

Weitere Studien zu SIDS Teil 3

48-55 **Für die Referenzen 48-55 zu "Der plötzliche Kindstod (SIDS)III ... sind keine Zusammenfassungen verfügbar**

55ff **Zusammenfassungen der Referenzen 85-83 und 86 zu SIDS_{III} [siehe Volltext zu 85 und Link zu 86]**

55: Adelson L, Kinney ER. Sudden and unexpected death in infancy and childhood. *Pediatrics*. 1956 May;17(5):663-99. No abstract available.

56: *Engl J Med*. 1991 Jun 27;324(26):1858-64. Comment in: *N Engl J Med*. 1991 Dec 19;325(25):1806-7. Sudden death in infants sleeping on polystyrene-filled cushions. Kemp JS, Thach BT

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BACKGROUND. Infants are at risk for both the sudden infant death syndrome (SIDS) and accidental suffocation. On postmortem examination, however, it is difficult to distinguish one from the other without information from the scene of death. Healthy infants are assumed to be able to turn their heads and, if not otherwise restrained, to obtain fresh air. We assessed this assumption in an investigation of infant deaths that occurred during sleep on cushions filled with polystyrene beads. **METHODS.** We obtained data on 25 deaths from the U.S. Consumer Product Safety Commission. We also used mechanical and animal models to study physiologic aspects of ventilation relevant to these results, by simulating the effects on an infant of breathing into a cushion. We measured the effects of softness, malleability (molding of the cushion about an infant's head), airflow resistance, and rebreathing of expired gases. **RESULTS.** All 25 study infants were prone when found dead, and at least 88 percent were face down with nose and mouth obstructed by the cushion. SIDS was the diagnosis in 19 of the 23 infants who underwent autopsy. Our findings show, however, that the cushion would have limited movement of the infant's head to obtain fresh air, and the amount of rebreathing we estimated to have occurred in the infants was lethal in a rabbit model. **CONCLUSIONS.** Accidental suffocation by rebreathing was the most likely cause of death in most of the 25 infants studied. Consequently, there is a need to reassess the cause of death in the 28 to 52 percent of the victims of SIDS who are found with their faces straight down. Safety regulations setting standards for softness, malleability, and the potential for rebreathing are needed for infant bedding.

57: *Am J Dis Child*. 1993 Jun;147(6):642-6. Erratum in: *Am J Dis Child* 1993 Aug;147(8):810. A sleep position-dependent mechanism for infant death on sheepskins. Kemp JS, Thach BT.

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OBJECTIVE--To determine whether rebreathing of expired air is a plausible lethal mechanism of sudden death in infants sleeping face down on sheepskins. **DESIGN--**Case reports of infants who died on sheepskins combined with a controlled study of effects of their respiratory microenvironment at death. **SETTING--**Research laboratory. **MATERIALS--**Rabbits used experimentally to assist in simulation of an infant's respiratory microenvironment. Rabbits breathed into sheepskin through the airway of an infant mannequin. **INTERVENTION--**None. **MEASUREMENTS/MAIN RESULTS--**Rebreathing of expired air was documented by carbon dioxide analysis of airway gas. Arterial blood gas analysis showed hypoxemia, hypercarbia, and acidosis in all experimental rabbits but not in controls. Rebreathing expired air was lethal for three of four experimental rabbits. **CONCLUSIONS--**Infants sleeping prone on a sheepskin, with their faces straight down, experience potentially lethal rebreathing of expired air. Avoidance of the prone position would markedly reduce the risk of rebreathing expired air. The pronounced decrease in sudden infant death syndrome in southern New Zealand that followed a campaign to eliminate prone sleeping may have been attributable to reduced fatal rebreathing, as deaths of infants sleeping face down on sheepskins were common before the campaign.

58: *J Pediatr*. 1993 Jun;122(6):874-80. Unintentional suffocation by rebreathing: a death scene and physiologic investigation of a possible cause of Sudden infant Death. Kemp JS, Kowalski RM, Burch PM, Graham MA, Thach BT.

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As part of a 1-year study of sudden unexpected infant deaths in St. Louis, we performed both a specialized death scene investigation and, in selected cases, a physiologic reconstruction of the death scene with a rabbit model. Those cases in which the infant was found face down with nose and mouth covered by bedding were chosen for the physiologic testing; our goal was to assess the potential for lethal rebreathing of expired air. The physiologic studies reconstructed the infants' premortem ventilatory environment by using the head from an infant mannequin positioned on the actual bedding on which an infant had died and a rabbit breathing through the mannequin's nares. The specialized scene investigation was carried out in 31 of 32 deaths from Sudden infant Death syndrome, diagnosed by the usual methods. Of 31 infants, eight died with their faces downward and covered by bedding, and the bedding was obtained for further study in seven of eight cases. The bedding had low resistance to airflow (6.25 to 22.6 cm H₂O/L per second), and caused considerable rebreathing that was lethal to the rabbits in five of seven cases. We conclude that items of bedding in common use are capable of causing lethal rebreathing by prone- sleeping infants whose nose and mouth become

covered; suffocation by rebreathing was the probable mechanism of death in a substantial number of these deaths that had been attributed to Sudden infant Death syndrome.

