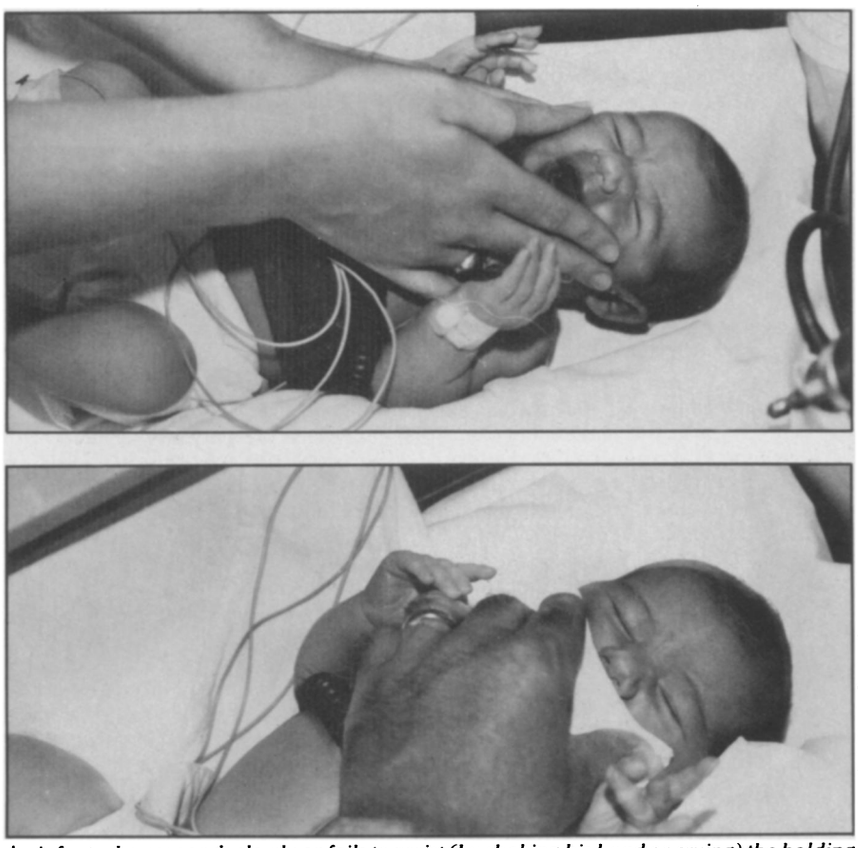
SUDDEN INFANT DEATH



Photos: Child Study Center, Brown University

An infant who responds slowly or fails to resist (by shaking his head or crying) the holding of his head (top) may be at greater risk for SIDS. *Similarly, a baby who does not respond properly (by a reflexive head movement and arm swinging, or crying) to a 10-second partial breathing blockage also appears at greater risk.*

Research results suggest that crib death — a chilling enigma until recently — may be a type of early learning disability. But prevention is still "down the road."

BY JOEL GREENBERG

This is the first of two articles on mysterious instances of infant death and disease.

It is early spring. The three-month-old child has had a runny nose for several days, but his parents aren't too concerned; he has no fever. It is probably the last in a normal string of winter sniffles, they reason. By the next morning the baby has died in his crib. It happened quietly, during sleep, without any apparent struggle.

If there is anything close to "death from natural causes" among infants it is sudden infant death syndrome (SIDS), or "crib death." Death, of course, for a two- to four-month-old (the most common age range of SIDS victims) is not natural, but until recently investigators have been nearly at a complete loss to explain the syndrome, which claims perhaps 10,000 lives a year in the United States alone.

"The terms crib death and SIDS are in fact diagnoses of essential ignorance ... the labels are applied when no discernible sign of pathology in the deceased infant is present," says psychologist Lewis P. Lipsitt, director of the Child Study Center at Brown University. "The terms ... would be more useful if it were generally appreciated that the diagnosis really means that the cause of the death is not yet known."

For the first time, however, Lipsitt and other researchers believe they are close to tracking the causes and mechanisms of the mysterious syndrome. Historically, most such deaths have been blamed on smothering — in a blanket, pillow or other external object. Lipsitt points out that such an explanation can foster parental guilt by suggesting negligence in providing for the baby's sleeping safety (at one time it was actually thought that the mother herself may have caused the death in some cases by rolling over on her infant during sleep).

