Abstract

ESPID--European Society for Study and Prevention of Infant Deaths
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Sudden Death Syndrome of Young Mammals; A Unifying Concept.

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When accumulated knowledge regarding the Sudden Death Syndrome (SDS) of the human infant and of the young of other mammalian species is carefully examined, the preponderance of cases of Sudden Infant Death Syndrome (SDS) no longer constitute a mystery. SDS of other young mammals including the calf, foal, piglet, rabbit, and monkey, has been determined to be not a separate etiologic entity but a peracute manifestation of the respiratory-enteric disease complex, or complexes . . . SDS is associated with a greatly increased numbers of E. coli in the proximal ileum and jejunum, as has been shown in diarrhea of various mammalian species, including the human infant. Studies in Germany (Bendig and Haenel) have shown similar increased E. coli in the proximal intestinal tract of 24 of 29 SIDS cases. Invasion of E. coli into these more absorptive portions of the small intestine results in absorption of increased amounts of lipopolysaccharides (LPS, or endotoxin) into the general blood circulation through a temporarily dysfunctional liver (RE system). Endotoxin causes decrease of phosphoenolpyruvate carboxykinase (PEPCK) in the liver, release of large amounts of serotonin from blood platelets, non-coagulability of blood, hyperkalemia, hyponatremia, acidosis, pulmonary edema and hemorrhage by diapedesis... Serotonin initiates in some cases the coronary chemoreflex (Bezold-Jarisch reflex) in which there is inhibition of sympathetic outflow and increased activity of the cardiac (efferent) vagus leading to profound bradycardia, hypotension and cardiac collapse . . . Triggering stressors include various viruses, chilling, overheating, lack of vitamins including A, C, B6, etc. . . Prevention includes feeding of breast milk only, to maximize immunologic defenses and minimize numbers of E. coli in the g.i. tract. The g.i. tract of the bottle-fed infant contains approximately 1,000 times the number of E. coli normal to the breast-fed infant.

Bendig, J. and Haenel, H.: ***Gastrointestinal Microecology of Sudden Unexpected Death of Infants.*** *Nutrition,* Proc. Eight Congress Nutr., Prague, Sept. 1969. (ed) Josef Masek, Prague, & Sir David P. Cuthbertson, Glasgow. Excerptica Medica, pub., Amsterdam. p.212-214-1970.

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Reisinger, R.C.: ***Pathogenesis and prevention of infectious diarrhea (scours) of newborn calves.*** *J Amer Vet Assoc* 147:1377-1386, 1965.

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Woodruff, P.W.H., O'Carroll, D.I., Koizumi, S., and Fine, J.: ***Role of Intestinal Flora in Major Trauma.*** *J. Inf. Dis.* 128 Supl: S290-294, July 1973)

Abstract

ESPID--European Society for Study and Prevention of Infant Deaths
Oxford, England August 27-30, 1993

Bacterial Endotoxins in Pathogenesis of Toxemia of Pregnancy, Neonatal Encephalopathies, and Cot Death.

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Toxemia of Pregnancy.

Blood flow from pregnant uterus (or hydatidiform mole) raises pressure in the inferior vena cava lessening flow through hepatic veins with variable stasis of hepatic circulation. This results in loss of hepatic function to adequately detoxify bacterial endotoxins of intestinal origin. Signs and pathology associated with toxemia of pregnancy in human and other animals are consistent with endotoxemia. Following birth, spontaneous or induced abortion, there is rapid lessening of blood flow from the uterus, drop in pressure in inferior vena cava, normalized blood flow through the liver which is then able to detoxify the build-up of circulating endotoxin. If the preceding events occur in a timely manner patients return to normal; if not, irreversible sequelae or death may ensue.

Neonatal Encephalopathy.

Reisinger, R.C.: *Discussion on Endotoxemia. J. Inf. Dis.* 128 (Supl): S303-305, July 1973.
Gilles, F.H. et al.: *Neonatal endotoxin encephalopathy. AM. Neurol.* 2:49, 1977.

Cot Death.

Endotoxemia is the major cause of Sudden Death Syndrome (SDS) in calves, foals, Rhesus monkeys, etc. The one published study in the human infant has yielded results similar to those found in other animals. (Bendig, J. and Haenel, H.: *Gastrointestinal Microecology of Sudden Unexpected Death of Infants. Nutrition,* Proc. Eight Congress Nutr., Prague, Sept. 1969. (ed) Josef Masek, Prague, and Sir David P. Cuthbertson, Glasgow.: Excerptica Medica, pub., Amsterdam. p.212-214, 1970.)

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Abstract

International Congress on SUDDEN INFANT DEATH SYNDROME

"The Role of Environmental Factors in Infant Morbidity and Mortality"

GRAZ-AUSTRIA May 24 to 27, 1995

SLEEPING POSITION, FORMULA FEEDING, ENDOTOXIN AND SIDS. R.C. Reisinger

Formula fed infants have 1,000 to 10,000 or more E. coli in their g.i. tracts than do breast fed infants. Numerous viruses, avitaminosis A, C, B-6, etc., chilling, overheating, and various other environmental stressors may make the infant 10,000 times more susceptible to E.coli endotoxin (lipopolysaccharide) . . . Raising ambient temperature increases susceptibility to endotoxin . . . Formula feeding increases body heat . . . Being in the prone position increases body heat . . . Prone positioning thus may increase susceptibility of formula fed infants to SIDS . . . Prone positioning would not equally increase susceptibility of the totally breast fed infant to SIDS . . .

Sudden Death Syndrome (SDS) in young of other mammalian species, including the calf and rhesus monkey, is associated with greatly increased numbers of E.coli in the proximal ileum and jejunum . . . J. Bendig and H. Haenel have reported similar increased E.coli in the proximal intestinal tract of 24 of 29 SIDS cases . . . Absorption of increased amounts of endotoxin into the general blood circulation through a temporarily dysfunctional reticuloendothelial system causes decrease of phosphoenolpyruvate carboxykinase (PEPCK) in the liver, release of large amounts of serotonin from blood platelets, non-coagulability of blood, pulmonary edema and hemorrhage by diapedesis. . . Serotonin initiates in some cases the coronary chemoreflex (Bezold-Jarisch reflex) in which there is inhibition of sympathetic outflow and increased activity of the cardiac (efferent) vagus leading to profound bradycardia, hypotension and cardiac collapse . . . Minimal lethal dose of endotoxin given over hours of time is so small as to be undetectable by any clinical test presently in use.

Bendig, J. and Haenel, H.: Gastrointestinal Microecology of Sudden Unexpected Death of InfantsNutrition, Proc. Eight Congress Nutr., Prague, Sept. 1969. (ed) Josef Masek, Prague, & Sir David P. Cuthbertson, Glasgow. Excerptica Medica, pub., Amsterdam. p.212-214-1970.

Reisinger, R.C.: ***A final mechanism of cardiac and respiratory failure.*** **Pub. in** ***SIDS 1974.*** **Proc. of Camps International. Symp. on SUD in Infancy. Pub:** ***Canadian Found. for Study of Infant Deaths.*** **4 Lawson Blvd., Toronto M4V 1Z4. 77-82-1974.**

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Wells,J.C.K., Davies,P.S.W.: ***Sudden Infant Death Syndrome.*** ***Archives of Disease in Childhood 1994;*** **70:252-253**

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