59: *Pediatrics*. 2000 Apr;105(4 Pt 1):774-9. Potential to prevent carbon dioxide rebreathing of commercial products marketed to reduce sudden infant death syndrome risk. Carolan PL, Wheeler WB, Ross JD, Kemp RJ.

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OBJECTIVE: Rebreathing of exhaled air is one proposed mechanism for the increased risk for sudden infant death syndrome among prone sleeping infants. We evaluated how carbon dioxide (CO₂) dispersal was affected by a conventional crib mattress and 5 products recently marketed to prevent prone rebreathing. **SETTING:** Infant pulmonary laboratory. **EQUIPMENT:** An infant mannequin with its nares connected via tubing to a 100-mL reservoir filled with 5% CO₂. The sleep surfaces studied included: firm mattress covered by a sheet, Bumpa Bed, Breathe Easy, Kid Safe/Baby Air, Halo Sleep System, and Sleep Guardian. The mannequin was positioned prone face-down or near-face-down. The sleep surfaces were studied with the covering sheet taut, covering sheet wrinkled, and with the mannequin arm positioned up, near the face. **MEASUREMENTS:** We measured the fall in percentage end-tidal CO₂ as the reservoir was ventilated with the piston pump. The half-time for CO₂ dispersal (t_{1/2}) is an index of the ability to cause or prevent rebreathing. **RESULTS:** Compared with the face-to-side control, 5 of 6 surfaces allowed a significant increase in t_{1/2} in all 3 prone scenarios. The firm mattress and 4 of the 5 surfaces designed to prevent rebreathing consistently allowed t_{1/2} above thresholds for the onset of CO₂ retention and lethal rebreathing in an animal model (*J Appl Physiol*. 1995; 78: 740). **CONCLUSIONS:** With very few exceptions, infants should be placed supine for sleep. For infants placed prone or rolling to the prone position, significant rebreathing of exhaled air would be likely on all surfaces studied, except one.

60: *Pediatr Res*. 1994 Jul;36(1 Pt 1):7-11. Physical properties of bedding that may increase risk of sudden infant death syndrome in prone-sleeping infants. Kemp JS, Nelson VE, Thach BT.

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Soft bedding has been shown in epidemiologic studies to increase the risk for sudden, unexpected death in prone-sleeping infants. We compared the physical properties of conventional bedding to bedding from two sources: 1) bedding that covered the airways of victims of sudden infant death syndrome (SIDS) lying prone and face down at the time of death; and, 2) bedding associated with increased risk for SIDS in case-control studies (i.e. bedding filled with ti tree bark). Using simple mechanical models and the head from an infant mannequin, we measured the resistance to airflow, malleability, and capacity to limit CO₂ dispersal of the bedding. We also describe a technique for quantifying bedding softness. The resistance and malleability were similar for the conventional bedding, the ti tree bedding, and the bedding from SIDS deaths (analysis of variance, $p = 0.85$ and 0.16). The ti tree bedding and the other bedding from SIDS cases differed from conventional bedding in two physical properties. Both groups were softer ($p < \text{or} = 0.005$) and limited CO₂ dispersal to a greater degree ($p < \text{or} = 0.009$). The finding that increased capacity to limit CO₂ dispersal is a consistent property of the bedding covering the airways of these SIDS victims and of bedding shown to be an epidemiologic risk factor for SIDS supports rebreathing of expired air as a mechanism underlying the association of certain kinds of bedding with SIDS.

61: *J Appl Physiol*. 1995 Feb;78(2):740-5. Quantifying the potential of infant bedding to limit CO₂ dispersal and factors affecting rebreathing in bedding. Kemp JS, Thach BT.

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Rebreathing may impair ventilation and lead to sudden death among sleeping infants. To estimate the potential for rebreathing imposed by an infant's sleep microenvironment, we developed a mechanical model to assess the rate of CO₂ dispersal away from an infant's face. We compared the mechanical model results with changes in arterial blood gases of rabbits. The rabbits breathed into the same microenvironments used for the model studies. In the rabbits, rebreathing (documented by capnometry) caused hypercarbia and in some cases death. The mechanical model consisted of the mannequin head positioned as in the rabbit studies and connected to a 100-ml syringe filled with CO₂. CO₂ was washed out of the system using 30-ml "breaths" (rate = 15/min). The half times (t_{1/2}) for CO₂ dispersal served to quantify the rebreathing potential of 16 items of bedding. The t_{1/2} values correlated with increments in the rabbits' arterial PCO₂ ($r = 0.789$). The threshold for the increase in the rabbits' arterial PCO₂ corresponded to t_{1/2} values of $> \text{or} = 18.7$ s; the 90% point for lethality in the rabbit model was 28.1 s. The mechanical model was also used to show the independent effects of softness and porosity of bedding on its rebreathing potential. By describing the potential for rebreathing within bedding, the mechanical model should be useful in future quantitative studies of infants' respiratory adaptation to sleep microenvironments.

62: Sleep. 1996 Dec;19(10 Suppl):S263-6. Rebreathing of exhaled gases: importance as a mechanism for the causal association between prone sleep and sudden infant death syndrome. Kemp JS.