More recently, though, research results point to the presence of some type of respiratory problem, such as apnea, that is *internal* rather than precipitated by an outside object. Studies by Lipsitt and several others "support the proposition that respiratory abnormalities were present at birth, months before the infants died," he says.

Further results from the nationwide Collaborative Perinatal Project of the National Institute of Neurological and Communicative Disorders and Stroke found multiple signs of possible neonatal brain dysfunction in future crib death victims. Abnormalities were documented in respiration, feeding and temperature regulation. Results of several neurological tests and Apgar scores (a numerical physical rating of the newborn infant) were significantly low, and birth weight was below average in many cases. Mothers of infants who succumbed had a greater incidence of influenza and cigarette smoking, tended to be of low socioeconomic and educational level and lived in crowded housing.

Perhaps the most significant of recent findings are coming from Providence, where Lipsitt has studied 15 SIDS cases (matched against two control groups of infants) as part of the collaborative study, and a total of 62 instances of crib death for his own ongoing project at Brown. His results have borne out a revolutionary theory of the syndrome's development and mechanism, which he believes could ultimately lead to early prediction and prevention techniques. Lipsitt's work is doubly important because, he says, it meshes closely with a SIDS prevention scale devised by English investigators R. G. Carpenter and J. L. Emery (SN: 9/10/77, p. 167) — a connection he revealed in March at a meeting in England.

Lipsitt's results suggest that sudden infant death is not merely a freak physical accident or the result of a major congenital defect. Instead, he says, crib death victims appear to have suffered from a subtle but complex "learning disability." Of course, learning at that age does not involve reading, speaking language or writing, but rather a developmental process by which totally biological, protective reflexes present at birth evolve into "learned, voluntary" responses, says the psychologist.

"Much brain tissue maturation occurs shortly after birth, and particularly in the first two months of human life," Lipsitt says. "If some behavioral patterns are not adequately learned by a certain age, coinciding with the time by which the un-

learned protective reflexes have diminished to an ineffectual level, the organism will not have been prepared adequately for survival."

In SIDS, most critical among the early reflexes is the "natural, defensive response of the normal neonate" to respiratory blockage or the threat of blockage. Lipsitt and his colleagues have observed this defensive "rage response" when a gauze pad is placed briefly over the mouth of an infant. His research also reveals that SIDS victims had a considerably higher incidence of respiratory abnormality at birth than did those of the control groups.

Lipsitt suggests that when subtle deficiencies exist in this and other innate responses (such as primitive grasping, reaching or visual attention), the "behavioral process of learning" that transforms such pure reflexes into more selective actions during the first two months may be inhibited. "If the newborn does not have a strong defensive response to threats to respiration, or to head restraint," says Lipsitt, "it is possible that the appropriate voluntary operant behaviors will not be learned which must ultimately supplant this congenital response by around two months of age."

Lipsett's findings corroborate the results of Emery and P.G.F. Swift, who observed that infants who respond poorly to respiratory blockage tend to lag behind others in overall development and are at greater risk for crib death. Earlier work in 1971 by researchers Rebecca Anderson and J.F. Rosenblith yielded similar results.

Crib death strikes primarily at night, most commonly during the winter and spring months and often after a cold or sniffles had been noticed by parents several days prior to death. Lipsitt suggests that respiratory blockage, combined with "failure of appropriate defensive behavior" and the infant's poor ability to compensate with mouth breathing for clogged nostrils "could conceivably lead to anoxia and a comatose state, and ultimately the infant's death.... It is quite possible that infants who have not learned to engage in responses necessary for clearing of the respiratory passages or clearing the way to those passages when threatened with occlusion will be those in particular jeopardy at the critical ages of two to four months," he says.

Other less direct but significant factors detected in the Brown research include:

