically matched controls without a history of cardiovascular, respiratory, or neurological disease. We compared the frequency of rare variants in SCN4A between groups (minor allele frequency <0·00005 in the Exome Aggregation Consortium). We assessed biophysical characterisation of the variant channels using a heterologous expression system.
Findings
Four (1·4%) of the 278 infants in the SIDS cohort had a rare functionally disruptive SCN4A variant compared with none (0%) of 729 ethnically matched controls (p=0·0057).
Interpretation
Rare SCN4A variants that directly alter NaV1.4 function occur in infants who had died from SIDS. These variants are predicted to significantly alter muscle membrane excitability and compromise respiratory and laryngeal function. These findings indicate that dysfunction of muscle sodium channels is a potentially modifiable risk factor in a subset of infant sudden deaths.
Funding
UK Medical Research Council, the Wellcome Trust, National Institute for Health Research, the British Heart Foundation, Biotronik, Cardiac Risk in the Young, Higher Education Funding Council for England, Dravet Syndrome UK, the Epilepsy Society, the Eunice Kennedy Shriver National Institute of Child Health & Human Development of the National Institutes of Health, and the Mayo Clinic Windland Smith Rice Comprehensive Sudden Cardiac Death Program. (45 references) (Author)

20180213-2*

National and State Trends in Sudden Unexpected Infant Death: 1990-2015. Lambert ABE, Parks SE, Shapiro-Mendoza CK (2018), Pediatrics vol 141, no 3, March 2018

Full URL: http://pediatrics.aappublications.org/content/early/2018/02/09/peds.2017-3519?utm_source=highwire&utm_medium=email&utm_campaign=Pediatrics_papetoc

BACKGROUND: Sharp declines in sudden unexpected infant death (SUID) in the 1990s and a diagnostic shift from sudden infant death syndrome (SIDS) to unknown cause and accidental suffocation and strangulation in bed (ASSB) in 1999-2001 have been documented. We examined trends in SUID and SIDS, unknown cause, and ASSB from 1990 to 2015 and compared state-specific SUID rates to identify significant trends that may be used to inform SUID prevention efforts.

METHODS: We used data from US mortality files to evaluate national and state-specific SUID rates (deaths per 100 000 live births) for 1990-2015. SUID included infants with an underlying cause of death, SIDS, unknown cause, or ASSB. To examine overall US rates for SUID and SUID subtypes, we calculated the percent change by fitting Poisson regression models. We report state differences in SUID and compared state-specific rates from 2000-2002 to 2013-2015 by calculating the percent change.

RESULTS: SUID rates declined from 154.6 per 100 000 live births in 1990 to 92.4 in 2015, declining 44.6% from 1990 to 1998 and 7% from 1999 to 2015. From 1999 to 2015, SIDS rates decreased 35.8%, ASSB rates increased 183.8%, and there was no significant change in unknown cause rates. SUID trends among states varied widely from 41.5 to 184.3 in 2000-2002 and from 33.2 to 202.2 in 2013-2015.

CONCLUSIONS: Reductions in SUID rates since 1999 have been minimal, and wide variations in state-specific rates remain. States with significant declines in SUID rates might have SUID risk-reduction programs that could serve as models for other states. (Author)

20180104-110*

Intestinal Microbiota Composition in Sudden Infant Death Syndrome and Age-Matched Controls. Leong LEX, Taylor SL, Shivasami A, et al (2017), The Journal of Pediatrics vol 191, December 2017, pp 63-68.e1

Objective

To assess whether features of the infant intestinal microbiome, including the carriage of toxigenic bacteria, are associated with sudden infant death syndrome (SIDS).

Study design

We undertook a case-controlled analysis of fecal microbiology in SIDS. Fecal material was obtained from 44 cases and 44 aged-matched controls. Microbiota composition was determined by 16S ribosomal RNA gene amplicon sequencing and comparisons between cases and controls made based on both bacterial alpha diversity measures and unconstrained ordination. Specific quantitative polymerase chain reaction assays were used to determine intestinal carriage of Staphylococcus aureus, toxigenic Clostridium difficile, and pathogenic and nonpathogenic Escherichia coli.

Results

The microbial composition for the study population as a whole was consistent with previous studies of infants <12 months of age, with a correlation between alpha diversity and age ($r^2 = 0.08$; $P = .007$). However, no difference was observed in alpha diversity between SIDS cases and controls ($P > .4$). Nonmetric multidimensional scaling also revealed no evidence of differences in microbiota dispersal between SIDS cases and controls ($P = .4$, permutational multivariate ANOVA test; Pseudo-$F = 0.9$), nor was a difference observed in microbiota dispersion ($P = .19$, PERMDISP test; $F = 1.9$). There were no significant intergroup differences in the carriage of S aureus, toxigenic C difficile, total E coli, or pathogenic E coli.

Conclusions

We found no evidence of an association between altered intestinal microbiology and SIDS, or to support the development of strategies to reduce the incidence of SIDS that target intestinal microbiology.

(48 references) (Author)

20171108-76*

Low cerebrospinal fluid hypocretin levels during sudden infant death syndrome (SIDS) risk period. Lancien M, Inocente CO, Dauvilliers Y, et al (2017), Sleep Medicine vol 33, May 2017, pp 57-60

OBJECTIVES:

The temporal association between sudden infant death syndrome (SIDS) and sleep suggests that the arousability from sleep provides a protective mechanism for survival. Recently, the hypocretin system, which promotes wakefulness, has been implicated in SIDS, since it has been reported that SIDS victims have fewer hypocretin neurons than infants who have died from other causes. To understand the role of hypocretin in SIDS, it is essential to better understand how this system matures. The present study compared cerebrospinal fluid(CSF) hypocretin in children aged 2-6 months, which is the age of peak incidence for SIDS, to both younger and older children.

METHOD:

Hypocretin levels were measured in CSF samples from 101 children who underwent a clinically relevant lumbar puncture. Children were separated into five age groups: 0-2 months, 2-6 months, 1-5 years, 5-10 years, and 10-18 years.

RESULTS:

Hypocretin levels were not significantly different between 1-5 years, 5-10 years, and 10-18 years. Therefore, these three groups were pooled into a single one (1-18 years) for further analysis. Between the 0-2 month, 2-6 month, and 1-18 year groups, a significant difference in CSF hypocretin levels existed ($p = 0.001$). Simple comparisons showed that CSF hypocretin levels in the 2-6 month age group were significantly lower than hypocretin levels in both the 0-2 month and 1-18 year group ($p < 0.001$ and $p = 0.008$, respectively), but not significantly between 0-2 month and 1-18 year children.

CONCLUSIONS:

The CSF hypocretin levels were lower at the age of peak incidence for SIDS. This could underlie an increased vulnerability to SIDS at this specific age. (Author)

20171031-10*

High serum serotonin in sudden infant death syndrome. Haynes RL, Frelinger AL 3rd, Giles EK, et al (2017), Proceedings of the National Academy of Sciences of the United States of America (PNAS) vol 114, no 29, 18 July 2017, pp 7695-7700

Sudden infant death syndrome (SIDS), the leading cause of postneonatal infant mortality, likely comprises heterogeneous disorders with the common phenotype of sudden death without explanation upon postmortem investigation. Previously, we reported that ?40% of SIDS deaths are associated with abnormalities in serotonin (5-hydroxytryptamine, 5-HT) in regions of the brainstem critical in homeostatic regulation. Here we tested the hypothesis that SIDS is associated with an alteration in serum 5-HT levels. Serum 5-HT, adjusted for postconceptional age, was significantly elevated (95%) in SIDS infants ($n = 61$) compared with autopsied controls ($n = 15$) [SIDS, 177.2 ± 15.1 (mean ± SE) ng/mL versus controls, 91.1 ± 30.6 ng/mL] ($P = 0.014$), as determined by ELISA. This increase was validated using high-performance liquid chromatography. Thirty-one percent (19/61) of SIDS cases had 5-HT levels greater than 2 SDs above the mean of the controls, thus defining a subset of SIDS cases with elevated 5-HT. There was no association between genotypes of the serotonin transporter promoter region polymorphism and serum 5-HT level. This study demonstrates that SIDS is associated with peripheral abnormalities in the 5-HT pathway. High serum 5-HT may serve as a potential forensic biomarker in autopsied infants with SIDS with serotonergic defects. (Author)

20171017-99

An Intuitive Approach to Understanding Infant Death. Kautz W (2017), Journal of Prenatal and Perinatal Psychology and Health (JPPPH) vol 32, no 1, Fall 2017, pp 16-53

The sudden, unexpected, and unexplained death of a healthy infant in its first year of life (nominally 2 to12 months) is surely one of the most tragic human experiences a parent can undergo. The shock of loss is commonly accompanied by extreme sorrow, grief, feelings of guilt, and the emergence of unanswerable questions on how such an event could possibly have occurred. Forty years of medical research to find the cause of Sudden Infant Death Syndrome (SIDS)

have found neither the cause of the phenomenon nor a means of predicting or preventing it-only a long list of secondary, unlikely, and non-causal 'risk factors' which offer no consolation to parents, no answers to their questions, and no substantial and trustworthy guidelines for action.
A novel investigation at the Center for Applied Intuition (CAI) utilized a systematic method of consensual intuitive inquiry to answer these questions. It sought to generate an explanation for the cause of SIDS and suggest how the disorder may best be handled by the parents and associated family members. A dozen 'expert intuitives,' whose skills had been verified for acquiring entirely new and correct knowledge in other areas, explained that a very young infant is sufficiently conscious to be able to choose 'at will' whether to continue its life or leave it-its own kind of suicide, just as adults may do. As the life force withdraws, the body succumbs to its weakest physical condition, which in the case of SIDS is not medically detectable.
Modern medical science possesses no means for investigating subjective information sources such as intuition, or even for testing whether proposed explanations are right or wrong. It has therefore disregarded non-physical approaches to understanding SIDS. However, corroboration of the intuitive findings is available from psychological sources. They show clearly that perinatal infants possess an active consciousness capable of sensation, memory, and some degree of choice, thereby adding credibility to the intuitive information.
SIDS can be seen as a natural occurrence, not a physical disorder or a medical disease and not a direct result of parental action or inaction. The usual grief, guilt, and confusion of the parents, while certainly understandable, arise from a misconception of the life process itself, which includes the possibility of premature death for infants just as it does for adults. These typical but mistaken responses by parents may be dispelled when they can achieve a fuller understanding and acceptance of the central place of loss and death in human life. The infant has the same choice as its parents to leave life at any time. The parents' love for their child is no less genuine by this revised understanding, though it applies more to the infant's consciousness, than to its body which can indeed be lost. Herein lies the meaning and the fundamental lesson parents may learn from losing a child to SIDS or any form of infant death. (Author)

20170919-23

Sudden Infant Death Syndrome (SIDS): our finest hour?. Morgan V (2017), The Practising Midwife vol 20, no 8, September 2017, pp 10-14

Unexplained infant deaths have reached the lowest level on record in England and Wales - 212 deaths in 2014, a rate of 0.3 deaths per 1,000 live births (Office for National Statistics [ONS] 2016).
Here, I trace this remarkable public health success story, consider the current situation and ask what midwives might learn from the SIDS story to help tackle current public health challenges. (10 references) (Author)

20170914-76*

Infection: the neglected paradigm in SIDS research. Goldwater PN (2017), Archives of Disease in Childhood vol 102, no 8, August 2017, pp 767-772

Full URL: http://adc.bmj.com/content/102/8/767

Despite decades of investigation and millions of dollars spent, the cause of sudden infant death syndrome (SIDS) eludes researchers. It is timely therefore to reconsider the reasons for this failure and to explore how research might go forward with better prospects. This review assesses SIDS research in the context of clinicopathological and epidemiological features and determines that only infection attains congruence. (98 references) (Author)

20170906-17*

Why are those most in need of Sudden Unexplained Infant Death (SUDI) Prevention information the least likely to receive it? A comment on unconscious bias and Māori health. Houkamau CA, Clarke K (2016), New Zealand Medical Journal vol 129, no 1440, August 2016, 6978

Full URL: https://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2016/vol-129-no-1440/6978

This paper expands discussions of unconscious bias into the New Zealand health care arena. We review international research which links health provider bias to inequitable health outcomes for ethnic minorities as well as local data

which indicates Māori may be vulnerable to bias. Our focus is on outcomes from the 2014 Well Child/Tamariki Ora (WCTO) Programme Delivery Report which shows Māori are less likely to receive SUDI prevention information from their Well-child health provider than other ethnic groups in New Zealand. (30 references) (Author)

20170816-120*

The gene encoding the inwardly rectifying potassium channel Kir4.1 may be involved in sudden infant death syndrome. Opdal SH, Vege A, Stray-Pedersen A, et al (2017), Acta Paediatrica vol 106, no 9, September 2017, pp 1474-1480

Aim

Disturbances in brain function and development may play a role in sudden infant death syndrome (SIDS). This Norwegian study aimed to test the hypothesis that specific variants of genes involved in water transport and potassium homeostasis would be predisposing factors for SIDS.

Methods

Genetic variation in the genes encoding aquaporin-4 (AQP4), Kir4.1 (KCNJ10) and α-syntrophin was analysed in 171 SIDS cases (62.6% male) with a median age of 15.5 (2-52) weeks and 398 adult controls (70.6% male) with a median age of 44 (11-91) years. All the subjects were Caucasians who were autopsied from 1988 to 2013.

Results

The CC genotype of rs72878794 in the AQP4 gene and a combination of the CC genotype in rs17375748, rs1130183, rs12133079 and rs1186688 in KCNJ10 (4xCC) were found to be associated with SIDS. The SIDS cases with the 4xCC SNP combination were younger than the SIDS cases with other genotype combinations ($p = 0.006$).

Conclusion

This study indicates that genetic variations in KCNJ10 and AQP4 may be predisposing factors for SIDS. Alterations in the expression of the AQP4/Kir4.1 complex can disrupt water and ion homeostasis, which may influence brain development and facilitate brain oedema formation This may be especially unfavourable during the first weeks of life. (Author) (28 references)

20170720-35*

My child cannot breathe while sleeping: a report of three cases and review. Seo WH, Park M, Eun S-H, et al (2017), BMC Pediatrics vol 17, no 169, 18 July 2017

Full URL: https://bmcpediatr.biomedcentral.com/articles/10.1186/s12887-017-0922-9

Background

Sudden breath-holding episodes during sleep in young children are potentially related to sudden infant death syndrome and other life-threatening events. Additionally, these episodes can negatively affect child's growth and development.

Case presentation

Here, we present 3 cases of preschool children with similar paroxysmal nocturnal waking events associated with choking that had different etiologies (nocturnal frontal lobe epilepsy, nocturnal gastroesophageal reflux disease, and parasomnia, respectively).

Conclusions

It is important to take into consideration the fact that breath spells during sleep can occur as a rare manifestation of parasomnia due to gastroesophageal reflux or as a symptom of nocturnal frontal lobe epilepsy. Full video electroencephalography, polysomnography, and simultaneous gastric pH monitoring should be used for the differential diagnosis of sleep-related disorders, such as breath spells, in children. (20 references) (Author) [Please note: BMC initially publishes articles in a provisional format. If there is a note on the document to indicate that it is still provisional, it may undergo minor changes]

20170420-43*

Sudden unexplained early neonatal death or collapse: a national surveillance study. Lutz TL, Elliott EJ, Jeffery HE (2016), Pediatric Research vol 80, no 4, October 2016, pp 493-498

Full URL: http://www.nature.com/pr/journal/v80/n4/full/pr2016110a.html

Background:
The incidence of sudden unexpected early neonatal death (SUEND) or acute life-threatening events (ALTEs) is reported as 0.05/1,000 to 0.38/1,000 live births. There is currently no national system in Australia for reporting and investigating such cases.
Methods:
A 3-y prospective, national surveillance study, run in collaboration with the Australian Pediatric Surveillance Unit (APSU). Data were provided by pediatricians reporting to APSU; and independently ascertained by the Coroner in two states (NSW and QLD) and the Newborn Early Transport Network in NSW. A detailed deidentified questionnaire was created.
Results:
In NSW and QLD, the incidence was 0.1 and 0.08/1,000 live births, respectively. Forty-eight definitive cases were identified. Common causes included accidental asphyxia, cardiac disease, persistent pulmonary hypertension of the newborn, and sudden infant death syndrome. Twenty-six babies collapsed on day 1 and 19 were found on the carer's chest.
Conclusion:
The incidence in NSW and QLD is higher than previously published. The first postnatal day is a vulnerable period for newborns, who require close observation particularly during skin-to-skin contact. Development and implementation of guidelines for safe sleeping in hospital are needed. Collaboration between obstetricians, midwives, and pediatricians is essential to ensure safety of the newborn. (22 references) (Author)

20170406-29*

Infant pacifiers for reduction in risk of sudden infant death syndrome

(Cochrane Review) (Last assessed as up-to-date: 25 February 2016). Psaila K, Foster JP, Pulbrook N, et al (2017), The Cochrane Database of Systematic Reviews Issue 4, 2017

Background
Sudden infant death syndrome (SIDS) has been most recently defined as the sudden unexpected death of an infant less than one year of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including the performance of a complete autopsy and a review of the circumstances of death and clinical history. Despite the success of several prevention campaigns, SIDS remains a leading cause of infant mortality. In 1994, a 'triple risk model' for SIDS was proposed that described SIDS as an event that results from the intersection of three factors: a vulnerable infant; a critical development period in homeostatic control (age related); and an exogenous stressor. The association between pacifier (dummy) use and reduced incidence of SIDS has been shown in epidemiological studies since the early 1990s. Pacifier use, given its low cost, might be a cost-effective intervention for SIDS prevention if it is confirmed effective in randomised controlled trials.
Objectives
To determine whether the use of pacifiers during sleep versus no pacifier during sleep reduces the risk of SIDS.
Search methods
We used the standard search strategy of the Cochrane Neonatal Review Group to search the Cochrane Central Register of Controlled Trials (CENTRAL 2016, Issue 2), MEDLINE via PubMed, Embase, and CINAHL to 16 March 2016. We also searched clinical trials databases, conference proceedings, and the reference lists of retrieved articles for randomised controlled trials and quasi-randomised trials.
Selection criteria
Published and unpublished controlled trials using random and quasi-random allocations of infants born at term and at preterm (less than 37 weeks' gestation) or with low birth weight (< 2500 g). Infants must have been randomised by one month' postmenstrual age. We planned to include studies reported only by abstracts, and cluster and cross-over randomised trials.
Data collection and analysis
Two review authors independently reviewed studies from searches. We found no eligible studies.
Main results
We identified no randomised controlled trials examining infant pacifiers for reduction in risk of SIDS.

Authors' conclusions
We found no randomised control trial evidence on which to support or refute the use of pacifiers for the prevention of SIDS. (Author)

20170329-97*

Altered gene expression and possible immunodeficiency in cases of sudden infant death syndrome. Ferrante L, Rognum TO, Vege A, et al (2016), Pediatric Research vol 80, no 1, April 2016, pp 77-84

Background:
A large number of studies have tried to uncover a genetic predisposition for sudden infant death syndrome (SIDS), but there is still uncertainty concerning the pathogenesis of these deaths. The purpose of this study was to investigate mRNA gene expression in SIDS cases and controls, in order to uncover genes that are differentially expressed in the two groups.
Methods:
Tissue from brain, heart, and liver from 15 SIDS cases and 15 controls were included in the study, and mRNA expression was determined using the Illumina whole genome gene expression DASL HT assay.
Results:
Seventeen genes showed significantly altered expression compared to controls, after correction for multiple testing. Three genes involved in the immune system were of particular interest, including the downregulation of MyD88 in tissue from SIDS brains, as well as the downregulation of the genes encoding CCL3 and UNC13 in the liver.
Conclusion:
These findings indicate that there is an altered expression of genes involved in the inflammatory process in a proportion of SIDS cases, which further strengthen the hypothesis that impaired immune response play a role in this syndrome. (42 references) (Author)

20170328-39*

Evolution and significance of the triple risk model in sudden infant death syndrome. Spinelli J, Collins-Praino L, Van Den Heuvel C, et al (2017), Journal of Paediatrics and Child Health vol 53, no 2, February 2017, pp 112-115

Sudden infant death syndrome (SIDS) is a leading cause of death in infants, although the mechanisms leading to death remain unclear. Multiple theories have emerged over time, with one of the most influential hypotheses being the triple risk model. This model, first devised in 1972 and later revised in 1994 by Filiano and Kinney, is still widely used in assisting with conceptualising and understanding sudden death in infancy. This model has evolved over time, with each version stressing that SIDS is likely to occur when certain risk factors coincide, suggesting that the lethal mechanisms in SIDS are likely to be multifactorial. All versions of the triplerisk model from 1972 to the present have emphasised the complexity of SIDS and serve as useful guides for current and future research into the enigma of sudden and unexpected death in infancy.
 (Author)

20170327-57*

Area-based study shows most parents follow advice to reduce risk of sudden infant death syndrome. Strömberg Celind F, Wennergren G, Möllborg, et al (2017), Acta Paediatrica vol 106, no 4, April 2017, pp 579-585

AIM:
Guidance on reducing the risk of sudden infant death syndrome (SIDS) was successfully introduced to a number of countries in the early 1990s. The most important recommendations were supine sleeping for infants and non-smoking for mothers. This 2012-2014 study examined adherence to the national Swedish SIDS advice.
METHODS:
We asked 1000 parents with infants registered at child healthcare centres in western Sweden to complete a questionnaire on infant care from birth to 12 months of age.
RESULTS:
We analysed 710 responses and found that, in the first three months, 1.3% of the infants were placed in the prone

sleeping position and 14.3% were placed on their side. By three to five months, this had risen to 5.6% and 23.6%. In the first three months, 83.1% were breastfed, 84.1% used a pacifier and 44.2% shared their parents' bed, while 5.8% slept in another room. Bed sharing was more likely if infants were breastfed and less likely if they used pacifiers. During pregnancy, 2.8% of the mothers smoked and the mothers who had smoked during pregnancy were less likely to bed share.
CONCLUSION:
Overall adherence to the SIDS advice was good, but both prone and side sleeping practices should be targeted.
 (26 references) (Author)

20170220-18*

Infants dying suddenly and unexpectedly share demographic features with infants who die with retinal and dural bleeding: a review of neural mechanisms. Squier W, Mack J, Jansen AC (2016), Developmental Medicine & Child Neurology vol 58, no 12, December 2016, pp 575-584

The cause of death in infants who die suddenly and unexpectedly (sudden unexpected death in infancy [SUDI]) remains a diagnostic challenge. Some infants have identified diseases (explained SUDI); those without explanation are called sudden infant death syndrome (SIDS). Demographic data indicate subgroups among SUDI and SIDS cases, such as unsafe sleeping and apparent life-threatening events. Infants dying suddenly with retinal and dural bleeding are often classified as abused, but in many there is no evidence of trauma. Demographic features suggest that they may represent a further subgroup of SUDI. This review examines the neuropathological hypotheses to explain SIDS and highlights the interaction of infant oxygen-conserving reflexes with the brainstem networks considered responsible for SIDS. We consider sex- and age-specific vulnerabilities related to dural bleeding and how sensitization of the dural innervation by bleeding may influence these reflexes, potentially leading to collapse or even death after otherwise trivial insults.
 (Author)

20170209-78*

Unexpected, unexplained and life-threatening events in infants are age-dependent descriptive syndromes with different risk and management. Herlenius E (2017), Acta Paediatrica vol 106, no 2, February 2017, pp 191-193

Editorial article examining the features between neonatal apparent life-threatening event (ALTE) and the proposed brief resolved unexplained event (BRUE) diagnosis for infants older than 60 days. (10 references) (Author, edited)

20170209-15

Metabolomic profiling of brain from infants who died from Sudden Infant Death Syndrome reveals novel predictive biomarkers. Graham SF, Chevallier OP, Kumar P, et al (2017), Journal of Perinatology vol 37, no 1, January 2017, pp 91-97

OBJECTIVE:
Sudden Infant Death Syndrome (SIDS) is defined as the sudden death of an infant <1 year of age that cannot be explained following a thorough investigation. Currently, no reliable clinical biomarkers are available for the prediction of infants who will die of SIDS.
STUDY DESIGN:
This study aimed to profile the medulla oblongata from postmortem human brain from SIDS victims (n=16) and compare their profiles with that of age-matched controls (n=7).
RESULTS:
Using LC-Orbitrap-MS, we detected 12 710 features in electrospray ionization positive (ESI+) mode and 8243 in ESI- mode from polar extracts of brain. Five features acquired in ESI+ mode produced a predictive model for SIDS with an area under the receiver operating characteristic curve (AUC) of 1 (confidence interval (CI): 0.995-1) and a predictive power of 97.4%. Three biomarkers acquired in ESI- mode produced a predictive model with an AUC of 0.866 (CI: 0.767-0.942) and a predictive power of 77.6%. We confidently identified 5 of these features (l-(+)-ergothioneine,

nicotinic acid, succinic acid, adenosine monophosphate and azelaic acid) and putatively identify another 4 out of the 15 in total.
CONCLUSIONS:
This study underscores the potential value of metabolomics for studying SIDS. Further characterization of the metabolome of postmortem SIDS brains could lead to the identification of potential antemortem biomarkers for novel prevention strategies for SIDS. (52 references) (Author)

20161121-37*

The high price of being labelled 'high risk': social context as a health determinant for sudden unexpected infant death in Māori communities. Houkamau C, Tipene-Leach D, Clarke K (2016), New Zealand College of Midwives Journal no 52, December 2016, pp 56-61

Background: For over 25 years, nationwide efforts to address sudden infant death in New Zealand have focused on advising parents to avoid four risk factors labelled as modifiable. But Māori infants still have sudden unexpected death in infancy (SUDI) at five times the rate of non-Māori.
Aim: This paper expands the conceptualisation of SUDI risk factors and suggests a reconsideration of the use of risk factor terminology.
Discussion: Working from the assumption that health outcomes are influenced by social determinants, we put forward two key propositions. Firstly, we argue (using maternal smoking as a case in point) that greater attention must be paid to the role of social and socio-economic factors in the prevention of SUDI in Māori communities. Secondly, we propose that the language of risk reduction impedes Māori engagement with health services because the discourse associated with being 'at risk' and 'vulnerable' casts Māori in a deficit framework affecting how Māori are perceived by health professionals and, more importantly perhaps, how Māori see themselves. (59 references) (Author) [Full article available online at: https://www.midwife.org.nz/resources-events/nzcom-journal/]

20161102-34*

The Disappearance of Sudden Infant Death Syndrome Has the Clock Turned Back?. Cutz E (2016), JAMA Pediatrics vol 170, no 4, April 2016, pp 315-316

Sudden Infant Death Syndrome (SIDS) is defined as a sudden unexpected death of an infant younger than 1 year who was previously well and in whom postmortem examination fails to identify the cause of death. The diagnostic term SIDS was first proposed at the Second International Conference on Causes of Sudden Infant Death in 1969, at a time when close to 9000 cases of sudden unexpected infant death (SUID) occurred in the United States alone.1The term SIDS served 3 main purposes: to encourage and focus research into these tragic deaths, to comfort parents with knowledge that the death was the result of a natural disease entity, and to absolve parents or caregivers of any blame for the death of their infant. Although considerable progress has been made since then, particularly in understanding the potential risk factors and biological underpinning, during the last decade the use of SIDS as a diagnosis has fallen out of favor. In many jurisdictions, the use of this term has been reduced considerably or abandoned altogether. For example, reports from the Office of the Chief Medical Examiner from Wayne County, Michigan, and New York, New York, indicate a 95% and 84% decline in SIDS diagnoses, respectively. In the province of Ontario (population 13.5 million, with about 128 cases of SUID recorded yearly), not a single case of SIDS has been diagnosed during the past 2 years.2,3 It appears that the clock has been turned backward and that the diagnostic term SIDS is disappearing. Instead, coroners and medical examiners are reverting to terminology employed in the past such as pneumonia, suffocation, and, most commonly, 'undetermined.' This diagnostic shift cannot help but have a profound effect on the affected families as well as dampen the enthusiasm for basic scientific research. (Author)

20161011-57*

Knowledge, Attitudes, and Risk for Sudden Unexpected Infant Death in Children of Adolescent Mothers: A Qualitative Study. Caraballo M, Shimasaki S, Johnston K, et al (2016), Journal of Pediatrics vol 174, July 2016, pp 78-83. e2

Objective

To investigate practices, knowledge, attitudes, and beliefs regarding infant sleep among adolescent mothers, a demographic at high risk for sudden unexpected infant death, and to identify novel public health interventions targeting the particular reasons of this population.
Study design
Seven targeted focus groups including 43 adolescent mothers were conducted at high school daycare centers throughout Colorado. Focus groups were recorded, transcribed, validated, and then analyzed in NVivo 10. Validation included coding consistency statistics and expert review.
Results
Most mothers knew many of the American Academy of Pediatrics recommendations for infant sleep. However, almost all teens reported bedsharing regularly and used loose blankets or soft bedding despite being informed of risks. Reasons for nonadherence to recommendations included beliefs that babies are safest and sleep more/better in bed with them, that bedsharing is a bonding opportunity, and that bedsharing is easier than using a separate sleep space. The most common justifications for blankets were infant comfort and concern that babies were cold. Participants' decision making was often influenced by their own mothers, with whom they often resided. Participants felt that their instincts trumped professional advice, even when in direct contradiction to safe sleep recommendations.
Conclusions
Among focus group participants, adherence with safe sleep practices was poor despite awareness of the American Academy of Pediatrics recommendations. Many mothers expressed beliefs and instincts that infants were safe in various unsafe sleep environments. Future study should investigate the efficacy of alternative educational strategies, including education of grandmothers, who have significant influence over adolescent mothers. (22 references) (Author)

20160811-1*

Cot deaths at lowest recorded level in England and Wales. Anon (2016), BBC News 11 August 2016

Full URL: http://www.bbc.co.uk/news/health-37033394

Reports that figures from the Office for National Statistics (ONS) show sudden infant deaths have reached the lowest level since 2004. (KM) [The full report is available from: http://www.bbc.co.uk/news/health-37033394] [Statistical bulletin for deaths in infancy available from: http://bit.ly/2bfH6o8]

20160729-10*

Reducing child mortality in London. Korkodilos M (2016), London: Public Health England July 2016

Full URL: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/541697/Reducing_child_deaths_in_London.pdf

Although there have been significant reductions in child deaths in the past 3 decades in England, too many children are still dying unnecessarily. This document includes: important child mortality statistics for children and young people (CYP) in London; national findings from Child Death Overview Panels (CDOP); actions to reduce child deaths. (Publisher)

20160322-15*

Sudden infant death syndrome and abnormal metabolism of thiamin. Lonsdale D (2015), Medical Hypotheses vol 85, no 6, December 2015, pp 922-926

Although it has been generally accepted that moving the infant from the prone to the supine position has solved the problem of sudden infant death syndrome (SIDS), it has been hypothesized that this is an insufficient explanation and that a mixture of genetic risk, some form of stressful incident and marginal brain metabolism is proportionately required. It is suggested that each of these three variables, with dominance in one or more of them, act together in the common etiology. Much has been written about the association of thiamin and magnesium but the finding of extremely high concentrations of serum thiamin in SIDs victims has largely caused rejection of thiamin as being involved in the etiology. The publication of abnormal brainstem auditory evoked potentials strongly suggests that there are electrochemical changes in the brainstem affecting the mechanisms of automatic breathing and the control

of cardiac rhythm. The brainstem, cerebellum and limbic system of the brain are known to be highly sensitive to thiamin deficiency (pseudo-hypoxia) and the pathophysiology is similar to a mild continued deprivation of oxygen. Little attention has been paid to the complex metabolism of thiamin. Dietary thiamin requires the cooperation of the SLC19 family of thiamin transporters for its absorption into cells and recent information has shown that transporter SNPs may be relatively common and can be expected to increase genetic risk. Thiamin must be phosphorylated to synthesize thiamin pyrophosphate (TPP), well established in its vital action in glucose metabolism. TPP is also a cofactor for the enzyme 2-hydroxyacyl-CoA lyase (HACL1) in the peroxisome, emphasizing its importance in alpha oxidation and plasmalogen synthesis in cell membrane physiology. The importance of thiamine triphosphate (TTP) in energy metabolism is still largely unknown. Thiamin metabolism has been implicated in hyperemesis gravidarum and iatrogenic Wernicke encephalopathy has been reported when the patient is treated with hyperalimentation, in spite of the pharmaceutical doses of thiamin in the intravenous fluid. Defective glucose metabolism, the vital fuel for energy synthesis, particularly in brain, must affect the developing fetus and the pattern of subsequent neonatal health. Sudden death in an apparently healthy infant, occurring at 3-4 months, has long been known to result from feeding the infant with thiamin deficient breast milk. The early investigators of the cause of beriberi considered that this form of sudden death was pathognomonic of the infantile form of the disease. (69 references) (Author)

20160311-27*

Why your baby's sleep matters. Ockwell-Smith S (2016), Hampshire: Pinter and Martin 2016. 160 pages

Research shows that 'normal' infant sleep is not what most experts claim it is. In fact, many of today's sleep 'problems' with young babies and children predominantly occur in the developed world.

In Why Your Baby's Sleep Matters, renowned gentle parenting expert Sarah Ockwell-Smith demonstrates how nurturing babies at night helps their brain development, and covers the topics every parent of a new baby will need to know about, including naps, SIDS, night weaning, and coping with your own exhaustion - and even dealing with advice and criticism from others. (publisher)

20160129-26*

Certain Causes of Neonatal Death. V. Cerebral Birth Trauma

V. Cerebral Birth Trauma. Fedrick J, Butler N (1971), Biol. Neonate vol 18, no 5-6, 1971, pp 321-329,

In this study, the authors follow continue their research in neonatal deaths - this time focusing on the causative factors that influence deaths, and comparing them with Cerebral Birth Trauma cases. The study identifies a range of factors that contribute to neonatal death, including the length of labour, membrane rupture the method of delivery and pre-eclampsia.

20151125-47*

Testing for infectious diseases in sudden unexpected infant death: a survey of medical examiner and coroner offices in the United States. Brooks EG, Gill JR, Buchsbaum R, et al (2015), Journal of Pediatrics vol 167, no 1, July 2015, pp 178-182.e1

Objectives

To determine interoffice variability in routinely performed sudden unexpected infant death (SUID) postmortem studies for infection and to assess availability and perceived utility of various tests of infectious diseases.

Study design

Online surveys were sent to all 154 offices of US medical examiners and coroners serving populations >300 000 people. Surveys included a set of potential laboratory tests for infectious disease. Respondents were asked to select which tests were available in their offices, and which tests were performed routinely in SUIDs vs which tests should be performed routinely.

Results

Of the 45 complete responses, 4.4% did not routinely perform histology, 8.9% did not routinely perform viral studies (ie, culture or molecular diagnostics), 22.2% did not routinely perform blood cultures, 26.7% did not routinely perform lung bacterial cultures, and 44.4% did not routinely perform cerebrospinal fluid cultures.

