INTRODUCTION

During my neurology training, one of my associates discovered his little son dead in the back of his car, after his daughter had cried out, "Why is brother so cold?" This was my first encounter with Sudden Infant Death. Since then I have been acutely aware of this tragic event which occurs in approximately 100 to 140 cases yearly in Virginia. It is amazing that with such frequency of incidence, this is only the second symposium held in Virginia. Hopefully, we will arouse more profound interest in the subject. Many theories have been expounded as to the cause of Sudden Infant Death, not the least of which are those reports in the newspapers. This symposium covers the problem of etiology, pathophysiology, and recognition of the at-risk infant, the management of the Sudden Infant Death victim, parents and the family, as well as efforts to prevent the occurrence of this event.

sult is yet to be discovered. (See Figure) If these brain derangements are in fact causative, it is essential to correlate clinical aspects with the pathological states. Neuropathological changes noted include impaired myelination of intrinsic and extrinsic nerve structures, leukomalacia of a specified area, dendritic abnormalities and astroglial proliferation, all affecting brain centers and resulting in dysfunction of the autonomic nervous system, respiratory and cardiac centers. The clinical correlates are the lack of respiratory drive, abnormal breathing patterns, and abnormal responses in organs under autonomic nervous system control, that is to say, the heart and gastrointestinal tract. Recently, a new syndrome in adults, sleep apnea, may well serve as a model for Sudden Infant Death in children.

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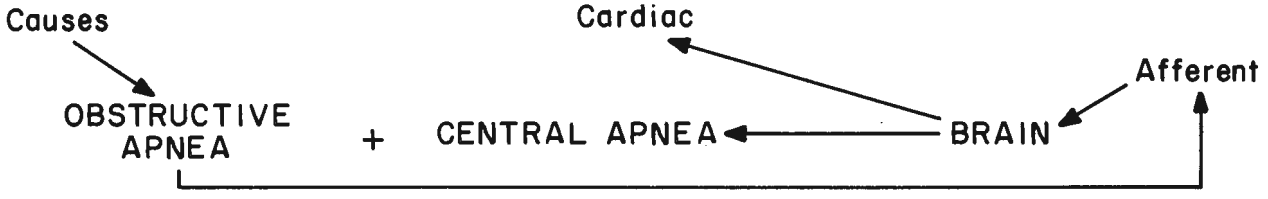

There is a growing feeling among neurologists and neuropathologists that whatever the multitude of environmental factors, afferent stimuli and end organ failure that occur, in at least 50% of the infants with Sudden Infant Death Syndrome (SIDS), there has been or is an old or continuing dysfunction in brain structures. How much of this is cause or result is yet to be discovered. (See Figure) If these brain derangements are in fact causative, it is essential to correlate clinical aspects with the pathological states. Neuropathological changes noted include impaired myelination of intrinsic and extrinsic nerve structures, leukomalacia of a specified area, dendritic abnormalities and astroglial proliferation, all affecting brain centers and resulting in dysfunction of the autonomic nervous system, respiratory and cardiac centers. The clinical correlates are the lack of respiratory drive, abnormal breathing patterns, and abnormal responses in organs under autonomic nervous system control, that is to say, the heart and gastrointestinal tract. Recently, a new syndrome in adults, sleep apnea, may well serve as a model for Sudden Infant Death in children.

At the Medical College of Virginia we have been aggressive in evaluating what we have diagnosed as near-miss SIDS and we believe that in many of our cases we have established treatable precipitating causes for acute episodes.

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