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Twenty to 52% of sudden infant death syndrome (SIDS) victims are found dead with their noses and mouths turned into underlying bedding. Several items of bedding have been shown to increase the risk for SIDS in case-control studies or to be associated with many SIDS deaths in case series. These items of bedding are after limit CO₂ dispersal more, and cause more rebreathing of exhaled gases than bedding infrequently associated with SIDS. Rebreathing of exhaled gases may explain some prone deaths, and avoiding rebreathing of these gases is one possible mechanism for the reduction in SIDS when infants avoid prone sleep. Results supporting these statements are reviewed and discussed.

63: J Pediatr. 1998 Feb;132(2):234-9. Comment in: J Pediatr. 1998 Feb;132(2):197-8. Softness and potential to cause rebreathing: Differences in bedding used by infants at high and low risk for sudden infant death syndrome. Kemp JS, Livne M, White DK, Arfken CL.

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OBJECTIVE: This study was carried out to determine whether bedding used by infants, who are at either high or low risk for sudden infant death syndrome (SIDS), differs in physical properties favoring rebreathing of exhaled gases. **STUDY DESIGN:** We compared softness and limitation of carbon dioxide dispersal by bedding, using a mechanical model. A questionnaire was used to describe sociodemographic risk factors and sleep practices; bedding was studied in homes with a model positioned where each infant was found sleeping that morning. **RESULTS:** The groups differed with respect to five sociodemographic risk factors (p values all < or = 0.0001). In addition, infants at higher risk were more likely to have been placed to sleep prone (46%, p = 0.02) by parents who were less likely to be aware of the risk associated with the prone position (62% aware, p = 0.005). Infants at higher risk had softer bedding (p < 0.0001, 54.1 +/- 17.2 cm² vs 33.7 +/- 7.7 cm² in contact with model), which caused more limitation of carbon dioxide dispersal (p = 0.008; CO₂ retained, 0.60% +/- 0.15% vs 0.34% +/- 0.05%). **CONCLUSIONS:** A series of infants who are at high risk for SIDS because of sociodemographic factors more often sleep on bedding that has physical properties favoring rebreathing, and their parents are less often aware of the risk associated with prone sleeping.

64: J Appl Physiol. 2001 Dec;91(6):2537-45. Inspired CO₂ and O₂ in sleeping infants rebreathing from bedding: relevance for sudden infant death syndrome. Patel AL, Harris K, Thach BT.

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Some infants sleep facedown for long periods with no ill effects, whereas others become hypoxemic. Rebreathing of expired air has been determined by CO₂ measurement; however, O₂ levels under such conditions have not been determined. To evaluate this and other factors influencing inspired gas concentrations, we studied 21 healthy infants during natural sleep while facedown on soft bedding. We measured gas exchange with the environment and bedding, ventilatory response to rebreathing, and concentrations of inspired CO₂ and O₂. Two important factors influencing inspired gas concentrations were 1) a variable seal between bedding and infants' faces and 2) gas gradients in the bedding beneath the infants, with O₂-poor and CO₂-rich air nearest to the face, fresher air distal to the face, and larger tidal volumes being associated with fresher inspired air. Minute ventilation increased significantly while rebreathing because of an increase in tidal volume, not frequency. The measured drop in inspired O₂ was significantly greater than the accompanying rise in inspired CO₂. This appears to be due to effects of the respiratory exchange ratio and differential tissue solubilities of CO₂ and O₂ during unsteady conditions.

65: Pediatrics. 2003 Apr;111(4 Pt 1):e328-32. Occurrence and mechanisms of sudden oxygen desaturation in infants who sleep face down. Patel AL, Paluszynska D, Harris KA, Thach BT.

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OBJECTIVE: Infants who sleep prone and face down on soft bedding are particularly vulnerable for sudden infant death syndrome. It has been suggested that 1 mechanism of death in this situation involves rebreathing of expired air. Many infants tolerate rebreathing while lying prone face down for long periods with stable saturations. Others occasionally have rapid desaturations and may require intervention to terminate rebreathing. The present study had 3 objectives: 1) to determine the frequency of rapid desaturations in a large group of healthy infants, 2) to elucidate the mechanism of these desaturations, and 3) to determine the timing of these events during periods of rebreathing. **METHODS:** We studied respiratory tracings and videotapes of 56 healthy 1- to 6-month-old infants who were sleeping face down and rebreathing on soft bedding in our laboratory. We compared the frequency of desaturations during rebreathing and nonrebreathing periods. We measured respiratory frequency and apnea occurrence before desaturation and

nonbreathing control episodes. We also measured minute ventilation during steady state before desaturation and just before desaturation. RESULTS: There were 25 desaturation episodes in infants while rebreathing, occurring in 11 (19.6%) of the 56 infants. Episodes were significantly more frequent during rebreathing than during nonbreathing periods. Three desaturation episodes reached <85%; 2 required intervention to terminate rebreathing. The respiratory frequency was not different between nonbreathing control and desaturation episodes. Brief apneas were significantly more frequent preceding desaturation than control episodes (44% vs 4%). Just before episodes, there was a transient decrease in minute volume despite increasing inspired carbon dioxide in 3 episodes. There was evidence of partial or complete pharyngeal airway obstruction in 3 episodes. Thirty-six percent of all episodes were immediately preceded by behavioral arousal. CONCLUSIONS: Rebreathing in prone sleeping infants is associated with an increased frequency of episodic desaturations. Desaturation may result from respiratory pattern changes such as brief apneas often associated with evidence of behavioral arousal or failure to increase ventilation in the face of rising inspired carbon dioxide, also associated with behavioral arousal.