Conclusions
Our findings suggest that there is considerable interoffice variability with testing for infectious diseases in SUIDs. This appeared to be largely the result of a perceived lack of testing utility rather than a lack of test availability.
Evidence-based practice guidelines regarding the interpretation of microbial testing results, as well as common testing protocols/algorithms, may lead to more accurate and standardized data, thus improving SUID investigation and surveillance. (25 references) (Author)

20151117-85*

Maternal obesity and gestational weight gain are risk factors for infant death. Bodnar LM, Siminerio LL, Himes KP, et al (2016), Obesity vol 24, no 2, February 2016, pp 490-498

Objective:
Assessment of the joint and independent relationships of gestational weight gain and prepregnancy body mass index (BMI) on risk of infant mortality was performed.
Methods:
This study used Pennsylvania linked birth-infant death records (2003-2011) from infants without anomalies born to mothers with prepregnancy BMI categorized as underweight (n = 58,973), normal weight (n = 610,118), overweight (n = 296,630), grade 1 obesity (n = 147,608), grade 2 obesity (n = 71,740), and grade 3 obesity (n = 47,277). Multivariable logistic regression models stratified by BMI category were used to estimate dose-response associations between z scores of gestational weight gain and infant death after confounder adjustment.
Results:
Infant mortality risk was lowest among normal-weight women and increased with rising BMI category. For all BMI groups except for grade 3 obesity, there were U-shaped associations between gestational weight gain and risk of infant death. Weight loss and very low weight gain among women with grades 1 and 2 obesity were associated with high risks of infant mortality. However, even when gestational weight gain in women with obesity was optimized, the predicted risk of infant death remained higher than that of normal-weight women.
Conclusions:
Interventions aimed at substantially reducing preconception weight among women with obesity and avoiding very low or very high gestational weight gain may reduce risk of infant death. (Author)

20151111-62

Sudden unexpected death in infancy: aetiology, pathophysiology, epidemiology and prevention in 2015. Fleming PJ, Blair PS, Pease A (2015), Archives of Disease in Childhood vol 100, no 10, October 2015, pp 984-988

Despite the fall in numbers of unexpected infant deaths that followed the 'Back to Sleep' campaigns in the early 1990s in the UK and many other countries, such deaths remain one of the largest single groups of deaths in the postneonatal period in many Western countries. Changes in the ways in which unexpected infant deaths are categorised by pathologists and coroners, and increasing reluctance to use the term 'sudden infant deathsyndrome', make assessment of nationally and internationally collected data on incidence potentially inaccurate and confusing. In this paper, we review current understanding of the epidemiology and aetiology of unexpected deaths in infancy, and current hypotheses on the pathophysiology of the processes that may lead to death. We also review interventions that have been adopted, with variable degrees of effectiveness in efforts to reduce the numbers of deaths, and new approaches that offer the possibility of prevention in the future.
 (37 references) (Author)

20150827-23

Neonatal nurses' beliefs, knowledge, and practices in relation to sudden infant death syndrome risk-reduction recommendations. Barsman SG, Dowling DA, Damato EG (2015), Advances in Neonatal Care vol 15, no 3, June 2015, pp 209-219

BACKGROUND:

Sudden infant death syndrome (SIDS) remains the third leading cause of infant death in the United States and the leading cause ofdeath beyond 1 month of age. In 2011, the American Academy of Pediatrics (AAP) released the newest SIDS risk-reduction recommendations, which address healthcare providers in neonatal intensive care units (NICUs). Little is known about neonatal nurses' SIDS prevention strategies since the release of these newest recommendations.
PURPOSE:
To assess neonatal nurses' beliefs, knowledge, and practices regarding SIDS prevention in both the NICU and step-down transitional care unit (TCU).
METHODS:
A prospective-descriptive design was used. The 33-item SIDS Risk-Reduction Questionnaire was distributed to a convenience sample of nurses in a level III NICU/TCU in the Midwest.
RESULTS:
Two hundred questionnaires were distributed; 96 (48%) were returned completed. Fifty-three percent of nurses strongly agreed that SIDS recommendations make a difference in preventing SIDS and 20% strongly believed that parents model SIDS prevention practices employed by staff. A majority of nurses correctly identified 2011 recommendations. Sixty-three percent of nurses often or always gave parents verbal information and 28% often or always gave parents written information regarding SIDS. Differences were seen between NICU and TCU nurses concerning beliefs andpractices, suggesting that TCU nurses more consistently follow SIDS recommendations.
IMPLICATIONS FOR PRACTICE:
Increased neonatal nursing and parental education regarding SIDS prevention and updated hospital policies promoting safe sleep are paramount.
IMPLICATIONS FOR RESEARCH:
Larger multicenter studies in level II/III NICUs are needed to provide further data on SIDS attitudes and practices. (30 references) (Author)

20150324-13*

Undiagnosed metabolic dysfunction and sudden infant death syndrome - a case-control study. Rosenthal NA, Currier RJ, Baer RJ, et al (2015), Paediatric and Perinatal Epidemiology vol 29, no 2, March 2015, pp 151-155

BACKGROUND: Decades of research has yielded few clues about causes of sudden infant death syndrome (SIDS). While some studies have shown a link to inborn errors of metabolism (IEMs), few have examined the link in a large population-based sample. This population-based case-control study assessed the association between undiagnosed IEMs and SIDS.
METHODS:
Children born in California during 2005-08 who died from SIDS were obtained from death records and linked to the newborn screening, birth certificate, and hospital discharge databases. Individuals with known chromosomal and neural tube defects, genetic disorders, and non-singleton births were excluded. Five controls were matched to each case on tandem mass spectrometry testing date and lab code. Rates of undiagnosed IEMs were compared between cases and controls using conditional logistic regression adjusting for known confounding factors.
RESULTS:
After adjusting for known confounding factors, SIDS cases had similar risk of having IEMs as controls (adjusted hazard ratio [HR] 1.3, 95% confidence interval [CI] 0.3, 5.5). Infants who were male, Black, and born preterm had higher risk of SIDS with the highest risk observed for those born preterm [adjusted HR = 1.7, 95% CI 1.3, 2.2]. Younger maternal age at delivery, mother being born in the US, parity after current birth >3, and delayed prenatal care were also significantly associated with higher risk of SIDS.
CONCLUSIONS:
While many maternal and infant factors are associated with an increased risk of SIDS, there is no evidence that undiagnosed IEMs are associated with increased risk. (Author)

20150302-64*

Could this little boy's tragic death provide clues about cot death? Tests reveal toddler had genetic heart condition never seen before in a child. Hull L, Davies M (2015), Daily Mail 27 February 2015

Full URL: http://www.dailymail.co.uk/health/article-2971965/Could-little-boy-s-tragic-death-provide-clues-cot-death-Tests-reveal-toddler-genetic-heart-condition-never-seen-child.html?ITO=1490&ns_mchannel=rss&ns_campaign=1490

Reports that scientists believe they are on the verge of a break-through into the causes of sudden infant death syndrome after a rare gene mutation that causes the heart to beat irregularly was discovered in the DNA of one-year-old Dexter Cook, whose parents found him dead in his cot. Explains that Gemma Littley and Graham Cook were so determined to find out why their son died that they agreed to participate in the pioneering genetic test, the results of which will be subject to further in-depth investigation. (Author)

20150123-21*

Sudden unexpected death in infancy - a collaborative thematic review 2010-2012. Child Death Review Programme and All Wales Perinatal Survey (2015), Swansea: Public Health Wales NHS Trust January 2015. 56 pages

Full URL: http://www.wales.nhs.uk/sitesplus/documents/888/Sudden%20Unexpected%20Deaths%20in%20Infancy%20-%20English.pdf

Report undertaken by the Child Death Review Programme and All Wales Perinatal Survey, which looks in detail at 45 unexplained sudden infant deaths in Wales. The report shows smoking to be a major risk factor, but premature birth, low birth weight, younger maternal age and parental alcohol consumption in the 24 hours prior to death were also found to be associated with the deaths reviewed. (CI)

20150107-11*

Sudden infant death syndrome: review for the obstetric care provider. Van Nguyen JM, Abenhaim HA (2013), American Journal of Perinatology vol 30, no 9, October 2013, pp 703-714

Sudden infant death syndrome (SIDS) is the leading cause of death among infants aged 1 to 12 months. In this article, we review risk factors that may predispose infants to increased vulnerability. Maternal characteristics, including nonmodifiable and modifiable factors, antenatal medical conditions, labor and delivery events, and infant characteristics, are reviewed, with the purpose of helping obstetric care providers target risk reduction efforts. We have reviewed over 85 case-control, retrospective, and prospective cohort studies published between 1975 and 2011. Major modifiable risk factors include maternal and paternal smoking, drug use, alcohol use, and insufficient prenatal care. Infants at increased risk include males, premature infants, infants of low birth weight or growth-restricted infants, and infants in multiple gestations. By targeting modifiable and nonmodifiable risk factors, it may be possible to decrease the incidence of SIDS. Efforts should be put on decreasing high-risk behaviors and encouraging sufficient antenatal follow-up. In view of recent increases in ethnic and social disparity with SIDS, it is essential that risk reduction guidelines, which have recently been expanded by the American Association of Pediatrics, be explained in a culturally sensitive manner. (Author)

20141217-3*

The role of physiological studies and apnoea monitoring in infants. Horne RSC, Nixon GM (2014), Paediatric Respiratory Reviews vol 15, no 4, 2014, pp 312-318

There is evidence that failure of cardio-respiratory control mechanisms plays a role in the final event of the Sudden Infant Death Syndrome (SIDS). Physiological studies during sleep in both healthy term born infants and those at increased risk for SIDS have been widely used to investigate how the major risk and protective factors for SIDS identified from epidemiological studies might alter infant physiology. Clinical polysomnography (PSG) in infants who eventually succumbed to SIDS however demonstrated abnormalities that were neither sufficiently distinctive nor predictive to support routine use of PSG for infants at risk for SIDS. PSG findings have also been shown to be not predictive of recurrence of Apparent Life Threatening Events (ALTE) and thus international guidelines state that PSG is not indicated for routine evaluation in infants with an uncomplicated ALTE, although PSG may be indicated when there

is clinical evidence of a sleep related breathing disorder. A decision to undertake home apnoea monitoring should consider the potential advantages and disadvantages of monitoring for that individual, in the knowledge that there is no evidence of the efficacy of such devices in preventing SIDS.
(Author)

20141217-2*

Infectious causes of sudden infant death syndrome. Alfelali M, Khandaker G (2014), Paediatric Respiratory Reviews vol 15, no 4, 2014, pp 307-311

Investigators have long suspected the role of infection in sudden infant death syndrome (SIDS). Evidence of infectious associations with SIDS is accentuated through the presence of markers of infection and inflammation on autopsy of SIDS infants and isolates of some bacteria and viruses. Several observational studies have looked into the relation between seasonality and incidence of SIDS, which often showed a winter peak. These all may suggest an infectious aetiology of SIDS. In this review we have summarised the current literature on infectious aetiologies of SIDS by looking at viral, bacterial, genetic and environmental factors which are believed to be associated with SIDS. (Author)

20141217-1*

Cardiac abnormalities and sudden infant death syndrome. Sweeting J, Semsarian C (2014), Paediatric Respiratory Reviews vol 15, no 4, 2014, pp 301-306

Many factors have been implicated in SIDS cases including environmental influences such as sleeping arrangements and smoking. Most recently, cardiac abnormalities have been hypothesised to play a role in some cases, particularly the primary genetic arrhythmogenic disorders such as familial long QT syndrome (LQTS). Both post-mortem and clinical studies of SIDS cases have provided supporting evidence for the involvement of cardiac genetic disorders in SIDS. This review provides a summary of this evidence focussing particularly on the primary hypothesis related to underlying familial LQTS. In addition, the current literature relating to other cardiac genetic conditions such as Brugada syndrome (BrS) and structural heart diseases such as hypertrophic cardiomyopathy (HCM) is briefly presented. Finally, the implications of a possible cardiac genetic cause of SIDS is discussed with reference to the need for genetic testing in SIDS cases and subsequent clinical and genetic testing in family members. (Author)

20141216-73*

Neurochemical abnormalities in the brainstem of the Sudden Infant Death Syndrome (SIDS). Machaalani R, Waters KA (2014), Paediatric Respiratory Reviews vol 15, no 4, 2014, pp 293-300

The brainstem has been a focus in Sudden Infant Death Syndrome (SIDS) research for 30 years. Physiological and animal model data show that cardiorespiratory, sleep, and arousal mechanisms are abnormal after exposure to SIDS risk factors or in infants who subsequently die from SIDS. As the brainstem houses the regulatory centres for these functions, it is the most likely site to find abnormalities. True to this hypothesis, data derived over the last 30 years shows that the brainstem of infants who died from SIDS exhibits abnormalities in a number of major neurotransmitter and receptor systems including: catecholamines, neuropeptides, acetylcholinergic, indole amines (predominantly serotonin and its receptors), amino acids (predominantly glutamate), brain derived neurotrophic growth factor (BDNF), and some cytokines. A pattern is emerging of particular brainstem nuclei being consistently affected including the dorsal motor nucleus of the vagus (DMNV), nucleus of the solitary tract (NTS), arcuate nucleus (AN) and raphe. We discuss the implications of these findings and directions that this may lead in future research. (Author)

20141216-72*

Sudden unexpected death in infancy: biological mechanisms. Galland BC, Elder DE (2014), Paediatric Respiratory Reviews vol 15, no 4, 2014, pp 287-292

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Sudden unexpected death in infancy (SUDI) covers both explained and unexplained deaths. Unexplained cases or SIDS are likely to have multiple neural mechanisms contributing to the final event. The evidence ranges from subtle physiological signs related to autonomic control, to findings at autopsy of altered neurotransmitter systems, including the serotonergic system, a network that has an extensive homeostatic role in cardio-respiratory and thermoregulatory control. Processes may be altered by the vulnerability of the infant due to age, poor motor ability, or a genetic predisposition. The fatal event may occur in response to an environmental stress. A single final physiological route to death seems unlikely. An understanding of the reasons for explained SUDI also reminds us that a thorough investigation is required after each death occurs. (Author)

20141117-3*

SIDS and infant sleep ecology. Ball HL, Russell CK (2014), Evolution, Medicine and Public Health 16 October 2014

Full URL: http://goo.gl/Jzq24a

Discusses whether Euro-American sleep ecology, which values early prolonged sleep with minimal awakenings used in conjunction with sleep aids such as dummies and swinging cradles or white noise, contributes to increased rates of sudden infant death in those societies. (5 references) (MB)

20141007-19*

Similarities and Differences in the Epidemiology of Pyloric Stenosis and SIDS. Lisonkova S, Joseph KS (2014), Maternal and Child Health Journal vol 18, no 7, 1 August 2014, pp 1721-1727

Similar temporal declines in infantile hypertrophic pyloric stenosis (IHPS) and sudden infant death syndrome (SIDS) and other common features have led to hypotheses about a shared etiology. We carried out a population-based study to highlight similarities and differences between IHPS and SIDS. We used vital statistics and hospitalization data on all live births in Washington State, USA (1987-2009). Changes in IHPS and SIDS rates over time were quantified using rate ratios with 95 % confidence intervals (CI). The duration between birth and diagnosis of IHPS or SIDS was examined as a function of gestational age at birth. Logistic regression analysis was used to identify risk factors and quantify adjusted temporal trends (2000-2008). Although both IHPS and SIDS rates declined significantly between 1987 and 2008, the patterns and magnitude of the declines (40 and 74 %, respectively) were different. IHPS and SIDS shared risk factors such as maternal smoking and single parent status but other factors showed qualitatively or and quantitatively different associations. Primiparity was a risk factor for IHPS [odds ratio (OR) 1.24, 95 % CI 1.09-1.41], and a protective factor for SIDS (OR 0.44, 95 % CI 0.36-0.55), while male sex had a stronger association with IHPS (OR 4.51, 95 % CI 3.85-5.28 vs 1.36, 95 % CI 1.13-1.64). Both IHPS and SIDS showed significant inverse associations between gestational age at birth and chronologic age at diagnosis/death. IHPS and SIDS share some epidemiologic features and risk factors but other risk factors have qualitatively or quantitatively different effects and recent temporal trends in the two diseases are dissimilar. (Author)

20140806-29

Long QT molecular autopsy in sudden infant death syndrome. Glengarry JM, Crawford J, Morrow PL, et al (2014), Archives of Disease in Childhood vol 99, no 7, July 2014, pp 635-640

OBJECTIVE:

To describe experience of long QT (LQT) molecular autopsy in sudden infant death syndrome (SIDS).

DESIGN:

Descriptive audit from two distinct periods: (1) A prospective, population-based series between 2006 and 2008 ('unselected'). (2) Before and after 2006-2008, with testing guided by a cardiac genetic service ('selected'). LQT genes 1, 2, 3, 5, 6 and 7 were sequenced. Next of kin were offered cardiac evaluation.

SETTING:

New Zealand.

PATIENTS:

102 SIDS cases.

INTERVENTIONS:

Nil.
MAIN OUTCOME MEASURES:
Detection of genetic variants.
RESULTS:
Maori 49 (47%), and Pacific island 24 (23%), infants were over-represented. Risk factors were common; bed sharing was reported in 49%. Rare genetic variants were commoner within the selected than unselected populations (5 of 31 infants (16%) vs 3 of 71 infants (4%) $p < 0.05$). In the selected population two infants had variants of definite or probable pathogenicity (KCNQ1, E146K; KCNH2, R1047L), two had novel variants of possible pathogenicity in SCN5A (I795F, F1522Y) and one had R1193Q in SCN5A, of doubtful pathogenicity. R1193Q was also the only variant in the three cases from the unselected population and occurred as a second variant with R1047L. Engaging families proved challenging. Only 3 of 8 (38%) variant-positive cases and 18 of 94 (19%) of variant-negative families participated in cardiac/genetic screening.
CONCLUSIONS:
LQT molecular autopsy has a very low diagnostic yield among unselected SIDS cases where risk factors are common. Diagnostic yield can be higher with case selection. Engagement of the family prior to genetic testing is essential to counsel for the possible uncertainty of the results and to permit family genotype-phenotype cosegregation studies. (38 references) (Author)

20140708-84*

Aquaporin-4 polymorphisms and brain/body weight ratio in sudden infant death syndrome (SIDS). Studer J, Bartsch C, Haas C (2014), Pediatric Research vol 76, no 1, 2014, pp 41-45

Background:Failure in the regulation of homeostatic water balance in the brain is associated with severe cerebral edema and increased brain weights and may also play an important role in the pathogenesis of sudden infant death syndrome (SIDS). We genotyped three single-nucleotide polymorphisms in the aquaporin-4 water channel-encoding gene (AQP4), which were previously shown to be associated with (i) SIDS in Norwegian infants (rs2075575), (ii) severe brain edema (rs9951307), and (iii) increased brain water permeability (rs3906956). We also determined whether the brain/body weight ratio is increased in SIDS infants compared with sex- and age-matched controls.
Methods:Genotyping of the three AQP4 single-nucleotide polymorphisms was performed in 160 Caucasian SIDS infants and 181 healthy Swiss adults using a single-base extension method. Brain and body weights were measured during autopsy in 157 SIDS and 59 non-SIDS infants. Results:No differences were detected in the allelic frequencies of the three AQP4 single-nucleotide polymorphisms between SIDS and adult controls. The brain/body weight ratio was similarly distributed in SIDS and non-SIDS infants. Conclusion:Variations in the AQP4 gene seem of limited significance as predisposing factors in Caucasian SIDS infants. Increased brain weights may only become evident in conjunction with environmental or other genetic risk factors. (Author)

20140702-23

Mitochondrial deoxyribonucleic acid may play a role in a subset of sudden infant death syndrome cases. Laer K, Vennemann M, Rothamel T, et al (2014), Acta Paediatrica vol 103, no 7, July 2014, pp 775-779

Aim:
It has been suggested that progressive adenosine triphosphate (ATP) depletion could play a key role in sudden infant death syndrome (SIDS). Because mitochondrial deoxyribonucleic acid (mtDNA) codes for a subset of essential genes for oxidative phosphorylation, we investigated 22 mtDNA polymorphisms in a large sample of Caucasian SIDS cases.
Methods:
A total of 774 samples were analysed, 365 from infant SIDS cases (mean age 131 days) and 409 from controls. These were investigated for the presence of 22 haplogroup-specific single nucleotide polymorphisms (SNPs), using a SNaPshot assay, a mini-sequencing assay that combines polymerase chain reaction (PCR) and sequencing.
Results:
No significant differences in assigned haplogroups could be detected between the groups. With regard to gender and

age, we found significant correlations for SNP positions 3010, 8251, 13 708, 14 470, 15 904 and 16 519. The most prominent result was the A allele in SNP 14 470 in male SIDS cases (p = 0.01).
Conclusion:
This is the largest study on mtDNA polymorphisms in SIDS to date, and our results indicate that mtDNA may play a role in a subset of SIDS cases. In order to complement these significant results, it is important to consider nuclear gene coding for mitochondrial proteins in future studies. (27 references) (Author)

20140603-74*

Cardio-respiratory control during sleep in infancy. Horne RSC (2014), Paediatric Respiratory Reviews vol 15, no 2, 2014, pp 163-169

During the first year of life and particularly the first 6 months autonomic control of the cardio-respiratory system is still undergoing maturation and infants are at risk of cardio-respiratory instability. These instabilities are most marked during sleep, which is important as infants spend the majority of each 24hours in sleep. Sleep state has a marked effect on the cardio-respiratory system with instabilities being more common in active sleep compared to quiet sleep. Responses to hypoxia are also immature during infancy and may make young infants more vulnerable to cardio-respiratory instability. It has been proposed that an inability to respond appropriately to a life threatening event underpins the Sudden Infant Death Syndrome (SIDS). The major risk factors for SIDS, prone sleeping and maternal smoking, both impair cardio-respiratory control in normal healthy term infants. (Author)

20140415-25

Genetic variation in the monoamine oxidase A and serotonin transporter genes in sudden infant death syndrome. Opdal SH, Vege A, Rognum TO (2014), Acta Paediatrica vol 103, no 4, April 2014, pp 393-397

AIM:
The purpose of this study was to investigate common polymorphisms in the genes encoding monoamine oxidase A (MAOA) and serotonin transporter (5-HTT) in Norwegian cases of sudden infant death syndrome (SIDS). This was done to further elucidate the role of genetic variation in these genes and SIDS.
METHODS:
A variable number of tandem repeat area in the promoter of the MAOA gene and rs25531 in the promoter region of the gene encoding 5-HTT were investigated in 193 SIDS cases and 335 controls. The methods used were polymerase chain reaction, restriction fragment analysis and gel electrophoresis.
RESULTS:
There were no differences between SIDS cases and controls for any of the investigated polymorphisms. This was also true when male and female SIDS cases were analysed separately.
CONCLUSION:
This article indicates that neither the VNTR in the promoter of the MAOA gene, nor rs25531 in the gene encoding 5-HTT, is involved in SIDS. However, as medullary serotonergic abnormalities most likely contribute to the death in at least some SIDS cases, it is important to investigate these genes, as well as other genes involved in the serotonergic network, in more detail. (30 references) (Author)

20140213-105

Is excess male infant mortality from sudden infant death syndrome and other respiratory diseases X-linked?. Mage DT, Donner EM (2014), Acta Paediatrica vol 103, no 2, February 2014, pp 188-193

AIM:
Male excess infant mortality is well known but unexplained. In 2004, we reported sudden infant death syndrome (SIDS) and other infant respiratory deaths showed a ~50% male excess in the United States between 1979 and 2002. This study analyses expanded US data from 1968 to 2010 to see whether infant respiratory deaths still show similar ~50% male excess and may be X-linked.
METHODS:
The analysis compared infant mortality data from the US Centers for Disease Control and Prevention, 1968-2010, with

11 World Health Organization International Classification of Diseases (ICD) rubric groups for respiratory deaths by accidents, congenital anomalies, respiratory diseases and causes unknown.
RESULTS:
The 11 ICD groupings presented male excesses of ~50% and combining the 453 953 US cases produced a male fraction of 0.6034, a 52.1% male excess. A further 72 380 non-US respiratory cases showed a similar 0.6055 male fraction, a 53.5% male excess.
CONCLUSION:
The constant ~50% male excess for quite different causes of respiratory death suggests they all have a common terminal event and that is acute anoxic encephalopathy. We hypothesise that this constant male excess phenomenon must be caused by a single X-linked gene, with a recessive condition, leading to a predisposition to succumb to acute anoxic encephalopathy. (30 references) (Author)

20140109-95*

QTc interval prolongation and severe apneas associated with a change in infant positioning. Ellsworth MA, Ulrich TJ, Carey WA, et al (2013), Pediatrics vol 132, no 6, December 2013. e1690-3

For more than a decade there has been considerable interest in the role of QT interval prolongation in the pathogenesis of sudden infant death syndrome. It has been proposed that the QT interval is a surrogate marker for autonomic instability and can be used to identify infants at risk for significant morbidity and mortality, including sudden infant death syndrome. We present the case of an infant that experienced a significant increase in his QTc, as detected by continuous QTc monitoring in the NICU after repositioning from a supine to prone position. This increase from a 413 ± 6 millisecond baseline average to 500 milliseconds was sustained for 2 hours and associated with clinically relevant apnea that ultimately required repositioning of the infant back to the supine position. Repositioning resulted in an immediate decrease of the QTc back to the previous baseline and termination of the apneic events. This case demonstrates an example of how the use of continuous QTc monitoring in the NICU setting may be used to detect QTc-accentuating factors in real time and identify situations that cause perturbations in an infant's autonomic nervous system. (Only the abstract is published in the print journal) (Author)

20140109-92*

Potential asphyxia and brainstem abnormalities in sudden and unexpected death in infants. Randall BB, Paterson DS, Haas EA, et al (2013), Pediatrics vol 132, no 6, December 2013. e1616-25

OBJECTIVE:
Sudden and unexplained death is a leading cause of infant mortality. Certain characteristics of the sleep environment increase the risk for sleep-related sudden and unexplained infant death. These characteristics have the potential to generate asphyxial conditions. We tested the hypothesis that infants may be exposed to differing degrees of asphyxia in sleep environments, such that vulnerable infants with a severe underlying brainstem deficiency in serotonergic, γ-aminobutyric acid-ergic, or 14-3-3 transduction proteins succumb even without asphyxial triggers (eg, supine), whereas infants with intermediate or borderline brainstem deficiencies require asphyxial stressors to precipitate death.
METHODS:
We classified cases of sudden infant death into categories relative to a 'potential asphyxia' schema in a cohort autopsied at the San Diego County Medical Examiner's Office. Controls were infants who died with known causes of death established at autopsy. Analysis of covariance tested for differences between groups.
RESULTS:
Medullary neurochemical abnormalities were present in both infants dying suddenly in circumstances consistent with asphyxia and infants dying suddenly without obvious asphyxia-generating circumstances. There were no differences in the mean neurochemical measures between these 2 groups, although mean measures were both significantly lower ($P < .05$) than those of controls dying of known causes.
CONCLUSIONS:

We found no direct relationship between the presence of potentially asphyxia conditions in the sleep environment and brainstem abnormalities in infants dying suddenly and unexpectedly. Brainstem abnormalities were associated with both asphyxia-generating and non-asphyxia generating conditions. Heeding safe sleep messages is essential for all infants, especially given our current inability to detect underlying vulnerabilities. (Only the abstract is published in the print journal) (Author)

20131104-10*

Functional single-nucleotide variant of HSPD1 in sudden infant death syndrome. Courts C, Grabmuller M, Madea B (2013), Pediatric Research vol 74, no 4, 2013, pp 380-383

Background: An insufficient stress response due to a genetically impaired heat shock protein (Hsp) could play a role in the pathogenesis in a subgroup of sudden infant death syndrome (SIDS) cases. Herein, we are the first to investigate whether a functionally impairing and thus pathogenic variant of the gene for Hsp60, encoded by HSPD1 (rs72466451), is correlated with the occurrence of SIDS. Methods:In a case-control study of a series of 133 cases of SIDS and 192 gender-matched German Caucasian control cases, the occurrence and distribution of the HSPD1 single-nucleotide variant (SNV) was analyzed using SNV genotyping by minisequencing. Results:The results show significantly increased frequency of the pathogenic variant of the HSPD1 SNV in a subgroup (4.5%) of SIDS cases. Conclusion:The results suggest that the pathogenic variant of rs72466451 may play a role in a subgroup of SIDS cases with impaired Hsp60-mediated stress response. (Author)

20130819-16*

Arousal from sleep pathways are affected by the prone sleeping position and preterm birth: Preterm birth, prone sleeping and arousal from sleep. Richardson HL, Horne SC (2013), Early Human Development vol 89, no 9, 2013, pp 705-711

OBJECTIVES:

Preterm infants exhibit depressed arousability from sleep when compared with term infants. As the final cortical element of the arousal process may be the most critical for survival, we hypothesized that the increased vulnerability of preterm infants to the Sudden Infant Death Syndrome (SIDS) could be explained by depressed cortical arousal (CA) responses. We evaluated the effects of preterm birth on stimulus-induced arousal processes in both the prone and supine sleeping positions.

STUDY DESIGN:

10 healthy preterm infants were studied with daytime polysomnography, in both supine and prone sleeping positions, at 36 weeks gestational age, 2-4 weeks, 2-3 months and 5-6 months post-term corrected age. Sub-cortical activations and cortical arousals (CA) were expressed as proportions of total arousal responses. Preterm data were compared with data from 13 healthy term infants studied at the same corrected ages.

RESULTS:

In preterm infants increased CAs were observed in the prone position at all ages studied. Compared to term infants, preterm infants had significantly fewer CAs in QS when prone at 2-3months of age and more CAs when prone at 2-4weeks in AS. There were no differences in either sleep state when infants slept supine.

CONCLUSIONS:

Prone sleeping promoted CA responses in healthy preterm infants throughout the first six months of post-term age. We have previously suggested that in term infants enhanced CA represents a critical protection against a potentially harmful situation; we speculate that for preterm-born infants the need for this protection is greater than in term infants. (Author)

20130730-43*

Sudden Infant Death Syndrome: cry characteristics. Robb MP, Crowell DH, Dunn-Rankin P (2013), International Journal of Pediatric Otorhinolaryngology vol 77, no 8, August 2013, pp 1263-1267

OBJECTIVE:

To acoustically evaluate the cries of SIDS infants and compare these cry features to a group of healthy term (HT)

infants, as well as previously published results for SIDS infants.
METHODS:
Pain-induced crying episodes were collected from four infants during the first weeks of life that later died of SIDS. Temporal and spectral features of each crying episode were characterized based on measures of cry duration, cry fundamental frequency (F0), and cry formant frequencies (F1 and F2).
RESULTS:
The SIDS infants were found to produce cries with longer duration compared to HT infants. The cries of SIDS infants also differed from HT infants in regard to the absolute difference in F2-F1 frequency.
CONCLUSIONS:
The acoustic features considered in the present study support the contention that the cries of SIDS infants are reflective of atypical respiratory-laryngeal control. Although research of this nature is rare, there is evidence to suggest an acoustic profile of crying that is specific to SIDS. (Author)

20130703-16*

Monoamine oxidase a gene polymorphism and the pathogenesis of sudden infant death syndrome. Courts C, Grabmuller M, Madea B (2013), Journal of Pediatrics vol 163, no 1, 2013, pp 89-93

OBJECTIVES:
To test the hypothesis that there is a significant association between functionally relevant allelic variants of the monoamine oxidase A (MAO-A) polymorphism and sudden infant death syndrome (SIDS).
STUDY DESIGN:
In a case-control study of 142 cases of SIDS and 280 sex-matched control cases, the distribution of allelic and genotype variants of a promoter polymorphism of the MAO-A gene was examined using polymerase chain reaction locus amplification and fluorescence based fragment length analysis.
RESULTS:
There was a significantly differential distribution of allelic and genotype variants between females with SIDS and controls. Moreover, there was a significant association between SIDS in females and allelic and genotype variants, each related to a higher transcriptional activity at the MAO-A locus.
CONCLUSIONS:
Our results suggest a role of MAO-A in female SIDS pathogenesis exerted by functionally relevant allelic and genotype variants of the MAO-A polymorphism. However, with the complex and inconsistent evidence available to date, the impact of the MAO-A promoter polymorphism on SIDS etiology remains unclear. (Author)

20130620-32

Prematurity and sudden infant death syndrome: United States 2005-2007. Malloy MH (2013), Journal of Perinatology vol 33, no 6, June 2013, pp 470-475

Objective:In 1987, the sudden infant death syndrome (SIDS) rate in the United States was 1.2 per 1000 live births. By the year 2005, the SIDS rate had dropped more than half to approximately 0.5 per 1000 live births. In 1987, the risk of SIDS was 2.32 times greater for extremely premature infants compared with term infants. The objective of this analysis was to determine if with the falling SIDS rate there has been a change in the risk for SIDS among preterm infants.Study Design:Data were obtained from the United States Linked Infant Birth and Death Certificate Public User Period files for the years 2005 to 2007. The adjusted odds ratios (ORs) for postneonatal out-of-hospital death by gestational age were determined by logistic regression modeling.Result:Over the 3-year period, there were 5203 postneonatal out-of-hospital deaths attributable to SIDS; 2010 attributable to other sudden deaths; 1270 attributable to suffocation in bed; and 3681 attributable to other causes. The adjusted OR for SIDS among the most preterm infants (24 to 28 weeks gestation) was significantly increased compared with term infants, ORadj=2.57 (95% confidence interval=2.08, 3.17), as were the adjusted ORs for the other causes of sudden infant death.Conclusion:Despite the marked drop in the incidence of SIDS since 1987, the risk for SIDS among preterm infants remains elevated. Other causes of sudden infant death for which SIDS is often mistaken reflect similar levels of increased risk among preterm infants. (39 references) (Author)

20130429-17*

Maternal alcohol use and sudden infant death syndrome and infant mortality excluding SIDS. O'Leary CM, Jacoby PJ, Bartu A, et al (2013), Pediatrics vol 131, no 3, March 2013. e770-8

BACKGROUND:
Improvements in the rate of infant mortality (death in first year of life) have not occurred in recent years. This study investigates the association between maternal alcohol-use disorder and sudden infant death syndrome (SIDS) and infant mortality not classified as SIDS using linked, population-based health and mortality data.
METHODS:
Exposed mothers were identified through the presence of an International Classification of Diseases 9/10 alcohol diagnosis, a proxy for alcohol-use disorder, recorded on health, mental health, and/or drug and alcohol datasets (1983-2005). Comparison mothers without an alcohol diagnosis were frequency matched to exposed mothers on maternal age within maternal race and year of birth of their children. All offspring with their birth recorded on the Midwives Notification System compose the exposed (n = 21 841) and comparison (n = 56 054) cohorts. Cases of SIDS (n = 303) and infant mortality excluding SIDS (n = 598) were identified through linkage with the Western Australian Mortality Register. Analyses were conducted by using Cox regression and results presented as adjusted hazard ratios (aHRs) and 95% confidence intervals (CIs).
RESULTS:
The highest risk of SIDS occurred when a maternal alcohol diagnosis was recorded during pregnancy (aHR 6.92, 95% CI 4.02-11.90) or within 1 year postpregnancy (aHR 8.61, 95% CI 5.04-14.69). An alcohol diagnosis recorded during pregnancy more than doubled the risk of infant deaths (excluding SIDS) (aHR 2.35, 95% CI 1.45-3.83). Maternal alcohol-use disorder is attributable for at least 16.41% (95% CI 9.73%-23.69%) of SIDS and 3.40% (95% CI 2.28%-4.67%) of infant deaths not classified as SIDS.
CONCLUSIONS:
Maternal alcohol-use disorder is a significant risk factor for SIDS and infant mortality excluding SIDS. (Author) (Only the abstract is published in the print journal)

20130403-21*

Midwives key to relaunched charity's life-saving ambitions. Bates F (2013), MIDIRS Essence April 2013

Full URL: http://www.midirs.org/2013/04/02/midwives-key-to-relaunched-charitys-life-saving-ambitions/

Francine Bates, chief executive of the Foundation for the Study of Infant Deaths (FSID) talks about why the charity is relaunching as The Lullaby Trust in April 2013 and explains how, with the support of the charity, midwives can play an invaluable role in helping to reduce the number of cot deaths. (MB)

20130307-12

Is there any correlation between HLA-DR expression in laryngeal mucosa and interleukin gene variation in sudden infant death syndrome?. Ferrante L, Opdal SH, Vege A, et al (2013), Acta Paediatrica vol 102, no 3, March 2013, pp 308-313

AIM: The mucosal immune system and cytokines are activated in a large proportion of cases of sudden infant death syndrome (SIDS). Our aim was to search for a possible association between cytokine polymorphisms and immune stimulation of the laryngeal mucosal in SIDS. METHODS: HLA-DR expression in laryngeal mucosal glands and surface epithelium in 97 SIDS victims was evaluated applying a semi-quantitative scoring system. The findings were related to cytokine gene polymorphisms as well as to the level of various cytokines in the cerebrospinal fluid (CSF). A risk score was established: a score of 0 prepresenting negative HLA-DR, supine position and no fever prior to death. RESULTS: The IL-6 -176CG/CC genotype was found in 92.3% of the SIDS cases with positive score for all risk factors (p = 0.01). Infants with high HLA-DR score had high levels of IL-6 in the cerebrospinal fluid (>30 µg/L) (p = 0.005). Furthermore, the IL-8 SNPs -781 CT/TT genotypes and -251 AA/AT genotypes were observed in 93% of the SIDS cases with one or more of the risk factors present compared with SIDS cases no risk factors reported (p = 0.003 and p = 0.016, respectively).

