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 the search phrase 'flame retardants.' The ISO indicated hyperventilation increased gaseous toxicity. Arousal, long QT, and sympathetic tone theories were incorporated. The vulnerable infant of Filiano and Kinney's Triple Risk Model of SIDS, was due to SIDS risks. The fetal hemoglobin switch at 2 to 6 months provided the developmental timing. Habituated hypoxia caused an increased arousal threshold and increased respiratory rate, in contrast, short-term hypoxia led to a decreased infant respiratory rate. QT prolonging gases, such as ammonia and combustion engine exhaust, were exogenous stressors. Acquired long QT syndrome (LQTS) was the lethal vector. Autoresuscitation from severe bradycardia is a long QT risk. Nicotine-withdrawal-induced bradycardia made 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 classified as one or more of these three components and composited to become lethal. All SIDS risks found location on Haber's Rule template. Anxiety was a fourth overarching SIDS factor that correlated directly with increased respiratory rate, heart rate, and corrected QT (QTc). Anxiety negated respiratory rate changes from prone rebreathing, as well as QTc effects from the fetal hemoglobin switch, and prone positioning.
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Multifactorial Cause of Sudden Infant Death