66: Pediatrics. 2004 Dec;114(6):1634-9. Influence of sleep position experience on ability of prone-sleeping infants to escape from asphyxiating microenvironments by changing head position. Paluszynska DA, Harris KA, Thach BT.

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OBJECTIVE: Several studies have found that back- or side-sleeping infants who are inexperienced in prone sleeping are at much higher risk for sudden infant death syndrome (SIDS) when they turn to prone or are placed prone for sleep compared with infants who normally sleep prone. Moreover, such inexperienced infants are more likely to be found in the face-down position at death after being placed prone compared with SIDS infants who are experienced in prone sleeping. We hypothesized that lack of experience in prone sleeping is associated with increased difficulty in changing head position to avoid an asphyxiating sleep environment. METHODS: We studied 38 healthy infants while they slept prone. Half of these were experienced and half were inexperienced in prone sleeping. To create a mildly asphyxiating microenvironment, we placed infants to sleep prone with their faces covered by soft bedding. We recorded inspired CO₂ (CO₂I), electrocardiogram, and respiration, and we videotaped head movements. Also, we assessed gross motor development (Denver Development Scale). RESULTS: When sleeping prone, with their faces covered by bedding, all infants experienced mild asphyxia as a result of rebreathing. All aroused and attempted escape from this environment. Infants used 3 stereotyped head-repositioning strategies. The least effective was nuzzling into the bedding with occasional brief head lifts. More effective were head lifts combined with a head turn. Some infants, however, could turn only to 1 side, right or left. Infants who were inexperienced in prone sleeping had less effective protective behaviors than experienced infants. Infant age did not correlate with efficacy of protective behaviors. Infants who were experienced in prone sleep had advanced gross motor development compared with inexperienced infants. CONCLUSION: Infants who are inexperienced in prone sleeping have decreased ability to escape from asphyxiating sleep environments when placed prone. These observations potentially explain the increased risk associated with prone sleep in infants who are inexperienced. The increased occurrence of the face-down position in such infants is also potentially explained. These findings suggest that airway protective behaviors may be acquired through the mechanism of operant conditioning (learning).

67: Australas Phys Eng Sci Med. 1991 Jun;14(2):112-8. Distribution of expired air in carry cots--a possible explanation for some sudden infant deaths. Ryan EL.

A possible explanation for the cause of some cot deaths is examined by placing an appropriate cot death model on a cold wet sheet so that its "breath" is directed downwards across the sheet, thereby being cooled and becoming heavy enough to stay trapped in the hollow of the mattress. The breath is then available for rebreathing by the model. The level of carbon dioxide (CO₂) "inhaled" is showed to be lower in nostril breathers, singletons and in bassinets and higher with mouth breathing models, in "twins", and in carry cots, especially if the model's head is accidentally covered. Under these conditions a living infant would become progressively acidotic due to inability to adequately excrete its metabolic CO₂. This must inevitably be accompanied by increasing hypoxia. If left undisturbed in this predicament, it would rebreathe its own breath for the period of time between feeds and develop increasing degrees of asphyxia, depending on the circumstances outlined.

68: Acta Paediatr. 1996 Mar;85(3):281-4. A potential danger of bedclothes covering the face. Campbell AJ, Bolton DP, Williams SM, Taylor BJ.

Department of Paediatrics and Child Health, Otago Medical School, Otago University, Dunedin, New Zealand.

Investigations of infants dying unexpectedly have reported up to 28% being found completely under bedding. No detailed physiological studies looking at the possibilities of asphyxia in this situation are available. The aim was to determine the potential for asphyxia under different types and thicknesses of bedding. A mechanical model of a 3-month-old infant's respiratory system was used. Bedding was positioned over the head in a supine position, and

inspired carbon dioxide recorded. With a fixed respiratory rate and tidal volume, carbon dioxide accumulation increased with increasing layers of blankets. Up to 8.3% inspired carbon dioxide was recorded with more than four layers of blankets. A cotton sheet between the face and blankets reduced the accumulation by half. An infant found dead under bedding may have been exposed to an asphyxial stress. Suffocation from rebreathing trapped, expired gases can be a cause of death in this situation.

69: *J Pediatr.* 1997 Feb;130(2):245-9. Comparison of two methods of determining asphyxial potential of infant bedding. Campbell AJ, Taylor BJ, Bolton DP.

Department of Physiology, Otago Medical School, Dunedin, New Zealand.

OBJECTIVE: To establish, with the use of live sedated piglets on a range of bedding surfaces, the possibility of asphyxia when an infant is breathing face down into infant bedding surfaces, and to compare the results with those obtained with a mechanical model. **METHODS:** Piglets underwent tracheotomy and were attached to a silicon rubber model head of a 3-month-old infant. This was placed face down on bedding surfaces, and respiratory and blood gas data were collected for a 60-minute period. **RESULTS:** All bedding surfaces but one showed some rebreathing. This was to lower levels than with the mechanical model, but the ranking of the surfaces by level of rebreathing was similar. Two piglets died within the 1-hour experimental time. **CONCLUSION:** It is confirmed that the data from a mechanical model of rebreathing on different bedding surfaces are matched by those derived from a piglet model that responds normally to asphyxia.

