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RESORPTIVE DEGENERATION OF THE HEART'S CONDUCTION SYSTEM AND SUDDEN INFANT DEATH SYNDROME: PRELIMINARY RESULTS

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The cardiac conduction system undergo remarkable morphological transformation beginning about 1 or 2 weeks after birth. Such molding, called "resorptive degeneration" consists of degeneration, cell death and replacing in an orderly programmed way. However, such process, if exaggerated, could provoke blocking disruption of the pathway itself, and if defective could leave in place some accessory communication between the atrio-ventricular pathway and the adjacent ordinary myocardium. The purpose of the study is to determine the presence of resorptive degeneration in the heart's conduction system in cases of sudden infant death syndrome (SIDS) and of explained death (ED). We analyzed 66 hearts from autopsied cases of SIDS (44 males and 22 females) and 12 hearts from cases of age-matched ED (6 males and 6 females). Histological observations were focused on the heart's conduction system. Histological examination of the cardiac conduction system has been carried out on serial sections, with the technique devised by one of the present authors (L. Rossi). We observed areas of resorptive degeneration in 96.96% of the SIDS cases and in 76.92% of the ED cases. Resorptive degeneration, if defective or exaggerated and/or if associated with particular neurovegetative stimuli, could have caused potentially malignant arrhythmias responsible for sudden and unexpected death. These data suggest the opportunity for a more accurate cardiac conduction system than the one currently performed in SIDS cases.

APOPTOSIS IN CARDIAC CONDUCTION SYSTEM IN SUDDEN INFANT DEATH SYNDROME (SIDS): PRELIMINARY RESULTS.

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Crib death is an old problem in medicine. The search for an explanation is both a medical and public concern. There is still no generally accepted explanation as to how and why it happens. New ingenious possibilities are frequently introduced. Some authors provide further evidence in support of a cardiac cause for crib death. It appears that final common pathway could be some form of fatal electrical instability of the heart. Postnatal morphogenesis of the human heart is an important part of its normal development. The term "resorptive degeneration" has been originally suggested by James in 1968 to indicate the normal process of cardiac molding. Such molding consists of degeneration, cell death and replacing in an orderly programmed way. The programmed cell death named apoptosis is of particular interest; its unpredictable but rapid occurrence could play a role in the pathogenesis of SIDS. The aim of this study is to determine the presence of apoptosis in the resorptive degeneration areas of the conduction system in SIDS and in explained death (ED) cases. We analyze 5 hearts from autopsied cases of SIDS (ranging in age from 2 to 12 months) and 5 from cases of ED with the same range of age. Histological examination of the cardiac conduction system was performed on serial sections, using in situ endolabeling of fragmented DNA (TUNEL) method. The apoptotic index in SIDS (range: 0% - 0.01%) was lower than in ED cases (range: 0 - 0.1%). Apoptosis of the cardiac conduction system is discussed as a process favoring electrical stability, but such process, if defective, could leave in place some accessory communication between the atrio-ventricular pathway and the adjacent ordinary myocardium.

MATERNAL SMOKING AND AORTOPULMONARY PARAGANGLIA IN SUDDEN INFANT DEATH SYNDROME.

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Infants of mothers who smoked during pregnancy have deficient hypoxic awakening responses, which may contribute to an increased risk of sudden infant death syndrome (SIDS). The exact mechanism remains to be determined. The aorticopulmonary paraganglia (APP), near the base of the heart, have a physiologically documented role in chemoreception, but their neuroanatomic complexity has limited the investigations in human diseases. Recently, we described APP hyperplasia in 24% of SIDS cases, but a maternal smoking correlation was not made. In this report, we studied the APP only in SIDS victims with (or not) clinical history of maternal smoking during pregnancy.

Using morphometric methods, we calculated the APP volumes from infants who died of SIDS and whose mothers smoked during pregnancy ($n=5$), infants who died of SIDS and whose mothers were nonsmokers ($n=17$), and age-matched control infants ($n=9$). APP hyperplasia (volume= 0.06 mm^3) was observed in infants who died of SIDS and who were born from smoking mothers, when compared with infants who died of SIDS and who were born to nonsmoking mothers (volume= 0.03 mm^3 ; $p<0.05$) and age-matched controls (volume= 0.02 mm^3 ; $p<0.05$).

Our findings suggest that maternal smoking can play a role in a dysfunction of the APP chemoreceptor system causing hyperplasia. The abnormality responsible could be primarily in the organ or it could reside in brain stem respiratory centers with efferent or afferent connections to the peripheral chemoreceptors. The study of the mechanisms of nicotine exposure on the development of the respiration and cardiovascular control may offer insights into the ultimate mechanism of SIDS.