CONCLUSION: This study adds further evidence to the hypothesis that there are genetically associated disturbances of immunological homoeostasis in SIDS. (30 references) (Author)

20130227-10*

Maternal alcohol use and sudden infant death syndrome and infant mortality excluding SIDS. O'Leary CM, Jacoby PJ, Bartu A, et al (2013), Pediatrics 25 February 2013. Online version ahead of print

BACKGROUND: Improvements in the rate of infant mortality (death in first year of life) have not occurred in recent years. This study investigates the association between maternal alcohol-use disorder and sudden infant death syndrome (SIDS) and infant mortality not classified as SIDS using linked, population-based health and mortality data.
METHODS: Exposed mothers were identified through the presence of an International Classification of Diseases 9/10 alcohol diagnosis, a proxy for alcohol-use disorder, recorded on health, mental health, and/or drug and alcohol datasets (1983-2005). Comparison mothers without an alcohol diagnosis were frequency matched to exposed mothers on maternal age within maternal race and year of birth of their children. All offspring with their birth recorded on the Midwives Notification System compose the exposed (n = 21 841) and comparison (n = 56 054) cohorts. Cases of SIDS (n = 303) and infant mortality excluding SIDS (n = 598) were identified through linkage with the Western Australian Mortality Register. Analyses were conducted by using Cox regression and results presented as adjusted hazard ratios (aHRs) and 95% confidence intervals (CIs).
RESULTS: The highest risk of SIDS occurred when a maternal alcohol diagnosis was recorded during pregnancy (aHR 6.92, 95% CI 4.02-11.90) or within 1 year postpregnancy (aHR 8.61, 95% CI 5.04-14.69). An alcohol diagnosis recorded during pregnancy more than doubled the risk of infant deaths (excluding SIDS) (aHR 2.35, 95% CI 1.45-3.83). Maternal alcohol-use disorder is attributable for at least 16.41% (95% CI 9.73%-23.69%) of SIDS and 3.40% (95% CI 2.28%-4.67%) of infant deaths not classified as SIDS.
CONCLUSIONS: Maternal alcohol-use disorder is a significant risk factor for SIDS and infant mortality excluding SIDS. (Author)

20130221-15*

The development of autonomic cardiovascular control is altered by preterm birth. Yiallourou SR, Witcombe NB, Sands SA, et al (2013), Early Human Development vol 89, no 3, 2013, pp 145-152

OBJECTIVES:
Autonomic dysfunction, either sympathetic or parasympathetic, may explain the increased incidence of Sudden Infant Death Syndrome (SIDS) among preterm infants, as well as their subsequent heightened risk of hypertension in adulthood. As little is known about the development of autonomic function in preterm infants, we contrasted autonomic cardiovascular control across the first 6months after term-corrected age (CA) in preterm and term infants.
STUDY DESIGN:
Preterm (n=25) and age matched term infants (n=31) were studied at 2-4weeks, 2-3months and 5-6months CA using daytime polysomnography. Blood pressure and heart rate were measured during quiet (QS) and active (AS) sleep. Autonomic control was assessed using spectral indices of blood pressure and heart rate variability (BPV and HRV) in ranges of low frequency (LF, reflecting sympathetic+parasympathetic activity), high frequency (HF, respiratory-mediated changes+parasympathetic activity), and LF/HF ratio (sympatho-vagal balance).
RESULTS:
In preterm infants, HF HRV increased, LF/HF HRV decreased and LF BPV decreased with age ($p<0.05$); these changes were most evident in AS. Compared to term infants, preterm infants in QS exhibited lower LF, HF and total HRV at 5-6months; higher HF BPV at all ages; and lower LF BPV at 2-4weeks ($p<0.05$).
CONCLUSIONS:
With maturation, in preterm infants, parasympathetic modulation of the heart increases while sympathetic modulation of blood pressure decreases. Compared to term infants, preterm infants exhibit lesser parasympathetic modulation of the heart along with greater respiratory-mediated changes and lower sympathetic modulation of blood pressure. Impaired autonomic control in preterm infants may increase their risk of cardiovascular dysfunction later in life. (Author)

20130214-79*

Endorsing safe infant sleep. Hitchcock S (2012), Nursing for Women's Health vol 16, no 5, October/November 2012, pp 386-397

The American Academy of Pediatrics (AAP) safe sleep recommendations are considered best practice and are effective in preventing sudden infant death syndrome (SIDS). Yet studies have found that nurses' practice in newborn nurseries and neonatal intensive care units is often inconsistent with safe sleep recommendations. Such inconsistencies cause confusion and hinder SIDS prevention efforts. In 2011, the AAP added significant content to its 2005 safe sleep recommendations and neonatal nurses are now being asked to endorse the recommendations from birth. This article reviews the recommendations, examines barriers and controversies and offers suggestions for how an organization might initiate change and move toward a unified endorsement of safe sleep strategies. [Erratum: Nursing for Woemn's Health, vol 17, no 1, 2013, p 12.] (19 references) (Author)

20130111-22*

Carnitine palmitoyltransferase I and sudden unexpected infant death in British Columbia First Nations. Sinclair GB, Collins S, Popescu O, et al (2012), Pediatrics vol 130, no 5, November 2012. e1162-9

OBJECTIVE:

Infant mortality in British Columbia (BC) First Nations remains elevated relative to other residents. The p.P479L (c.1436C>T) variant of carnitine palmitoyltransferase 1 (CPT1A) is frequent in some aboriginal populations and may be associated with increased infant deaths. This work was initiated to determine the performance of acylcarnitine profiling for detecting this variant, to determine its frequency in BC, and to determine if it is associated with sudden infant deaths in this population.

METHODS:

Newborn screening cards from all BC First Nations infants in 2004 and all sudden unexpected deaths in BC First Nations infants (1999-2009) were genotyped for the CPT1A p.P479L variant and linked to archival acylcarnitine data.

RESULTS:

The CPT1A p.P479L variant is frequent in BC First Nations but is not evenly distributed, with higher rates in coastal regions (up to 25% homozygosity) with historically increased infant mortality. There is also an overrepresentation of p.P479L homozygotes in unexpected infant deaths from these regions, with an odds ratio of 3.92 (95% confidence interval: 1.69-9.00). Acylcarnitine profiling will identify p.P479L homozygotes with a 94% sensitivity and specificity.

CONCLUSIONS:

The CPT1A p.P479L variant is common to some coastal BC First Nations, and homozygosity for this variant is associated with unexpected death in infancy. The high frequency of this variant in a wide range of coastal aboriginal communities, however, suggests a selective advantage, raising the possibility that this variant may have differing impacts on health depending on the environmental or developmental context. (Author) (Only the abstract is published in the print journal)

20121109-97

Risk factors for early sudden deaths and severe apparent life-threatening events. Poets A, Urschitz MS, Steinfeldt R, et al (2012), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 97, no 6, November 2012, pp F395-F397

OBJECTIVE: To identify potential risk factors for unexpected sudden infant deaths (SID) and severe apparent life-threatening events (S-ALTE) within 24 h of birth. DESIGN: Case-control study embedded in an epidemiological survey over a 2-year period. PATIENTS AND METHODS: Throughout 2009, every paediatric department in Germany was asked to report cases of unexplained SID or S-ALTE in term infants with a 10-min Apgar score ≥8 to the Surveillance Unit for Rare Pediatric Conditions. Throughout 2010, the inclusion criteria were extended to infants ≥35 week gestational age and those where an explanation for the deterioration had been found. For each unexplained case, hospitals were asked to fill in a questionnaire for 3 (near-)term controls with good postnatal adaptation at the age (in minutes) when the event had occurred in the case under study. RESULTS: Of the 85 cases reported, 34 fulfilled the entry criteria; of these, two were near-term newborns and, in three cases, a cause had been identified for the event.

For the 31 cases with unknown cause for the event (13 males; mean (SD) gestational age 38.9 (1.7) week), the authors gathered 93 controls (51 male infants; 38.9 (1.4) week). As significant risk factors for S-ALTE and SID, the authors could identify primipara (OR 6.22; 95% CI 2.11 to 18.32) and potentially asphyxiating position (OR 6.45; 95% CI 1.22 to 34.10). CONCLUSIONS: Close observation of newborns seems necessary, particularly in primipara; a potentially asphyxiating position should be avoided. (8 references) (Author)

20121109-104

Organ volume measurements: comparison between MRI and autopsy findings in infants following sudden unexpected death. Prodhomme O, Seguret F, Martrille L, et al (2012), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 97, no 6, November 2012, pp F434-F438

OBJECTIVE: To assess the accuracy of a semiautomated 3D volume reconstruction method for organ volume measurement by postmortem MRI. METHODS: This prospective study was approved by the institutional review board and the infants' parents gave their consent. Postmortem MRI was performed in 16 infants (1 month to 1 year of age) at 1.5 T within 48 h of their sudden death. Virtual organ volumes were estimated using the Myrian software. Real volumes were recorded at autopsy by water displacement. The agreement between virtual and real volumes was quantified following the Bland and Altman's method. RESULTS: There was a good agreement between virtual and real volumes for brain (mean difference: -0.03% (-13.6 to +7.1)), liver (+8.3% (-9.6 to +26.2)) and lungs (+5.5% (-26.6 to +37.6)). For kidneys, spleen and thymus, the MRI/autopsy volume ratio was close to 1 (kidney: 0.87±0.1; spleen: 0.99±0.17; thymus: 0.94±0.25), but with a less good agreement. For heart, the MRI/real volume ratio was 1.29±0.76, possibly due to the presence of residual blood within the heart. The virtual volumes of adrenal glands were significantly underestimated (p=0.04), possibly due to their very small size during the first year of life. The percentage of interobserver and intraobserver variation was lower or equal to 10%, but for thymus (15.9% and 12.6%, respectively) and adrenal glands (69% and 25.9%). CONCLUSIONS: Virtual volumetry may provide significant information concerning the macroscopic features of the main organs and help pathologists in sampling organs that are more likely to yield histological findings. (15 references) (Author)

20121106-22*

Carnitine palmitoyltransferase I and sudden unexpected infant death in British Columbia First Nations. Sinclair GB, Collins S, Popescu O, et al (2012), Pediatrics 22 October 2012. Online version ahead of print

Full URL: http://pediatrics.aappublications.org/content/130/5/e1162

OBJECTIVE: Infant mortality in British Columbia (BC) First Nations remains elevated relative to other residents. The p.P479L (c.1436C>T) variant of carnitine palmitoyltransferase 1 (CPT1A) is frequent in some aboriginal populations and may be associated with increased infant deaths. This work was initiated to determine the performance of acylcarnitine profiling for detecting this variant, to determine its frequency in BC, and to determine if it is associated with sudden infant deaths in this population. METHODS: Newborn screening cards from all BC First Nations infants in 2004 and all sudden unexpected deaths in BC First Nations infants (1999-2009) were genotyped for the CPT1A p.P479L variant and linked to archival acylcarnitine data. RESULTS: The CPT1A p.P479L variant is frequent in BC First Nations but is not evenly distributed, with higher rates in coastal regions (up to 25% homozygosity) with historically increased infant mortality. There is also an overrepresentation of p.P479L homozygotes in unexpected infant deaths from these regions, with an odds ratio of 3.92 (95% confidence interval: 1.69-9.00). Acylcarnitine profiling will identify p.P479L homozygotes with a 94% sensitivity and specificity. CONCLUSIONS: The CPT1A p.P479L variant is common to some coastal BC First Nations, and homozygosity for this variant is associated with unexpected death in infancy. The high frequency of this variant in a wide range of coastal aboriginal communities, however, suggests a selective advantage, raising the possibility that this variant may have differing impacts on health depending on the environmental or developmental context. (Author)

20120919-68

No association of SIDS with two polymorphisms in genes relevant for the noradrenergic system: COMT and DBH. Klintschar M, Heimbold C (2012), Acta Paediatrica vol 101, no 10, October 2012, pp 1079-1082

Aim: Recent research suggests that genetic variance determining the strength of noradrenergic transmitting might contribute to the aetiology of SIDS. We have typed 2 functional polymorphisms of relevance for both biosynthesis and catabolism of noradrenalin: The Val158Met single-nucleotide polymorphism (SNP) of the Catechol-O-methyl transferase gene (COMT) and the 1021C/T SNP of the dopamine dehydroxylase gene (DBH). Methods: COMT and DBH were typed in 171 and 196 SIDS cases and 213 and 244 controls, respectively, using PCR followed by digestion with restriction enzymes. Typing was performed using a QIAxcel automatic electrophoresis unit. Results: Both SNPs were in Hardy-Weinberg equilibrium, and for none of these polymorphisms, an association with SIDS could be demonstrated. The allelic frequencies of the DBH locus were C: 78.32% and T: 21.68% in SIDS and C: 77.66% and T: 22.34% in controls. For the COMT locus, the allelic frequencies were A: 51.17% and G: 48.83% in SIDS and A: 52.82% and G: 47.18% in controls. Conclusion: Despite these negative results, the noradrenergic system is still an attractive candidate as modulator of SIDS risk to our eyes. There are several genes involved in this system that have not been studied up to now. (25 references) (Author)

20120904-19*

Altered placental development in pregnancies resulting in sudden infant death syndrome (SIDS). Widdows K, O'Malley A, O'Neill B, et al (2012), Early Human Development vol 88, no 10, 2012, pp 805-811

BACKGROUND: Sudden infant death syndrome (SIDS) is postulated to be a developmental disorder originating during fetal life in utero. Knowledge regarding the intrauterine environment in which SIDS infants develop is, however, inadequate and how the placenta develops prior to a SIDS event has not been studied. AIM: To investigate the morphological development of the placenta obtained from full-term infants who subsequently succumbed to SIDS. STUDY DESIGN: To estimate the percentage and total volumes of the chorionic villi and villous trophoblast membrane using stereological techniques. SUBJECTS: Placentas were obtained retrospectively from normal birthweight (SIDS-NBW n=18) and small-for-gestational age (SIDS-SGA, n=14) infants who had succumbed to SIDS, and compared to either control (n=8) or SGA placentas (n=7), respectively. RESULTS: SIDS-NBW placentas displayed evidence of augmented villous growth shown by significantly greater volumes of placental chorionic villi (gas-exchanging (GE) villi) in comparison to controls; this was not observed for SIDS-SGA placentas. However, both SIDS-NBW and SIDS-SGA placentas displayed significantly greater volumes of the cytotrophoblast (CT) (SIDS-NBW only), syncytiotrophoblast (SIDS-SGA only) and syncytial knots (SCT-K) and those displaying apoptotic syncytial nuclei (AP SCT-K). In contrast, SGA placentas displayed significantly reduced volumes of chorionic villi, GE villi and the villous trophoblast indicating a SIDS-specific effect associated with augmented placental growth. CONCLUSIONS: Our findings provide initial evidence that placental abnormality, although not necessarily causative, may precede a subset of SIDS cases supporting the hypothesis that the origins of SIDS begin during fetal life in utero. (Author)

20120711-34

Infant gender, shared sleeping and sudden death. Byard RW, Elliott J, Vink R (2012), Journal of Paediatrics and Child Health vol 48, no 6, June 2012, pp 517-519

Aim: To determine whether there is a gender imbalance in infant deaths in shared sleeping compared to solitary sleeping situations. Methods: Examination of autopsy reports of 133 infants aged between 7 and 364 days autopsied over a 19-year period from January 1991 to December 2009 was undertaken where death had either been attributed to SIDS, or had been classified as undetermined or unascertained. Cases were divided into two groups of solitary sleepers and shared sleepers, and the ratio of male to female cases was compared. Results: Ninety-five solitary sleepers were aged from 1 to 11 months (average 4.1 months) and consisted of 63 males (age range 1 to 11 months) and 32 females (age range 1 to 10 months). The 38 shared sleepers were aged from 1 week to 12 months (average 2.6 months) and consisted of 17 males (age range 2 weeks to 5 months) and 21 females (age range 1 week to 10 months). The male to female ratio in the solitary sleepers was approximately 2:1 and in the shared sleepers was 0.8:1, a statistically significant difference (p = 0.02). Conclusion: The lack of a male predominance typical of SIDS cases in infants who were sleeping with others, compared to those who were sleeping alone, suggests that these situations may be different. It is possible, therefore, that different lethal mechanisms may be involved in some shared sleeping situations. (22 references) (Author)

20120516-11*

Risk factor changes for sudden infant death syndrome after initiation of Back-to-Sleep campaign. Trachtenberg FL, Haas EA, Kinney HC, et al (2012), Pediatrics vol 129, no 4, April 2012, pp 630-638

OBJECTIVE: To test the hypothesis that the profile of sudden infant death syndrome (SIDS) changed after the Back-to-Sleep (BTS) campaign initiation, document prevalence and patterns of multiple risks, and determine the age profile of risk factors. METHODS: The San Diego SIDS/Sudden Unexplained Death in Childhood Research Project recorded risk factors for 568 SIDS deaths from 1991 to 2008 based upon standardized death scene investigations and autopsies. Risks were divided into intrinsic (eg, male gender) and extrinsic (eg, prone sleep). RESULTS: Between 1991-1993 and 1996-2008, the percentage of SIDS infants found prone decreased from 84.0% to 48.5% (P < .001), bed-sharing increased from 19.2% to 37.9% (P < .001), especially among infants <2 months (29.0% vs 63.8%), prematurity rate increased from 20.0% to 29.0% (P = .05), whereas symptoms of upper respiratory tract infection decreased from 46.6% to 24.8% (P < .001). Ninety-nine percent of SIDS infants had at least 1 risk factor, 57% had at least 2 extrinsic and 1 intrinsic risk factor, and only 5% had no extrinsic risk. The average number of risks per SIDS infant did not change after initiation of the BTS campaign. CONCLUSIONS: SIDS infants in the BTS era show more variation in risk factors. There was a consistently high prevalence of both intrinsic and especially extrinsic risks both before and during the Back-to-Sleep era. Risk reduction campaigns emphasizing the importance of avoiding multiple and simultaneous SIDS risks are essential to prevent SIDS, including among infants who may already be vulnerable. (Author) (Full article available online at http://www.pediatrics.org/cgi/doi/10.1542/peds.2011-1419)

20120417-28

Temporal trends in sudden infant death syndrome in Canada from 1991 to 2005: contribution of changes in cause of death assignment practices and in maternal and infant characteristics. Gilbert NL, Fell DB, Joseph KS, et al (2012), Paediatric and Perinatal Epidemiology vol 26, no 2, March 2012, pp 124-130

The rate of sudden infant death syndrome (SIDS) declined significantly in Canada and the US between the late 1980s and the early 2000s. In the US, this decline was shown to be due in part to a shift in diagnosis, as deaths from accidental suffocation and strangulation in bed and from other ill-defined and unspecified cause increased concurrently. This study was undertaken to determine whether there was such a shift in diagnosis from SIDS to other causes of death in Canada, and to quantify the true temporal decrease in SIDS. Cause-specific infant death rates were compared across three periods: 1991-95, 1996-2000 and 2001-05 using the Canadian linked livebirth-infant death file. The temporal decline in SIDS was estimated after adjustment for maternal and infant characteristics such as maternal age and small-for-gestational age using logistic regression. Deaths from SIDS decreased from 78.4 [95% confidence interval (CI) 73.4, 83.4] per 100 000 livebirths in 1991-95, to 48.5 [95% CI 44.3, 52.7] in 1996-2000 and to 34.6 [95% CI 31.0, 38.3] in 2001-05. Mortality rates from other ill-defined and unspecified causes and accidental suffocation and strangulation in bed remained stable. The temporal decline in SIDS between 1991-95 and 2001-05 did not change substantially after adjustment for maternal and infant factors. It is unlikely that the temporal decline of SIDS in Canada was due to changes in cause-of-death assignment practices or in maternal and infant characteristics. (20 references) (Author)

20120125-21

Infant care practices related to sudden infant death syndrome in South Asian and White British families in the UK. Ball HL, Moya E, Fairley L, et al (2012), Paediatric and Perinatal Epidemiology vol 26, no 1, January 2012, pp 3-12

In the UK, infants of South Asian parents have a lower rate of sudden infant death syndrome (SIDS) than White British infants. Infant care and life style behaviours are strongly associated with SIDS risk. This paper describes and explores variability in infant care between White British and South Asian families (of Bangladeshi, Indian or Pakistani origin) in Bradford, UK (the vast majority of which were Pakistani) and identifies areas for targeted SIDS intervention. A cross-sectional telephone interview study was conducted involving 2560 families with 2- to 4-month-old singleton infants enrolled in the Born in Bradford cohort study. Outcome measures were prevalence of self-reported practices in infant sleeping environment, sharing sleep surfaces, breast feeding, use of dummy or pacifier, and life style

behaviours. We found that, compared with White British infants, Pakistani infants were more likely to: sleep in an adult bed (OR = 8.48 [95% CI 2.92, 24.63]); be positioned on their side for sleep (OR = 4.42 [2.85, 6.86]); have a pillow in their sleep environment (OR = 9.85 [6.39, 15.19]); sleep under a duvet (OR = 3.24 [2.39, 4.40]); be swaddled for sleep (OR = 1.49 [1.13, 1.97]); ever bed-share (OR = 2.13 [1.59, 2.86]); regularly bed-share (OR = 3.57 [2.23, 5.72]); ever been breast-fed (OR = 2.00 [1.58, 2.53]); and breast-fed for 8+ weeks (OR = 1.65 [1.31, 2.07]). Additionally, Pakistani infants were less likely to: sleep in a room alone (OR = 0.05 [0.03, 0.09]); use feet-to-foot position (OR = 0.36 [0.26, 0.50]); sleep with a soft toy (OR = 0.52 [0.40, 0.68]); use an infant sleeping bag (OR = 0.20 [0.16, 0.26]); ever sofa-share (OR = 0.22 [0.15, 0.34]); be receiving solid foods (OR = 0.22 [0.17, 0.30]); or use a dummy at night (OR = 0.40 [0.33, 0.50]). Pakistani infants were also less likely to be exposed to maternal smoking (OR = 0.07 [0.04, 0.12]) and to alcohol consumption by either parent. No difference was found in the prevalence of prone sleeping (OR = 1.04 [0.53, 2.01]). Night-time infant care therefore differed significantly between South Asian and White British families. South Asian infant care practices were more likely to protect infants from the most important SIDS risks such as smoking, alcohol consumption, sofa-sharing and solitary sleep. These differences may explain the lower rate of SIDS in this population. (28 references) (Author)

20111031-20*

SIDS-related knowledge and infant care practices among Māori mothers. Tipene-Leach D, Hutchison L, Tangiora A, et al (2010), New Zealand Medical Journal vol 123, no 1326, 26 November 2010, u 4445

Aim: Māori have high SIDS rates and relevant information is needed to craft appropriate prevention strategies. The aim of the study was to determine what Māori mothers know about SIDS prevention, and to determine their SIDS-related child care practices. Methods: Māori mothers who gave birth in the Counties Manukau District Health Board area were surveyed about their SIDS related knowledge, and infant care practices and their reasons for using and their concerns about these practices. Results were compared with a similar 2005 survey of a largely European sample. Results: Knowledge of Māori mothers about SIDS prevention was much lower than for European mothers. More Māori infants slept prone and Māori mothers stopped breastfeeding significantly earlier. Although co-sleeping rates were similar, bedsharing increased to 65% for some part of the night. In addition, more than half of the Māori mothers had smoked in pregnancy and 21% of them were sharing a bed with their infant. Potentially unsafe soft objects such as rolled blankets or pillows were used by a third of mothers to help maintain the sleep position. Conclusions: Māori mothers have a poorer knowledge of SIDS prevention practices. The high rate of maternal smoking, the early cessation of breastfeeding, and co-sleeping where there was smoking in pregnancy were also areas of concern. Appropriate health promotion measures need to be developed for the high-risk Māori community. (18 references) (Author)

20111003-12313*

Risk factors for SIDS. Results of the National Institute of Child Health and Human Development SIDS Cooperative Epidemiological Study. Hoffman HJ, Damus K, Hillman L, et al (1988), Annals of the New York Academy of Sciences vol 533, 1988, pp 13-30

No abstract available.

20111003-11675*

The role of infection and inflammation in sudden infant death syndrome. Blood-Siegfried J (2009), Immunopharmacology and Immunotoxicology vol 31, no 4, 2009, pp 516 -23

Sudden Infant Death Syndrome (SIDS) is the most common cause of post-neonatal mortality in the developed world. The exact cause of SIDS is likely to be multifactorial involving a critical developmental period, a vulnerable infant, and one or more triggers. Many SIDS infants have a history of viral illness preceding death. Prone sleep position, one of the leading risk factors, can increase airway temperature, as well as stimulate bacterial colonization and bacterial toxin production. Markers of infection and inflammation are often found on autopsy along with microbial isolates. Although the causal link between infection and SIDS is not conclusive, there is evidence that an infectious insult could be a likely trigger of SIDS in some infants

20110909-16

Breastfeeding and reduced risk of sudden infant death syndrome: a meta-analysis. Hauck FR, Thompson JMD, Tanabe KO, et al (2011), Pediatrics vol 128, no 1, July 2011, pp 103-110

Full URL: http://pediatrics.org/cgi/doi/10.1542/peds.2010-3000

CONTEXT: Benefits of breastfeeding include lower risk of postneonatal mortality. However, it is unclear whether breastfeeding specifically lowers sudden infant death syndrome (SIDS) risk, because study results have been conflicting. OBJECTIVE: To perform a meta-analysis to measure the association between breastfeeding and SIDS. METHODS: We identified 288 studies with data on breastfeeding and SIDS through a Medline search (1966-2009), review articles, and meta-analyses. Twenty-four original case-control studies were identified that provided data on the relationship between breastfeeding and SIDS risk. Two teams of 2 reviewers evaluated study quality according to preset criteria; 6 studies were excluded, which resulted in 18 studies for analysis. Univariable and multivariable odds ratios were extracted. A summary odds ratio (SOR) was calculated for the odds ratios by using the fixed-effect and random-effect inverse-variance methods of meta-analysis. The Breslow-Day test for heterogeneity was performed. RESULTS: For infants who received any amount of breast milk for any duration, the univariable SOR was 0.40 (95% confidence interval [CI]: 0.35-0.44), and the multivariable SOR was 0.55 (95% CI: 0.44-0.69). For any breastfeeding at 2 months of age or older, the univariable SOR was 0.38 (95% CI: 0.27-0.54). The univariable SOR for exclusive breastfeeding of any duration was 0.27 (95% CI: 0.24-0.31). CONCLUSIONS: Breastfeeding is protective against SIDS, and this effect is stronger when breastfeeding is exclusive. The recommendation to breastfeed infants should be included with other SIDS risk-reduction messages to both reduce the risk of SIDS and promote breastfeeding for its many other infant and maternal health benefits. (54 references) (Author) (Full article available online at http://pediatrics.org/cgi/doi/10.1542/peds.2010-3000)

20110614-3*

Protect your children from SIDS by breast feeding new study shows. (2011), Medical News Today 13 June 2011

Reports that new research has shown that infants who receive any amount of breast milk have a significantly reduced risk of Sudden Infant Death Syndrome. Among infants who were exclusively breastfed, the risk was reduced by 73%. (CI)

20110330-48

The brain-derived neutrophic factor val66met polymorphism and sudden unexpected infant death. Opdal H, Melien O, Hynnekleiv T, et al (2011), Acta Paediatrica vol 100, no 1, January 2011, pp 86-89

AIM: Findings of hypoxia prior to death and involvement of a dysregulation of the serotonergic network in sudden infant death syndrome (SIDS) may indicate that brain-derived neutrophic factor (BDNF) also is of importance with regard to sudden unexpected infant death. Based on this, the purpose of this study was to investigate the BDNF val66met polymorphism in SIDS cases, cases of infectious death and controls. METHODS: The polymorphism was investigated in 163 SIDS cases, 34 cases of infectious death and 121 controls, using real-time PCR and fluorescence melting curve analysis. RESULTS: There were no differences in val66met genotype distribution between neither the SIDS cases nor the cases of infectious death and controls ($p = 0.95$ and $p = 0.52$, respectively). CONCLUSION: The study indicates that the val66met polymorphism is not important for sudden unexpected infant death. However, several other SNPs in the BDNF gene, as well as in other genes involved in this pathway, including G-protein, have to be investigated to fully exclude any involvement of BDNF in SIDS. (24 references) (Author)

20110318-15*

[Foundation for the Study of Infant Deaths (FSID) responds to criticism of Eastenders' baby swap plotline]. (2011), FSID E-Newsletter 18 March 2011

Defends the FSIDs decision to assist the BBC with the Eastenders' controversial storyline involving the swapping of a baby who had suffered sudden infant death with a live baby. Explains that the FSID accepted the BBCs invitation to

give advise on the plotline, which had been fixed by the BBC months before the FSIDs involvement, because of the importance of an accurate portrayal of the grief suffered by Kat, one of the central characters in the story, and the practical events and professional interventions which would inevitably take place. (JSM)

20110215-25

Variant interleukin 1 receptor antagonist gene alleles in sudden infant death syndrome. Highet AR, Gibson CS, Goldwater PN (2011), Archives of Disease in Childhood vol 95, no 12, December 2010, pp 1009-1012

OBJECTIVE: To investigate if carriage of interleukin 1 (IL-1) receptor antagonist gene variants are associated with sudden infant death syndrome (SIDS) in a large cohort of case-control demographically matched infants. DESIGN: 118 SIDS and 233 control infants, who were matched to each SIDS infant by date of birth, sex, birth weight (±500 g), gestational age and ethnicity, were genotyped for an IL-1RN 89 bp tandem repeat polymorphism and analysed for significant associations. RESULTS: No significant difference in genotype frequencies was observed between low and normal birthweight infants and year of birth (1987-1994, when the SIDS incidence was higher). In infants born between 1987 and 1994, an association was observed with SIDS and allele 2 where 18% of SIDS infants carried the 2/2 genotype compared with 9% of controls (χ(2) p=0.026, OR 2.46). Allele 3 was found at a low frequency, but was significantly more common in SIDS infants (3.1%) compared with controls (0.9%, Fisher's exact p=0.04, OR 3.76). CONCLUSION: The higher prevalence of IL-1RN allele 2, which predisposes to poor outcomes from infection, in SIDS infants born between 1987 and 1994 (ie, prior to the dramatic decrease in SIDS incidence) suggests that the high incidence during this period could point to infection playing a role in aetiology. An association of IL-1RN allele 3 with SIDS was also found, but should be interpreted with caution due to the low frequency of this variant. The consequence of allele 3 carriage is currently unknown in the absence of functionality studies for this isoform. (26 references) (Author)

20101209-24*

Serotonin May Be the Key to SIDS. Thompson D (2010), Healthfinder 8 December 2010

Suggests that various recent research projects indicate that a lack of the hormone serotonin may be a key factor in Sudden infant death syndrome (SIDS) as it is thought that it is involved in waking a baby if it is becoming hypoxic. Lists common contributing factors for SIDS and recommendations which may help to prevent it. (JR)

20101202-31*

Investigating sudden unexpected death in infancy and early childhood. Cote A (2010), Paediatric Respiratory Reviews vol 11, no 4, 2010, pp 219-225

Sudden unexpected death is one of the most frequent ways of dying in the first year of life after the neonatal period. It is however, much less frequent after the first birthday. Investigations into the cause of death are very important, for a significant proportion of these sudden deaths can be explained only after a thorough investigation. Of the causes identified, infection is the most frequent cause; metabolic disorders and cardiovascular diseases play a role as well, although the proportion of cases is much smaller. There is now evidence that cardiac channel gene mutations also play an important role; however, identification of these conditions relies on costly testing that is not readily or widely available. The physician's role as primary care provider is critical in ensuring that families understand the results of the investigation into their child's death. It is important that everything be done to identify the cause of death so that no such tragedy recurs in the same family. (Author)

20101201-15*

Lack of association of the serotonin transporter polymorphism with the sudden infant death syndrome in the San Diego Dataset. Paterson DS, Rivera KD, Broadbelt KG, et al (2010), Pediatric Research vol 68, no 5, 2010, pp 409-413

Dysfunction of medullary serotonin (5-HT)-mediated respiratory and autonomic function is postulated to underlie the pathogenesis of the majority of sudden infant death syndrome (SIDS) cases. Several studies have reported an increased frequency of the LL genotype and L allele of the 5-HT transporter (5-HTT) gene promoter polymorphism

(5-HTTLPR), which is associated with increased transcriptional activity and 5-HT transport in vitro, in SIDS cases compared with controls. These findings raise the possibility that this polymorphism contributes to or exacerbates existing medullary 5-HT dysfunction in SIDS. In this study, we tested the hypothesis that the frequency of LL genotype and L allele are higher in 179 SIDS cases compared with 139 controls of multiple ethnicities in the San Diego SIDS Dataset. We observed no significant association of genotype or allele with SIDS cases either in the total cohort or on stratification for ethnicity. These observations do not support previous findings that the L allele and/or LL genotype of the 5-HTTLPR are associated with SIDS. (Author)

20100805-81

Teaching resuscitation to parents. Warwood G (2010), In: Lumsden H and Holmes D editors. Care of the newborn by ten teachers. London: Hodder Arnold 2010, pp 168-177

Discusses the provision and timing of training for parents and carers in basic infant life support. Includes the guidelines for parents issued by the Foundation for the Study of Infant Deaths, which are designed to reduce the risks thought to be associated with unexplained infant deaths. Details the four stages of training that should be included in parent education sessions. (7 references) (CR)

20100802-5*

Surprising gender difference discovered In SIDS study. Medical News Today (2010), Medical News Today 2 August 2010

News item reporting on a study published in the journal Sleep [1] which has found that male infants are easier to arouse from quiet sleep at 2 to 4 weeks of age than female infants, but that by 2 to 3 months of age, there are no differences in arousability. The findings suggest that there are no gender difference in arousability that make male infants more likely susceptible than females to sudden infant death. 1. Richards HL. Sleeping like a baby - does gender influence infant arousability? Sleep, vol 33, no 8, 2010, pp 1055-1060. (MB)

20100802-3*

Sleeping like a baby - does gender influence infant arousability?. Richards HL, Walker AM, Horne RSC (2010), Sleep vol 33, no 8, 2010, pp 1055-1060

Introduction: Victims of the sudden infant death syndrome (SIDS) may have preexisting abnormalities in their arousal pathways, inhibiting the progression of subcortical activation (SCA) to full cortical arousal (CA). Approximately 60% of SIDS victims are male, and it has been suggested that male infants have delayed cortical maturation compared to females. We hypothesized that CA frequency would be lower and CA threshold would be higher in male infants during both active (AS) and quiet (QS) sleep. Methods: 50 healthy term infants (21 male, 29 female) were studied with daytime polysomnography at 2-4 weeks and 2-3 months after birth. Arousal from sleep was induced using a pulsatile air-jet to the nostrils at increasing pressures. Results: At 2-4 weeks, arousability from AS was similar in males and females, however during QS, male infants required a lower stimulus to induce SCA and CA. This gender difference in arousal threshold was not observed at 2-3 months. CA frequencies were similar between genders during both sleep states at both ages, though overall, CA was more frequent in AS than in QS. Conclusions: This study demonstrated that at 2-4 weeks, male infants were easier to arouse than female infants during QS. There were no significant effects of gender on total arousability or SCA and CA frequencies at 2-3 months, the age of peak SIDS incidence. Thus, although male infants are at greater risk of SIDS than female infants, this difference is unlikely to be associated with gender differences in CA threshold or frequency. (Author)

20100723-46*

Aquaporin-4 gene variation and sudden infant death syndrome. Opdal SH, Vege A, Stray-Pedersen A, et al (2010), Pediatric Research vol 68, no 1, 2010, pp 48-51