70: *Arch Dis Child.* 1998 Apr;78(4):323-8. Mechanical model testing of rebreathing potential in infant bedding materials. Carleton JN, Donoghue AM, Porter WK.

Directorate for Laboratory Sciences, US Consumer Product Safety Commission, Washington DC, USA.

Rebreathing of expired air may be a lethal hazard for prone sleeping infants. This paper describes a mechanical model to simulate infant breathing, and examines the effects of bedding on exhaled air retention. Under simulated rebreathing conditions, the model allows the monitoring of raised carbon dioxide (CO₂) inside an artificial lung-trachea system. Resulting levels of CO₂ (although probably exaggerated in the mechanical model compared with an infant, due to the model's fixed breathing rate and volume) suggest that common bedding materials vary widely in inherent rebreathing potential. In face down tests, maximum airway CO₂ ranged from less than 5% on sheets and waterproof mattresses to over 25% on sheepskins, bean bag cushions, and some pillows and comforters. Concentrations of CO₂ decreased with increasing head angle of the doll, away from the face down position. Recreations of 29 infant death scenes also showed large CO₂ increases on some bedding materials, suggesting these infants could have died while rebreathing.

71: *Tohoku J Exp Med.* 1998 May;185(1):55-65. Inhaled air trapping effect of Japanese bedding as a risk of sudden unexpected death in infancy. Funayama M, Mimasaka S, Iwashiro K, Nozawa R.

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We assessed some Japanese bedding on the assumption of the effects of air trapping using an infant mannequin. The change of CO₂ concentration in the airway of a mannequin head placed on bedding was continuously monitored using a CO₂ analyzer during simulated breathing. To compare the level of CO₂ dispersal among different items of bedding, CO₂ half time (t_{1/2}) values were used. The t_{1/2} values were calculated by measuring the time required for the expired percent CO₂ to reach 1/2 the initial percent end-tidal PCO₂. We also measured softness and resistance to airflow (R) of the same items. As for the bedding, 4 types of futon and several types of bottom sheets/towels were combined. The t_{1/2} value in supine position was 9.8 seconds. When the model was placed prone on futon, the t_{1/2} values increased to 14.1 seconds (hard mattress type)--17.2 seconds (soft cotton-like futon). With respect to present Japanese baby futon (hard mattress type), there may be a relatively low potential for rebreathing to occur, compared with soft futon. In every case, the t_{1/2} value was prolonged by the use of a towel spread on the futon. CO₂ dispersal may depend not only on the softness of the futon, but also on the combination of bottom sheet/towel and mattress. There was no relationship between R values and t_{1/2} values. The potential of rebreathing increased in face down position among all bedding, and supine position was the best CO₂ dispersal position.

72: *Acta Otolaryngol Suppl.* 2000;543:183-5. Computational simulation of accumulation of expired air in the infant cot. Djupesland PG, Borresen BA.

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Accumulation and re-breathing of CO₂ in expired air has been suggested as one possible explanation for the strong association between prone sleeping position and Sudden infant Death syndrome (SIDS). This preliminary study applying a modern computational fluid dynamics (CFD) program to simulate the aerodynamics in an infant cot supports the idea that accumulation of expired air may occur in the prone position. The literature dealing with the potential association

between re-breathing of accumulated CO₂ and SIDS is briefly reviewed.

73: J Paediatr Child Health. 2002 Apr;38(2):192-5. Rebreathing potential of infant mattresses and bedcovers. Colditz PB, Joy GJ, Dunster KR.

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OBJECTIVE: To establish the CO₂ dispersion and retention properties of some mattresses and bed coverings commercially available in Australia. **METHODS:** Five mattresses were studied in (i) an in vivo model in which an infant's head was covered by a headbox, rebreathing was allowed to occur, and the final steady state CO₂ concentration was measured; and (ii) an in vitro model in which 5% CO₂ in a headbox was allowed to disperse, and the time taken for the concentration to reach 1% was measured. Five types of bedcover were studied in (i) an in vivo model in which an infant's head was covered by a bedcover and the final steady state CO₂ concentration was measured; and (ii) an in vitro model in which 5% CO₂ under a bedcover was allowed to disperse, and the time taken for the concentration to reach 1% was measured. **RESULTS:** The steady state CO₂ concentrations ranged from 0.6% to 3.0% for the mattresses ($P < 0.05$). The time for CO₂ to disperse ranged from 5.5 min to 30.4 min ($P < 0.05$). Steady state CO₂ concentrations ranged from 2.5% to 3.6% for the bedcoverings ($P > 0.05$). The time for CO₂ to disperse ranged from 5.4 min to 7.7 min ($P > 0.05$). **CONCLUSIONS:** Some commercial cot mattresses and bedcoverings allow high concentrations of CO₂ to accumulate in rebreathing environments. Some mattress types studied were more diffusive to CO₂, whereas there was no difference between the bedcovers studied. This may have implications for vulnerable infants at risk of sudden infant death syndrome.

74: Forensic Sci Int. 2006 Apr 16; [Epub ahead of print] The relationship between bedding and face-down death in infancy: Mathematical analysis of a respiratory simulation system using an infant mannequin to assess gas diffusibility in bedding. Sakai J, Funayama M, Kanetake J.

