

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 review articles were searched within a medical school's and a private college's digital subscriptions. The ISO 13344:2015 fire-effluent-simulation equation was found searching the phrase 'flame retardants.' The ISO demonstrated hyperventilation increases gaseous toxicity. Arousal, long QT, and sympathetic tone theories were added sequentially.

Results: 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. SIDS-risks categorize as Haber's Rule's three components: concentration (respiratory rate), time (arousal), and dose endpoint (long QT syndrome, LQTS). Multiple SIDS-risk factors 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 decreased arousal. The lethal vector is a prolonged corrected QT. Prone sleeping decreases arousal and theoretically causes hyperventilation due to rebreathing. The ventricular repolarization reserve is least in the early morning, both in cardiac events and SID. Tachycardic autoresuscitation from severe bradycardia is a corrected QT risk. Nicotine-withdrawal-induced bradycardia is smoking's SIDS-risk. Sympathetic tone in the forms of underlying anxiety versus a sudden increase in tone are a fourth factor affecting SIDS-risk. Propranolol is used to treat prolonged QT and Omega-3's have likely potential to prevent the lethality of LQTS.

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

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