Sudden Death of Babies

There are few things in life so sad as the sudden death of a baby. It is not surprising then that the subject has long been one of medical and public interest, and the scholarly historical account by Froggatt and his colleagues includes biblical and other ancient descriptions. Today certain publicized efforts have brought new attention to research on the subject, which is as yet largely unexplained despite a wide spectrum of proposed theories. One theory has suggested that the final common pathway may be mediated by electrical instability of the heart in young babies, a situation in part the consequence of a normal developmental process occurring in all infants but at the same time rendering the behavior of the conduction system capricious. In this issue of Circulation Drs. J. T. Lie, H. S. Rosenberg and E. E. Erickson offer some important additional observations on the morphology of the conduction system in this crucial period of life.

As indicated by Lie and his colleagues, there have now been several such studies of the cardiac conduction system of human infants, but while it seems to me that most of the anatomical descriptions are quite similar or identical, the interpretations of what the findings mean have been at variance. In my original report there was no suggestion that the finding of an active molding and shaping of the atrioventricular (A-V) node and His bundle represented a pathological process. On the contrary, I emphasized that the process was ubiquitous at the newborn period, that it was in this sense a normal phenomenon rather than a pathological one, but that ubiquity could not be read as synonymous with safety or stability. Despite my precautionary phrases, Valdes-Dapena and her colleagues took me to task about the "histopathologic changes" (her quotation marks, not mine) even though I had assiduously avoided calling these pathological in nature. In her study with Ray Truex and others they concluded that there were no dead cells, no phagocytosis, no replacement fibrosis, nor any evidence of rapid (sic) remodeling, and therefore they questioned the validity of attributing malfunction of this or any other anatomical system to features of its normal development. In the article she did admit that our differences were largely ones of interpretation, although in her own accompanying editorial she was a bit more critical. R. H. Anderson and his colleagues essentially agreed with Valdes-Dapena. On the other side of the argument, both W. R. Anderson and his associates and J. A. J. Ferris have supported the concept that the cardiac conduction system does undergo postnatal molding and shaping in the vicinity of the central fibrous body.

The major issue is whether the normal A-V node and His bundle of the newborn infant do undergo active molding and shaping, a process which the late Robert Grant said occurred directly in the region of the central fibrous body and several other parts of the human heart. In their new report Lie et al. say that an "apparent" molding process involved the A-V node and His bundle in all the hearts they studied, and that it was most evident the first year of life, being complete not long after that. This is exactly what I saw and said as well, and I am delighted with what I interpret as confirmation. However, Lie et al. make a different interpretation, saying that their inability to find cell death, degeneration, inflammation or macrophage infiltration makes them doubt a functional significance for the molding process, although they admit that electrophysiological studies during life are merited.

The thing which Valdes-Dapena, R. H. Anderson and Lie et al. most criticize is the use of terms such as cell death, degeneration, fibrosis, necrosis, inflammation and active destruction. At the same time, they have been the principal users of such terms, a gambit well known among debaters as the straw man approach. All of these terms conjure images of some marked abnormality rather than normal development, and it is not an image I ever intended. Many normal developmental processes in the human body are very much associated with transient instability of performance, particularly in infancy and childhood.

There is no particular reason to be so resistive to the concept of cell death and degeneration as a part of normal evolutionary development, and abundant examples are known from the work of others on this matter. To expect that extensive or massive sections of cell death or necrosis are to be found is not what the process of molding means. Molding means that cells are gradually replaced in an orderly programmed way, which is why I suggested the term resorptive degeneration. Dr. Lie's excellent drawings in figure 3 represent things exactly as I saw them, and as Professor Navaratnam of Cambridge University also drew them. However, our interpretations differ in that the islands in my archipelago are said to disappear because of resorptive degeneration, whereas Lie and his colleagues do not suggest just what happens to theirs, although they admit...
that adult hearts are different. There is so little in the report by Lie et al. with which I could quarrel substantively that I could equally well consider it a confirmation of my own observations instead of their implied refutation. Or as they say in the computer world, one man’s noise is another man’s signal.

Some of the other matters are of less importance in my opinion than is the one of whether active molding and shaping occurs, and what its cytological mechanism may be. For example, focal hemorrhages, the occurrence of cartilaginous islands and narrowing of the A-V node artery are not ubiquitous and no one has claimed them to be. However, to say that they are not functionally significant because some such changes were found in “control” cases (also dead) presupposes that more was known about the exact mechanism of death in those cases than is usually the case; this particularly refers to knowledge of the electrical activity of the heart in the terminal period, information which is rarely available. In fact, it is the difficulty of getting just such information on a sufficiently large number of cases which has left the functional question about cardiac electrical stability in the newborn period still unsettled. While this type of work will be inordinately difficult, requiring prolonged periods of 24-hour noise-free electrical monitoring in normally restless infants, it nevertheless remains a legitimate question and the difficulty in getting an answer to it does not weaken its importance.

It would indeed be regrettable if the interpretations offered by Lie et al. discouraged others from investigating the functional aspect of the question. The possibility of electrical instability of the heart in the newborn period is an important matter not only relative to the pathogenesis of crib death, but also for other disabling problems short of death, such as the consequences of cerebral hypoxia secondary to an arrhythmia or conduction disturbance. But easily the most persuasive argument for determining whether or not cardiac electrical instability of a dangerous degree occurs in babies is that most forms of it are either treatable or preventable.

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