The purpose of this study was to investigate the aquaporin-4 (AQP4) gene in cases of sudden infant death syndrome (SIDS) and controls and to elucidate the hypothesis that a genetically determined disturbed water homeostasis in the brain is involved as a predisposing factor in SIDS. The single nucleotide polymorphisms (SNPs) rs2075575, rs4800773,

rs162004, and rs3763043 in the AQP4 gene were investigated in 141 SIDS cases and 179 controls. For each SIDS case, a brain/body weight ratio was calculated. The study revealed an association between the T allele and the CT/TT genotypes of rs2075575 and SIDS (C versus T, $p < 0.01$; CC versus CT/TT, $p = 0.03$). For the other three investigated SNPs, there were no differences in genotype frequencies between SIDS cases and controls. For the SNP rs2075575, it was also found an association between brain/body weight ratio and genotype in the SIDS cases aged 0.3-12 wk ($p = 0.014$, median ratio CC 10.6, CT/TT 12.1). In conclusion, this study indicates that rs2075575 may be of significance as a predisposing factor for SIDS, and that the CT/TT genotypes are associated with an increased brain/body weight ratio in infants dying from SIDS during the vulnerable period from birth up to 3 mo of age. (Author)

20100719-3*

Breathing regulated by bright stars of the brain. (2010), Medical News Today 17 July 2010

Explains how a new research finding may have significance in understanding respiratory failure in sudden infant deaths. Describes how brains cells called Astrocytes, so named because of their characteristic star shape, have a central role in breathing regulation, sensing the levels of carbon dioxide in the blood and then causing breathing to increase in accord with prevailing metabolism and activity, by activating brain neuronal respiratory networks. (JSM)

20100623-6

Sudden unexpected deaths in infancy part I: the phenomena of sudden and unexplained infant death. Crawford D (2010), Journal of Neonatal Nursing vol 16, no 3, June 2010, pp 104-110

The phenomena of sudden and unexpected infant deaths has been extensively studied and is informed by an increasingly large literature base. This article has reviewed and summarized some of the more credible areas of study and seeks to make the evidence more accessible to influence practices within the nursing domain. (61 references) (Author)

20100325-84

Cytokine gene polymorphisms and sudden infant death syndrome. Ferrante L, Opdal SH, Vege A, et al (2010), Acta Paediatrica vol 99, no 3, March 2010, pp 384-388

Aim: Several studies indicate that the mucosal immune system is stimulated in cases of sudden infant death syndrome (SIDS), and our hypothesis is that this immune reaction is because of an unfavourable combination of functional polymorphisms in the cytokine genes. Methods: Thus, in this study, single nucleotide polymorphisms (SNPs) in the genes encoding IL-6, IL-8, IL-12, IL-13, IL-16, IL-18 and IFNγ were investigated in 148 SIDS cases, 56 borderline SIDS cases, 41 cases of infectious death and 131 controls. Results: Regarding genotype distribution, no differences between the investigated groups were found. However, in the SIDS group, the genotypes IL-8 −251AA/AT and IL-8 −781CT/TT were significantly more frequent in the SIDS cases found dead in a prone sleeping position, compared with SIDS cases found dead in other sleeping positions. In addition, there was an association between fever prior to death and the genotype IL-13 +4464GG in the cases of infectious death. Conclusion: This study indicates that specific interleukin genotypes are a part of a genetic make up that make infants sleeping prone at risk for SIDS. (28 references) (Author)

20100308-19*

Cot death breakthrough could help pinpoint babies at risk. Willsher K (2010), Guardian 5 March 2010

Full URL: http://www.guardian.co.uk/lifeandstyle/2010/mar/05/cot-death-breakthrough

News item reporting research from France which claims that sudden infant death syndrome could be caused by a fault in the regulation of the heartbeat, causing it to slow down to the point where it stops altogether. The researchers believe this could lead to a blood test to identify the newborn infants most at risk of cot death. (TC)

20100211-15

Impact of sodium/proton exchanger 3 gene variants on sudden infant death syndrome. Poetsch M, Nottebaum BJ, Wingenfeld L, et al (2010), Journal of Pediatrics vol 156, no 1, January 2010, pp 44-48

OBJECTIVE: To determine the contribution of variations in the sodium/proton exchanger 3 (NHE3) gene in sudden infant death syndrome (SIDS). STUDY DESIGN: Variations in the exons and promoter of the NHE3 gene were analyzed with direct sequencing analysis and mini sequencing (SNaPshot analysis) in 251 cases of SIDS, plus 50 infant control subjects who had died of other causes, and 170 healthy adults. RESULTS: The C2405T variant (exon 16) and 2 polymorphisms in the promoter (G1131A and C1197T) were encountered significantly more frequently in cases of SIDS than in control subjects. At least 1 of these 3 variants was detected in 49% of SIDS cases, but only in 30% of control subjects. CONCLUSIONS: Our findings suggest the involvement of polymorphisms in the NHE3 gene and promoter in cases of SIDS, which may result in an overexpression of NHE3 in the medulla oblongata and which possibly leads to a disturbance in breathing control. Furthermore, our results underline the heterogeneous character of SIDS. (33 references) (Author)

20100210-10

Sudden infant death syndrome during low incidence in Sweden 1997-2005. Mollborg P, Alm B (2010), Acta Paediatrica vol 99, no 1, January 2010, pp 94-98

BACKGROUND: Following the change from prone to supine in preferred sleeping position, the incidence of Sudden Infant Death Syndrome (SIDS) in Sweden fell from 1.1 per 1000 live births in 1992 to 0.41 in 1995. After a further small decline, we have been experiencing a plateau at around 0.25 since 2000. AIM: To identify the changes that have occurred in the epidemiology of SIDS since the end of the Nordic Epidemiological SIDS Study in 1995. METHODS: Data from the Medical Birth Register of Sweden, covering the years 1995-2005, were used. Sleeping position is not included in the register. Results: The incidence of SIDS has remained low in Sweden. Independent risk factors were smoking during early pregnancy, parents not living together, low maternal age, high parity and short gestational age. The odds ratio for smoking has continued to increase and the median age of death has continued to decrease since the previous study. We found no signs of seasonality in the current material. CONCLUSIONS: Age at death continued to decrease. The high incidence during weekends persisted. Seasonality was not significant. There was no evidence of a changing effect from risk factors in the studied period. (21 references) (Author)

20100204-35*

Mood controller linked to cot death. Callaway E (2010), New Scientist 3 February 2010

Explains that scientists have that serotonin, the chemical which controls sleep, breathing and heart-rate in babies, is found in lower levels in those who have died of cot death. Reports that the researchers who made this discovery have offered hope of a test to predict which infants are at greater risk of cot death. (JSM)

20100204-19*

SIDS babies have low serotonin levels, study finds. Bonfield J (2010), CNN 3 February 2010

Full URL: http://edition.cnn.com/2010/HEALTH/02/03/sids.serotonin/index.html

Reports on a study which has found that babies who died from cot death have 26% lower levels of the brain chemical serotonin in their brainstems than babies who died from other causes, and also had lower levels of tryptophan hydroxylase, the enzyme necessary to produce serotonin. Explains that serotonin regulates sleep, heart-rate and breathing in babies. Gives recommendations on sleeping position to promote uninterrupted breathing, and lessen the chances of cot death: babies should be placed on their backs to sleep so that they don't roll over, they should not be over 'bundled', and no loose bedding or stuffed toys should be left in the cot. (JSM)

20100204-13*

Low serotonin linked to infants' sudden cot death: study. Anon (2010), The Independent 4 February 2010

Reports that researchers in Boston, USA have found that sudden infant death syndrome (SIDS) may be linked to low levels of the neurotransmitter serotonin which wakes babies up when breathing is disrupted, and the enzyme that

helps transport it to babies' brainstems. States that in addition to these findings, babies who had died of SIDS had fewer serotonin receptors in their brainstems. (JSM)

20091027-49

Sleep-related respiratory abnormalities and arousal pattern in achondroplasia during early infancy. Ednick M, Tinkle BT, Phromchairak J, et al (2009), Journal of Pediatrics vol 155, no 4, October 2009, pp 510-515

OBJECTIVE: To assess sleep-disordered breathing (SDB), sleep architecture, and arousal pattern in infants with achondroplasia and to evaluate the relationship between foramen magnum size and the severity of SDB. STUDY DESIGN: A retrospective review of polysomnographic recordings and medical records was performed in infants with achondroplasia and in aged-matched control subjects. All studies were re-scored with the emphasis on respiratory events, sleep state, and arousals. In addition, the neuroimaging study of the brain (magnetic resonance imaging) was reviewed to evaluate foramen magnum diameters and to assess their relationship to SDB. RESULTS: Twenty-four infants met the criteria for entry into analysis, 12 infants with achondroplasia (A) and 12 control infants (C). There was no significant difference in age or sex. Infants with achondroplasia had a significant increase in total respiratory disturbance index (RDI; A, 13.9 +/- 10.8 versus C, 2.0 +/- 0.9; P < .05). However, there was no significant difference in percentages of active sleep, quiet sleep, or sleep efficiency. Analysis of arousals demonstrated that infants with achondroplasia had a significant decrease in both spontaneous arousal index (A, 10.5 +/- 3.5/hr versus C, 18.6 +/- 2.7; P < .0001) and respiratory arousals (A, 10.3% +/- 6.3% versus C, 27.5 +/- 9.5%; P < .0001). Evaluation of foramen magnum dimensions demonstrated smaller foramen magnum size, but there were no significant correlations between anteroposterior or transverse diameters and RDI. CONCLUSION: Infants with achondroplasia have significant SDB during early infancy. SDB in infants with achondroplasia is not associated with alteration in sleep architecture, possibly because of attenuation of the arousal response. We speculate that the concomitant increased apneic events and decreased arousal response will lead to vulnerability in these infants and may underlie the pathophysiologic mechanism of sudden unexpected death in this population. (46 references) (Author)

20091027-43

Minimizing the risks of sudden infant death syndrome: to swaddle or not to swaddle?. Richardson HL, Walker AM, Horne RSC (2009), Journal of Pediatrics vol 155, no 4, October 2009, pp 475-481

OBJECTIVE: To evaluate the effects of swaddling on infant arousability, particularly the progression of subcortical activation (SCA) to full cortical arousal (CA), because impaired arousal may contribute to sudden infant death syndrome. STUDY DESIGN: Healthy term infants, who were routinely swaddled (n = 15) or unswaddled (n = 12) at home, were studied with daytime polysomnography at 3 to 4 weeks and 3 months after birth. When both swaddled and unswaddled, arousability was assessed with a pulsatile jet of air at the nostrils. RESULTS: Larger increases in overall arousal thresholds (SCA plus CA) with swaddling were observed in infants who were easiest to arouse when unswaddled. Swaddling did not alter SCA or CA frequencies of routinely swaddled infants at either age. In infants who were naïve to swaddling, arousal thresholds were increased and CA frequency decreased during swaddled quiet sleep at 3 months. CONCLUSIONS: This study provides a scientific basis for assessing the safety of swaddling in infant care practice. The decreased cortical arousals observed in infants unfamiliar with swaddling may correspond to the increased risk of sudden infant death syndrome for inexperienced prone sleepers. (35 references) (Author)

20091022-62

Cholinergic and oxidative stress mechanisms in sudden infant death syndrome. Dick A, Ford R (2009), Acta Paediatrica vol 98, no 11, November 2009, pp 1768-1775

AIM: To determine whether biochemical parameters of cholinergic and oxidative stress function including red cell acetylcholinesterase (AChE), serum/plasma thyroglobulin, selenium, iron, ferritin, vitamins C, E, and A affect risk in apparent life-threatening event (ALTE), sudden infant death syndrome (SIDS), and sudden unexpected death in infancy (SUDI). To assess these biochemical parameters as a function of age; and for influence of pharmacology and epidemiology, including infant health, care, and feeding practices. METHODS: A multicentre, case-control study with blood samples from 34 ALTE and 67 non-ALTE (control) infants matched for age, and 30 SIDS/SUDI and four

non-SIDS/non-SUDI (post-mortem control) infants. RESULTS: Levels/activity of the biochemical parameters were not significantly different in ALTE vs. control infants, with the exception of higher vitamin C levels in the ALTE group (p = 0.009). In ALTE and control groups, AChE and thyroglobulin levels increased and decreased respectively from birth to attain normal adult levels from 6 months. Levels of iron and ferritin were higher in the first 6 month period for all infant groups studied, intersecting with vitamin C levels peaking around 4 months of age. CONCLUSION: Lower AChE levels and higher combined levels of iron and vitamin C in the first 6 months of life may augment cholinergic and oxidative stress effect, particularly at the age when SIDS is most prevalent. This may contribute to risk of ALTE and SIDS/SUDI events during infancy. (30 references) (Author)

20090904-46

Who is blaming the baby?. Peters C, Becher JC, Lyon AJ, et al (2009), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 94, no 5, September 2009, pp F377-F378

Sudden unexplained collapse within the first 12 h of life is a rare but recognised event. Over a 2-year period, five infants, previously assessed as healthy, were found collapsed in our maternity unit in the care of their primiparous mothers. Two were found prone on their mother's chest, and two were in their mother's bed. The outcomes were poor, with four neonatal deaths and one death at 18 months. The rate of sudden unexplained neonatal collapse was 0.4 per 1000 live births. No cause for collapse was identified despite extensive investigations, which included postmortem in all the neonatal deaths. One infant, however, showed widespread antenatal brain damage at postmortem. It is postulated that some infants with an underlying vulnerability may maladapt to extrauterine life following an hypoxic stressor possibly caused by positional airway obstruction. (7 reference) (Author)

20090825-39

The Sudden Infant Death Syndrome. Kinney HC, Thach BT (2009), The New England Journal of Medicine vol 361, no 8, 20 August 2009, pp 795-805

This article looks in detail at the aetiology of sudden infant death syndrome. Placing an infant to sleep in the supine position reduced the incidence of SIDS drastically but it is still more prevalent in certain ethnic populations althougth how much poverty - a known risk factor - contributes to this is difficult to quantify. There are higher rates of prematurity, alcohol use and smoking in lower socioeconomic populations, all of which are risk factors for SIDS. Various causes of SIDS are identified including infection and asphyxia which are discussed in detail, as are known contributory factors such as maternal smoking in pregnancy, smoking around the infant. (110 references) (VDD)

20090804-53*

Sudden infant death syndrome and sudden intrauterine unexplained death: correlation between hypoplasia of raphé nuclei and serotonin transporter gene promoter polymorphism. Lavezzi AM, Casale V, Oneda R, et al (2009), Pediatric Research vol 66, no 1, July 2009, pp 22-27

This study, besides to delineate the cytoarchitecture and the localization in the brainstem of the human raphé nuclei, aims to evaluate the correlation between neuropathological raphé defects and serotonin transporter gene (5-HTT) promoter region polymorphisms in a cohort of 28 SIDS victims, 12 sudden intrauterine unexplained deaths (SIUD), and 17 controls. Hypoplasia of one or more nuclei of both the rostral and caudal raphé groups was found in 57% of SIDS, in 67% of SIUD, and only in 12% of controls. Furthermore, a significant correlation among 5-HTT Long (L) allele, hypoplasia of the raphé nuclei, and maternal smoking in pregnancy was observed in sudden fetal and infant deaths. The presence of the L allele represents a predisposing factor for sudden fetal and infant death in association with morphologic developmental defects of the raphé nuclei and prenatal smoke exposure. A further consideration of the authors is that SIUD should not be regarded as a separate entity from SIDS, given the potentially shared neuropathological and genetic bases. (Author)

20090701-65

Sudden unexpected neonatal death in the first week of life: autopsy findings from a specialist centre. Weber MA,

Ashworth MT, Risdon RA, et al (2009), Journal of Maternal-Fetal and Neonatal Medicine vol 22, no 5, May 2009, pp 398-404

OBJECTIVE: Sudden unexpected early neonatal death (SUEND) in the first week of life shares features with sudden unexpected death in infancy (SUDI) but is not included as SUDI, which is limited to post-perinatal deaths. The aim of this study was to review SUEND autopsies performed in a single specialist centre over a 10-year period, (1996-2005). METHODS: Retrospective analysis of >1500 consecutively performed paediatric autopsies performed by paediatric pathologists at one centre conducted according to a standard protocol including ancillary investigations. SUENDs were identified and autopsy findings reviewed. RESULTS: Of 1516 post-mortem examinations, 180 were first-week neonatal deaths, 55 (31%) presenting as SUEND. Thirty-two (58%) were explained following autopsy, whilst the remainder were unexplained; most deaths during sleep were associated with adult co-sleeping. Around 40% of explained deaths were associated with undiagnosed congenital abnormalities, mainly congenital heart disease. In addition, there were nine infection-related deaths and three deaths from unsuspected metabolic disease (fatty acid oxidation defects). CONCLUSION: There are distinct differences between SUEND and SUDI, with significantly more explained deaths in the former and a much greater proportion due to congenital abnormalities and metabolic disease. (22 references) (Author)

20090618-96*

Placental transfusion insult in the predisposition for SIDS: a mathematical study. Alastruey J, Sherwin SJ, Parker KH, et al (2009), Early Human Development vol 85, no 7, July 2009, pp 455-459

A difference has been observed between the newborn hearing screening tests of thirty-one SIDS cases versus control infants that survived the first year of life [Rubens DD, Vohr BV, Tucker R, O'Neil CA, Chung W. Newborn oto-acoustic emission hearing screening tests. Preliminary evidence for a marker of susceptibility to SIDS. Early Hum Dev 2008;84(4);225-9]. This study is motivated by the hypothesis that the predisposition for SIDS may be caused by inner ear and brainstem damage from a high venous pressure insult at birth that disrupts an infant's ability to detect rising CO(2) levels following the first month of life. The injury is not immediately lethal due to the persistence of fetal physiological responses during the early postnatal period [Guntheroth WG. Crib death, the Sudden Infant Death Syndrome. Armonk NY: Futura Publishing Co.; 1995. p. 291]. Elastic vessels are assumed in the umbilical vein and newborn venous circulation at the time of a potential high pressure placental transfusion insult and pulse wave propagation is simulated using the nonlinear one-dimensional equations of blood flow in elastic vessels. Peak pressures in the auricular veins increase with the amplitude and length of the umbilical surge, reaching over 60 mm Hg when two consecutive surges separated by 100 ms, of a peak pressure of 100 mm Hg, and a pulse interval of 200 ms are propagated in a network with low peripheral reflections. Our findings support the proposed mechanism for inner ear damage in SIDS and the potential benefit of a newborn hearing screening test in identifying susceptibility and early preventative measures following birth. (Author)

20090604-26

Developmental alterations of the prefrontal cerebral cortex in sudden unexplained perinatal and infant deaths. Lavezzi AM, Mauri M, Mecchia D, et al (2009), Journal of Perinatal Medicine vol 37, no 3, 2009, pp 297-303

The aim of this study was to investigate the developmental patterns of the human prefrontal cortex involved in breathing control in a wide cohort of fetal and infant death victims, aged from the 22(nd) gestational week to 10 months of life, and to evaluate whether morpho-functional disorders are present in this specific cortical area in victims of sudden unexplained death. A further aim was to determine whether prenatal absorption of nicotine could also affect the maturational processes of the prefrontal cortex. A pronounced radial organization of the cerebral wall was evident from the 26(th) gestational week. By 36 gestational weeks this columnar structure disappeared, coinciding with the formation of a laminar cytoarchitecture. The mature cortex, observable from the 4(th) month of life, was organized horizontally into six laminae. In 33% of the sudden death victims the prefrontal cortex showed morphological alterations with anomalous laminar patterns and delayed neuronal maturation. A significant correlation with prenatal cigarette exposure was found. (39 references) (Author)

20090602-56*

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Role of congenital long-QT syndrome in unexplained sudden infant death: proposal for an electrocardiographic screening in relatives. Baruteau AE, Baruteau J, Joomye R, et al (2009), European Journal of Pediatrics vol 168, no 7, July 2009, pp 771-777

INTRODUCTION: Congenital long-QT syndrome (LQTS) is a sporadic or familial inherited arrhythmia. It can lead to sudden death by ventricular fibrillation which occurs at any age but particularly during infancy. Recent studies of postmortem molecular analysis in infants who died of unexplained sudden infant death syndrome (SIDS) showed abnormal mutations to LQTS in 10% to 12%. Current methods of etiologic investigation of sudden infant death syndrome do not allow the diagnosis of LQTS. A targeted anamnesis together with systematic electrocardiograms of first- and second-degree relatives could be an efficient LQTS diagnostic tool. Therefore, we propose to include them in screening procedures for SIDS etiologies. CONCLUSION: LQTS accounts for a significant number of unexplained SIDS. We suggest adding a systematic familial electrocardiographic screening to the current etiologic investigations in order to track congenital LQTS in relatives.
(Author)

20090430-8

Sterile site infection at autopsy in sudden unexpected deaths in infancy. Goldwater PN (2009), Archives of Disease in Childhood vol 94, no 4, April 2009, pp 303-307

OBJECTIVE: To examine and compare bacteriological findings at autopsy of cases of sudden unexpected infant death and those of deaths from other cause. DESIGN: Autopsy report review of 130 sudden infant death syndrome (SIDS) cases (2004 definition), 32 cases of sudden unexpected death in infancy (SUDI) due to infection and 33 cases of non-infectious sudden deaths. SETTING: Qualitative assessment of normally sterile site (NSS; heart blood, spleen or cerebrospinal fluid) bacteriology in SIDS and age-matched comparison deaths that occurred in the late 1980s and early 1990s. MAIN OUTCOME MEASURES: Comparative sterile site bacteriological findings. RESULTS: Sterile site infection was rare in cases of sudden accidental death (eg, motor vehicle accident or drowning); however, the finding of true pathogens such as Staphylococcus aureus in sterile sites in SIDS and deaths associated with infection was relatively common. 10.76% of SIDS had S aureus present in a sterile site, compared with 18.75% of cases of infection-related deaths. S aureus was not found in sudden accidental deaths. The incidence of coliform bacteria in NSS in SIDS was not significantly different from that seen in deaths from other cause. NSS bacteriology yielded no growth in 45.4% of sudden accidental deaths, 43% of SIDS and 28.1% of infectious causes of death. CONCLUSIONS: The finding of S aureus in NSS in a large proportion of cases of SIDS would indicate that a proportion of these babies died of staphylococcal disease. Although the differences in NSS isolation of S aureus in the three infant groups did not quite achieve significance, on the basis of these findings and the characteristic virulence of S aureus, it is recommended that sudden unexpected deaths from which S aureus is isolated from NSS be considered for reclassification. The incidence of coliform bacteria in NSS in SIDS is not significantly different from that in deaths from another cause (both accidental and infectious). From these findings it is recommended that the opinion of a consultant microbiologist be sought to interpret microbiological findings prior to finalising autopsy reports on SUDI. (14 references (Author)

20090211-37

Surfactant protein A and D gene polymorphisms and protein expression in victims of sudden infant death. Stray-Pedersen A, Vege A, Opdal SH, et al (2009), Acta Paediatrica vol 98, no 1, January 2009, pp 62-68

AIM: To investigate the innate immune components surfactant protein A (SP-A) and D (SP-D) in victims of sudden infant death syndrome (SIDS). METHODS: Ten common single nucleotide polymorphisms (SNPs) in the exons of SP-A1, SP-A2 and SP-D genes were analysed in 42 cases of SIDS and 46 explained sudden infant deaths. SP-A and SP-D protein expression in tissue from the aerodigestive tract was semi-quantitatively evaluated by immunohistochemistry. RESULTS: SP-D immunoreactivity was found in lungs and tissue from submandibular gland, palatine tonsils and duodenum. Positive SP-A immune staining was found exclusively in lung tissue. Neither the allele nor the haplotype distribution of the SP-A and SP-D genes was significantly different in SIDS compared to explained deaths. The most common SP-A haplotype, 6A2/1A0, tended to be overrepresented in the cases with low immunohistochemical SP-A expression (61%) compared to cases with high expression (49%), p = 0.08. The SP-D expression was not influenced by

the 11 C/T or 160 A/G polymorphisms. CONCLUSION: No significant association between the common genetic variants of SP-A and SP-D and SIDS is disclosed by the present study. However, low SP-A protein expression may possibly be determined by the 6A2/1A0 SP-A haplotype, this should be subject for further investigation. (32 references) (Author)

20090211-36

HTR2A variation and sudden infant death syndrome: a case-control analysis. Rand CM, Berry-Kravis EM, Fan W, et al (2009), Acta Paediatrica vol 98, no 1, January 2009, pp 58-61

AIM: The serotonergic (5-HT) system functions in central autonomic regulation with homeostatic roles in cardiorespiratory control, thermoregulation, arousal and sleep-wake cycling. Altered function and development of this system in cases of sudden infant death syndrome (SIDS) have been established, but the aetiology of these disturbances remains unclear. The serotonin receptor, HTR2A, functions within this system with roles in the homeostatic response to hypoxia including excitatory effects on respiration, gasping and rhythm generation, all functions potentially compromised in SIDS. The objective of this study was to examine the relationship between SIDS risk and HTR2A variation. METHODS: All coding regions, intron-exon boundaries and the promoter region of HTR2A were PCR amplified and analysed by standard sequencing in 96 SIDS cases and 96 matched controls. RESULTS: Twenty-one HTR2A variations were identified in this case-control cohort, including four novel variations (c.C-1185A, c.T-923C, c.T-17C and c.C50T). None of the variations identified showed a significant association with SIDS. CONCLUSION: This report provides evidence that despite known alterations of the 5-HT system in SIDS, and the logical role for the HTR2A receptor, genetic variation of HTR2A as studied in our cohort is not responsible for these alterations. These results represent a further step in the investigation of the aetiology of the altered serotonin system in SIDS cases. (31 references) (Author)

20090129-29*

Blood pressure and heart rate patterns during sleep are altered in preterm-born infants: implications for sudden infant death syndrome. Witcombe NB, Yiallourou SR, Walker AM, et al (2008), Pediatrics vol 122, no 6, December 2008. e1242-1248

OBJECTIVE: Preterm infants are at an increased risk of sudden infant death syndrome, which may result from immature autonomic control of heart rate and blood pressure. Previous studies have demonstrated that preterm infants have altered heart rate and blood pressure control at term-equivalent age; however, little information is available beyond this age. The aim of this study was to determine the effect of preterm birth on heart rate and blood pressure control over the first 6 months of life after reaching term-equivalent age, including the age at which sudden infant death syndrome risk is increased, to understand the pathogenesis of sudden infant death syndrome. METHODS: Preterm (n=25) and term (n=20) infants were studied longitudinally at 2 to 4 weeks', 2 to 3 months', and 5 to 6 months' term-corrected age by using daytime polysomnography. A photoplethysmographic cuff (Finometer) around the infant's wrist measured blood pressure during quiet and active sleep. RESULTS: Blood pressure was lower in the preterm group during both quiet and active sleep at all ages studied. In contrast, there were no differences between groups in heart rate. Within the infants in the preterm group, blood pressure averaged lower at 2 to 3 months' corrected age compared with both 2 to 4 weeks' and 5 to 6 months' corrected age and was lower in quiet sleep compared with active sleep at all ages studied. Heart rate decreased with increasing age and was lower in quiet sleep compared with active sleep at 5 to 6 months' corrected age. CONCLUSIONS: Sleep state and age affect heart rate and blood pressure patterns in prematurely born infants over the first 6 months of term-corrected age. It is notable that preterm infants had persistently lower blood pressure compared with age-matched term infants, signifying long-term alterations in cardiovascular control in infants born prematurely. (Only the abstract is published in the print journal. Full article available online at http://www.pediatrics.org/cgi/doi/10.1542/peds.2008-1400) (Author)

20081218-109

The role of post-mortem investigations in determining the cause of sudden unexpected death in infancy. Weber MA, Ashworth MT, Risdon RA, et al (2008), Archives of Disease in Childhood vol 93, no 12, December 2008, pp 1048-1053

INTRODUCTION: Several autopsy protocols have been suggested for investigating sudden unexpected deaths in

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infancy (SUDI). The aim of this study is to provide data on the utility of such post-mortem investigations from a large paediatric autopsy series to inform future policy. METHODS: Retrospective analysis of >1500 consecutive post-mortem examinations carried out by specialist paediatric pathologists at a single centre during a 10-year period according to a common autopsy protocol that included the use of detailed ancillary investigations. SUDI was defined as the sudden unexpected death of an infant aged from 7 to 365 days. All data capture and cause of death classification were carried out according to defined criteria. RESULTS: Of 1516 paediatric post-mortem examinations, 546 presented as SUDI. In 202 infants (37%), death was explained by the autopsy findings. The other 344 cases (63%) remained unexplained. Of the explained deaths, over half (58%) were infective, most commonly due to pneumonia (22%). The component of the post-mortem examination that primarily determined the final cause of death was histological examination in 92 infants (46%), macroscopic examination in 61 (30%), microbiological investigations in 38 (19%) and clinical history in 10 (5%). CONCLUSION: This constitutes the largest single-institution autopsy study of SUDI. Ten years on from the Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) SUDI studies, the ascertainment of a cause of death at autopsy has improved. However, with almost two thirds of SUDI remaining unexplained, alternative and/or additional diagnostic techniques are required to improve detection rates of identifiable causes of death at autopsy. (12 references) (Author)

20081218-104

The changing epidemiology of infantile hypertrophic pyloric stenosis in Scotland. Sommerfield T, Chalmers J, Youngson G, et al (2008), Archives of Disease in Childhood vol 93, no 12, December 2008, pp 1007-1011

BACKGROUND: The aetiology of infantile hypertrophic pyloric stenosis (IHPS) has not been fully elucidated. Since the 1990s, a sharp decline in IHPS has been reported in various countries. Recent research from Sweden reported a correlation between falling rates of IHPS and of sudden infant death syndrome (SIDS). This was attributed to a reduction in the number of infants sleeping in the prone position following the 'Back to Sleep' campaign. OBJECTIVES: To describe the changing epidemiology of IHPS in Scotland, to examine the relationship between IHPS and SIDS rates and to examine trends in other factors that may explain the observed reduction in IHPS incidence. DESIGN: Incidence rates of IHPS and SIDS were derived from routine data and their relationship analysed. Trends in mean maternal age, maternal smoking, mean birth weight and breastfeeding rates were also examined. SETTING: The whole of Scotland between 1981 and 2004. RESULTS: IHPS incidence fell from 4.4 to 1.4 per 1000 live births in Scotland between 1981 and 2004. Rates were consistently higher in males, although the overall incidence patterns in males and females were similar. Rates showed a positive relationship with deprivation. The fall in the incidence of IHPS preceded the fall in SIDS by 2 years and the incidence of SIDS displayed less variability than that of IHPS. Significant temporal trends were also observed in other maternal and infant characteristics. CONCLUSION: There has been a marked reduction in Scotland's IHPS incidence, but this is unlikely to be a consequence of a change in infant sleeping position. (15 references) (Author)

20081126-17*

Cardiac ion channel gene mutations in sudden infant death syndrome. Otagiri T, Kijima K, Osawa M, et al (2008), Pediatric Research vol 64, no 5, November 2008, pp 482-487

Sudden infant death syndrome (SIDS) is multifactorial and may result from the interaction of a number of environmental, genetic, and developmental factors. We studied three major genes causing long QT syndrome in 42 Japanese SIDS victims and found five mutations, KCNQ1-K598R, KCNH2-T895M, SCN5A-F532C, SCN5A-G1084S, and SCN5A-F1705S, in four cases; one case had both KCNH2-T895M and SCN5A-G1084S. All mutations were novel except for SCN5A-F532C, which was previously detected in an arrhythmic patient. Heterologous expression study revealed significant changes in channel properties of KCNH2-T895M, SCN5A-G1084S, and SCN5A-F1705S, but did not in KCNQ1-K598R and SCN5A-F532C. Our data suggests that nearly 10% of SIDS victims in Japan have mutations of the cardiac ion channel genes similar to in other countries. (Author)

20081112-53

Recurrence rates for sudden infant death syndrome (SIDS): the importance of risk stratification. Campbell MJ, Hall D,

Stephenson T, et al (2008), Archives of Disease in Childhood vol 93, no 11, November 2008, pp 936-939

OBJECTIVE: To investigate the importance of stratification by risk factors in computing the probability of a second death from sudden infant death syndrome (SIDS) in a family. DESIGN: Simulation study. BACKGROUND: The fact that a baby dies suddenly and unexpectedly means that there is a raised probability that the baby's family have risk factors associated with SIDS. Thus one cannot consider the risk of a subsequent death to be that of the general population. The Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) identified three major social risk factors: smoking, age<27 and parity>1, and unemployed/unwaged as major risk factors. It gave estimates of risk for families with different numbers of these risk factors. We investigate whether it is reasonable to assume that, conditional on these risk factors, the risk of a second event is independent of the risk of the first and as a consequence one can square the risks to get the risk of two SIDS in a family. We have used CESDI data to estimate the probability of a second SID in a family under different plausible scenarios of the prevalence of the risk factors. We have applied the model to make predictions in the Care of Next Infant (CONI) study. RESULTS: The model gave plausible predictions. The CONI study observed 18 second SIDS. Our model predicted 14 deaths (95% prediction interval 7 to 21). CONCLUSION: When considering the risk of a subsequent SIDS in a family one should always take into account the known risk factors. If all risks have been identified, then conditional on these risks, the risk of two events is the product of the individual risks. However, for a given family we cannot quantify the magnitude of the increased risk because of other possible risk factors not accounted for in the model. (9 references) (Author)

20081104-46*

Helicobacter pylori antigen in stool is associated with SIDS and sudden infant deaths due to infectious disease. Brown RE, Rimsza LM, Pastos K, et al (2008), Pediatric Research vol 64, no 4, 2008, pp 405-410

Infection with Helicobacter pylori has been proposed to be a common cause of Sudden Infant Death Syndrome, SIDS. We investigated the frequency of H. pylori infection in 160 infant deaths and 156 live controls by means of the HpSA immunoassay. Histology was performed in 26 randomly selected cases. H. pylori antigen was detected in 8% (12/156) of the live controls compared to 25% (30/122) of SIDS cases (p<0.001), 53% (9/17) of deaths due to infection (p<0.001) and 9% (1/11) of accidental/violent deaths (p=0.60). In the classic age peak for SIDS, 1-5 months, 31 % (21/67) of SIDS cases were HpSA positive compared to 1.5 % (1/68) of live controls (p<0.001). Rod-like immunoperoxidase positive H. pylori organisms were identified in 7/12 HpSA positive gastric antrum sections compared to 2/14 HpSA negative (p=0.038). Significantly elevated IL-6 levels in cerebrospinal fluid representing signs of central immune stimulation were demonstrated in HpSA positive SIDS victims compared to HpSA negative victims (p=0.045). Detection of H. pylori antigen in stool is associated with SIDS and deaths due to infections. We hypothesize that H. pylori infection in infancy may be involved as the triggering pathogen for sudden death during the first five months after birth. (Author)

20081023-6

Impact of changes in infant death classification on the diagnosis of sudden infant death syndrome. Moore BM, Fernbach KL, Finkelstein MJ, et al (2008), Clinical Pediatrics vol 47, no 8, October 2008, pp 770-776

This study evaluates the hypothesis that a decline in sudden infant death syndrome in Minnesota is associated with increases in other categories of sudden unexpected infant death. Matched birth and death certificates, autopsy reports, and home visit questionnaires were reviewed for 722 sudden unexpected infant deaths that occurred from January 1, 1996 through December 31, 2002. Descriptive data and cause of death were recorded. Cause of death was compared for 2 periods: early (1996-1998) and late (2000-2002). The age of the infant at death, sex, race, and infant death rates were similar between the 2 periods (P = .637). Sudden infant death syndrome declined by 50.1% (P < .001). Overlay deaths increased 235.5% (P < .01). Asphyxia related deaths increased 259.6% (P < .001). Injury-related deaths increased 840.0% (P < .001). A decline in sudden infant death syndrome in Minnesota was associated with increased deaths in categories that are asphyxial in nature and are potentially preventable. (23 references) (Author)

20081023-52*

Informed consent for the study of retained tissues from postmortem examination following sudden infant death.

