Abstract

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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.

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