Respiratory Rate (Concentration), Arousal (Exposure Time), and Long QT (Lethal Dose) Associated Sudden Infant Death Risk Factors are Altered by a Fourth Factor, Anxiety.

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Abstract

Sudden Infant Death Syndrome (SIDS) theories, from over 50+ years of research, were found to be incomplete solutions to the syndrome. This author sought to resolve their partial inadequacies. SIDS review articles were searched from a medical school and a private college's digital subscriptions. The ISO 13344:2015 fire-effluent-simulation equation was found by searching the phrase 'flame retardants.' The ISO indicated hyperventilation to increase gaseous toxicity. Arousal, long QT, and sympathetic tone theories were incorporated. The Triple Risk Model of SIDS by Filiano and Kinney identified SIDS risks as a vulnerable infant. The fetal hemoglobin switch at 2 to 6 months provided the developmental timing. Habituated hypoxia caused an increased arousal threshold and increased respiratory rate, contrasted with short-term hypoxia's leading to a decreased infant respiratory rate. QT interval prolonging gases, such as ammonia or combustion engine exhaust, were possible exogenous stressors. Acquired long QT syndrome (LQTS) was the lethal vector. Severe bradycardia is indirectly a long QT risk. Nicotine withdrawal induces bradycardia, making smoking a SIDS risk. Known SIDS risks are categorized by Haber's Rule into three components: concentration (respiratory rate), time (arousal threshold), and dose endpoint (LQTS).The SIDS risks fall into one or more of these three components and composite to become lethal. All SIDS risks were accommodated on the Haber's Rule template. Anxiety was a fourth overarching SIDS factor correlating directly with increased respiratory rate, heart rate and QTc. Anxiety negates respiratory rate changes from prone rebreathing, and QTc effects on fetal hemoglobin switch and from prone positioning.
Preprints DOI: 10.13140/RG.2.2.18116.60805/1

Keywords: cardiovascular electrophysiology, Haber's Rule, pediatric sleep medicine, respiratory rate, sudden infant death syndrome.

Multifactorial Cause of Sudden Infant Death