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Rebreathing is a model for the relationship between a prone sleeping position and sudden infant death syndrome. This study used a mechanical simulation model to establish the relationship between types of bedding and rebreathing potential for an infant placed prone (face down) at different postnatal ages. The infant mannequin was connected to a respirator set to deliver physiologically appropriate combinations of tidal volume (V(T)) and respiratory rates (RR) across a range of postnatal ages (0-18 months). Before measurements were made, CO₂ flow was regulated to 5±0.1% of end-tidal PCO₂ (EtCO₂). After the model was placed in a prone position, any increase in the fractional concentration of inspired CO₂ (FiCO₂) was measured. FiCO₂ increased immediately and rapidly, and reached a maximum value within a few minutes. The maximum FiCO₂ ranged from under 2% to over 10%, depending on the bedding. FiCO₂ was also affected by V(T) and RR. This model is not applicable to actual infants because of the large tissue stores of CO₂ in infants; however, it is useful for evaluation of gas diffusibility of bedding and will simplify the investigation of sleeping environments when a baby is found dead with its face covered by soft bedding. In general, the higher the FiCO₂, the greater the rebreathing potential. Theoretically, considering the paucity of body stores of O₂, changes in FiO₂ would be affected not by changes in FiCO₂, but by CO₂ production and gas movement around the infant's face. The rapid decrease of FiO₂ is approximated at the inverse of the FiCO₂ timecourse, suggesting the significance of not only CO₂ accumulation but also O₂ deprivation in the potential space around the baby's face.

75: Arch Dis Child. 1993 Aug;69(2):187-90. Comment in: Arch Dis Child. 1993 Dec;69(6):711. Arch Dis Child. 1994 Apr;70(4):354-5. Rebreathing expired gases from bedding: a cause of cot death? Bolton DP, Taylor BJ, Campbell AJ, Galland BC, Cresswell C.

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The reported association of cot death and sleeping prone could be due to rebreathing of expired gases. A mechanical model simulating the respiratory system of an infant, exhaling warm humidified air with an end tidal carbon dioxide of 5%, has been used to investigate this. Some commonly used bedding materials caused an accumulation of carbon dioxide of 7% to over 10% with the model lying face down. This phenomenon persisted even with the head inclined at 45 degrees, but only on very soft materials, and could be a cause of cot death in a baby unresponsive to asphyxial blood gas changes. A coir fibre mattress allowed complete dispersal of exhalate as did a rubber sheet between any mattress and the covering sheet.

76: J Paediatr Child Health. 1994 Feb;30(1):45-9. Comment in: J Paediatr Child Health. 1994 Aug;30(4):371-2. J Paediatr Child Health. 1995 Jun;31(3):258-9. Carbon dioxide concentrations in the environment of sleeping infants. Malcolm G, Cohen G, Henderson-Smart D.

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South Wales, Australia.

In 22 infants continuous measurements were made of the concentration of carbon dioxide (CO₂) in inspired air during sleep. Evidence was found of CO₂ enrichment of inspired air in certain environmental conditions. The levels achieved were not sufficiently high to acutely endanger an infant. Carbon dioxide concentrations as high as 2-3% were observed in the prone position when the infant's head was under a blanket and when the lower face was obscured by bedding. Sleeping prone on a sheepskin also resulted in an increased concentration of CO₂ but to a lesser extent than being under a blanket. In awake infants the presence of a pacifier also promoted an excess of CO₂ in the inspired air, both in the prone and supine positions. The physiological and clinical implications of these findings, in relation to the Sudden Infant Death Syndrome (SIDS), are unknown and warrant investigation.

77: *Acta Paediatr.* 1995 Sep;84(9):988-95. CO₂ rebreathing: a possible contributory factor to some cases of sudden infant death? Skadberg BT, Oterhals A, Finborud K, Markestad T.

Department of Paediatrics, University Hospital of Bergen, Norway.

Physical and geometrical conditions influencing carbon dioxide (CO₂) accumulation near the face of a sleeping infant positioned deep in a cot or pram (open cot shaft) or underneath bedding (closed cot shaft) were investigated. By means of mathematical and data-based simulation, and an experimental rebreathing model, both hypothetical (dry, exhaled air +20 degrees C) and more physiological conditions (heated, humidified exhaled air, room temperature +20 degrees C; with and without pooling of cold air within the shaft) were tested. With exhaled air at +20 degrees C, the CO₂ concentration increased to about 10% within 5 min. The increase was faster the smaller the volume, and the smaller the opening of the cot shaft. When expiratory air was heated, the CO₂ concentration increased with the same speed as when the shaft was closed, but to only 0.1-0.3% when the shaft was open. Pooling of cold air in the shaft increased CO₂ accumulation 70-200 times the concentration in air (to <5.5%) when the shaft was open. Turbulence of the air outside the open shaft reduced the increase in CO₂ concentration. The experiments imply that CO₂ may accumulate around an infant's head when placed deep in a cot or pram with the bedding and walls creating a narrow, vertical, shaft-like tunnel to the surrounding air. Although the CO₂ concentration may theoretically attain dangerous levels in such circumstances, a rapid equilibrium between the air within and outside the cot usually occurs due to convection of the expiratory air and turbulence from drafts, the infant's body movements and breathing. Such factors will largely eliminate any significant rebreathing with the exception of the extreme situation when expired air is contained within a closed space.

