

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 (SIDS) 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.

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.

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

Reisinger, R.C.: *Studies on the pathogenesis of diarrhea of newborn calves.* M.S. thesis. U Wisconsin, Madison, 1957.

Reisinger, R.C.: *Parainfluenza 3 virus in cattle.* *Ann NY Acad Sci* 101: 576-582, 1962.

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)

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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.)

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.

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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