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| Topics: **SIDS/SUID** |
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| **Etiopatogenesis and prevention of SIDS** |
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| **Background:**The CNS maturation is genetically determined and is linked to the contribution of glutamate to dendritic synapses. All neurones, except ganglionic neurones, need dendrites to function. The dendritic synapses are formed and increase in number only in presence of glutamate and, thus, when to the CNS arrive stimuli from the periphery. At birth these synapses are relatively few and are even less before birth.  **Objectives:**We aim to explain how the pacifier use can prevent the SIDS. We propose an etiopathogenesis model  which ties together what is known about the brain regions affected in SIDS and a documented preventative factor in infants. We propose that SIDS occurs due to a lack of sufficient development and plasticity of glutamatergic synapses in the mesencephalic nucleus of the trigeminal nerve (Me5) and the reticular formation (RF) of the brainstem.  **Methods:** We reviewed the CNS maturation, the glutamate as responsible for the synapses maturations, and how an anatomical formation in the CNS, with very singular characteristics, can supply the brainstem of glutamate. This structure is the Me5 and reviewing its anatomy we found out that it is the only intra-neuraxial ganglion, mainly made up of pseudo-unipolar neurones whose efferent branches collect stimuli from the periodontium, while the afferents go to the others trigeminal nuclei and especially the RF, to which Me5 releases glutamate; many studies show the relationship between the Me5 and the RF. We studied, then, the Me5 cells: they don’t possess dendrites, therefore, they don’t need graded potential and their action is very powerful, as demonstrated by the investigations on pacifier use and SIDS (up to 90% survival) and researches on the correlation between the use the chewing gum and the Arousal.  **Results**: The pacifier use can prevent SIDS since it stimulates the Me5, which stimulates the RF and increases the number of dendritic synapses on these cells; this way more graded potential are formed, is realised the action potential in the cell soma and the RF cells can continue to operate normally.  **Conclusion**: Our research suggests a plausible model by which SIDS occurs, while also explaining the lack of major neuropathological findings in post-mortem brains. It’s in agreement with previous studies that identify the brainstem as the primary site of dysfunction in SIDS, and provides a means to understand why these infant deaths almost exclusively occur during sleep in children who show no signs of cardiac or respiratory distress during wakefulness. According to this model the lack of glutamate to the dendrites of the RF cells could hamper its proper functioning both in regards with the regulation of vital functions both for the intrinsic functionality of the RF specific cells (eg. production of serotonin, acetylcholine etc.). The failure in the supply of glutamate to the dendrites of the RF cells during sleep could be compensated by activating the Me5 through the pacifiers use. This has the potential to be a low cost means to significantly reducing infant death worldwide and warrants further scientific attention.  **Funding source:**none. |
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