78: *Pediatr Pulmonol.* 1996 Dec;22(6):335-41. Are bedding and rebreathing suffocation a cause of SIDS? Guntheroth WG, Spiers PS.

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Suffocation by bedclothes became a popular diagnosis in the 1940s but gradually became replaced with the diagnostic label of Sudden Infant Death Syndrome (SIDS). In 1991 a paper purported that, instead of SIDS, pillows filled with polystyrene beads had caused death by rebreathing suffocation; this conclusion was reached on the basis of experiments with anesthetized rabbits breathing through a doll's head that was placed face down on the pillow. Because of the anesthesia, rabbits could not change their face down position. The doll's nares could not collapse, which would have resulted in rapid death due to conventional suffocation. The rabbits required up to 3 hours or more to die of hypercarbia and hypoxia. Studies in normal infants revealed that they turned from the face-down position after only 2 minutes. (The only infant who retained CO₂ soon died of a fatal neurologic disorder, with central hypoventilation). Using the rabbit/doll's head and mechanical models, a wide range of bedding was indicted, including cushions, sheepskins, pillows, comforters, foam mattresses, and even simple blankets and sheets as potentially causing fatal rebreathing. Except for the use of pillows in general, as well as mattresses filled with kapok and bark, there has been no epidemiologic support for these indictments. Although normal infants are unlikely to succumb to rebreathing suffocation, infants with blunted ventilatory responsiveness and delayed arousal due to prior hypoxia were hypothesized to be at increased risk. Support for this concept was found in the pathology of the brain stem in victims of SIDS that was attributed to prior hypoxic injury. In infants who survived prolonged apnea, less than 20% have demonstrated a diminished ventilatory responsiveness to hypercarbia, but, more significantly, none had an absent response. Arousal to hypercarbia, an abnormality which is crucial to the hypothesis of rebreathing suffocation, is regularly present in normal subjects, but the threshold is higher in near-SIDS infants; however, no instances of failure to arouse have been reported in near-SIDS. If the infant is placed on his or her back or side, the issue of bedding could become moot; unfortunately, a sizable percentage of infants are still being placed prone for sleep. Instead of confusing parents with an ever-expanding list of "dangerous bedding," the message "Back to Sleep" should be emphasized.

79: *Arch Dis Child.* 2000 Nov;83(5):423-8. Ventilatory sensitivity to mild asphyxia: prone versus supine sleep position. Galland BC, Bolton DP, Taylor BJ, Sayers RM, Williams SM.

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AIMS: To compare the effects of prone and supine sleep position on the main physiological responses to mild asphyxia: increase in ventilation and arousal. METHODS: Ventilatory and arousal responses to mild asphyxia (hypercapnia/hypoxia) were measured in 53 healthy infants at newborn and 3 months of age, during quiet sleep (QS) and active sleep (AS), and in supine and prone sleep positions. The asphyxial test mimicked face down rebreathing by slowly altering the inspired air: CO₂, maximum 5% and O₂, minimum 13.5%. The change in ventilation with inspired CO₂ was measured over 5-6 minutes of the test. The slope of a linear curve fit relating inspired CO₂ to the logarithm of ventilation was taken as a quantitative measure of ventilatory asphyxial sensitivity (VAS). Sleep state and arousal were determined by behavioural criteria. RESULTS: At 3 months of age, prone positioning in AS lowered VAS (0.184 prone v 0.269 supine, $p = 0.050$). At newborn age, sleep position had no effect on VAS. Infants aged 3 months were twice as likely to arouse to the test than newborns ($p = 0.013$). Placing infants prone as opposed to supine increased the chances of arousal 1.57-fold ($p = 0.035$). CONCLUSION: Our findings show 3 month old babies sleeping prone compared to supine have poorer ventilatory responses to mild asphyxia, particularly in AS, but the increased prevalence of arousal is a protective factor.

80: Pschyrembel Klinisches Wörterbuch. Berlin 2002

81: *Pediatr Res.* 1999 Mar;45(3):350-4. Gasping and other cardiorespiratory patterns during sudden infant deaths. Poets CF, Meny RG, Chobanian MR, Bonofiglio RE.

Department of Pediatrics, Medical School, Hannover, Germany.

To gain information on the cardiorespiratory changes occurring immediately before sudden infant death (SID), recordings of heart rate and chest wall impedance were analyzed in nine infants who had died at a median age of 4.8 mo (range 1-6 mo) while attached to a memory monitor. Postmortem diagnoses were sudden infant death syndrome in seven infants and mild bronchopulmonary dysplasia in two infants. Primary cause of the monitor alarm was bradycardia in all but two infants. Heart rate fell to $< \text{or} = 15 \text{ bpm}$ 7.5 min (range 1.4-25.2 min) after the first alarm; there was no indication of heart block or ventricular tachycardia. Apnea ($> 20 \text{ s}$) began 0.3 to 13.7 min (median 2.7 min) after this alarm in five infants and 7 to 20 s before it in three infants; in the remaining infant, stimulation occurred before any apnea. Gasping was already present at the time of the first monitor alarm in three infants and occurred within 2.7 min after it in a further four infants. One infant only began to gasp 13 min after the first monitor alarm. The duration of gasping ranged from 3 s to 11 min in those five infants in whom it was not interrupted by resuscitation. The latter was given to three infants 4, 21, and 228 s after the monitor alarm but had no effect on the ongoing decrease in heart rate. Since gasping only occurs if PaO₂ is $< 5\text{-}15 \text{ mm Hg}$, it is most likely that the seven infants who gasped at or shortly after the first monitor alarm were already severely hypoxemic at that time. This hypoxemia developed in the absence of prolonged central apnea. The role of other mechanisms potentially resulting in severe hypoxemia, such as upper airway obstruction or rebreathing, remains to be determined.

