Serotonin and SIDS

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TO THE EDITOR: The article by Cummings et al. (2) emphasizes the vital role of serotonin in cardiovascular homeostasis and respiratory control, using mice with a genetic deficiency, challenged by severe and repeated anoxia. [Their model of an heritable defect as the cause of sudden infant death syndrome (SIDS) is not consistent with SIDS with a familial recurrence of only 16/1000 (4) and the incidence of all deaths after a SIDS case was 21/1000, indicating the lack of specificity of the recurrence.]

Although the authors demonstrate that depletion of serotonin would increase the risk of death in these mice and that it may apply to SIDS, they do not discuss the possibility that an infant’s failed efforts to survive would consume this essential substance. In short, are the serotonin abnormalities in the brain stem of SIDS victims the cause or the effect?

Cann-Moison et al. (1) found that the levels of metabolites of serotonin and other neurotransmitters were elevated in cerebrospinal fluid of SIDS victims, as well as in infants who had survived severe asphyxia.

Kinney, a coauthor of this paper, has postulated since 1994 that SIDS can be explained by a triple-risk hypothesis (3). These authors conclude that SIDS victims develop an abnormality of serotonergic cells during fetal life and are then born with a vulnerability that does not kill until 2–6 mo of age by a “stressor.” But Waggener (5) and three other groups prospectively tested a group of neonates who subsequently died of SIDS and found their respiratory function was no different from their controls.

Finally, since the triple-risk hypothesis posits a defect originating in fetal life, by definition it is a congenital defect. The peak age of death for congenital defects is the first month of life; at that age the rate of SIDS is the lowest of the first year. The authors have performed their experiment well, but their extrapolation of the results to SIDS is unwarranted.

REFERENCES