Elliot JG, Ford DL, Beard JF, et al (2008), Journal of Medical Ethics vol 34, no 10, October 2008, pp 742-746

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OBJECTIVE: To develop an approach for seeking informed consent to examine tissues retained from a previous study of sudden infant death syndrome (SIDS) as part of a study on asthma, and to document responses and participation rate. DESIGN: Pilot open-ended approach to 10 volunteer SIDS parents, followed by staged approach (newsletter, mail and telephone call) to seek consent from the target SIDS families for the asthma study. PARTICIPANTS: Parents (n = 10) of SIDS infants known to SIDS and Kids Victoria and parents of SIDS infants (n = 107) from the 1991-2 SIDS in Victoria case-control study. MAIN OUTCOMES: Qualitative responses of the piloted parents and study parents, and participation rates. RESULTS: The pilot group responses were used to refine the written material to be provided. Of the 72 families for which contact details were available, 45 gave verbal consent for contact by the Victorian Institute of Forensic Medicine regarding the asthma study, three refused and 24 did not respond to two letters. Thirty-three completed consent forms, all positive for participation in the asthma study, giving a positive response rate of 73% (33/45). CONCLUSIONS: The use of postmortem tissue for research is acceptable to the next of kin when an approach is sensitive to their concerns and needs and is made by experienced counsellors from a familiar organisation. Despite the painful memories evoked by the approach of the research group, the acceptance rate among those who could be contacted was high. (Author)

20081007-33*

Sleep fragmentation and evidence for sleep debt in alcohol-exposed infants. Troese M, Fukumizu M, Sallinen BJ, et al (2008), Early Human Development vol 84, no 9, September 2008, pp 577-585

BACKGROUND:: Infants exposed prenatally to alcohol are at increased risk for poor neurodevelopmental outcome including Sudden Infant Death Syndrome. AIM:: To examine the relationship between prenatal alcohol exposure, sleep, arousal and sleep-related spontaneous motor movements in early infancy. STUDY DESIGN:: Low-income women (N=13) were interviewed regarding pre- and pregnancy rates of alcohol, cigarette smoking and other substance use in the perinatal period. Infants were examined in a laboratory nap study using EEG, videography and actigraphy at 6-8 weeks of age. Estimates of maternal pre- and pregnancy alcohol use were used to divide infants into high vs. low maternal alcohol use groups. SUBJECTS:: Mother-infant dyads recruited from a family practice clinic. OUTCOME MEASURES:: Sleep-related spontaneous movements, behavioral state, and maternal assessments of infant alertness and irritability. RESULTS:: Pre-pregnancy rates of alcohol consumption including binge drinking correlated with maternal report of poor infant alertness, and increased irritability. High maternal exposure groups showed increased sleep fragmentation, e.g. frequency and duration of wakefulness following sleep onset and decreased active sleep. Bout analysis of the temporal structure of sleep-related spontaneous movements showed significantly reduced bout duration associated with high maternal alcohol use. CONCLUSION:: These results present evidence that prenatal alcohol exposure disrupts postnatal sleep organization and suppresses spontaneous movements during sleep, and increased sleep fragmentation promotes sleep deprivation. Results are consistent with the SIDS model of chronic sleep debt and suggest that attenuated sleep-related movements should be examined as an important modulator of cardiorespiratory functions during sleep in high-risk groups. (Author)

20081001-18*

Unexpected infant deaths associated with use of cough and cold medications. Rimsza ME, Newberry S (2008), Pediatrics vol 122, no 2, August 2008. e318-22

OBJECTIVE: The objective of this study was to determine whether caregivers had given infants who died unexpectedly over-the-counter cough and cold medications before the infant deaths to identify sociodemographic risk factors for their use. METHODS: The Arizona Child Fatality Review Program reviews the circumstances surrounding every child death that occurs in the state each year. By statute, the multidisciplinary review teams have access to all medical charts, autopsy reports, law enforcement reports, and other records for their review and use these data to determine the cause of death and its preventability. The data on all infants who died unexpectedly in 2006 and had an autopsy and postmortem toxicologic studies were reviewed for this analysis. RESULTS: Ten unexpected infant deaths that were associated with cold-medication use were identified. The infants ranged in age from 17 days to 10 months. Postmortem toxicology testing found evidence of recent administration of pseudoephedrine, antihistamine, dextromethorphan, and/or other cold-medication ingredients in these infants. The families who used these

medications were poor and publicly insured, and 50% of them had limited English proficiency. Only 4 of these infants had received medical care for their current illness before their death. The over-the-counter cough and cold medication had been prescribed by a clinician for only 1 of these infants. CONCLUSIONS: Review of these infants' deaths raises concern about the role of the over-the-counter cough and cold medications in these deaths. These findings support the recommendation that such medications not be given to infants. In addition, these findings suggest that warnings on these medications 'to consult a clinician' before use are not being followed by parents. Educational campaigns to decrease the use of over-the-counter cough and cold medications in infants need to be increased. (Only the abstract is published in the print journal. Full article available online at http://www.pediatrics.org/cgi/doi/10.1542/peds.2007-3813) (Author)

20080912-1*

Bacterial infections linked to cot deaths. Gray L (2008), The Telegraph 11 September 2008

News item reporting that a new study has identified a bacterial infection that appears to contribute to some cases of Sudden Infant Death Syndrome. (CR)

20080910-47

A functional polymorphism in the tyrosine hydroxylase gene indicates a role of noradrenalinergic signaling in sudden infant death syndrome. Klintschar M, Reichenpfader B, Saternus KS (2008), Journal of Pediatrics vol 153, no 2, August 2008, pp 190-193

OBJECTIVES: Catecholamines may contribute to the cause of sudden infant death syndrome (SIDS). TH01, a tetrameric short tandem repeat marker in the tyrosine hydroxylase gene, regulates gene expression and catecholamine production. STUDY DESIGN: We investigated TH01 in 172 German Caucasian SIDS cases and 390 sex- and age-matched control subjects. RESULTS: The *9.3 alleles were more frequent in patients with SIDS than in control subjects (40.12% vs 31.15%; P = .006). For homozygotes the odds ratio was 1.83 (95% confidence interval: 1.09-3.05), for carriers 1.58 (1.09-2.28). Moreover, *9.3 alleles were significantly more frequent during the winter (47.73% vs 35.38% in the warmer seasons), and the frequency of *9.3 alleles varied significantly with the age at death (weeks 7 to 12: 49.04% vs 29.63% within the first 6 weeks). Other risk factors (sleeping position, gestation, smoking) had no significant impact on the frequency of *9.3. CONCLUSIONS: Our results indicate a relationship between SIDS and TH01 genotype, presumably caused by an impairment of breathing regulation or arousal. We propose that noradrenalinergic neuronal activity contributes to the cause of a major subset of SIDS victims. Moreover, the results further stress that SIDS is a highly heterogenic group. (28 references) (Author)

20080904-62*

Apolipoprotein E e4 and its prevalence in early childhood death due to sudden infant death syndrome or to recognised causes. Becher JC, Keeling JW, Bell J, et al (2008), Early Human Development vol 84, no 8, August 2008, pp 549-554

BACKGROUND:: Specific genetic polymorphisms have been shown to be more common in unexplained infant death. The APOE genotype exhibits opposite effects at the extremes of age with protective effects of e4 on perinatal mortality but detrimental effects as age progresses. OBJECTIVE:: To determine whether the APOE e4 allele is associated with early childhood (1 week-2 years) unexplained death ('sudden infant death syndrome', SIDS) or with recognised causes (non-SIDS) and to compare these cohorts with published perinatal and adult data. METHODS:: DNA was extracted from spleen tissue of children dying in South East Scotland between 1990 and 2002. APOE alleles (e2, e3, e4) were determined using PCR. Comparisons of allele frequencies between groups were made. RESULTS:: There were 167 SIDS cases and 117 non-SIDS cases. Allele distributions of SIDS cases were similar to healthy newborns. Allele distributions of non-SIDS cases were more similar to adults than to healthy newborns. The percentage of children with at least one e4 allele was significantly lower in non-SIDS compared to SIDS (p=0.016). Non-SIDS cases had a higher frequency of e3 compared to SIDS cases (p=0.01) and to healthy newborns (0.005). CONCLUSIONS:: Children dying from identified causes have different APOE allele distributions from SIDS cases, but are similar to adults. Children dying from SIDS have an allele distribution comparable to healthy newborns. The prevalence of e4 in SIDS is

not of an order to contribute significantly to the age-related decline in e4. (Author)

20080611-41

Extreme and conventional cardiorespiratory events and epidemiologic risk factors for SIDS. Hoppenbrouwers T, Hodgman JE, Ramanathan A, et al (2008), Journal of Pediatrics vol 152, no 5, May 2008, pp 636-641

OBJECTIVE: To test the hypotheses that there is a lack of correlation between extreme events and epidemiologic risk factors for sudden infant death syndrome (SIDS), and if conventional events are normal, their numbers should increase once a circadian decrease in breathing rate is established. In addition, the number of events should decrease with maternal smoking. STUDY DESIGN: Three outcome variables were derived from the Collaborative Home Infant Monitoring Evaluation (CHIME) of 1082 infants: (1) at least 1 extreme event lasting > or = 30 seconds, (2) at least 1 conventional event lasting > or = 20 seconds, and (3) being part of the 50% of infants with the most events. RESULTS: Multivariate logistic regression analyses found that extreme events were not statistically associated with any known SIDS risk factors and occurred less often during the early morning. Healthy term infants had significantly fewer of these events compared with preterm infants, subsequent siblings of infants with SIDS, and infants with an apparent life-threatening event, a finding that was not evident after 43 weeks (3 weeks postterm). Conventional events increased during the night, whereas maternal smoking was associated with a decrease in conventional events. Apneic episodes persisting for > or = 40 seconds occurred in 1.8% of the infants. CONCLUSIONS: Extreme events are associated with immaturity and do not seem to be immediate precursors of or causally related to SIDS. (26 references) (Author)

20080603-49

Infection and sudden unexpected death in infancy: a systematic retrospective case review. Weber MA, Klein NJ, Hartley JC, et al (2008), The Lancet vol 371, no 9627, 31 May 2008, pp 1848-1853

BACKGROUND: The cause and mechanism of most cases of sudden unexpected death in infancy (SUDI) remain unknown, despite specialist autopsy examination. We reviewed autopsy results to determine whether infection was a cause of SUDI. METHODS: We did a systematic retrospective case review of autopsies, done at one specialist centre between 1996 and 2005, of 546 infants (aged 7-365 days) who died suddenly and unexpectedly. Cases of SUDI were categorised as unexplained, explained with histological evidence of bacterial infection, or explained by non-infective causes. Microbial isolates gathered at autopsy were classified as non-pathogens, group 1 pathogens (organisms usually associated with an identifiable focus of infection), or group 2 pathogens (organisms known to cause septicaemia without an obvious focus of infection). FINDINGS: Of 546 SUDI cases, 39 autopsies were excluded because of viral or pneumocystis infection or secondary bacterial infection after initial collapse and resuscitation. Bacteriological sampling was done in 470 (93%) of the remaining 507 autopsies. 2079 bacteriological samples were taken, of which 571 (27%) were sterile. Positive cultures yielded 2871 separate isolates, 484 (32%) of which showed pure growth and 1024 (68%) mixed growth. Significantly more isolates from infants whose deaths were explained by bacterial infection (78/322, 24%) and from those whose death was unexplained (440/2306, 19%) contained group 2 pathogens than did those from infants whose death was explained by a non-infective cause (27/243, 11%; difference 13.1%, 95% CI 6.9-19.2, p<0.0001 vs bacterial infection; and 8.0%, 3.2-11.8, p=0.001 vs unexplained). Significantly more cultures from infants whose deaths were unexplained contained Staphylococcus aureus (262/1628, 16%) or Escherichia coli (93/1628; 6%) than did those from infants whose deaths were of non-infective cause (S aureus: 19/211, 9%; difference 7.1%, 95% CI 2.2-10.8, p=0.005; E coli: 3/211, 1%, difference 4.3%, 1.5-5.9, p=0.003). INTERPRETATION: Although many post-mortem bacteriological cultures in SUDI yield organisms, most seem to be unrelated to the cause of death. The high rate of detection of group 2 pathogens, particularly S aureus and E coli, in otherwise unexplained cases of SUDI suggests that these bacteria could be associated with this condition. FUNDING: Foundation for the Study of Infant Deaths. (21 references) (Author)

20080530-3*

'Infections could be major cause of cot death'. Smith R (2008), The Telegraph 30 May 2008

News item reporting that a study carried out at Great Ormond Street hospital has indicated that some unexplained

sudden infant deaths could be linked to common bacterial infections. (CR)

20080530-1*

Cot death link to bacteria. Randerson J (2008), Guardian 30 May 2008

Reports that a 10-year study of sudden infant deaths has revealed a link with bacterial infection. Although the study goes some way to confirm suspicions that bacteria such as Staphylococcus aureus and Escherichia coli are sometimes the cause of sudden infant death, researches have cautioned that, at present, the link is merely an association. (CR)

20080222-68

SIDS is not a disease entity. Williams LH (2008), BMJ vol 336, no 7641, 23 February 2008, pp 404-405

A letter discussing sudden infant death, the terminology used and measures that could be taken monitor sleeping babies more closely. (1 reference) (CB)

20080213-8

Does cot death still exist?. Gornall J (2008), BMJ vol 336, no 7639, 9 February 2008, pp 302-304

With leading researchers saying smoking and other modifiable factors account for most sudden infant deaths, Jonathan Gornall asks whether it is time to put the diagnosis to bed. (23 references) (Author)

20071120-34

Size for gestational age at birth: impact on risk for sudden infant death and other causes of death, USA 2002. Malloy MHY (2007), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 92, no 6, November 2007, pp F473-F478

BACKGROUND: Small for gestational age (SGA) infants have been reported to be at higher risk for sudden infant death syndrome (SIDS). OBJECTIVE: To compare the risk of SIDS among SGA and large for gestational age (LGA) infants with that of death from other causes of sudden unexpected deaths in infancy (SUDI) and the residual 'other' causes of infant death. METHODS: The 2002 US period infant birth and death certificate linked file was used to identify infant deaths classified as SIDS (ICD-10 code R95), SUDI (ICD-10 codes R00-Y84 excluding R95) or all other residual codes. The 2002 race and sex-specific birth cohorts were used to generate the 10th and 90th percentiles of birth weight for each gestational age week from 24 to 42 weeks' gestation. Demographic variables previously identified as associated with SIDS were used in multiple logistic regression equations to determine the risk for death among SGA and LGA infants (birth weight <10th percentile and >90th percentile, respectively) independent of other potentially confounding variables. RESULTS: Complete data on 1956 SIDS deaths, 2012 SUDI, and 11 592 other deaths were available. The adjusted OR for SIDS, SUDI and 'other' causes for SGA infants was 1.65 (95% CI 1.47 to 1.85), 1.78 (1.59 to 2.00) and 4.68 (4.49 to 4.88), respectively. The adjusted OR for LGA infants was reduced for SIDS (0.73 (0.60 to 0.89)), SUDI (0.81 (0.68 to 0.98)) and 'other' (0.42 (0.38 to 0.46)). CONCLUSION: Although SGA infants seem to be at slightly increased risk for SIDS or SUDI their risk for 'other' residual causes is about 2.5 times higher. LGA infants seem to be at reduced risk of mortality for all causes. The mechanisms by which restricted intrauterine growth increases risk of mortality and excessive intrauterine growth offers protective effects are uncertain. (28 references) (Author)

20071120-23

Small for gestational age infants and sudden infant death syndrome: a confluence of complex conditions. Hunt CE (2007), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 92, no 6, November 2007, pp F428-F429

Comments on a paper in the same issue of the journal [1], which examines whether small for gestational age infants are at increased risk of sudden infant death. 1. Malloy MHY. Archives of Disease in Childhood: Fetal and Neonatal Edition, vol 92, no 6, November 2007, pp F473-F478. (14 references) (MB)

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HRH The Princess Royal

Sudden infant death syndrome. Moon RY, Horne RSC, Hauck FR (2007), The Lancet vol 370, no 9598, 3 November 2007, pp 1578-1587

Despite declines in prevalence during the past two decades, sudden infant death syndrome (SIDS) continues to be the leading cause of death for infants aged between 1 month and 1 year in developed countries. Behavioural risk factors identified in epidemiological studies include prone and side positions for infant sleep, smoke exposure, soft bedding and sleep surfaces, and overheating. Evidence also suggests that pacifier use at sleep time and room sharing without bed sharing are associated with decreased risk of SIDS. Although the cause of SIDS is unknown, immature cardiorespiratory autonomic control and failure of arousal responsiveness from sleep are important factors. Gene polymorphisms relating to serotonin transport and autonomic nervous system development might make affected infants more vulnerable to SIDS. Campaigns for risk reduction have helped to reduce SIDS incidence by 50-90%. However, to reduce the incidence even further, greater strides must be made in reducing prenatal smoke exposure and implementing other recommended infant care practices. Continued research is needed to identify the pathophysiological basis of SIDS. (176 references) (Author)

20071008-46

Marked obesity in infancy and relationship to sudden infant death. Byard RW (2007), Journal of Paediatrics and Child Health vol 43, no 9, September 2007, pp 649-650

Letter reporting on the case of the death of a previously well 15-week old baby. The baby was overweight and the author suggests that this may have contributed to her death. (5 references) (MB)

20070918-66*

Sudden infant death syndrome in twins and singletons. Pharoah PO, Platt MJ (2007), Twin Research and Human Genetics vol 10, no 4, August 2007, pp 644-648

Twins compared with singletons and monozygous (MZ) compared with dizygous (DZ) twins are at increased risk of fetal and infant death, cerebral palsy and many congenital anomalies. The aim of this study is to investigate whether zygosity is a risk factor for the sudden infant death syndrome (SIDS). Birth registration data and draft infant death certificates for all multiple births in England and Wales 1993 to 2003 were provided by the Office for National Statistics. As a partial proxy for zygosity, same-sex was compared with opposite-sex twins for birthweight-specific mortality and mortality attributed to SIDS. Data on singleton infants were obtained by subtraction of multiple births from routinely published population births and infant deaths. SIDS mortality among low birthweight infants was significantly less in twins than singletons. The twin-singleton relative risk was reversed in infants of normal birthweight. Among infants of normal birthweight, neonatal SIDS was significantly more common in same- compared with opposite-sex pairs. Among infants of low birthweight, postneonatal SIDS was significantly more common in same- compared with opposite-sex pairs. The difference in birthweight distribution of same- compared with opposite-sex twins for neonatal SIDS suggests that zygosity is a risk factor for SIDS. As congenital cerebral anomalies are a feature of many monozygous twin conceptions, a detailed macro- and microscopical examination of the brain in twin SIDS may indicate an otherwise unrecognised pathology. (Author)

20070918-24*

Sudden infant death syndrome: rare mutation in the serotonin system FEV gene. Rand CM, Berry-Kravis EM, Zhou L, et al (2007), Pediatric Research vol 62, no 2, August 2007, pp 180-182

Recent studies have identified abnormalities in the development and function of medullary serotonin (5-HT) pathways in postmortem brain from sudden infant death syndrome (SIDS) cases, suggesting 5-HT-mediated dysregulation of the autonomic nervous system (ANS) in SIDS. The human fifth Ewing variant (FEV) gene is specifically expressed in central 5-HT neurons in the brain, with a predicted role in specification and maintenance of serotonergic neuronal phenotype. We hypothesized that variations of FEV may underlie abnormalities of the 5-HT system in SIDS cases and thus may be associated with SIDS risk. To elucidate the relationship between variation in FEV and SIDS, DNA was prepared from 96 African American and Caucasian SIDS cases and 96 gender- and ethnicity-matched controls. Standard sequencing and analysis of FEV revealed a heterozygous insertion mutation (IVS-191_190insA) upstream of

the 5' exon 3 splice site occurring more frequently in SIDS cases (6/96) compared with controls (0/96; p = 0.01) and in the overall African American group (6/98) compared with the Caucasian group (0/94; p = 0.03). Identification of a variation in a gene responsible for 5-HT neuronal development, exclusively in a subset of African American SIDS cases in this cohort, may help explain both the observed abnormalities of this system in some SIDS cases and the ethnic disparity observed in SIDS. (Author)

20070817-22*

Comparison of heart rate responses during cortical and subcortical arousals in term and preterm infants. Hanzer M, Kerbl R, Urlesberger B, et al (2007), Early Human Development vol 83, no 8, August 2007, pp 511-515

The aim of this study was to determine whether prematurity affects heart rate responses during spontaneous arousals. Polygraphic recordings were performed during undisturbed daytime naps in 35 preterm infants (gestational age at birth 32+/-2 weeks) and 35 term infants. Arousals were scored according to the recommendations of the International Paediatric Work Group on Arousals and categorized either as cortical arousals (CA) or subcortical arousals (SCA). Heart rate (HR) and respiratory frequency (RF) were measured during arousal and during the 10-s and 30-s period before and after arousal. Changes in HR and RF were expressed as the percentage of modification normalized for the 30-s period preceding arousal. Altogether, 122 arousals in preterm infants (66 CA, 56 SCA) and 105 arousals in term infants (57 CA, 48 SCA) were scored. Mean duration of the arousal period was 9+/-4 s and 8+/-3 s, respectively. In term infants, a significant increase in HR during arousal could be shown (11.3+/-8.2%; p<0.001), whereas this increase was significantly greater during CA compared to SCA (13.7+/-6.2% versus 8.4+/-9.4%; p<0.001). In contrast, HR decreased during arousal in preterm neonates (-3.9+/-19.3%; p<0.05). These findings suggest that cardiovascular control seems to be maturationally delayed in preterm infants, which may contribute to their increased risk for Sudden Infant Death Syndrome (SIDS). (Author)

20070809-46

Diagnostic utility of soluble fms-like tyrosine kinase 1 and soluble endoglin in hypertensive diseases of pregnancy.. Salahuddin S, Lee Y, Vadnais M, et al (2007), American Journal of Obstetrics & Gynecology (AJOG) vol 197, no 1, pp 28-29

OBJECTIVE: The objective of this pilot study was to evaluate the clinical utility of soluble fms-like tyrosine kinase 1 (sFlt 1) and soluble endoglin (sEng) in the differential diagnosis of hypertension in late pregnancy. STUDY DESIGN: We analyzed serum levels of sFlt 1 and sEng in women with gestational hypertension (GHTN; n = 17), chronic hypertension (CHTN; n = 19), preeclampsia (n = 19), and normal pregnancy (n = 20) in the third trimester. We calculated the sensitivity, specificity, and positive and negative likelihood ratio (LR) for each factor in diagnosing preeclampsia. RESULTS: The sensitivity and specificity of sFlt 1 in differentiating preeclampsia from normal pregnancy were 90% and 90%, respectively, and 90% and 95% for sEng. In women with GHTN, they were 79% and 88% for sFlt 1; 84% and 88% for sEng; 90% and 63% for uric acid. In women with CHTN, they were 84% and 95% for sFlt 1; 84% and 79% for sEng; 68%; and 78% for uric acid. The positive LR for preeclampsia was 9 for sFlt 1 and 7 for sEng in women with normal pregnancy; in women with GHTN; 6.7 for sFlt 1 and 7.2 for sEng; in CHTN, 16 for sFlt 1 and 4 for sEng. Serum uric acid had a positive LR of only 2.4 in women with GHTN and 3.1 in women with CHTN. CONCLUSION: Both sFlt 1 and sEng may prove useful in differentiating preeclampsia from other hypertensive diseases of pregnancy. A prospective cohort study should be performed determine the clinical utility of measuring these proteins.

20070803-75

Investigation of unexplained infant deaths in Jerusalem, Israel 1996-2003. Eisenstein EM, Haklai Z, Schartz S, et al (2007), Archives of Disease in Childhood vol 92, no 8, August 2007, pp 697-699

BACKGROUND: Sudden infant death syndrome (SIDS) is a diagnosis of exclusion that may be assigned only after investigations including a forensic autopsy are performed to exclude possible organic and environmental causes of death. Israeli society is influenced by the Jewish and Islamic faiths, which permit autopsy only under selected circumstances. Against this background, we carried out a study to determine what examinations are performed to investigate unexplained infant deaths in Jerusalem, Israel. METHODS: We examined hospital, Ministry of Health and Ministry of Interior records of unexplained infant deaths in the Jerusalem district from the years 1996-2003. RESULTS:

Ninety six cases were identified from all sources. Forty nine (51%) infants were brought to a hospital at or near the time of death. Studies to determine the cause of death were performed in 54% of cases for which medical records were available for review. These studies included bacterial cultures (44%), skeletal surveys (12%), computerised tomography (3%) and metabolic studies (3%). Only one forensic autopsy was performed, and in no instance was the death site examined by medical personnel. There was a high rate of retrospective review by district health physicians. The most frequently assigned cause of death was SIDS. CONCLUSIONS: The capacity of public health officials and forensic pathologists to investigate unexplained infant deaths is strongly affected by the legal, religious and political milieu in which they work. Efforts should be made to develop socially acceptable methods of improving the quality of infant death investigations in Jerusalem. (26 references) (Author)

20070730-1

Biomarker for hypertension-preeclampsia: are we close yet?. Sibai BM (2007), American Journal of Obstetrics & Gynecology (AJOG) vol 197, no 1, July 2007, pp 1-2

Editorial which presents an overview of hypertensive disorders in pregnancy and highlights the need for biochemical markers to identify women who are at risk. Comments on an article in this issue (1) that looks at the predictive value of serum angiogenic factors in the prediction of preeclampsia. 1. Salahuddin S et al. Diagnostic utility of soluble fms-like tyrosine kinase 1 and soluble endoglin in hypertensive diseases of pregnancy. Am J Obstet Gynecol, vol 197, no 1, July 2007, pp 28-29. (11 references) (CR)

20070727-7

Efficacy of a SIDS risk factor education methodology at a native American and Caucasian site. Burd L, Peterson M, Face GC, et al (2007), Maternal and Child Health Journal vol 11, no 4, July 2007, pp 365-371

OBJECTIVE: To complete a community based efficacy study of a SIDS risk reduction methodology. METHODS: We utilized two community sites for this study: 1) a Native American home visiting program for pregnant and young mothers; and 2) an obstetrics department in a community hospital. Pre and posttests were used to measure learning. The risk reduction intervention was delivered by hospital nurses or the home visiting staff and required about 20 minutes. Each of the nine risk factors was discussed. RESULTS: We completed paired pre and post testing with 341 women. The pre tests found substantial knowledge deficits about SIDS risk factors in both groups. The pre and posttest changes for the nine risk factors ranged from 5% to 74%. Participants from both groups demonstrated nearly equivalent rates of learning for all nine of the risk concepts. CONCLUSION: This study demonstrated the efficacy of this brief intervention program. The program was effective in increasing parental knowledge of the risk factors targeted by this study in both settings. The magnitude of change supports additional research with this program in other settings and with additional populations. (16 references) (Author)

20070720-26*

Paediatricians argue second cot death is rarer than study says. Boseley S (2007), Guardian 20 July 2007

Reports on a study published in the BMJ that has found that two cot deaths in a family may be rarer than claimed. (SB)

20070711-87

Educating parents about the risk factors of sudden infant death syndrome: the role of neonatal intensive care unit and well baby nursery nurses. Esposito L, Hegyi T, Ostfeld BM (2007), Journal of Perinatal and Neonatal Nursing vol 21, no 2, April/June 2007, pp 158-164

Nurses in newborn nurseries and neonatal intensive care units are instrumental in educating parents about reducing the risk for SIDS. Nurse participation is acknowledged and encouraged in the current policy statement on SIDS Risk Reduction put forth by the American Academy of Pediatrics. Despite the decline in SIDS, it remains the leading cause of postneonatal infant mortality, and despite greater public compliance with the risk reduction guidelines there is room for improvement in how effectively and consistently they are disseminated. To facilitate nursing participation as educators, role models, and collaborators in the development of relevant hospital policies and procedures, we review

the current recommendations, addressing issues that may serve as barriers to participation, describing the biological plausibility underlying risk-reducing practices, and presenting resources from which nurses may obtain teaching materials and model policies. (46 references) (Author)

20070611-36*

Inappropriate mediastinal baroreceptor reflex as a possible cause of sudden infant death syndrome - is thorough burping before sleep protective?. Flaig C (2007), Medical Hypotheses vol 68, no 6, 2007, pp 1276-1286

Despite extensive research, a link between the assumed mechanisms of death and known risk factors for sudden infant death syndrome (SIDS) has not yet been established. Modifiable risk factors such as prone sleeping position, nicotine exposure and thermal stress and non-avoidable risk factors like male gender and some risky socio-economic conditions could be detected, but the etiology of SIDS remains unknown. In many SIDS cases histopathological findings suggest an involvement of vital autonomic control functions and unidentified trigger factors seem to play a role. From a hypothetical point of view, a developmental sympatheticovagal imbalance of the cardiovascular reflex control could cause a predisposition for SIDS. An assumed gastroesophageal trigger impulse is possibly developed during the first weeks of life and could lead to the infant's vagal reflex death. Air swallowed during feeding escapes through the esophagus while the infant is sleeping. The temporarily bloated esophagus exerts pressure on neighboring mediastinal baroreceptors, which is potentially misinterpreted as a rise in arterial pressure. The following cardiodepressoric baroreceptor reflex could lead to arterial hypotension, bradycardia and cardiac arrest. Sleeping in prone position may create an increased thoracic pressure on mediastinal baroreceptors, causing a more pronounced vagal reflex and an increased likelihood of SIDS. Prone position in connection with soft objects in the infant's sleeping environment potentially generates an increased oculobulbar pressure, resulting in an additional cardiodepressoric condition (Aschner-Dagnini phenomenon). From the sixth month of life onwards the sympatheticovagal balance seems to have matured sufficiently to compensate the life-threatening challenges in most infants. Insufficient postprandial burping could either create another independent modifiable risk factor or present the missing link to a common trigger mechanism for SIDS. Further investigations may possibly lead to the explicit recommendation to burp all infants sufficiently and repeatedly before sleep. (Author)

20070607-47*

Identifying infants at risk for sudden infant death syndrome. Sahni R, Fifer WP, Myers MM (2007), Current Opinion in Pediatrics vol 19, no 2, April 2007, pp 145-149

PURPOSE OF REVIEW: This review examines recent research relevant to the underlying pathophysiology and risk factors for sudden infant death syndrome. RECENT FINDINGS: Current research focuses on the linkage between known risk factors and vulnerability, genetic contributions, and the role of dysfunctional brainstem neurotransmission in the pathogenesis of this syndrome. While social inequalities, prematurity, maternal smoking, infant sleeping practices and sleep environment, arousal failures and environmental pollutants remain important risk factors, new evidence is emerging that certain genetic polymorphisms may contribute to vulnerability. New neuropathological studies have provided strong support for abnormal brainstem serotonergic function. Since serotonin influences a wide range of physiological systems including breathing, the cardiovascular system, temperature, and sleep-wake cycles, this finding strongly supports the hypothesis that sudden infant death syndrome is the result of dysregulation of the autonomic nervous system and provides biological plausibility for certain risk reduction strategies. SUMMARY: Despite a putative diagnostic shift, sudden infant death syndrome remains the most common cause of death from 1 month to 1 year of age. Recent studies confirmed established risk factors and have suggested new genetic vulnerabilities. Finally, new evidence supports a key role for abnormalities in brainstem serotonin systems in the pathophysiology of this syndrome. (Author)

20070607-164

Mitochondrial tRNA genes and flanking regions in sudden infant death syndrome. Opdal SH, Vege A, Arnestad M, et al (2007), Acta Paediatrica vol 96, no 2, February 2007, pp 211-214

AIM: Mitochondrial DNA (mtDNA) mutations have been proposed as a genetic risk factor for sudden infant death syndrome (SIDS). The aim of this study was to further investigate this issue, by sequencing the mitochondrial tRNA genes with flanking regions in SIDS cases and controls. METHOD: The selected genes were investigated in 24 cases of SIDS and 10 controls, the method used were direct sequencing. In addition, the A10398G mutation in the ND3 gene was investigated in 220 SIDS cases, 26 cases of infectious death and 93 controls, using allele-specific PCR. RESULTS: Mutations, recorded as differences from the revised Cambridge sequence, were found in 32 different sites in the coding regions investigated. There was no difference in mutation frequency between SIDS cases and controls, and no single mutation was found associated with SIDS. CONCLUSION: The present study does not indicate an association between a specific mitochondrial tRNA gene mutation and SIDS, nor a higher mtDNA tRNA mutation frequency in SIDS cases than in controls. (30 references) (Author)

20070607-152

A mitochondrial DNA polymorphism associated with cardiac arrhythmia investigated in sudden infant death syndrome. Arnestad M, Opdal SH, Vege A, et al (2007), Acta Paediatrica vol 96, no 2, February 2007, pp 206-210

AIM: Long QT syndrome (LQTS) has been shown to be the cause of death in some cases originally diagnosed as sudden infant death syndrome (SIDS). Such cardiac arrhythmias have also been noted in families with mitochondrial disease, and studies indicate that mitochondrial disease could be involved in SIDS. This makes the mtDNA polymorphism T3394C interesting, as a previous study has shown it to be associated with electrocardiographic (ECG) changes after exercise in a family with LQTS, where some members harboured a KCNH2 mutation. SUBJECTS: A total of 245 SIDS cases and 176 control cases. METHODS: DNA was prepared from blood/tissue samples. Polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP) were performed to search for the mtDNA polymorphism and KCNH2 mutation. Differences were confirmed by sequencing. RESULTS: The T3394C polymorphism was found in 3 pure SIDS cases (1.5%), 2 borderline SIDS cases (4.4%), 1 case of explained death (1.6%) and 2 living control cases (1.8%) (p = 0.62). The KCNH2 mutation was not found in cases or controls. CONCLUSION: The mtDNA polymorphism studied was found in a small number of SIDS cases and the frequency did not differ statistically from control subjects, making an association with increased SIDS risk unlikely. (30 references) (Author)

20070607-113

Another new theory explaining the cause of SIDS. Lagercrantz H, Wennergren G (2007), Acta Paediatrica vol 96, no 2, February 2007, pp 151-152

Editorial commenting on a new study [1] which found that infants that died from SIDS have a decreased binding of serotonin in the brain nuclei involved in sleep and breathing. 1. Paterson DS. JAMA, vol 296, 2006, pp 2124-32. (11 references) (MB)

20070514-5*

Scientists find blood glucose link to cot death. Sheerin J (2007), Independent 7 May 2007

News item reporting on research that has found that defects in glucose production, particularly in low birth weight babies, may lead to sudden infant death. (MB)

20070116-60*

Sudden infant death syndrome. Learning from stories about SIDS, motherhood and loss. Gurbutt DJ (2007), Abingdon: Radcliffe Publishing March 2007. 115 pages

This insightful guide is based on real life accounts from mothers who have experienced Sudden Infant Death Syndrome. Focusing on grief, motherhood and maternal identify, the book is an intriguing read - often upsetting, yet desperately compelling. the stories and poignant memories bring the subject to life. Health and social care professionals will find a wealth of emotional and practical insights to improve professional practice, as will psychiatrists, psychologists, counsellors and therapists. (Publisher)

20061201-1*

Cot deaths 'expert' Sir Roy may not have been wrong. Akbar A (2006), Independent 1 December 2006. 1 page

Brief news item reporting on a new report that challenges the findings of a cot death study that helped to discredit Sir Roy Meadow. (MB)

20061107-9*

Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. Paterson DS, Trachtenberg FL, Thompson EG, et al (2006), JAMA (Journal of the American Medical Association) vol 296, no 17, November 2006, pp 2124-2132