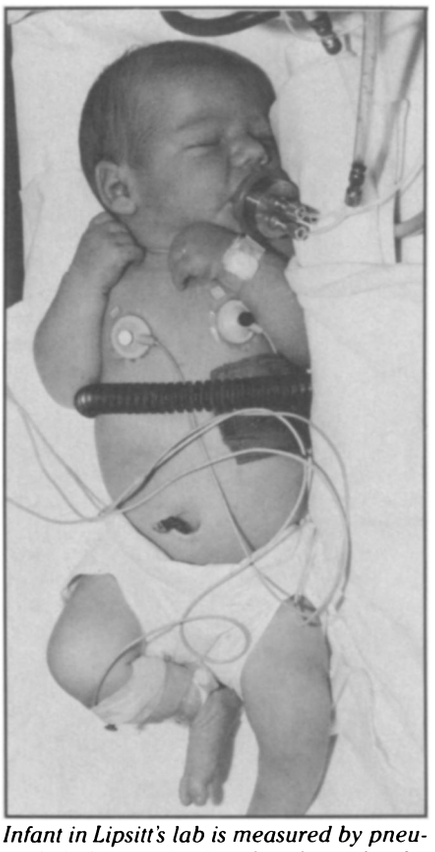
- The retrospective Apgar scores of the SIDS victims measured lower than those of controls at each of three times tested; the SIDS group at 5 minutes had barely reached the level of controls at 1 minute. The Apgar system assesses heart and respiratory function, muscle tone and reflexes.
- Mothers of SIDS victims had anemia during pregnancy twice as frequently as did mothers of surviving children.
- In addition to exhibiting respiratory abnormalities, crib death infants were born with somewhat lower birth weights and shorter body lengths and higher serum billirubin levels.
- The SIDS group required intensive care and was hospitalized significantly longer than controls.

"The literature on crib death in professional journals and in public documents tends to emphasize the prior normality of SIDS victims," Lipsitt notes. "It appears that while SIDS victims are essentially normal infants, on a group basis they do seem to be a risk population that can be identified on the basis of their biographies. It seems clear that infants who ultimately succumbed at two, three, four or five months of age were already showing, in general and on the average, that they were beginning life with some fragility."

In the latest portion of his work, Lipsitt has applied the SIDS risk scale of British scientists Carpenter and Emery to the case histories of Rhode Island's last 62 crib death victims. The results, which he reported in March at a special study group meeting on the syndrome, constitute an almost perfect match, and essentially corroborate the ability of the scale to predict potential SIDS victims, says Lipsitt. Included in the Carpenter-Emery SIDS risk factors are: younger mothers; smaller or premature babies; infants further along in the birth order than first or second born; urinary tract infection in the mother during pregnancy; and bottle-fed, rather than breast-fed children. The only factor that did not correlate with the Carpenter-Emery findings was blood group, according to Lipsitt. The British researchers found that infants of mothers with types A, B or AB appeared more susceptible than those of mothers with type A. Lipsitt's results did not suggest such a match.

Researchers are still a long way from pinpointing exactly what prenatal factors can contribute to the set of deficiencies that now appear inherent in sudden infant death syndrome. But there are some general indicators. "We can dare say that there are a number of fetal conditions that are definitely related to the welfare of the baby," says Lipsitt. Effects on the fetus of maternal smoking, drinking and drug use have been documented in a number of studies. There are also indications that certain pollutants, other environmental factors and the mother's emotional state can also influence fetal development.

Among the most frightening possibilities is the suggestion by a group of state of California researchers that some SIDS cases might result from "infantile botulism" — a newly detected phenomenon in which some infants appear to grow the potentially lethal spores in their intestines (see second article in this series). And University of Michigan scientists report "limited evidence for respiratory viral antigen" as another potential cause of crib death. The researchers have discovered certain antibodies in the lung tissue of some apparent SIDS infants, indicating "a possible hypersensitivity reaction" to disease just before death.



Infant in Lipsitt's lab is measured by pneumobelt for respiration, by electrodes for heart rate and by automatic nipple for sucking behavior. The baby is lying on a stabilimeter which records general body movement. Lipsitt's theory of the importance of "learning" to cope with respiratory blockage came originally from pediatrician Mavis Gunther, who first observed that infants often "struggle" while feeding.

"All factors that affect the baby's well-being are likely to have influences in the first couple of months of life," Lipsitt says. The ultimate aim of the research is to not only identify risk factors but, ideally, to take steps to prevent the syndrome from claiming an infant's life. "That's down the road a piece," Lipsitt concedes. Eventually, he adds, "we should be able to take remedial measures ... [to] administer certain kinds of experiences that obviate crib deaths. Such techniques are not available, yet." But, he says, prevention methods may come from more knowledge about the apparent learning deficiency in susceptible infants. "We are supposing that very important learning processes go on at this age," says Lipsitt. "[It is] a behavioral process that capitalizes on built-in responses. In the first two months, the infant *has to* learn." □