Respiratory rate, arousal, and long QT risk factors for Sudden Infant Death follow Haber's Rule

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Abstract

Introduction: Sudden Infant Death Syndrome (SID[S]) theories, from over 50+ years of research, were each an incomplete solution. This author sought to resolve their partial inadequacies.

Methods: SIDS articles were searched within a medical school's and a private college's subscriptions. The ISO 13344:2015 fire-effluent-simulation equation was found searching the phrase 'flame retardants.' Arousal, long QT, and sympathetic tone along with ketogenesis, theories were added sequentially.

Results: The ISO demonstrated hyperventilation increases gaseous toxicity. The SIDS-risks create infant vulnerability. The fetal hemoglobin switch at 2 to 6 months provides the developmental timing. QT prolonging gases, such as ammonia and combustion engine exhaust, are exogenous stressors with prolonged corrected QT the lethal vector. SIDS-risks categorize as Haber's Rule's three components: concentration (respiratory rate), time (arousal threshold), and dose endpoint (long QT syndrome, LQTS). SIDS-risk factors accumulate to facilitate lethality. An infant's respiratory rate is 3.6 times that of an adult. Habituated hypoxia during the fetal hemoglobin switch causes an increased respiratory rate and arousal threshold. Prone sleeping increases arousal threshold and rebreathing theoretically hyperventilation. The ventricular repolarization reserve is least in the early morning, both in cardiac events and SID. Autoresuscitation from severe bradycardia *via* tachycardia is a corrected QT SID-risk. Smoking's nicotine-withdrawal-induced bradycardia is SID-risk. Sympathetic tone in the forms of underlying anxiety versus a sudden increase in tone are a fourth factor affecting SID-risk. Propranolol is used to treat prolonged QT and Omega-3s have potential to prevent the lethality of LQTS.

Conclusion: This proposed SID-hypothesis incorporates all SIDS-risk factors.

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Multifactorial Cause of Sudden Infant Death