CONTEXT: The serotonergic (5-hydroxytryptamine [5-HT]) neurons in the medulla oblongata project extensively to autonomic and respiratory nuclei in the brainstem and spinal cord and help regulate homeostatic function. Previously, abnormalities in 5-HT receptor binding in the medullae of infants dying from sudden infant death syndrome (SIDS) were identified, suggesting that medullary 5-HT dysfunction may be responsible for a subset of SIDS cases. OBJECTIVE: To investigate cellular defects associated with altered 5-HT receptor binding in the 5-HT pathways of the medulla in SIDS cases. DESIGN, SETTING, AND PARTICIPANTS: Frozen medullae from infants dying from SIDS (cases) or from causes other than SIDS (controls) were obtained from the San Diego Medical Examiner's office between 1997 and 2005. Markers of 5-HT function were compared between SIDS cases and controls, adjusted for postconceptional age and postmortem interval. The number of samples available for each analysis ranged from 16 to 31 for SIDS cases and 6 to 10 for controls. An exploratory analysis of the correlation between markers and 6 recognized risk factors for SIDS was performed. MAIN OUTCOME MEASURES: 5-HT neuron count and density, 5-HT(1A) receptor binding density, and 5-HT transporter (5-HTT) binding density in the medullary 5-HT system; correlation between these markers and 6 recognized risk factors for SIDS. RESULTS: Compared with controls, SIDS cases had a significantly higher 5-HT neuron count (mean [SD], 148.04 [51.96] vs 72.56 [52.36] cells, respectively; P<.001) and 5-HT neuron density (P<.001), as well as a significantly lower density of 5-HT(1A) receptor binding sites (P<or=.01 for all 9 nuclei) in regions of the medulla involved in homeostatic function. The ratio of 5-HTT binding density to 5-HT neuron count in the medulla was significantly lower in SIDS cases compared with controls (mean [SD], 0.70 [0.33] vs 1.93 [1.25] fmol/mg, respectively; P = .001). Male SIDS cases had significantly lower 5-HT(1A) binding density in the raphe obscurus compared with female cases (mean [SD], 16.2 [2.0] vs 29.6 [16.5] fmol/mg, respectively; P = .04) or with male and female controls combined (mean [SD], 53.9 [19.8] fmol/mg; P = .005). No association was found between 5-HT neuron count or density, 5-HT(1A) receptor binding density, or 5-HTT receptor binding density and other risk factors. CONCLUSIONS: Medullary 5-HT pathology in SIDS is more extensive than previously delineated, potentially including abnormal 5-HT neuron firing, synthesis, release, and clearance. This study also provides preliminary neurochemical evidence that may help explain the increased vulnerability of boys to SIDS. (Author)

20061101-3*

Scientists identify brain condition that may solve mystery of cot deaths. Laurance J (2006), Independent 1 November 2006. 2 pages

News item reporting on research that has identified a brain abnormality in victims of sudden infant death. (MB)

20061101-2*

Scientists find the key to cot deaths. Hawkes N (2006), Times 1 November 2006. 2 pages

News item reporting on research that has identified a brain abnormality in victims of sudden infant death. (MB)

20060920-82

The G protein beta3 subunit 825C allele is associated with sudden infant death due to infection. Hauge Opdal S, Melien O, Rootwelt H, et al (2006), Acta Paediatrica vol 95, no 9, September 2006, pp 1129-1132

Aim: To investigate the Gbeta3 subunit C825T polymorphism with regard to sudden unexpected infant death. The reported association between the Gbeta3s protein and increased immune cell function in humans makes this

polymorphism highly interesting both with regard to sudden infant death syndrome (SIDS) and deleterious infectious disease. Methods: The cases investigated in the present study consist of 250 SIDS cases, 38 cases of sudden unexpected infant death due to infection and 99 living infant controls. Typing of the C825T polymorphism was performed by real-time PCR with allele-specific probes and melting curve analyses. Results: The cases of infectious death have a higher percentage of both the C allele (p=0.037 compared to the SIDS cases, p=0.022 compared to the controls) and the CC genotype (p=0.05 compared to the SIDS cases, p=0.016 compared to the controls). There were no differences between SIDS cases and controls.Conclusion: The observed association between the 825C allele and infectious death may indicate that the presence of the 825T allele exerts a protective effect towards serious infection, possibly through enhanced G protein signalling. The C allele, on the other hand, appears to represent a disadvantage in this regard. (27 references) (Author)

20060825-29*

Lung flaw link to cot death risk. BBC News (2006), BBC News 23 August 2006. 2 pages

Babies born with faults in three key lung genes have 14 times the risk of dying from cot death, researchers say. (Author)

20060825-20*

Gene variations linked to cot deaths. Fleming N (2006), The Telegraph 24 August 2006. 1 page

Abnormal lung development caused by genetic differences could be responsible for many cases of cot death, according to new research. (Author)

20060711-42

Sudden infant death syndrome (SIDS) shortly after hexavalent vaccination: another pathology in suspected SIDS?.
Ottaviani G, Matturri L, Bruni B, et al (2005), Journal of Perinatal Medicine vol 33, no 2, 2005, pp 165-169

Experts from panels of the European Agency for the Evaluation of Medical Products have investigated whether there might be a link between hexavalent vaccines and some cases of deaths that occurred. Participants included pathologists with experience in the field of vaccines and sudden infant death syndrome who conducted autopsies. However, to the best of our knowledge, little, if any, attention was paid to examination of the brainstem and the cardiac conduction systems on serial sections, nor was the possibility of a triggering role of the vaccine in these deaths considered. Herein we report the case of a 3-month-old female infant dying suddenly and unexpectedly shortly after being given a hexavalent vaccination. Examination of the brainstem on serial sections revealed bilateral hypoplasia of the arcuate nucleus. The cardiac conduction system presented persistent fetal dispersion and resorptive degeneration. This case offers a unique insight into the possible role of hexavalent vaccine in triggering a lethal outcome in a vulnerable baby. Any case of sudden unexpected death occurring perinatally and in infancy, especially soon after a vaccination, should always undergo a full necropsy study according to our guidelines. (21 references) (Author)

20060515-7*

Postnatal hypokinesia and the delayed time frame of sudden infant death syndrome. Reid GM (2006), Medical Hypotheses vol 67, no 1, 2006, pp 143-145

The sudden infant death syndrome peaks in the second and third month of life. This is the period of the 'two-month transformation of the central nervous system' in the human infant. Studies of 120 days of imposed hypokinesia in man demonstrated that the maximum period of autonomic dysfunction was delayed until the beginning of the second month through to the fourth month of the experiment. Hypokinesia also impaired sleep mechanisms and induced polymorphic changes in almost all systems of the human body. These studies suggest that prolonged postnatal hypokinesia in infants may induce autonomic dysfunction in the CNS, especially during the 'two-month transformation period' of major postnatal neural development. (Author)

20060406-14

Fetal heart rate patterns and sudden infant death syndrome. Menihan CA, Phipps M, Weitzen S (2006), JOGNN: Journal of Obstetric, Gynecologic and Neonatal Nursing vol 35, no 1, January/February 2006, pp 116-122

Objective: To determine differences in electronic fetal monitoring patterns between infants who died of sudden infant death syndrome and controls. Design: Case-control study (N= 127). Setting: A tertiary-level women's hospital in Providence, Rhode Island. Participants: Infants born between 1990 and 1998 who subsequently died of sudden infant death syndrome and controls. Demographic and clinical data included medical maternal charts and fetal monitoring records. Results: Compared with controls (n= 98), the mothers whose infants subsequently died of sudden infant death syndrome (n= 29) had lower birthweight babies (sudden infant death syndrome 2,840 vs. controls 3,385 g; $p < .01$), were younger (22 vs. 28 years; $p < .01$), were more likely to receive Medicaid health insurance (odds ratio 4.6; confidence interval 1.9-11.2), were more likely to be unmarried (odds ratio 5.2; confidence interval 2.1-12.8), had less intention to breastfeed (26% vs. 57%), and were more likely to smoke (odds ratio 4.6; confidence interval 9-11.2). Main outcome measures: There were no statistical differences in fetal heart rate variability or sleep/wake cycles detected between groups. Conclusion: Statistical differences were found in demographic characteristics between sudden infant death syndrome mother-infant couples and their controls. However, no differences were detected in the intrapartum electronic fetal monitoring records, specifically in variability and sleep/wake cycles. (14 references) (Author)

20060222-50*

Sudden infant death syndrome (SIDS): Microgravity and inadequate sensory stimulation. Reid GM (2006), Medical Hypotheses vol 66, no 5, 2006, pp 920-924

In early gestation, the human foetus develops in a buoyant environment, which is similar to the near-weightlessness of space flight. After the 26th week of gestation, the foetus gradually becomes exposed to gravitational forces. The influence of fluid immersion declines as the weight of the foetus increases. In this way, the foetus adapts and trains for the gravity environment after birth. Failure of gravitational loading in the last trimester of pregnancy delays development and maintains the pathophysiological environment of microgravity as experienced by the astronaut in space flight. The deconditioning effects of microgravity during space flight are the reverse processes of intrauterine development after the 26th week when the foetus begins training body processes for adaptation to an earthly environment. Growth requires space and movement, which suggests that a growth-retarded foetus may have been deprived of the mechanical dimension of uterine wall pressure, and, in twins, the smaller sibling may have been deprived of space. The behaviour of a study group of sudden infant death syndrome infants suggested a continuation of the effects of the foetal akinesia syndrome during the third trimester period of gestation. NASA research into the pathophysiology of microgravity was based on a simple insight: that the physiological effects of human space travel were virtually identical to the adjustments the body makes when lying down. This is the same environment as that of the human foetus in the first 22 weeks of gestation after which the uterine environment becomes a prelude to adaptations to the force of gravity. (Author)

20060213-20*

Gasping cells theory of cot death. BBC News (2006), BBC News 13 February 2006. 2 pages

Cot death could be caused by a genetic defect which means babies who stop breathing cannot kick-start their lungs, research suggests. (Author)

20060115-34*

Sudden infant death syndrome: the colon connection. Mann NS, Rossaro L (2006), Medical Hypotheses vol 66, no 2, 2006, pp 375-379

The etiology of sudden infant death syndrome (SIDS) is not known. Various maternal and infant risk factors have been identified. Adoption of the non-prone position has reduced the incidence of SIDS but has not eliminated the problem. Some sulfate reducing bacteria in the colon produce hydrogen sulfide (H(2)S) which is as toxic as hydrogen cyanide. Normally, the colonic mechanism for metabolizing and detoxifying H(2)S is very effective and no H(2)S appears in the

exhaled breath although small amounts are present in the flatus. We are putting forth the hypothesis that in some cases of SIDS colonocytic mechanism for detoxifying H(2)S may not have matured by the age of 3 months and H(2)S may be absorbed resulting in SIDS. The hypothesis can be tested by in vitro evaluation of colonic tissue from SIDS cases for its ability to detoxify H(2)S. (Author)

20051219-55

Sudden infant death syndrome and complications in other pregnancies. Smith GCS, Wood AM, Pell JP, et al (2005), Lancet vol 366, no 9503, 17 December 2005, pp 2107-2111

Background: The likelihood of recurrence of sudden infant death syndrome (SIDS) is an issue of biological, clinical, and legal interest. Obstetric complications are associated with an increased risk of SIDS and are likely to recur in subsequent pregnancies. We postulated that women whose infants died from SIDS would be more likely to have had obstetric complications in their other pregnancies. Methods: We linked national UK databases of maternity-hospital discharges, perinatal deaths, and death certifications. We studied 258 096 women who had consecutive births in Scotland between 1985 and 2001. Findings: Women who had an infant who died from SIDS were at increased risk in their next pregnancy of delivering an infant small for gestational age (odds ratio 2·27, 95% CI 1·54-3·34, p<0·0001) and of preterm birth (2·53, 1·82-3·53, p<0·0001). The risk of SIDS was higher for the children of women whose previous infant had been small for gestational age (1·87, 1·19-2·94, p=0·007) or preterm (1·93, 1·24-3·00, p=0·004). Multivariate analysis showed that all associations were explained by common maternal risk factors for SIDS and obstetric complications and by the likelihood of recurrence of fetal growth restriction and preterm birth. Interpretation: Women whose infants die from SIDS are more likely to have complications in their other pregnancies. Recurrence of pregnancy complications predisposing to SIDS could partly explain why some women have recurrent SIDS. (25 references) (Author)

20051214-26

Serum testosterone and estradiol in sudden infant death. Emery MJ, Krous HF, Nadeau-Manning JM, et al (2005), Journal of Pediatrics vol 147, no 5, November 2005, pp 586-591

OBJECTIVE: To test the hypothesis that among infants who die unexpectedly, testosterone and/or estradiol levels are elevated in those diagnosed with SIDS versus those with known causes of death (controls). STUDY DESIGN: Postmortem blood was collected and coded from infant autopsies, and serum was prepared and frozen until assayed for total testosterone and estradiol by fluoroimmunoassay. Subject information was then collected from the medical examiner's report. RESULTS: Testosterone, but not estradiol, was significantly higher in 127 SIDS cases versus 42 controls for both males (4.8 +/- 0.4 vs 2.2 +/- 0.4 nmol, respectively; P < .005) and females (2.4 +/- 0.2 vs 1.6 +/- 0.2 nmol, respectively; P < 0.03). CONCLUSIONS: Higher testosterone levels in infant victims of unexpected, unexplained death may indicate a role for testosterone or related steroids in SIDS. Further research is needed to understand the potential utility of testosterone as an indicator of SIDS risk. (34 references) (Author)

20051025-61

Acute idiopathic pulmonary haemorrhage in infancy: case report and review of the literature. Habiba A (2005), Journal of Paediatrics and Child Health vol 41, no 9/10, September/October 2005, pp 532-533

This report presents the case of a 4-month-old male infant with recurrent bouts of haemoptysis for which no cause could be detected after extensive investigation. Literature reports of this condition from other geographic locations around the world are reviewed, together with epidemiologic studies attempting to provide a link with certain environmental exposures, toxic and infectious. A diagnostic entity of acute idiopathic pulmonary haemorrhage in infancy has recently been proposed. To my knowledge, this is the first case reported from New Zealand. Although the incidence of such reported cases appears to be rare, they constitute an interesting public health problem, particularly because some of the risk factors appear to overlap with risk factors for sudden infant death. They can therefore trigger an investigation into the home and outdoor environments, and may provide valuable insights into a possible underlying genetic factor and potentially harmful exposures in the modern urban or rural settings. (10 references) (Author)

20050928-5*

Sudden infant death syndrome: how significant are the cardiac channelopathies?. Tester DJ, Ackerman MJ (2005), Cardiovascular Research vol 67, no 3, August 2005, pp 388-396

Having an apparently healthy, thriving infant fail to reach his/her first birthday is profoundly tragic. This tragedy is compounded when the infant's death is unexpected and unexplained, signed out as sudden infant death syndrome (SIDS). Despite impressive success and welcome reductions in these tragic deaths due in large measure to 'Back-to-Sleep' campaigns, the fundamental pathogenic mechanisms precipitating such deaths remain dimly exposed. Here, we review the causal link between SIDS and mutations involving the SCN5A-encoded cardiac sodium channel, provide new findings following extensive postmortem genetic testing of long QT syndrome (LQTS)-associated potassium channel genes in a population-based cohort of SIDS, and summarize the current understanding regarding the spectrum and prevalence of cardiac channelopathies in the pathogenesis of SIDS. (Author)

20050809-26

Unexpected but not unexplained: investigating a case of sudden unexpected death in infancy. Sidebotham P (2005), Archives of Disease in Childhood: Education & Practice Edition vol 90, no 2, August 2005, pp ep40-ep45

Discusses a case of sudden infant death in a 10 day old boy. Outlines the care and resuscitation provided at the time of admission of the infant and the investigations made into the cause of death. After all investigations, the final cause of death was attributed to a disorder of mitochondrial beta oxidation. (14 references) (SB)

20050802-66*

Comparison of postnatal development of heart rate responses to trigeminal stimulation in sleeping preterm and term infants. Tuladhar R, Harding R, Adamson M, et al (2005), Journal of Sleep Research vol 14, no 1, March 2005, pp 29-36

Autonomic dysfunction has been regarded as a possible cause of the sudden infant death syndrome (SIDS) and it has been suggested that preterm infants, who are at a greater risk of SIDS than term infants, may have immature autonomic control. Our aim was to compare the maturation of cardiac autonomic control during sleep in preterm and term infants by examining heart rate responses to arousing and non-arousing trigeminal stimuli. Preterm infants (n = 15) and term infants (n = 24) were studied longitudinally with daytime polysomnography. Air-jet stimulation of the nares was delivered in both active sleep (AS) and quiet sleep (QS), and heart rate (HR) changes recorded for both arousal and non-arousal responses. Changes in HR (DeltaHR%) were calculated as the relative differences between baseline HR (BHR) and either MaxHR (arousal) or MinHR (non-arousal). Comparisons of HR changes between sleep states and postnatal ages were made with two-way anova for repeated measures and between groups with two-way anova. The increase in HR (DeltaHR%) was greater in term than preterm infants ($P < 0.05$), but only at 2-3 weeks corrected postnatal age (CPA). In preterm infants, there were no differences in BHR between sleep states, whereas in term infants, BHR was higher in AS than in QS at 2-3 weeks and 2-3 months of age. The smaller DeltaHR% to arousing stimuli in preterm infants compared with term infants at 2-3 weeks suggests that cardiac sympathetic activity in preterm infants may be lower than in term infants. This mechanism may account for the increased risk for SIDS of preterm infants. (Author)

20050524-27

Epidemiology and apparent life threatening events. Kiechl-Kohlendorfer U, Hof D, Peglow UP, et al (2005), Archives of Disease in Childhood vol 90, no 3, March 2005, pp 297-300

AIMS: To investigate the epidemiology and risk factors of apparent life threatening events (ALTE). METHODS: A prospective study enrolled all live-born infants in the Tyrol (1993-2001). Information on pregnancy, sociodemographic characteristics, child care practices, and infant's behaviour in the first four to six weeks of life was collected with a standardised questionnaire, and was available for 44,184 infants. ALTE was identified from hospital admission records. RESULTS: During the study period 164 ALTE cases were identified, corresponding to an incidence of 2.46/1000 live births. In 73 of these infants no cause for the event and no comorbidity could be found (idiopathic ALTE). On average

ALTE manifested ten weeks earlier than SIDS. Of various SIDS risk factors in the survey area, the prone sleeping position, smoking during pregnancy, low gestational age, profuse night sweating, and family history of infant death showed a moderate relation to the risk of overall ALTE, but only smoking maintained significance in the multivariate risk model. None of these variables was associated with idiopathic ALTE. In contrast to SIDS the frequency of ALTE did not change during the study period. None of the ALTE infants experienced SIDS later in life. Behavioural abnormalities such as feeding difficulties, episodes of pallor, cyanotic episodes, and repeated apnoea episodes were strongly associated with an increased risk of overall and idiopathic ALTE. CONCLUSIONS: Although there are some similarities in the clinical presentation and epidemiology of SIDS and ALTE, differences clearly predominate. Accordingly, ALTE and SIDS should not be considered different manifestations of the same disease process. (24 references) (Author)

20050513-33*

Current controversies in the pathophysiology and prevention of sudden infant death syndrome. Spitzer AR (2005), Current Opinion in Pediatrics vol 17, no 2, April 2005, pp 181-185

PURPOSE OF REVIEW: To examine recent research relevant to sudden infant death syndrome (SIDS) to determine whether there is a place for home monitoring in the care of children believed to be at risk. RECENT FINDINGS: Current SIDS research has focused on the genetics of SIDS, brainstem abnormalities and arousal failures, the effects of tobacco smoke and other environmental agents, the role of infectious diseases, and prenatal factors that may contribute to SIDS. Investigations have suggested that there are infants who appear to respond less effectively when challenged by certain environmental or infectious agents. These infants have blunted responses to stress and diminished arousal to hypoxemia, in part because of failures in genetically determined brainstem function. It is unclear at this time whether home monitoring would offer protection in all circumstances, but it may be helpful in certain patients. SUMMARY: There appears to be progress in understanding the causes of SIDS. As additional studies emerge, the optimal approaches to care will become more apparent, with home monitoring one of the possible interventions. (Author)

20050429-2

Is there a relation between SIDS and long QT syndrome?. Skinner JR (2005), Archives of Disease in Childhood vol 90, no 5, May 2005, pp 445-449

Reviews the possible relationship between sudden infant death and long QT syndrome. (51 references) (SB)

20050429-10

Infection, health problems, and health care utilisation, and the risk of sudden infant death syndrome. Vennemann MMT, Findeisen M, Butterfaß-Bahloul T, et al (2005), Archives of Disease in Childhood vol 90, no 5, May 2005, pp 520-522

AIM: To examine whether symptoms suggestive of infection, health problems, and health care utilisation are risk factors for SIDS. METHODS: Matched case-control study with 333 SIDS infants and 998 control infants matched for region, age, gender, and reference sleep. Information was obtained by parental interview, paediatrician completed questionnaire, and hospital admission data. RESULTS: No symptoms were associated with SIDS after adjustment for potential confounders. Illness in the last four weeks as reported by the paediatrician did not differ between cases and controls. Developmental problems and special investigations at any stage of life significantly increased the risk of SIDS (adjusted OR = 2.14 and 2.07). Admission to hospital after the first week of life was associated with an increased risk of SIDS (adjusted OR = 1.88). CONCLUSION: Symptoms of infection and illness are no longer risk factors for SIDS in communities such as Germany where few infants sleep prone. The increased risk of SIDS with developmental problems may indicate that infants which subsequently die of SIDS are abnormal or in some way vulnerable. (10 references) (Author)

20050321-27*

A comparison of the effects of parental risk markers on pre- and perinatal variables in multiple patient cohorts with fetal alcohol syndrome, autism, Tourette syndrome, and sudden infant death syndrome: an enviromic analysis. Klug MG, Burd L, Kerbeshian J, et al (2003), Neurotoxicology and Teratology vol 25, no 6, November-December 2003, pp 707-717

The prevalence and magnitude of effect of individual risk markers for specific developmental disorders vary widely across diagnostic category. The four study cohorts for this project were patients from four diagnostic registries in North Dakota for fetal alcohol syndrome (FAS), autism, sudden infant death syndrome (SIDS), and Tourette syndrome. These four cohorts were used to estimate prevalence and magnitude of effect of parental risk markers in patients with developmental disabilities. Cases with North Dakota birth certificates were matched with controls. Using birth certificate data, we then examined five parental risk markers for each cohort and estimated direct and indirect effects for each risk marker by cohort. The authors found two significant paternal risk markers (age in SIDS and education in FAS). Significant maternal markers were age in SIDS, education in FAS, autism, and SIDS. Marital status was a significant risk marker in FAS. Effect sizes were estimated using paired t tests, odds ratios, and population attributable risk (PAR) for both direct and indirect effects for each marker. We estimated both direct and indirect effects to allow for direct comparisons of the differential effect estimates of each of these markers. The direct effect of parental markers differs across diagnostic cohorts of patients. Use of cohorts from similar denominator populations obtained from prevalence studies is a useful methodological tool for estimating the prevalence and magnitude of effect of risk markers. (Author)

20050114-61

Gene-environment interactions: implications for sudden unexpected deaths in infancy. Hunt CE (2005), Archives of Disease in Childhood vol 90, no 1, January 2005, pp 48-53

From the perspective of systems biology, genes and proteins interact to produce complex networks, which in turn interact with the environment to influence every aspect of our biological lives. Recent advances in molecular genetics and the identification of gene polymorphisms in victims of sudden infant death syndrome (SIDS) are helping us better to understand that SIDS, like all other human conditions in health and disease, represents the confluence of specific environmental risk factors interacting in complex ways with specific polymorphisms to yield phenotypes susceptible to sudden and unexpected death in infancy. Failure to consider both genetic and environmental risk factors will impede research progress. (39 references) (Author)

20040903-15

New insight into sudden infant-death syndrome. Opdal SH, Rognum TO (2004), Lancet vol 364, no 9437, 4 September 2004, pp 825-826

Outlines the theories which have been posited for the aetiology of sudden infant death syndrome (SIDS). (16 references) (RM)

20040831-48

Second-trimester maternal serum levels of alpha-fetoprotein and the subsequent risk of sudden infant death syndrome. Smith GCS, Wood AM, Pell JP, et al (2004), The New England Journal of Medicine vol 351, no 10, 2 September 2004, pp 978-986

Background: Unexplained stillbirth and the sudden infant death syndrome (SIDS) share some features. A raised maternal serum level of alpha-fetoprotein during the second trimester of pregnancy is a marker of placental dysfunction and a strong predictor of the risk of unexplained stillbirth. It is unknown whether alpha-fetoprotein levels also predict the risk of SIDS. Methods: We linked a prenatal-screening database for women in western Scotland with databases of maternity, perinatal death, and birth and death certifications to assess the association between second-trimester levels of maternal serum alpha-fetoprotein and the subsequent risk of SIDS. Results: Among 214,532 women with singleton births, there were 114 cases of SIDS (incidence, 2.7 per 10,000 births among women with alpha-fetoprotein levels in the lowest quintile and 7.5 per 10,000 births among those with levels in the highest quintile). When the lowest quintile was used as a referent, the unadjusted odds ratios for SIDS for the second through fifth quintiles were 1.7 (95 percent confidence interval, 0.8 to 3.5), 1.8 (95 percent confidence interval, 0.9 to 3.7), 2.5 (95 percent confidence interval, 1.3 to 4.8), and 2.8 (95 percent confidence interval, 1.4 to 5.4), a respectively (P for trend = 0.001). The risk of SIDS varied inversely with the birth-weight percentile and the gestational age at delivery; after adjustment for these factors, the odds ratios for SIDS were 1.7 (95 percent confidence interval, 0.8 to 3.5), 1.7 (95

percent confidence interval, 0.8 to 3.5), 2.2 (95 percent confidence interval, 1.1 to 4.4), and 2.2 (95 percent confidence interval, 1.1 to 4.3), respectively (P for trend = 0.01). Conclusions: There is a direct association between second-trimester maternal serum alpha-fetoprotein levels and the risk of SIDS, which may be mediated in part through impaired fetal growth and preterm birth. (23 references) (Author)

20040819-10

A multivariate 'time based' analysis of SIDS risk factors. Matthews T, McDonnell M, McGarvey C, et al (2004), Archives of Disease in Childhood vol 89, no 3, March 2004, pp 267-271

AIMS: To investigate the influence of analytical design on the variability of published results in studies of sudden infant death syndrome (SIDS). METHODS: The results of a prospective case-control study, of 203 cases of SIDS, and 622 control infants are presented. All variables significant on univariate analysis were included in a multivariate model analysed in nine stages, starting with sociodemographic variables, then sequentially and cumulatively adding variables relating to pregnancy history, current pregnancy, birth, the interval from birth to the week prior to death, the last week, the last 48 hours, and the last sleep period. A ninth stage was created by adding placed to sleep prone for the last sleep period. RESULTS: As additional variables are added, previously published SIDS risk factors emerged such as social deprivation, young maternal age, > or =3 previous live births, maternal smoking and drinking, urinary tract infection in pregnancy, reduced birth weight, and the infant having an illness, regurgitation, being sweaty, or a history of crying/colic in the interval from birth to the week before death, with co-sleeping and the lack of regular soother use important in the last sleep period. As the model progressed through stages 1-9, many significant variables became non-significant (social deprivation, young maternal age, maternal smoking and drinking) and in stage 9 the addition of placed to sleep prone for the last sleep period caused > or =3 previous live births and a reduced birth weight to become significant. CONCLUSION: The variables found to be significant in a case-control study, depend on what is included in a multivariate model. (21 references) (Author)

20040812-40

Research and sudden infant death syndrome: definitions, diagnostic difficulties and discrepancies. Byard RW, Krous HF (2004), Journal of Paediatrics and Child Health vol 40, no 8, August 2004, pp 419-421

The diagnosis of causes of sudden infant death is an often complex and difficult process. Variable standards of autopsy practice and the use of different definitions for entities such as sudden infant death syndrome (SIDS) have also contributed to confusion and discrepancies. For example, the term SIDS has been used when the requirements of standard definitions have not been fulfilled. In an attempt to correct this situation recent initiatives have been undertaken to stratify cases of unexpected infant death and to institute protocols that provide frameworks for investigations. However, if research is to be meaningful, researchers must be scrupulous in assessing how extensively cases have been investigated and how closely cases fit with internationally recognized definitions and standards. Unless this approach is adopted, evaluation of research findings in SIDS will be difficult and the literature will continue to be beset by contradictions and unsubstantiated conclusions. (13 references) (Author)

20040709-52*

Role of virus-induced myocardial affections in sudden infant death syndrome: a prospective postmortem study. Dettmeyer R, Baasner A, Schlamann M, et al (2004), Pediatric Research vol 55, no 6, June 2004, pp 947-952

The cause of sudden infant death syndrome (SIDS) is an unresolved problem of high relevance. Previous studies indicate a role of infections. In our prospective study, we investigated the frequency of virus-induced myocardial affections in SIDS. Postmortem samples from SIDS victims and control subjects were investigated prospectively. Pediatric cases of unnatural death served as controls. Samples were studied for enteroviruses, adenoviruses, parvovirus B19, and Epstein-Barr virus applying PCR. Immunohistochemical investigations for inflammatory cells, the necrosis marker C5b-9((m)) complement complex, and the enteroviral capsid protein VP1 were performed. Overall, 62 SIDS victims were studied. As controls, 11 infants were enrolled. Enteroviruses were detected in 14 (22.5%), adenoviruses in 2 (3.2%), Epstein-Barr viruses in 3 (4.8%), and parvovirus B19 in 7 (11.2%) cases of SIDS. Control group samples were completely virus negative. Compared with controls, immunohistochemical investigations partially

revealed a significant increase in the number of T lymphocytes in SIDS myocardial samples ($p < 0.05$). Furthermore, cases with elevated numbers of leukocytes and macrophages, microfocal C5b-9((m))(+) necroses, and enteroviral VP1 capsid protein within the myocardium were detected. Applying a comprehensive combination of molecular and immunohistochemical techniques, our results demonstrate a clearly higher prevalence of viral myocardial affections in SIDS. Our results emphasize the importance of PCR-based diagnosis of viral myocardial affections. We suggest preliminary criteria for cellular immunohistochemical diagnosis of viral myocardial affections derived from our findings. For future investigations in SIDS, we suggest a comprehensive approach that includes PCR and immunohistochemistry. Our results offer novel strategies for diagnosis of pediatric myocardial viral affections. (Author)

20040709-34

Unexpected infant death: lessons from the Sally Clark case. Byard RW (2004), Medical Journal of Australia vol 181, no 1, 5 July 2004, pp 52-54

In November 1999, in the United Kingdom, a woman was convicted of the murder of her two infant sons. An appeal against the conviction was dismissed in October 2000, but the conviction was quashed by a second court of appeal in January 2003. Review of the autopsy findings showed that standard procedures had not always been followed, thus limiting verification of the alleged findings. Some potentially important diagnoses and conclusions were also altered over time. This case and its sequelae demonstrate the difficulties that may arise if cases are not fully investigated by pathologists with specific training or experience in paediatric forensic pathology, with all of the Results being clearly summarised and discussed in autopsy reports. Trying to clarify findings, diagnoses and circumstances of death at a later stage may simply not be feasible, owing to a wide variety of possibilities other than inflicted injury. This type of case has unfortunately led to mistrust of the medical and legal systems and has made the investigation of such emotive and tragic cases all the harder. (15 references) (Author)

20040512-7

Clinical correlates, natural history and outcome of neonatal apnoea. Baird TM (2004), Seminars in Neonatology vol 9, no 3, June 2004, pp 205-211

Apnoea is common in the newborn period and especially in preterm newborns. Bradycardia and desaturation of oxyhaemoglobin typically occur with apnoea. These abnormalities reflect an immature cardiorespiratory system and resolution of this immaturity can be expected within a predictable time frame. Infants who have apnoea in the newborn period are thought not to be at higher risk for sudden infant death syndrome (SIDS). Whether apnoea episodes are associated with a higher incidence of long-term handicap for these infants is not yet clear. (47 references) (Author)

20040506-77*

Is sudden infant death syndrome (SIDS) an autoimmune disorder of endogenous vasoactive neuropeptides?. Staines DR (2004), Medical Hypotheses vol 62, no 5, 2004, pp 653-657

Sudden infant death syndrome (SIDS) remains a perplexing diagnosis with conflicting laboratory investigation and lack of a biologically plausible aetiology. Investigations into the endogenous vasoactive neuropeptides, including pituitary adenylate cyclase-activating polypeptide (PACAP) and vasoactive intestinal peptide (VIP) are revealing the critical role these substances have in homeostasis including thermo- and cardiovascular regulation. For example, studies in PACAP receptor-deficient mice have revealed sudden neonatal death attributed to respiratory control defects, possibly due to mutations in genes encoding components of PACAP signalling pathways. PACAP and VIP belong to the secretin/glucagon superfamily of hormones and function as vasoactive neuropeptides. They act as hormones, neurotransmitters, immune modulators and neurotrophes. They are readily catalysed to small peptide fragments. They and their binding sites are immunogenic and are known to be associated with a range of autoimmune conditions. Vasoactive neuropeptides have a known role in thermoregulation and deficiency states are associated with higher neonatal death rates in rats. PACAP plays a significant role in carbohydrate and lipid metabolism and impairment of functioning has potentially serious consequences. It is postulated PACAP and VIP receptors in brain may become

compromised through autoimmune phenomena resulting in cardio-respiratory dysfunction and death. This paper discusses the potential role of certain vasoactive neuropeptides in causing autoimmune responses in susceptible infants predisposing them to SIDS. (Author)

20040427-77*

Apparent life-threatening events and sudden infant death on a monitor. Poets CF (2004), Paediatric Respiratory Reviews vol 5, suppl 1, 2004, pp S383-S386

This review summarises recent data on mechanisms for apparent life-threatening events (ALTE) and sudden infant death (SID) which show that (i). recordings obtained during ALTE allow the detection of previously unrecognised but preventable mechanisms in a significant proportion of infants and should thus be performed routinely in infants with such a history, (ii). in recordings obtained during SID and idiopathic ALTE, prolonged apnoea was found in only a minority, while severe hypoxaemia appeared to the common mechanism, (iii). it remains yet unclear by which mechanism this hypoxaemia develops, with upper and/or lower airway obstruction, rebreathing of expired air and intrapulmonary shunting being potential candidates, (iv). there is evidence that arousal fails during SID, which could be related to known risk factors such as tobacco smoke exposure, whereas (v). gasping occurred during the majority of SID cases where respiratory patterns have been analysed, but it remains unclear why gasping remains ineffective in resuscitating the infant from hypoxaemia. (Author)

20040423-64

Adult hemoglobin levels at birth and risk of sudden infant death syndrome. Richardson DB, Wing S, Lorey F, et al (2004), Archives of Pediatrics and Adolescent Medicine vol 158, no 4, April 2004, pp 366-371

BACKGROUND: During the final weeks of gestation, infants normally begin a transition from the production of fetal to adult hemoglobin. Delayed or faulty transition to the production of adult hemoglobin might play a role in the etiology of sudden infant death syndrome (SIDS). OBJECTIVE: To examine the association between adult hemoglobin levels measured at birth and the subsequent risk of SIDS. DESIGN AND SETTING: Cohort study of all infants born in California between March 1, 1990, and December 31, 1997, who were enrolled in the state's Newborn Screening Program and followed up during the first year of life to identify deaths attributed to SIDS. PARTICIPANTS: Population-based sample of 3.2 million infants. MAIN OUTCOME MEASURE: Risk of death attributed to SIDS. RESULTS: The study included 2425 infants whose deaths were attributed to SIDS. There was an inverse relationship between adult hemoglobin level, expressed as a percentage of total hemoglobin, and the subsequent incidence of SIDS. After adjustment for infant sex, race/ethnicity, length of gestation, maternal age, maternal education, maternal smoking, intrauterine growth restriction, and preeclampsia/eclampsia, the relative risks of SIDS for infants in the lower 4 quintiles of adult hemoglobin level were, in descending order, 1.12 (95% confidence interval [CI], 0.96-1.32), 1.38 (95% CI, 1.19-1.59), 1.55 (95% CI, 1.34-1.80), and 2.15 (95% CI, 1.87-2.47) compared with infants in the highest quintile. CONCLUSIONS: These findings suggest that infants with low levels of adult hemoglobin in the first hours after birth are at elevated risk of SIDS. Delayed maturation in production of adult hemoglobin may play a role in the etiology of some SIDS cases. (26 references) (Author)