82: *Pediatr Pulmonol.* 2003 Aug;36(2):113-22. Characterization of successful and failed autoresuscitation in human infants, including those dying of SIDS. Sridhar R, Thach BT, Kelly DH, Henslee JA.

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Our purpose was to identify and further characterize physiologic mechanisms relevant to autoresuscitation from hypoxic apnea in infants dying suddenly and unexpectedly. We studied cardiorespiratory recordings of 24 infants (age range, 0.8-21 months) who died suddenly while being monitored at home. These recordings were analyzed for features indicated by studies in animal models to be characteristic of hypoxic gasping, and of recovery from bradycardia and apnea associated with gasping (e.g., autoresuscitation). Findings in 5 infants diagnosed as having sudden infant death syndrome were compared with 6 non-SIDS infants whose deaths resulted from other conditions. Additionally, we studied 15 healthy infants during sleep, using home monitor and other respiratory recording techniques, in order to obtain comparison data. We found in recordings from 23 of 24 subjects that hypoxic gasps with characteristic features occurred immediately preceding death. A unique pattern of complex, closely spaced gasps ("double" or "triple" gasps) was present in many subjects. Evidence of partially successful autoresuscitation closely following one or more gasps occurred in 11 subjects, while another 4 had evidence of complete autoresuscitation with return of normal heart rate and resolution of apnea on one or more occasions. Significant differences between SIDS infants and those dying from other causes included increased occurrence of complex gasps and decreased occurrence of partial or complete autoresuscitation in the SIDS infants. The non-SIDS cases were different from the SIDS cases in that only one had "double" gasps ($n = 7$), while none had "triple" gasps, as compared with 4 out of 5 SIDS cases with these patterns ($P < 0.05$, chi-square). Also, in contrast with the SIDS cases, more of the cases with specific postmortem diagnoses had evidence of partial (5 out of 6 cases) or complete (1 out of 6 cases) autoresuscitation ($P < 0.05$, chi-square). We conclude that partial or complete autoresuscitation by gasping is not uncommon in moribund infants during the first year of life. Failure of autoresuscitation mechanisms other than failure to initiate gasping may be characteristic of infants dying of SIDS. Some SIDS infants appear to be different from infants dying with other diagnoses with respect to efficacy and characteristics of hypoxic gasping. Copyright 2003 Wiley-Liss, Inc.

83: Paediatr Respir Rev. 2004;5 Suppl A:S383-6. Apparent life-threatening events and sudden infant death on a monitor. Poets CF.

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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 be 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.

84: Sprott J. Cot Death Controversies. S Afr Med J. 2006 Jul;96(7):568; author reply 568-9. No abstract available. **[siehe englischen und deutschen Volltext zu 84]** Comment on: S Afr. Med J. 2005 Nov;95(11):853-7. **[siehe Vollext 43]**

85: Jorch H. Vizepräsidentin der GEPS- Deutschland eV, Vorsitzende der GEPS NRW. Forum-Diskussion: "was ist von diesen Matratzenüberzügen zu halten". **Siehe:** http://www.sids.de/forum/board_entry.php?id=1075#p1268

86: Pediatrics. 2005 Nov;116(5):1245-55. Epub 2005 Oct 10. Comment in: Pediatrics. 2006 Mar;117(3):990-1; author reply 994-6. Pediatrics. 2006 Mar;117(3):991-2; author reply 994-6. Pediatrics. 2006 Mar;117(3):992-3; author reply 994-6. Pediatrics. 2006 Mar;117(3):993-4; author reply 994-6. Pediatrics. 2006 May;117(5):1850; author reply 1850-3. The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk.

American Academy of Pediatrics Task Force on Sudden Infant Death Syndrome.

There has been a major decrease in the incidence of sudden infant death syndrome (SIDS) since the American Academy of Pediatrics (AAP) released its recommendation in 1992 that infants be placed down for sleep in a nonprone position. Although the SIDS rate continues to fall, some of the recent decrease of the last several years may be a result of coding shifts to other causes of unexpected infant deaths. Since the AAP published its last statement on SIDS in 2000, several issues have become relevant, including the significant risk of side sleeping position; the AAP no longer recognizes side sleeping as a reasonable alternative to fully supine sleeping. The AAP also stresses the need to avoid redundant soft bedding and soft objects in the infant's sleeping environment, the hazards of adults sleeping with an infant in the same bed, the SIDS risk reduction associated with having infants sleep in the same room as adults and with using pacifiers at the time of sleep, the importance of educating secondary caregivers and neonatology practitioners on the importance of "back to sleep," and strategies to reduce the incidence of positional plagiocephaly associated with supine positioning. This statement reviews the evidence associated with these and other SIDS-related issues and proposes new recommendations for further reducing SIDS risk.