Multifactorial Causes of Sudden Infant Death--Respiratory Rate with Arousal and Long QT vs. with Sympathetic Tone: A Review

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Abstract

Aim: Sudden Infant Death Syndrome (SIDS) theories, from over 50+ years of research, were each found lacking. This author sought to resolve their inadequacies.

Methods: SIDS review articles were searched from a medical school library's subscriptions. Further searching found the ISO 13344:2015 fire-effluent-simulation equation where hyperventilation increases gaseous-toxicity. Arousal, long QT, and sympathetic tone theories were incorporated sequentially.

Results: Haber's Rule and the Triple Risk Model of SIDS help to understand SIDS risks and cause. Haber's Rule categorizes SIDS risks into three components: concentration (respiratory rate), time (arousal), and endpoint (acquired long QT syndrome). The fetal hemoglobin switch at 2 to 6 months is SIDS's developmental timing. Habituated hypoxia causes an increased arousal threshold and an increased respiratory rate, contrasted with short-term hypoxia causes a decreased respiratory rate. QT interval prolonging gases, such as ammonia or combustion engine exhaust, may be exogenous stressors. Hypercapnia/hypoxia causes the 3-months-old infants' respiratory rates to increase. Increased anxiety overrides prone rebreathing's and fetal hemoglobin switch's respiratory rate changes, as well as the prone position's effect on QTc. Severe bradycardia plots on the QT nomogram as likely causing *torsade de pointes*.

Conclusion: Haber's Rule components categorize the SIDS risks and in composite they are lethal.

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Respiratory Rate and Sudden Infant Death Risk