20040423-52

Sudden unexpected death in infancy and socioeconomic status: a systematic review. Spencer N, Logan S (2004), Journal of Epidemiology and Community Health vol 58, no 5, May 2004, pp 366-373

This paper aimed to systematically review observational studies documenting the relation between sudden unexpected death in infancy and socioeconomic status. A search of two electronic databases (Medline 1966 to November 2002; Embase 1981 to November 2002) yielded 52 case-control or cohort studies meeting the inclusion criteria. An increased risk of sudden unexpected death in infancy was reported in 51 studies and 32 of 33 studies reporting graded measures of socioeconomic status showed a dose-response relation of sudden death with socioeconomic status. Of the 10 studies in which adjustment was made for maternal smoking, socioeconomic status retained an independent effect on infant death in nine. The effect of socioeconomic status was also independent of birth weight in 10 of 11 studies and independent of sleeping position in two. The included studies reported a

significant association of socioeconomic status with sudden unexpected death in infancy with risk of infant death increasing with greater exposure to adverse social circumstances. The findings support a significant role for adverse social circumstances in the pathways to sudden unexpected death in infancy. (76 references) (Author)

20040414-42

Maternal and Obstetric Risk Factors for Sudden Infant Death Syndrome in the United States. Getahun D, Amre D, Rhoads GG, et al (2004), Obstetrics & Gynecology vol 103, no 4, April 2004, pp 646-652

OBJECTIVE: The objectives of this study were to 1) study the incidence of sudden infant death syndrome (SIDS) among singleton births in the United States and 2) identify maternal and obstetric risk factors for SIDS. METHODS: A cohort of all live births in the United States from 1995 to 1998, formed the source population (n = 15,627,404). The data were obtained from the National Centers for Health Statistics Linked Births and Infant Deaths File. A nested case-control study was used to examine risk factors for SIDS. From this birth cohort, all SIDS deaths (n = 12,404) were first identified (case group). From the remaining non-SIDS births, a 4-fold larger sample (n = 49,616) was randomly selected as a control group. RESULTS: The overall incidence of SIDS was 81.7 per 100,000 live births. More mothers in the case group than in the control group were reported to have placenta previa (odds ratio [OR]: 1.70; 95% confidence interval [CI] 1.24, 2.33), abruptio placentae (OR 1.57; 95% CI 1.24, 1.98), premature rupture of membranes (OR 1.48; 95% CI 1.33, 1.66), or small for gestational age (OR 1.40; 95% CI 1.30, 1.50 for the 10th percentile). SIDS cases were also more likely to be male. Mothers of cases were more likely to be younger, less educated, and nonwhite, and more of them smoked during pregnancy and did not attend prenatal care. CONCLUSION: This analysis confirms the importance of several well known demographic and lifestyle risk factors for SIDS. In addition, placental abnormalities were risk factors for SIDS. LEVEL OF EVIDENCE: II-2 (27 references) (Author)

20040126-26

Sudden infant death syndrome: a critical review of approaches to research. Goldwater PN (2003), Archives of Disease in Childhood vol 88, no 12, December 2003, pp 1095-1100

This review explores the various research approaches taken attempting to solve the problem of SIDS. It would appear that major clues provided by pathological findings have been largely overlooked and as a consequence much effort, time, and money has been wasted on projects that satisfy only sub-specialty and political needs. Close examination of the pathological clues would provide better insights into the mechanisms underlying this enigmatic and heartbreaking problem. (85 references) (Author)

20040119-9*

Questionnaire study to Japanese SIDS families. Sawaguchi T, Yokota S, Nishimaki S, et al (2003), Early Human Development vol 75, suppl, December 2003, pp 181-192

OBJECTIVES: To clarify the situation of the incidence of the sudden infant death syndrome (SIDS) in Japan to provide the basis for health administration training. METHOD: The questionnaire study about the circumstances and responses in discovering the death of a SIDS infant was carried out by the SIDS Family Association Japan. The bereaved parents were asked to reply as to whether or not the SIDS was the cause of their children's death, carried out a cross tabulation and a chi(2) test of significance. RESULT: We found differences between SIDS and non-SIDS infants in respect to the place of death, the health condition up to death, the sleeping position at the time of death and room sharing. With regards to the responses on the death of a child, we found differences between the SIDS and non-SIDS infants in respect to whom the infant was in contact with at the time of death and especially with the responses of the paramedics. Other differences found were in respect to whether or not considerations were given to the family in informing them about procedures and treatment, in informing them of the procedures and medical information or to the family's feelings in advising an autopsy and whether or not a sufficient explanation was provided before the autopsy. CONCLUSION: To lighten the trauma suffered by a SIDS family, it is necessary that we pay adequate attention to giving consideration to the family through general informed consent including giving thoughtful consideration in advising an autopsy. (Author)

20040119-8*

Recent trend of the incidence of sudden infant death syndrome in Japan. Sawaguchi T, Namiki M (2003), Early Human Development vol 75, suppl, December 2003, pp 175-179

OBJECTIVES: To clarify the trend of the incidence of SIDS in the last 20 years in Japan to provide the basis for health administration training. METHOD: We have studied the SIDS incidence rate, the infant mortality rate, the neonatal mortality rate and perinatal deaths of the last 20 years in Japan and calculated the rate at which SIDS has contributed to infant mortality. RESULT: We found that the 2001 SIDS incidence rate in Japan was 0.24 per 1000 births, which had taken a downturn since its upturn of around 1995. The rate of SIDS incidence as a part of the infant mortality rate in 2001 in Japan was 7.7%, which had taken a downturn since its upturn of around 1997. CONCLUSION: The SIDS incidence rate in Japan in recent years is on the decline. (Author)

20031217-35*

Sudden infant death syndrome: a cybernetic etiology. ben-Aaron M (2003), Medical Hypotheses vol 61, no 5-6, November/December 2003, pp 601-604

The brain's processes, by hypothesis, involve information processing by an extraordinarily complex, highly sophisticated, self-organizing cybernetic system embedded in the central nervous system. This cybernetic system generates itself in successive stages. Breathing is, by default, an autonomous function, but breath control is learned. If there is not a smooth transfer of function at the time when a successor system (one that enables autonomous breathing to be overridden by voluntary control) takes over, breathing may cease, without any overt cause being detectable, even with a thorough postmortem examination. If conditions are such that, at that point, the infant's body lacks the strength to resume breathing again under autonomic control, Sudden Infant Death Syndrome may result. The theory explains why infants are at greater risk if they sleep face down. (Author)

20031114-29*

The triple risk hypotheses in sudden infant death syndrome. Guntheroth WG, Spiers PS (2002), Pediatrics vol 110, no 5, November 2002

Sudden infant death syndrome (SIDS) victims were regarded as normal as a matter of definition (Beckwith 1970) until 1952 when Kinney and colleagues argued for elimination of the clause, 'unexpected by history.' They argued that 'not all SIDS victims were normal,' and referred to their hypothesis that SIDS results from brain abnormalities, which they postulated 'to originate in utero and lead to sudden death during a vulnerable postnatal period.' Bergman (1970) argued that SIDS did not depend on any 'single characteristic that ordains a infant for death,' but on an interaction of risk factors with variable probabilities. Wedgwood (1972) agreed and grouped risk factors into the first 'triple risk hypothesis' consisting of general vulnerability, age-specific risks, and precipitating factors. Raring (1975), based on a bell-shaped curve of age of death (log-transformed), concluded that SIDS was a random process with multifactorial causation. Rognum and Saugstad (1993) developed a 'fatal triangle' in 1993, with groupings similar to those of Wedgwood, but included mucosal immunity under a vulnerable developmental stage of the infant. Filiano and Kinney (1994) presented the best known triple risk hypothesis and emphasized prenatal injury of the brainstem. They added a qualifier, 'in at least a subset of SIDS,' but, the National Institute of Child Health and Development SIDS Strategic Plan 2000, quoting Kinney's work, states unequivocally that 'SIDS is a developmental disorder. Its origins are during fetal development.' Except for the emphasis on prenatal origin, all 3 triple risk hypotheses are similar. Interest in the brainstem of SIDS victims began with Naeye's 1976 report of astrogliosis in 50% of all victims. He concluded that these changes were caused by hypoxia and were not the cause of SIDS. He noted an absence of astrogliosis in some older SIDS victims, compatible with a single, terminal episode of hypoxia without previous hypoxic episodes, prenatal or postnatal. Kinney and colleagues (1983) reported gliosis in 22% of their SIDS victims. Subsequently, they instituted studies of neurotransmitter systems in the brainstem, particularly the muscarinic (1995) and serotenergic systems (2001). The major issue is when did the brainstem abnormalities, astrogliosis, or neurotransmitter changes occur and whether either is specific to SIDS. There is no published method known to us of determining the time of origin of these markers except that the injury causing astrogliosis must have occurred at least 4 days before death (Del Bigio

and Becker, 1994). Because the changes in neurotransmitter systems found in the arcuate nucleus in SIDS victims were also found in the chronic controls with known hypoxia, specificity of these markers for SIDS has not been established. It seems likely that the 'acute control' group of Kinney et al (1995) died too quickly to develop gliosis or severe depletion of the neurotransmitter systems. We can conclude that the acute controls had no previous episodes of severe hypoxia, unlike SIDS or their 'chronic controls.' Although the average muscarinic cholinergic receptor level in the SIDS victim was significantly less than in the acute controls, the difference was only 27%, and only 21 of 41 SIDS victims had values below the mean of the acute controls. The study of the medullary serotonergic network by Kinney et al (2001) revealed greater reductions in the SIDS victims than in acute controls, but the questions of cause versus effect of the abnormalities, and whether they occurred prenatally or postnatally, remain unanswered. Hypoplasia of the arcuate nucleus was stated to occur in 5% of their SIDS cases by Kinney et al (2001), but this is a 'primary developmental defect' according to Matturri et al (2002) with a larger series, many of whom were stillbirths. These cases should not be included under the rubric of SIDS, by definition. There are difficulties with Filiano and Kinney's (1994) explanation of the age at death distribution of SIDS. They postulate that the period between 1 and 6 months represents an unstable time for virtually all physiologic systems. However, this period demonstrates much less instability than does the neonatal period, when most deaths from congenital defects and severe maternal anemia occur. We present data for infants born to mothers who were likely to have suffered severe anemia as a consequence of placenta previa, abruptio placentae, and excessive bleeding during pregnancy; these infants presumably are at increased risk of hypoxia and brainstem injury. The total neonatal mortality rate in these 3 groups of infants is 4 times greater than the respective postneonatal mortality, and in the postneonatal period the non-SIDS mortality rate is between 14 and 22 times greater than the postneonatal SIDS rate in these 3 groups. A preponderance of deaths in the neonatal period is also found for congenital anomalies, a category that logically should include infants who experienced prenatal hypoxia or ischemia; this distribution of age of death is very different from that for SIDS, which mostly spares the first month and peaks between 2 and 3 months of age. Finally, evidence inconsistent with prenatal injury as a frequent cause of SIDS comes from prospective studies of ventilatory control in neonates who subsequently died of SIDS; no significant respiratory abnormalities in these infants have been found (Waggener et al 1990; Schectman et al 1991). We conclude that none of the triple risk hypotheses presented so far have significantly improved our understanding of the cause of SIDS. Bergman's and Raring's concepts of multifactorial causation with interaction of risk factors with variable probabilities is less restrictive and more in keeping with the large number of demonstrated risk factors and their varying prevalence. If prenatal hypoxic damage of the brainstem occurred, it seems likely that the infant so afflicted would be at risk for SIDS, but it is even more likely that their death would occur in the neonatal period, as we have demonstrated in infants who have known maternal risk factors that involve severe anemia. This is in contrast to the delay until the postneonatal period of most SIDS deaths. A categorical statement that the origin of SIDS is prenatal is unwarranted by the evidence. Brainstem abnormalities have not been shown to cause SIDS, but are more likely a nonspecific effect of hypoxia. (Only the abstract is published in the print journal. Full article available online at http://www.pediatrics.org/cgi/content/full/111/4/e64) (Author)

20031031-31

Circadian variations in sudden infant death syndrome: associations with maternal smoking, sleeping position and infections. The Nordic Epidemiological SIDS Study. Daltveit AK, Irgens LM, Oyen N, et al (2003), Acta Paediatrica vol 92, no 9, September 2003, pp 1007-1013

Aim: To study circadian variation in the sudden infant death syndrome (SIDS) and possible associations with risk factors for SIDS. Methods: A questionnaire-based case-control study matched for place of birth, age and gender was conducted in Denmark, Norway and Sweden: The Nordic Epidemiological SIDS Study. The study comprised 244 SIDS victims and 869 control infants between September 1992 and August 1995. The main outcome was hour found dead. Results: A significant circadian pattern was observed among the 242 SIDS victims with a known hour found dead, with a peak at 08.00-08.59 in the morning (n = 33). Of the SIDS victims, 12% were found dead at 00.00-05.59,58% at 06.00-11.59,21% at 12.00-17.59 and 9.0% at 18.00- 23.59. When comparing night/morning SIDS and day/evening SIDS (found dead 00.00-11.59 and 12.00-23.59, respectively), the proportion of night/morning SIDS was high among infants of smoking mothers (81 % vs 53%, p < 0.00 I), infants with a reported cold (82% vs 64%, p = 0.007) and infants sleeping side/supine (81 % vs 60%, p < 0.001). No associations were observed between hour found dead and other

sociodemographic risk factors for SIDS. Risk (odds ratio and 95% confidence interval) of night/morning SIDS and day/evening SIDS was 7.0 (4.5-10.9) and 1.5 (0.8-2.5), respectively, for maternal smoking, 2.2 (1.5-3.1) and 0.6 (0.3-1.3), respectively, if the infant had a reported cold, 3.7 (2.1-6.6) and 3.1 (1.1-8.4), respectively, if the infant was put to sleep in the side position (supine reference), and 11.0 (5.9-20.2) and 21.6 (7..6-60.8), respectively, if the infant was put to sleep in the prone position.
Conclusion: The observed higher proportion of night/morning cases in SIDS if the mother smoked, if the infant was reported to have a cold and if the infant was sleeping side/supine may contribute to the understanding of some epidemiological characteristics of SIDS. (26 references) (Author)

20031031-28

Does circadian variation in risk factors for sudden infant death syndrome (SIDS) suggest there are two (or more) SIDS subtypes?. Mitchell EA, Williams SM (2003), Acta Paediatrica vol 92, no 9, September 2003, pp 991-993

Sudden infant death syndrome (SIDS) is known to occur more frequently at night. In two studies it has now been shown that a prone sleep position is more strongly associated with SIDS occurring during the day, whereas night-time deaths are more strongly associated with maternal smoking and illness. Conclusion: This variation, although unexplained, does suggest at least two SIDS subtypes: one related to sleep position and possibly a thermal mechanism, and one related to an uncontrolled inflammatory response to infection predominantly occurring at night. In addition, there are probably other mechanisms that do not show a circadian variation. (27 references) (Author)

20030828-6*

Viruses and sudden infant death. Samuels M (2003), Paediatric Respiratory Reviews vol 4, no 3, September 2003, pp 178-183

Viral respiratory infections are the most likely trigger for sudden infant death syndrome (SIDS). SIDS cases commonly have evidence of respiratory tract inflammation, a preceding history of symptoms of minor illness and occur in winter peaks coinciding with respiratory viral epidemics. Respiratory infections are a common cause for infants presenting with sudden events, involving apnoea and hypoxaemia and there are physiological mechanisms by which infants may develop sudden and severe, potentially life-threatening hypoxaemia. The rate of SIDS has fallen in the last 15 years. This is probably more to do with the reasons for the fall in deaths from respiratory causes rather than changes in sleeping position. Further falls in SIDS death rates may occur with reductions in cigarette smoking, encouragement of breastfeeding and minimising the potential for young infants to acquire respiratory infections. Early identification and recognition of life-threatening features of infections may further minimise the risks of sudden death. (Author)

20030711-30

Apnea, sudden infant death syndrome, and home monitoring. COMMITTEE ON FETUS AND NEWBORN (2003), Pediatrics vol 111, no 4, April 2003, pp 914-917

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. (35 references) (Author)

20030612-36

Analysis of the mitochondrial genome in sudden infant death syndrome. Divne AM, Rasten-Almqvist P, Rajs J, et al (2003), Acta Paediatrica vol 92, no 3, 2003, pp 386-388

AIM: To investigate the mitochondrial genome and its association with sudden infant death syndrome (SIDS).
METHODS: Twenty SIDS infants were screened for previously reported mitochondrial DNA mutations using direct

sequencing. The whole mitochondrial genome was also sequenced for six of the infants. RESULTS: Three substitutions, A11467G, A12308G and G12372A, comprising a haplogroup were present in four infants diagnosed as pure SIDS. This haplogroup was also common in a control group. CONCLUSIONS: No specific mutation or polymorphism was found in association with SIDS. (12
references) (Author)

20030529-50

Sudden infant death due to disseminated pneumococcal infection. Thayyil S, Murthy VN, Thompson F (2003), Archives of Disease in Childhood vol 88, no 2, February 2003, p 157

Case study report on a 6-month old infant who died very suddenly as a result of pneumococcal disease. (6 references) (MS)

20030425-5

Vagal overactivity: a risk factor of sudden infant death syndrome?. Shojaei-Brosseau T, Bonaiti-Pellie C, Lyonnet S, et al (2003), Archives of Disease in Childhood vol 88, no 1, January 2003, p 88

Correspondence which briefly outlines research that found that vagal overactivity may be a risk factor for sudden infant death. (4 references) (SB)

20030425-1

The epidemiology of sudden infant death syndrome. Platt MJ, Pharoah PO (2003), Archives of Disease in Childhood vol 88, no 1, January 2003, pp 27-29

Background: Twins compared to singletons are at increased risk of sudden infant death syndrome (SIDS). Aims: To compare the epidemiology of SIDS in twins and singletons and to test the hypothesis that monozygous (MI) were at greater risk of SIDS than dizygous (DI) twins. Methods: Data from the Office for National Statistics on all registered live births and infant deaths with registered cause of death 'sudden unexpected death in infancy' in England and Wales from 1993 to 1998 were obtained, together with the registered birth weight and, for twins, whether they were of like or unlike sex. Results: The crude relative risk of SIDS in twins is twice that in singletons. There has been a significant temporal decline in SIDS mortality. There is also a significant increase in risk with decreasing birth weight for both twins and singletons. The birth weight specific risk of SIDS in all except for those >/= 3000 g is greater in singletons than in twins. There is no significant difference in risk of SIDS in like compared with unlike sex twins. Conclusions: In spite of a lower risk of SIDS in twins compared with singletons for each birth weight group <3000 g, one component of the higher crude relative risk of SIDS in twins is attributable to the higher proportion of twins that are of low birth weight. A second component is the higher risk in twins compared with singletons for those of birth weight >/= 3000 g. Like sex are at no greater risk than unlike sex twins, which suggests that zygosity is not a significant factor in SIDS. (18 references) (Author)

20030422-39

Defining the sudden infant death syndrome. Beckwith JB (2003), Archives of Pediatrics and Adolescent Medicine vol 157, no 3, March 2003, pp 286-290

Sudden infant death syndrome (SIDS) is a term that was first proposed in 1969 for a distinctive subgroup of unexpected infant deaths that occur during the postneonatal period with relatively consistent clinical, epidemiological, and pathological features. This term played an important role by focusing attention on a major category of postneonatal infant death, providing support to grieving families, and diminishing the guilt and blame characteristic of these deaths. Unfortunately, the application of this term has become increasingly controversial. Some have applied it too liberally, and others not at all. According to the definition proposed in 1969, despite slight changes suggested in 1989, SIDS remains a diagnosis of exclusion. Although this syndrome has several distinctive features, including age distribution and apparent occurrence during sleep, there has been reluctance to include these features in the definition. The problems created by the lack of an adequate definition are discussed. A 2-tiered approach is suggested,

with a more general definition intended primarily for case management and death administration, and a more restrictive one intended primarily for research purposes, which distinguishes those deaths closely fitting the classic SIDS profile from those with one or more less typical features. (15 references) (Author)

20021223-65*

Quality of diet, body position, and time after feeding influence behavioral states in low birth weight infants. Sahni R, Saluja D, Schulze KF, et al (2002), Pediatric Research vol 52, no 3, 2002, pp 399-404

The effects of variations in carbohydrate and fat intake and body position on behavioral activity states were evaluated in 64 healthy, growing low birth weight infants (birth weight, 750- 1600 g). The infants, enrolled in a prospective, randomized, double-blind, controlled study of effects of quality of dietary energy, were fed one of the five formulas. These formulas contained fixed intakes of protein (4 g/kg per day) but different intakes of carbohydrate (9.1 to 20.4 g/kg per day) and fat (4.3 to 9.5 g/kg per day). Six-hour daytime sleep studies were performed at 2-wk intervals from time of full enteral intake until discharge (mean postconceptional age at first study, 33.2 ±1.8 wk). Infants were randomly assigned to the prone or supine position for the first 3-h postprandial period; the position was reversed during the second 3 h. Behavioral activity state, i.e. quiet sleep (QS), active sleep, indeterminate sleep, awake, or crying was coded each minute throughout the postprandial period. The overall incidence of QS was almost double in the prone position versus the supine ($p < 0.0001$). In contrast, the probability of being in either of the two wakeful states (awake and crying) was increased when infants were placed in supine position ($p < 0.0001$). Increased likelihood of being in QS while prone was found only during the 30 min after and before feeding in a 150-min prandial cycle. In contrast, increased amounts of awake and crying in supine position were observed throughout the feeding interval. As carbohydrate intake increased, time spent in QS in supine position increased (from 8.6% to 12.5%, $p < 0.02$), and a trend in the same direction was noted for the prone position ($p = 0.06$). However, during postprandial minutes 10-100, when QS is likely to be entrained by the nutrient intake, enhancement of QS was found in the prone position only ($p < 0.02$). Carbohydrate intake influences the total time spent and the distribution of behavioral activity states within the postprandial period in low birth weight infants. The effect of nutrient intake on sleep profile is dependent on body position and time after feed. Mechanistic hypotheses relating sudden infant death syndrome to sleeping position may need to take these observations into account. (35 references) (Author)

20021216-11

Cardiac pathology in sudden unexpected infant death. Dancea A, Cote A, Roblicek C, et al (2002), Journal of Pediatrics vol 141, no 3, September 2002, pp 336-342

Objectives: To determine the spectrum of cardiac pathology and circumstances of death in infants with sudden unexpected death and to define the impact of sudden cardiac deaths to overall sudden infant death. Study design: Retrospective analysis of all autopsies of infants with sudden death 7 days to 2 years of age between January 1987 and December 1999 in the province of Quebec (Canada). Results: Eighty-two cases of sudden death with cardiac pathology were found, representing 10% of the total number of sudden infant deaths. A structural malformation was present in the majority of cases (54%); however, cardiac pathology in anatomically normal hearts was also common (46%). Most (64%) anatomic malformations were detected before death compared with 13% of nonstructural heart disease. Although a major proportion of children were found dead during sleep, a significant number were described as being awake at time of death (32%). Conclusions: Heart disease is present in a significant percentage of autopsies of infants with sudden death. Structural heart malformations predominate, although nonstructural pathologic features of the heart are common and usually unrecognized before an autopsy is performed. Cardiac pathologic features are frequent when the child is witnessed to be awake at the time of sudden death. (48 references) (Author)

20020905-11

Effects of maternal tobacco smoking, sleeping position, and sleep state on arousal in healthy term infants. Horne RSC, Ferens D, Watts AM, et al (2002), Archives of Disease in Childhood: Fetal and Neonatal Edition vol 87, no 2, September 2002, pp F100-F105

Objectives: To investigate whether a history of maternal tobacco smoking affected the maturation of arousal

responses and whether sleeping position and infant age alters these relations. Design: Healthy term infants (13 born to mothers who did not smoke and 11 to mothers who smoked during pregnancy) were studied using daytime polysomnography on three occasions: (a) two to three weeks after birth, (b) two to three months after birth, and (c) five to six months after birth. Multiple measurements of arousal threshold in response to air jet stimulation were made in both active sleep (AS) and quiet sleep (QS) when infants slept both prone and supine. Results: Maternal smoking significantly elevated arousal threshold in QS when infants slept supine at 2-3 months of age ($p<0.05$). Infants of smoking mothers also had fewer spontaneous arousals from QS at 2-3 months in both prone ($p<0.051$ and supine ($p<0.001$) sleeping positions. In infants of non-smoking mothers, arousal thresholds were elevated in the prone position in AS at 2-3 months ($p<0.001$) and QS at 2-3 weeks ($p<0.051$ and 2-3 months ($p<0.001$). Conclusions: Maternal tobacco smoking significantly impairs both stimulus induced and spontaneous arousal from QS when infants sleep in the supine position, at the age when the incidence of sudden infant death syndrome is highest. (32 references) (Author)

20020829-20

Increased facial temperature as an early warning in sudden infant death syndrome. Russell MJ, Vink R (2001), Medical Hypotheses vol 57, no 1, 2001, pp 61-63

The promotion of supine sleeping position in young infants has resulted in significant declines in the incidence of Sudden Infant Death Syndrome although little is understood in terms of mechanisms. We hypothesize that supine sleeping position promotes appropriate thermal regulation via the face and head which is the major source of infant heat loss. By facilitating temperature regulation, the supine position ensures that the centre for thermoregulation in the hypothalamus does not become dysfunctional due to local temperature fluctuations. Because these hypothalamic, thermoregulatory neurones are synaptically linked to those regulating respiration in the medulla, adequate temperature control by the infant maintains normal respiration. In contrast, an increase in face and head temperature over and above core temperature would suggest thermoregulatory stress and an increased likelihood of respiratory apnoea. (23 references) (Author)

20020828-56

Is there a link between infant botulism and sudden infant death? Bacteriological results obtained in Central Germany. Bohnel H, Behrens S, Loch P, et al (2001), European Journal of Pediatrics vol 160, no 10, 2001, pp 623-628

Despite the fact that botulism was described in I Germany for the first time by Kerner in 1820, the disease is almost forgotten in this country. Only about 10-20 cases of classical botulism (intoxication) are recorded every year, including 1-2 cases of clinical infant botulism. As we assumed a high incidence of botulism to be connected with cases of sudden infant death (SID), we undertook the research work presented here. From every case of unexpected infant death up to 12 months of age, standardised specimens (blood, liver and intestine) were taken at autopsy. They were tested for the presence of botulinum neurotoxin (BoNT) and/or bacterial forms of Clostridium botulinum with subsequent BoNT neutralisation tests by the international standard mouse bioassay. Age, sex, pathological findings and season were recorded. Over a 5-year period, 75 samples including 57 SID cases were tested. Free toxin was found in nine and bacterial forms were detected in six samples. Toxin neutralisation revealed the definite presence of BoNT/ BoNT producing bacteria (mainly type E), whereas another 11 toxin tests were inconclusive. According to international literature, these 15 cases are to be interpreted as infant botulism. Conclusion: the results show a remarkable incidence of infant botulism without any known previous medical history, partly hidden as sudden infant death. We propose to systematically search for botulism in connection with sudden infant death. (40 references) (Author)

20020828-24

Sudden infant death syndrome requires genetic disposition, some form of stress and marginal malnutrition. Lonsdale D (2001), Medical Hypotheses vol 57, no 3, 2001, pp 382-386

Over the past 30 years or more, the problem of sudden, unexplained death in infants (8108) has made little headway.

Many hypotheses have been offered but the basic cause remains elusive. The only successful prevention has been made by the supine sleeping posture. There is still, however, a hard core of unexplained incidents. There is evidence that certain stress factors are involved, and there is good evidence that the tragedy has a familial or genetic tendency. The third factor necessary for the event is inefficient oxidation in brain cells induced most commonly by marginal malnutrition in pregnancy or after birth. The absence of anyone or more of these three factors decreases risk to the point of extinction. Anything that impedes healthy oxidation, or accelerates energy utilization through responding to stress, increases the risk greatly. Improving the biochemical mechanisms through appropriate nutrition is by far the best defense. (44 references) (Author)

20020722-34

A review of the anatomy of the upper airway in early infancy and its possible relevance to SIDS. Tonkin SL, Gunn TR, Bennet L, et al (2002), Early Human Development vol 66, 2002, pp 107-121

Background: Since the danger of prone sleeping in the first 6 months of life has been publicised, there has been a dramatic and consistent reduction in the incidence of sudden infant death syndrome (SIDS). However, unexpected infant deaths and apparent life-threatening events (ALTEs) continue to occur that are clearly not associated with known epidemiological risk factors. Aims: To review the unique features of the anatomy and function of the upper airway of the young infant which contribute to increased vulnerability to hypoxia in this age group. We discuss the clinical identification of those infants at risk of obstruction or restriction of the upper airway and the management of the 'at risk' infant. Conclusions: In the era after the 'back to sleep' campaigns, it is likely that an increasing proportion of cases of ALTEs and SIDS will be related to obstruction or limitation of upper airway size leading to sleep hypoxia/asphyxia. This type of problem may be anticipated by evaluation and investigation of infants with signs or a clinical history consistent with possible upper respiratory tract compromise, including micrognathia. (61 references) (Author)

20020705-14

Breast feeding and the sudden infant death syndrome in Scandinavia, 1992-95. Alm B, Wennergren G, Norvenius SG, et al (2002), Archives of Disease in Childhood vol 86, no 6, June 2002, pp 400-402

Aims: To assess the effects of breast feeding habits on sudden infant death syndrome (SIDS). Methods: The analyses are based on data from the Nordic Epidemiological SIDS Study, a case-control study in which parents of SIDS victims in the Scandinavian countries between 1 September 1992 and 31 August 1995 were invited to participate, each with parents of four matched controls. The odds ratios presented were computed by conditional logistic regression analysis. Results: After adjustment for smoking during pregnancy, paternal employment, sleeping position, and age of the infant, the adjusted odds ratio (95% CI) was 5.1 (2.3 to 11.2) if the infant was exclusively breast fed for less than four weeks, 3.7 (1.6 to 8.4) for 4-7 weeks, 1.6 (0.7 to 3.6) for 8-11 weeks, and 2.8 (1.2 to 6.8) for 12-15 weeks, with exclusive breast feeding over 16 weeks as the reference. Mixed feeding in the first week post-partum did not increase the risk. Conclusions: The study is supportive of a weak relation between breast feeding and SIDS reduction. (22 references) (Author)

20020703-2

Sudden infant death syndrome risk factors in north Queensland: a survey of infant-care practices in Indigenous and non-Indigenous women. Panaretto KS, Smallwood VE, Cole P, et al (2002), Journal of Paediatrics and Child Health vol 38, no 2, April 2002, pp 129-134

Objective: To assess the prevalence of sudden infant death syndrome (SIDS) risk factors in the Indigenous and non-Indigenous community of Townsville, a large remote urban centre in north Queensland, Australia. Methods: Thirty Indigenous and 30 non-Indigenous women with young children were surveyed using sections of the West Australian Infancy and Pregnancy Survey 1997-1998. The prevalence of SIDS risk factors was compared between the two groups and medians and univariate associations were generated where appropriate. Results: The Indigenous women were significantly younger and more likely to be single. The median age of the infants was 8 months (range 0.3-26 months) with no difference between the two groups. Thirty-seven per cent of Indigenous infants slept prone

(cf. 17% of non-Indigenous infants; P = 0.03), and 77% shared a bed (cf. 13% of non-Indigenous infants; P < 0.001). The Indigenous households had significantly more members, with 57% including extended family members (cf. 20% non-Indigenous group; P = 0.003). Fifty-three per cent of the Indigenous women smoked during pregnancy (cf. 23% of non-Indigenous women; P = 0.017), 60% were smokers at the time of the interview, and smoking occurred inside 40% of Indigenous houses (cf. 20% and 20% for non-Indigenous women, respectively; P < 0.001,0.09).
Conclusion: This small survey suggests that the prevalence of SIDS risk factors is higher in the Indigenous population, and a new approach to education is needed urgently to promote SIDS awareness among Indigenous women. (31 references) (Author)

20020619-47

Awareness of sudden infant death syndrome risk factors among mothers of Pacific infants in New Zealand. Paterson J, Tukuitonga C, Butler S, et al (2002), New Zealand Medical Journal vol 115, 8 February 2002, pp 33-35

Aim. To describe the awareness of Sudden Infant Death Syndrome (SIDS) risk factors among mother of Pacific infants in New Zealand. Methods. The data were gathered as part of the Pacific Islands Families Study in which 1376 mothers were interviewed when their infants were six weeks old. Included in this interview were questions designed to examine the mothers' awareness of SIDS risk factors. Results. Over one third (38.8%) of mothers were unable to accurately report a SIDS risk factor, 53.4% reported the risk associated with putting the baby to sleep in a prone position, 31.5% maternal smoking, and 19.5% correctly reported other SIDS risk factors. Lack of awareness of SIDS risk factors was significantly associated with Samoan and Cook Islands Maori ethnicity, being Pacific Islands born, having no post school qualifications, lower household income, not being fluent in English, having more than five children, and not attending antenatal classes. Conclusions. Despite SIDS prevention efforts, a considerable number of mothers in this cohort reported no awareness of SIDS risk factors. More effective methods are needed to provide consistent SIDS prevention information across Pacific ethnic groups. (24 references) (Author)

20020613-5

Increased inspiratory effort in infants with a history of apparent life-threatening events. Samuels M (2002), Acta Paediatrica vol 91, no 3, 2002, pp 260-261

Commentary on research evidence for the relationship between apparent life threatening events in infants and respiratory problems in infants. The possible increased risks of sudden infant death are examined. (17 references) (KL)

20020523-80

Is a rare gene behind cot deaths?. Polak M (2002), General Practitioner 13 May 2002, p 63

Researchers at Edinburgh University and the Western General Hospital, led by neuropathologist Jeanne Bell, are investigating whether babies who die of sudden infant death carry a particular gene. The apolipoprotein E gene has been implicated in Alzheimer's and with poor recovery in cases of brain and head injuries. (Author)

20020516-8

Parallel incidences of sudden infant death syndrome and infantile hypertrophic pyloric stenosis: a common cause?. Persson S, Ekbom A, Granath F, et al (2001), Pediatrics vol 108, no 4, October 2001. 5 pages

Objective. To determine whether there was a correlation between the incidence of infantile hypertrophic pyloric stenosis (IHPS) and the incidence of sudden infant death syndrome (SIDS) during the period 1970 to 1997 and to discuss different causative factors that could be influencing the changing trend in incidence. Methods. We compared the incidence of IHPS in the Stockholm Health Care Region with the incidence of SIDS in Sweden each year between 1970 and 1997. First, the relation was assessed by calculation of a correlation coefficient; second, the relative linear decrease was estimated for the time period 1990 to 1997. Results. The incidence of IHPS increased steadily during the 1970s, from 0.5 per 1000 live births in 1970 to 2.7 in 1979. During the 1980s, the average incidence was 2.8. During the 1990s, there was a significant decrease in the number of IHPS cases in Stockholm. The incidence rate of IHPS parallels

the incidence of SIDS during the study period (r = 0.58). The incidence of SIDS dropped after the risk-reduction campaign in the beginning of the 1990s, which recommended that infants sleep on their back. We could not identify any other changes of behavioral risk factors in early exposures that could explain the temporal trends. Conclusions. The statistical findings suggest that IHPS and SIDS have causative factors in common. We suggest that prone sleeping is one of those factors. (Only the abstract is published in the print journal. Full article available online at www.pediatrics.org/cgi/content/full/108/4/e70 or from MIDIRS subject to usual copyright restrictions). (33 references) (Author)

20020425-75

Febrile convulsions and sudden infant death syndrome. Vestergaard M, Basso O, Henriksen TB, et al (2002), Archives of Disease in Childhood vol 86, no 2, February 2002, pp 125-126

It has been suggested that sudden infant death syndrome (SIDS) and febrile convulsions are related aetiologically. We compared the risk of SIDS in 9877 siblings of children who had had febrile convulsions with that of 20 177 siblings of children who had never had febrile convulsions. We found no support for the shared susceptibility hypothesis. (6 references) (Author)

20020411-51

Prenatal and intrapartum events and sudden infant death syndrome. Klonoff-Cohen HS, Srinivasan IP, Edelstein SL (2002), Paediatric and Perinatal Epidemiology vol 16, no 1, January 2002, pp 82-89

The purpose of this study was to evaluate specific pregnancy and labour and delivery events that may increase the risk of sudden infant death syndrome (SIDS). A matched case-control study was conducted in five counties in southern California, using California death certificate records. The sample consisted of 239 Caucasian, African-American, Hispanic and Asian mothers of SIDS infants and 239 mothers of control infants matched on sex, race, birth hospital and date of birth. Mothers participated in a detailed telephone interview and provided access to obstetric and paediatric records. More case than control mothers reported a family history of anaemia (OR = 2.12, p< 0.001). Placental abruptions were strongly associated with SIDS (unadjusted OR = 7.94, [95% CI 1.34,47.12]). There was an increased risk of SIDS death associated with maternal anaemia during pregnancy (OR = 2.51, [95% CI 1.25,5.03]), while simultaneously adjusting for maternal smoking during pregnancy, maternal years of education and age, parity, infant birthweight, gestational age, medical conditions at birth, infant sleep position and post-natal smoking. Interactions of anaemia and pre-natal smoking as well as anaemia and post-natal smoking were not statistically significant. There were no other statistically significant differences between case and control mothers for pregnancy conditions, labour and delivery events (e.g. caesarean sections, anaesthesia, forceps) or newborn complications (e.g. nuchal cord, meconium aspiration). Anaemia and placental abruptions were significantly associated with an increased risk of SIDS; both are circumstances in which a fetus may become hypoxic, thereby compromising the subsequent growth, development and ultimate survival of the infant. (26 references) (Author)

20020205-27

Understanding the child killer. Munro R (2002), Nursing Times vol 98, no 2, 10-16 January 2002, p 12

Sudden infant death syndrome claims the lives of seven UK babies a week, with few warning signs. Robert Munro explains how specialist investigation and coordination between health professionals is vital to understand and prevent it. (Author)

20011213-8

Ventilatory responses to rebreathing in infants exposed to maternal smoking. Campbell AJ, Galland BC, Bolton DPG, and others (2001), Acta Paediatrica vol 90, no 7, July 2001, pp 793-800

This study assessed the effect of maternal smoking during pregnancy on ventilatory and waking responses of infants to a respiratory challenge. This challenge mimicked the time-course and concentration of gases that an infant would experience rebreathing face-down into soft bedding. Control (C n = 97) and smokers' infants (SM; n = 96) were studied

at ages 1 and 3 mo. Asphyxial gas (hypercapnia/hypoxia) was delivered to the supine sleeping baby via a hood by slowly altering the inspired air: C02 maximum 5% and 02 minimum 13.5%. Respiratory pattern was recorded by inductive plethysmography as the sum of ribcage and abdominal movements. The change in ventilation with inspired C02 was measured over 5-6 min of the test. The slope of a linear curve fit relating inspired C02 to the logarithm of ventilation was taken as a quantitative measure of ventilatory asphyxial sensitivity (VAS). Protective responses were graded from 1: no waking and an estimated arterial carbon dioxide tension (PaC02) >-60 mmHg (least protective), to 4: fully awake (most protective). The results showed VAS was higher in SM infants than controls: +0.03 (p = 0.04). The oxygen saturation (SaO2) of SM infants fell -0.4% (p = 0.02) more than SaO2 Of controls despite a greater tidal volume increase: + 13.0% (p = 0.04). Overall protective responses were the same between groups, but grade 1, although rare, was found in 7 SM infants and only 4 control infants; this difference was not significant. Conclusion: The study did not confirm the postulate that infants of mothers who smoked during pregnancy have a reduced ventilatory response or raised waking thresholds. (31 references) (Author)

20011106-9

Sudden infant death syndrome: neonatal hypodynamia (reduced exercise level). Reid GM (2001), Medical Hypotheses vol 56, no 3, 2001, pp 280-285

Sudden infant death syndrome (SIDS) has been described as a silent unexpected death during sleep. Infants with near-miss SIDS have shown a higher heart rate and diminished heart rate variability during sleep. Non-rapid-eye-movement (NREM) sleep rate variability was related to respiration. A decreased heart rate variability was also observed in infants with respiratory distress syndrome (RDS) or prenatal hypoxia. It was hypothesized that decreased heart rate variability and decreased body measurement during sleep were related to a decreased arousal response. Cardiac output is greater in the supine position. Acetylcholine slows the heart beat. Postural changes modify the acute baroreflex control of the heart rate. The cerebellum also contributes to the reflex anti-orthostatic (supine) cardiovascular response to postural change. Delayed myelination of various areas of the brain occurred in SIDS victims and it was suggested that the defect in central respiratory control could be a motor rather than a sensory problem, and that the search for abnormalities should be extended to regions in the cerebellum and pre-frontal-temporal-limbic systems. The cerebellum exercises control over motor neuron impulses from the cerebral cortex to lower structures. An extended period of neonatal decreased body movement has its counterpart in the astronaut exposed to the deconditioning effect of zero gravity. Hypodynamia induces hyperglycemia, insulin resistance, renal inositoluria and impaired nerve conduction. Myoinositol is 20 times higher in fetal-like tissue than in adults. The insecticide lindane (gammexane) is an inositol antagonist. Lindane administration to neonatal rats induced low levels of specific components of myelin proteins in oligodendrocytes in the brain. The activity of these specific enzymes was reduced in oligodendrocytes in the brain of SIDS victims. It is hypothesized that lindane administration to laboratory neonatal animals is a laboratory model for studying delayed development of the brain in SIDS. (56 references). (Author).

20011106-3

Sudden infant death syndrome (SIDS): T-cell immunodeficiency - part 1. Reid GM (2001), Medical Hypotheses vol 56, no 2, 2001, pp 256-258

It is hypothesized that SIDS mimics AIDS and atopic eczema in that defective T lymphocytes and overactive B cells overstimulate pro-inflammatory cytokines in the mucosal immune system. Virally infected cells are unable to convert linoleic acid (LA) into gamma-linolenic acid (GLA) which eventually leads to defective T lymphocyte production. Abnormal lung cytokine synthesis by virus-induced immunodeficient T lymphocytes is associated with the murine AIDS-related complex (ARC). Adenosine triphosphate (ATP) deficient anaerobic cells cannot convert LA to GLA. It is hypothesized that, in SIDS victims, elevated levels of hypoxanthine and immunoglobulins are evidence of chronic hypoxemia and ATP catabolism, and an inability to convert LA to GLA, leading to defective T lymphocytes in the mucosal immune system. (30 references). (Author).

20011106-15

Siblings of sudden infant death syndrome and near miss in about 30 families: is there a genetic factor?. Calenda PL, Mallet E, Calenda E (2000), Medical Hypotheses vol 54, no 3, 2000, pp 408-411

The purposes of this study were: (1) to compare our data with those reported in the general population; (2) to find a factor implicated in repetitive accidents; (3) to look for a possible genetic factor; and (4) to evaluate whether or not the risk of recurrence was the same in a family with two or more infants having died of sudden infant death syndrome (SIDS) as in the general population. We study retrospectively 77 files which constitute a group of 30 families which was analysed with reference to a list of data laid out in the shape of genealogical trees. Our study shows that risk factors are similar to those described previously and repetitive among sibling or cousins. The rate of recurrence is not available for the general population. On studying some family trees, we can speculate on the existence of an autosomal dominant gene with incomplete penetrance. (5 references). (Author).

20010831-2

Sleep, sleep position, and the sudden infant death syndrome: to sleep or not to sleep? That is the question. Thach BT (2001), Journal of Pediatrics vol 138, no 6, June 2001, pp 793-795

Editorial commentary on an article published in this journal issue (1) which investigates a potential cause of sudden infant death - failure to arouse from sleep under a life-threatening situation. 1. Horne RSC, Ferens, D, Watts A, and others. The prone sleeping position impairs arousability in healthy term infants. Journal of Pediatrics, vol 138, 2001, pp 811-816. (20 references) (KL)

20010822-4

Cot death confusion: explaining the unexplainable. Jackson T (2001), BMJ vol 323, no 7308, 11 August 2001, p 347

Commentary on recent news items reporting on the case of solicitor Sally Clark, convicted in November 1999 of murdering two of her children. Misgivings about the safety of the conviction have been expressed by many, including prominent doctors. Elements of the debate are discussed. (KL)

20010703-2

The influence of race and gestational age on the age of maximum risk of SIDS in infancy. Spiers PS (2000), Medical Hypotheses vol 55, no 1, July 2000, pp 51-55

Risk of sudden infant death syndrome (SIDS) reaches a maximum in the third month. Thereafter, it decreases by half every 40 days or so. It is proposed that the relative sparing of the very young infant is a consequence of an innate (but temporary) characteristic possessed by the newborn infant. Interpretation of available data suggests that this innate characteristic is negatively associated with the infant's level of maturity. This is the basis for the hypothesis that the age at which the risk of SIDS begins to decline at a uniform rate decreases as the infant's gestational age increases. Because of a greater level of maturity at birth, the age at which this occurs in the black infant should be earlier than average. An analysis of the data on 32 573 instances of SIDS within the United States between 1985 and 1991 provides support for the hypothesis. (29 references) (Author)

20010627-15

Serotonin transporter gene variation is a risk factor for sudden infant death syndrome in the Japanese population. Narita N, Narita M, Takashima S, and others (2001), Pediatrics vol 107, no 4, April 2001, pp 690-692

Objective. Serotonin (5-HT) in the nervous system is a major factor in facilitation of the brain center for respiration. Variations in the promoter region of the 5-HT transporter (5-HTT) gene have been shown to potentially regulate 5-HT activity in the brain. Therefore, we aimed to identify the possibility that specific allele variants of the 5-HTT gene can be found as a genetic background for sudden infant death syndrome (SIDS). Methods. Polymorphisms in the 5' regulatory region of the 5-HTT gene were determined in genomic DNA obtained from 27 SIDS victims and 115 age-matched health control participants.

Results. There were significant differences in genotype distribution and allele frequency of the 5-HTT promoter gene between SIDS victims and age-matched control participants. The L and XL alleles were more frequently found in SIDS

victims than in age-matched control participants. Conclusion. Efficiency in the transportation of 5-HTT with the L allele is known to be higher than that with the S allele. The excitatory function by 5-HT is considered to be lower in the respiratory center of individuals with the L allele compared with those with S allele. The XL allele variant has shown another novel biological risk factor for SIDS. (19 references) (Author)

20010626-28

A competing risk model of sudden infant death syndrome incidence in two US birth cohorts. Pollack HA, Frohna JG (2001), Journal of Pediatrics vol 138, no 5, May 2001, pp 661-667

Objectives: To compare changing incidence and changing risk factors associated with sudden infant death syndrome (SIDS) in the 1989 and 1996 US birth cohorts. Study Design: All available singleton births over 500 g from the 1989 linked birth-infant death file and the 1996 and 1997 Perinatal Mortality files were examined. A log-logistic survival model was used to explicitly account for declining competing risks among low birth weight infants. Results: Controlling for maternal prenatal smoking and other confounders, SIDS incidence declined by >33% between the 2 survey years (adjusted odds ratio = 0.628 with 95% CI [0.598, 0.660]). Self-reported declines in maternal prenatal smoking were also associated with significantly declines in SIDS incidence. African American infants and infants born weighing <100 g experienced relative risk compared with non-Hispanic white infants born weighing <2500g. Hispanic/Latino infants had significantly lower SIDS risk than non-Hispanic white infants in both years. Accounting for declining competing risks and other factors, relative SIDS risks among infants born between 500 and 1000 g increased over the study period. Conclusions: SIDS incidence sharply declined between 2989 and 1996. High incidence of SIDS in African Americans and increased relative SIDS risk for infants born weighing <1000g require increased attention from clinicians and public health policy makers. (32 references) (Author)

20010621-24

Review of risk factors for sudden infant death syndrome. Sullivan FM, Barlow SM (2001), Paediatric and Perinatal Epidemiology vol 15, no 2, April 2001, pp 144-200

Sudden infant death syndrome (SIDS) accounts for the largest number of deaths during the first year of life in developed countries. The possible causes of SIDS are numerous and, to date, there is no adequate unifying pathological explanation for SIDS. Epidemiological studies have played a key role in identifying risk factors, knowledge of which has underpinned successful preventive programmes. This review critically assesses information on the main risk factors and causal hypotheses put forward for SIDS, focusing on research published since 1994. The overall picture that emerges from this review is that affected infants are not completely normal in development, but possess some inherent weakness, which may only become obvious when the infant is subjected to stress. Initially there may be some minor impairment or delay in development of respiratory, cardiovascular or neuromuscular function. None of these is likely to be sufficient, in isolation, to cause death and, provided the infant survives the first year of life, may no longer be of any significance. However, when a compromised infant is confronted with one or more stressful situations, several of which are now clearly identified as risk factors, and from which the majority of infants would normally escape, the combination may prove fatal. (322 references) (Author)

20010530-3

Parental reported apnoea, admissions to hospital and sudden infant death syndrome. Mitchell EA, Thomson JMD (2001), Acta Paediatrica vol 90, no 4, April 2001, pp 417-422

Three studies were undertaken: (i) a nation-wide case-control study for sudden infant death syndrome (SIDS), with 393 cases and 1592 controls, examined the association between parental reported apnoea and SIDS; (ii) a case-cohort study, with 84 cases of parental reported apnoea and 1502 controls, aimed to identify risk factors for apnoea; and (ii) national hospital admission data for ALTE and national SIDS mortality data were compared for the years 1986 to 1994. Parental reported apnoea was associated with a significant increased risk of SIDS [adjusted odds ratio (OR) 1.86; 95% confidence interval (CI) 1.12, 3.09]. The population attributable risk was 8%. There was a significant increased risk for parental reported apnoea in infants who did not die after adjustment for potential confounders with maternal smokers, short gestation and admission to the neonatal unit. There was no association with prone sleeping position,

co-sleeping and bottle feeding. The mean annual admission rate for ALTE was 9.4/1000 live births. This did not change significantly over the study period (1986-1994). In contrast, the SIDS mortality rate decreased from over 4/1000 to 2.1/1000. Admission rates were higher for Maori infants and boys. Conclusion: It may be concluded that the relationship between parental reported apnoea and SIDS is tenuous. (16 references) (Author)

20010503-37

SIDS: more facts and controversies. Goldwater PN (2001), Medical Journal of Australia vol 174, no 6, 19 March 2001, pp 302-304

A more robust theory of the causation of sudden infant death syndrome (SIDS) is needed. The asphyxial theory of SIDS, which encompasses the prone sleeping position, relies on contradictory pathological evidence and fails to explain infants with SIDS who are found in the supine or lateral position. Many of the risk factors for SIDS point to an infective cause. The relative risks of these infection-related factors differ from study to study, as does the relative risk of prone sleeping position. I present the case for an infection model for SIDS causation, which has largely been neglected by mainstream SIDS researchers. (36 references) (Author)

20010409-13

The UK accelerated immunisation programme and sudden unexpected death in infancy: case-control study. Fleming PJ, Blair PS, Platt MW, and others (2001), BMJ vol 322, no 7290, 7 April 2001, pp 822-825

Subjects: Immunisation details were available for 93% (303/325) of infants whose deaths were attributed to the sudden infant death syndrome (SIDS); 90% (65/72) of infants with explained sudden deaths; and 95% (1515/1588) of controls. Results: After all potential confounding factors were controlled for, immunisation uptake was strongly associated with a lower risk of SIDS (odds ratio 0.45 (95% confidence interval 0.24 to 0.85)). This difference became non-significant (0.67 (0.31 to 1.43)) after further adjustment for other factors specific to the infants sleeping environment. Similar proportions of SIDS deaths and reference sleeps (corresponding to the time of day during which the index baby had died) among the controls occurred within 48 hours of the last vaccination (5% (7/149) v 5% (41/822)) and within two weeks (21% (31/149) v 27% (224/822)). No longer term temporal association with immunisation was found (P = 0.78). Of the SIDS infants who died within two weeks of vaccination, 16% (5/31) had signs and symptoms of illness that suggested that medical contact was required, compared with 26% (16/61) of the non-immunised SIDS infants of similar age. The findings for the infants who died suddenly and unexpectedly but of explained causes mirrored those for SIDS infants. Conclusions: Immunisation does not lead to sudden unexpected death in infancy, and the direction of the relation is towards protection rather than risk. (19 references) (Author)

20010402-23

The black infant's susceptibility to sudden infant death syndrome and respiratory infection in late infancy. Spiers PS, Guntheroth WG (2001), Epidemiology vol 12, no 1, January 2001, pp 33-37

Between 2 and 11 months of age, the risk of sudden infant death syndrome (SIDS) declines more slowly in black infants than in infants of other races. This phenomenon might also be a feature of certain non-SIDS causes of death. Identifying these causes may through analogy provide support for the theory that SIDS is a disease of the central nervous system, an unusual consequence of respiratory infection, or a form of suffocation. We used logistic regression analysis on details of infant deaths in the United States, 1985-1991, to examine the difference between the rates of decline with increasing age in the mortality rates of black infants and infants of other races. We defined slower rate of decline in black infants as a positive difference. The magnitude and direction (positive) of the difference for deaths due to respiratory infection were similar to those for SIDS. It is unlikely that this difference in the rates of decline for respiratory infection can be explained by diagnostic cross-misclassification between respiratory infection and SIDS. SIDS appears to be a disease of the respiratory system caused by infection that affects that system's control centers. (26 references) (Author)

20010317-20

Electrocardiography first for reducing cot death. Bonati M, Rocchi F, Pirola M (2001), Lancet vol 357, no 9259, 17 March 2001, p 889

Brief letter commenting critically on the recent suggestion by the Italian Superior Council of Health that routine electrocardiographic screening should be offered free of charge to all neonates to prevent sudden infant death syndrome. This is based on evidence that sudden infant death syndrome is linked to an extended QT interval in neonates. (5 references) (RGW)

20010305-32

How reliable are cot death statistics?. (2000), Foundation for the Study of Infant Deaths (FSID) News Autumn 2000, p 3

The Foundation for the Study of Infant Deaths (FSID) questions the accuracy of recent cot death figures in the United Kingdom and offers a new definition of the term 'cot death' to provide greater accuracy and consistency in its investigation and certification. (KL)

20010302-11

An association between sudden infant death syndrome (SIDS) and helicobacter pylori infection. Kerr J R, Al-Khattaf A, Barson A J, and others (2000), Archives of Disease in Childhood vol 83, no 5, November 2000, pp 429-434

Background: Helicobacter pylori has recently been detected in the stomach and trachea of cases of sudden infant death syndrome (SIDS) and proposed as a cause of SIDS. Aims: To establish the incidence of H pylori in the stomach, trachea, and lung of cases of SIDS and controls. Methods: Stomach, trachea, and lung tissues from 32 cases of SIDS and eight control cases were examined retrospectively. Diagnosis of SIDS was based on established criteria. Controls were defined by death within 1 year of age and an identifiable cause of death. Tissues were examined histologically for the presence of bacteria. Extracted DNA from these tissues was tested for Hpylori ureC and cagA sequences by nested polymerase chain reaction and amplicons detected by enzyme linked immunosorbent assay (ELISA). The cut off for each ELISA for each of the tissue types was taken as the mean optical density plus two times the standard deviation of a range of negative controls. Results-Ages of SIDS cases ranged from 2 to 28 weeks. Ages of controls ranged from 3 to 44 weeks. For the ureC gene, 25 SIDS cases were positive in one or more tissues compared with one of the controls. For the cagA gene, 25 SIDS cases were positive in one or more tissues compared with one of the controls. Conclusions: There is a highly significant association between H pylori ureC and cagA genes in the stomach, trachea, and lung of cases of SIDS when compared with controls. (60 references) (Author)

20001212-02

Investigation of sudden unexpected deaths in infancy. Moore A, Debelle G, Symonds L, and others (2000), Archives of Disease in Childhood vol 83, no 3, September 2000, pp 276-277

The authors discuss the need to ascertain incidence of inherited metabolic diseases, infection, and non-accidental injury in cases of sudden unexpected deaths in infancy, and include the guidelines which they have developed in the West Midlands for managing such cases. (KL)

20001117-09

Heart weight in infants - a comparison between sudden infant death syndrome and other causes of death. Rasten-Almqvist P, Eksborg S, Rajs J (2000), Acta Paediatrica vol 89, no 9, September 2000, pp 1062-1067

Heart and body weights were compared with regard to heart pathology and cause of death in well-defined groups of infants under 1 y of age. In the period 1980 to 1998, out of 468 infants autopsied at the Department of Forensic Medicine in Stockholm, Sweden, 331 died of sudden infant death syndrome (SIDS), while 137 died of other causes. Physical violence was the known cause of death in 30 infants and cardiovascular malformations in another 19. Inflammatory alterations of the myocardium. were found in 74 cases (16%): in 17 cases (3.6%) myocarditis was interpreted as the main cause of death; in 45 (10%) it was interpreted as contributing to SIDS and in 12 cases (2.5%) it was observed but judged not to be a contributory cause of death in non-SIDS victims. Two of these infants died as a result of physical violence. Body weight was the best predictor for heart weight as analysed by multiple regression,

including age, sex, body weight, length, BMI and birth weight. An equation for estimating heart weight from body weight gave an accuracy within the range 0.75-1.25 in 89.2% and 85.0% of the SIDS and non-SIDS groups, respectively. Conclusion: Body weight is the best predictor for estimating heart weight. No evidence supported the notion that heart weight, body weight or birth weight of SIDS victims differs from non-SIDS, although heart weight in infants with cardiovascular malformations deviated from observations in the other groups. (24 references) (Author)

20001112-47

Gastroesophageal reflux and infant apnea. Amin RS (2000), Journal of Pediatrics vol 137, no 3, September 2000, pp 298-300

Examines possible links between gastroeosophageal reflux and infant apnoea, and the apparent lack of any connection to sudden infant death syndrome. (17 references) (JAL)

20001108-24

Cot death: are we further forward, or just re-mixing the message?. Crawford D (2000), Paediatric Nursing vol 12, no 8, October 2000, pp 20-22

Doreen Crawford summarises the evidence around SIDS and discusses the implications for professionals of updated guidance. (35 references) (Author)

20001011-32

Abnormal brain pathways discovered in SIDS victims: health care providers influential in promoting back sleeping. (2000), AWHONN Lifelines vol 4, no 4, August/September 2000, pp 15-16

A team of researchers has found that infants who die of sudden infant death syndrome (SIDS) have abnormalities in several parts of the brain stem. These findings build upon the results of an earlier study that found abnormalities in a brain region (the arcuate nucleus) in children who died of SIDS. (Author)

20000912-02

Sudden infant death syndrome: oxidative stress. Reid GM, Tervit H (1999), Medical Hypotheses vol 52, no 6, 1999, pp 577-580

In studies of oxidative stress in sudden infant death syndrome (SIDS) there were two major findings: (1) During normal post-natal development, there was a gradual decline in the number of Cu/Zn superoxide dismutase (SOD) and glutathione peroxidase (GSHPx) immunoreactive neurons in the hippocampus and parahippocampus gyrus in the brain; (2) The total number of immunoreactive neurons was elevated in SIDS victims compared to age matched controls in infants 6 months of age and under (1). SOD and neuronal aging and degeneration in the hippocampus and neocortex were features of SIDS, Alzheimer's disease and Down's syndrome. In the SIDS study of infants from 3~6 months of age, the elevation of SOD in SIDS victims was significant, whereas no significant elevation of GSHPx was detected. An imbalance between SOD and GSHPx was said to be crucial in the prevention of toxicity of free radicals (1). Zinc-deficient cells cannot up-regulate gene expression of the scavenger enzymes SOD and GSHPx in cells exposed to high levels of superoxide and hydrogen peroxide (2). GSHPx coupled to reduced nicotine adenine diphosphate (NADPH) regenerating systems via glutathione reductase is virtually able to guarantee an effective protection of biological structures against oxidative attack (22). When the capacity of the cell to regenerate GSH is exceeded - primarily due to an insufficient supply of NADPH oxidised glutathione (GSSG) is released from the cell and protein synthesis turns off (20). We hypothesize that the increased incidence of aging and neuronal death and increased incidence of SOD and GSHPx reactive neurons in early post-natal development indicates an increased up-regulation of gene expression of scavenger enzymes during high exposure to oxidative stress after birth. GSH-dependent peroxide metabolism is linked to the pentose phosphate shunt via NADPH-dependent glutathione reductase (GR). GSHPx is a selenium containing enzyme which together with catalase (CAT) SOD and vitamin E protects cells in the free radical chain. Zinc upregulates gene expression of these antioxidants. (24 references) (Author)

20000816-20

Sudden infant death syndrome: a preconditioning approach to acute arterial hypoxemia. Reid GM (2000), Medical Hypotheses vol 54, no 6, 2000, pp 987-989

The blood hemoglobin F (HbF) concentration increases in response to chronic arterial hypoxemia and is abnormally elevated in sudden infant death syndrome (SIDS) post-mortem indicating a need for greater oxygen affinity of hemoglobin (Hb) or diminished oxygen usage by tissues or both. Modifying Hb oxygen affinity in rats revealed that increased, rather than decreased, hemoglobin-oxygen affinity permitted survival at greatly reduced environmental oxygen pressures equivalent to high altitude. Decreased Hb-oxygen affinity resulted in bradycardia 5-10 minutes before death. Cardiorespiratory recordings from infants dying suddenly and unexpectedly at home demonstrated cardiovascular failure with hypotension and bradycardia, rather than a cessation of breathing. A fall in blood pressure and acidosis due to hypoxemia in combination with reduced arterial oxygen saturation leads to circulatory failure, heart failure and death. It is speculated that the final mechanism of SIDS mimics failure to survive at high altitudes and very low environmental oxygen pressures when low arterial oxygen pressures combine with decreased Hb-oxygen affinity lead to severe hypoxemia and death. (18 references) (Author)

20000815-24

A molecular link between the sudden infant death syndrome and the long-QT syndrome. Schwartz PJ, Priori SG, Dumaine R, and others (2000), The New England Journal of Medicine vol 343, no 4, 27 July 2000, pp 262-267

Case report illustrating the authors hypothesis of a link between the long-QT syndrome and the risk of sudden infant death. (32 references).

20000810-31

Birth weight- and gestational age-specific sudden infant death syndrome mortality: United State, 1991 versus 1995. Malloy MH, Freeman DH (2000), Pediatrics vol 105, no 6, June 2000, pp 1227-1231

Objective. To estimate the changes in birth weight- and gestational age-specific sudden infant death syndrome (SIDS) mortality rates since the publication of the sleep-positioning recommendations by the American Academy of Pediatrics Task Force on Infant Positioning and SIDS. Methods. This is a historical cohort study using US vital statistic linked birth and infant death certificate files for the years 1991 and 1995. SIDS deaths were identified as any death attributed to International Classification of Diseases, Ninth Revision code 7980, occurring between the 28th and 365th days of life. Results. There were 4871 deaths attributed to SIDS in 1991 for a postneonatal mortality rate of 1.2/1000 postneonatal survivors compared with 3114 deaths in 1995 for a rate of .8/1000. This represents a 33% drop in the post-neonatal SIDS mortality from 1991 to 1995. Between 1991 and 1995, SIDS rates declined 38%, 38%, 35%, and 32% for birth weight groupings of 500 to 999 g, 1000 to 1499 g, 1500 to 2499 & and >/=2500 g, respectively. There were no SIDS deaths attributed to infants weighing <500 g. The SIDS rates declined 27%, 21%, 40%, and 23% for gestational age groups of <29 weeks, 29 to 32 weeks, 33 to 36 weeks, and >/= 37 weeks. The rate of decline did not differ significantly across birth weight- or gestational age-specific categories. There was a significant increase in the black: non-black postneonatal SIDS mortality ratio from 2.00 to 2.28, reflecting a smaller decline in birth weight and gestational age-specific mortality for blacks than observed for the non-black population. Conclusion. Postneonatal SIDS mortality decreased significantly across all broad birth weight and gestational age categories. If the decline in the prevalence of prone positioning that has been reported since 1992 has occurred across all birth weight and gestational age, these data support the hypothesis that supine or side sleep positioning is effective in preterm/low birth weight infants as well as term infants. (27 references) (Author)

20000613-46$

Weight gain and sudden infant death syndrome: changes in weight z scores may identify infants at increased risk. Blair PS, Nadin P, Cole TJ, and others (2000), Archives of Disease in Childhood vol 82, no 6, June 2000, pp 462-469

Aims: To investigate patterns of infant growth that may influence the risk of sudden infant death syndrome (SIDS). Design: Three year population based case control study with parental interviews for each death and four age matched controls. Growth was measured from prospective weight observations using the British 1990 Growth Reference. Setting: Five regions in England (population greater than 17 million, more than 470 000 live births over three years).

Subjects: 247 SIDS cases and 1110 controls. Results: The growth rate from birth to the final weight observation was significantly poorer among the SIDS infants despite controlling for potential confounders (SIDS mean change in weight z score (szw) = -0.38 (SD 1.40) v controls = + 0.22 (SD 1.10), multivariate: $p < 0.0001$). Weight gain was poorer among SIDS infants with a normal birth weight (above the 16th centile: odds ratio (OR) = 1.75, 95% confidence interval (CI) 1.48-2.07, $p < 0.0001$) than for those with lower birth weight (OR = 1.09, 95% CI 0.61-1.95, $p = 0.76$). There was no evidence of increased growth retardation before death. Conclusions: Poor postnatal weight gain was independently associated with an increased risk of SIDS and could be identified at the routine six week assessment. (10 references) (Author)

20000607-31$

Cost-effectiveness and implications of newborn screening for prolongation of QT interval for the prevention of sudden infant death syndrome. Zupancic JAF, Triedman JK, Alexander M, and others (2000), Journal of Pediatrics vol 136, no 4, April 2000, pp 481-489

Objective: To determine the cost-effectiveness of universal and high-risk neonatal electrocardiographic (ECG) screening for QT prolongation as a predictor of sudden infant death syndrome (SIDS) risk in a theoretical group of neonates. Study design: Incremental cost-effectiveness analysis with decision analytic modeling. A hypothetical cohort of healthy, term infants was modeled, comparing options of no screening, high-risk neonate screening, and universal screening. The high-risk strategy is speculative, because no currently accepted methodology is known for identifying infants at high risk for SIDS. Given the uncertain mechanisms of association between prolonged corrected QT interval (QTc) and SIDS, analyses were repeated under different assumptions. Sensitivity analyses were also performed on all input variables for both costs and effectiveness. Results: Under the assumption that neonatal electrocardiographic screening detects long QT syndrome responsive to conventional therapy, the cost-effectiveness of high-risk screening was $3403 per life year gained, whereas universal screening cost $18,465 per additional life year gained. However, if the effectiveness of SIDS therapy falls below 10%, the cost-effectiveness deteriorates to $28,376 per life year saved for the high~risk strategy and $118,900 for universal screening. The analyses were robust to a broad array of sensitivity analyses. Conclusions: The acceptability of the cost-effectiveness of neonatal electrocardiographic screening is heavily dependent on the pathophysiologic mechanism of SIDS and on the efficacy of monitoring and antiarrhythmic treatment. The nature of this association must be elucidated before routine neonatal electrocardiographic screening is warranted. (31 references) (Author)

20000513-35$

Sudden infant death syndrome: unravelling a mystery. Pearn JH (2000), Journal of Paediatrics, Obstetrics and Gynaecology vol 26, no 2, March/April 2000, pp 5-8

Considers the possible causes of sudden infant death syndrome, its diagnosis, children who are particularly at risk, prevention strategies and management, including an illustration of how to make up the cot to help keep the baby's head uncovered. (4 references) (JAL)

20000408-09$

The role of auditory neural input in sudden infant death syndrome: a brief review and hypothesis. Stewart, MW, Smith, RS, Ribichini, F, and others (2000), Prenatal and Neonatal Medicine vol 5, no 1, February 2000, pp 9-16

We present the hypothesis that auditory neural input is important to maintain diencephalically mediated sleep respiratory function during a critical period in infant development, and that attenuation of these stimuli due to sleep practices, sleep environment, or auditory system dysfunction may contribute to the development of sudden infant death syndrome (SIDS). It is hypothesized that severely attenuated auditory neural input leads to respiratory deregulation and/or bradycardia in the at-risk infant, leading to subsequent intermittent and chronic sleep hypoxia. The hypoxia leads to damage of the auditory system, particularly the cochlea, which further attenuates auditory neural input, leading to a potentially fatal vicious cycle. This hypothesis integrates a wide variety of epidemiological, psychophysical, acoustic and pathophysiological research related to SIDS. (80 references). (Author).

20000403-02*

Sudden unexpected deaths in infancy: the CESDI SUDI studies 1993-1996. Fleming P, Blair P, Bacon C, and others, editors (2000), London: The Stationery Office 2000. 160 pages

The studies of Sudden Unexpected Deaths in Infancy (SUDI) conducted as part of the Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) in England and Wales, constitute the most comprehensive investigation of such deaths carried out to date. Parallel studies incorporating a multidisciplinary confidential enquiry, a case-control study and a review of the pathology were conducted for 450 unexpected deaths of infants aged between one and 52 weeks occurring over three years in a defined geographical area. In total 470,000 births were included in the study. The results of these studies add significantly to our understanding of the factors that contribute to both explained and unexplained infant deaths. The report also deals with the needs of bereaved families and makes recommendations about how unexpected infant deaths should be investigated. The information in this report will be of interest and importance to all those who advise families with young infants, and to those who may have to deal with unexpected deaths. (Publisher)

20000314-13$

A clinical comparison of SIDS and explained sudden infant deaths: how healthy and how normal?. Platt MW, Blair PS, Fleming PJ, and others (2000), Archives of Disease in Childhood vol 82, no 2, February 2000, pp 98-106

Objectives: To compare the clinical characteristics associated with sudden infant death syndrome (SIDS) and explained sudden unexpected deaths in infancy (SUDI). Design: Three year population based, case control study with parental interviews for each death and four age matched controls. Setting: Five regions in England (population, > 17 million; live births, > 470 000). Subjects: SIDS: 325 infants; explained SUDI: 72 infants; controls: 1588 infants. Results: In the univariate analysis , all the clinical features and health markers at birth, after discharge from hospital, during life, and shortly before death, significant among the infants with SIDS were in the same direction among the infants who died of explained SUDI. In the multivariate analysis, at least one apparent life threatening event had been experienced by more of the infants who died than in controls (SIDS: 12% v 3% controls; odds ratio (OR) = 2.55; 95% confidence interval (CI), 1.02 to 6.41; explained SUDI: 15% v 4% controls; OR = 16.81; 95% CI, 2.52 to 112.30). Using a retrospective illness scoring system based on 'Baby Check', both index groups showed significant markers of illness in the last 24 hours (SIDS: 22% v 8% controls; OR = 4.17; 95% CI, 1.88 to 9.24; explained SUDI: 49% v 8% controls; OR = 31.20; 95% CI, 6.93 to 140.5). Conclusions: the clinical characteristics of SIDS and explained SUDI are similar. Baby check might help identify seriously ill babies at risk of sudden death, particularly in high risk infants. (31 references) (Author)

20000311-16

Sudden unexpected deaths in infancy: the CESDI SUDI studies. Ferguson P (2000), RCM Midwives Journal vol 3, no 3, March 2000, pp 86-87

Despite the fall in sudden infant death syndrome in the last decade, sudden unexpected deaths in infancy (SUDI) are still the largest single group of deaths in the post-neonatal period. Because of this, CESDI decided to look at why apparently normal healthy babies sometimes fail to survive. Causes investigated include infections, accidental death, non-accidental injuries, congenital abnormalities, intestinal obstructions, and metabolic disorders. (3 references) (Author)

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