J Forensic Sci, Jan. 1977, Vol. 22, No. 1

L. R. Simson, Jr.,[1] *M.D. and R. E. Brantley,*[1] *M.D.*

Postural Asphyxia as a Cause of Death in Sudden Infant Death Syndrome

Many investigators have considered the possible role of asphyxia in sudden infant death syndrome (SIDS). Suggested mechanisms have included those which might mechanically compromise the airway, such as smothering, laryngospasm [*1*], and nasal obstruction from upper respiratory tract infection in the obligate nasal breathing infant [*2*]. Much current research focuses on the possibility of centrally mediated respiratory failure related to sleep apnea, functional abnormality of the ponto-medullary respiratory centers [*3*], chemically induced laryngeal apnea [*4*], and inappropriate operation of the oxygen-conserving reflex ("diving reflex") [*5*].

Roentgenographic observations in the following cases of unexpected infant death caused us to reconsider the anatomic and functional relationships of infantile airway structures which might be implicated in mechanical airway obstruction. These observations, interpreted in the context of infantile airway anatomy and respiratory physiology, prompt us to offer this hypothesis: One of the causes of SIDS is postural asphyxia. We propose that mechanical obstruction of the "normal" infant airway occurs at the pharyngeal level by either of two mechanisms. First, posterior displacement of the mandible and tongue may elevate the soft palate against the posterior pharyngeal wall. Second, upward displacement of the floor of the mouth and larynx may either elevate the soft palate against the posterior pharyngeal wall or force the epiglottis against the soft palate to obstruct the airway. The weight of an infant's head, pressing on the tip of his chin, is sufficient to displace the mandible. Flexion of an infant's head so that the chin approximates the sternum elevates both the floor of the mouth and the larynx. If the pharyngeal airway should be significantly obstructed by either of these mechanisms, and the infant is unable to restore normal anatomic relationships, he will die quietly of asphyxia.

Methods and Observations

Three infants were studied. Two had been found unexpectedly dead in their cribs. The third was found dead in a semireclining infant chair where she had been placed for the night. The scenes of death were thoroughly investigated. All three subjects were products of uncomplicated full-term pregnancies and had exhibited normal growth and development. Two of the infants were thought to be entirely well prior to death. The third infant had been undergoing treatment for an upper respiratory tract infection.

Roentgenographic examination of the upper airway of each infant was performed under the direction of a radiologist (R. E. B.). With the infant lying on his back, the

Received for publication 30 April 1976; accepted for publication 4 June 1976.

[1] Departments of Pathology and Radiology, respectively, Edward W. Sparrow Hospital, Lansing, Mich. 48902.

head hyperextended over a large block beneath the neck, about 3 ml of warmed contrast medium (proplyiodone oil suspension) were instilled, with a medicine dropper, into each nostril. Using standard techniques, roentgenograms in the anterior-posterior, lateral, and Towne's projections were made. Then, while manual pressure was applied to the tip of the jaw to displace the mandible posteriorly, another lateral projection roentgenogram was obtained. In one case an additional lateral projection roentgenogram was made after 2 ml of contrast medium had been instilled into the infant's mouth and while his head was sharply flexed forward. An autopsy was then performed on each subject, and representative sections of all major organs were examined microscopically (L. R. S.).

Case 1

This 3-week-old female infant was found lying facedown in her crib. She appeared to have been behaving normally when fed and put to bed about 9:00 a.m. She was found dead at 10:30 a.m. At autopsy no pathologic abnormalities were demonstrated except for numerous petechial hemorrhages in the visceral pleura, pericardium, and thymus. This case fulfilled the criteria for SIDS [6].

Roentgenograms in the anterior-posterior and Towne's projections demonstrated a normally contoured upper airway without evidence of obstruction. Similarly, the lateral projection roentgenogram (Fig. 1) showed contrast medium outlining the normal nasal passages, nasopharynx, oropharynx, and the superior surface of the posterior half of the

FIG. 1—*Case 1. The infant is supine with the neck extended. Oily contrast medium demonstrates a widely patent nasal airway.*

tongue. The upper airway was widely patent. However, when gentle pressure was applied to displace the mandible posteriorly (Fig. 2), contrast medium was seen to have spread anteriorly over the tongue which was forced against the palate. The posterior

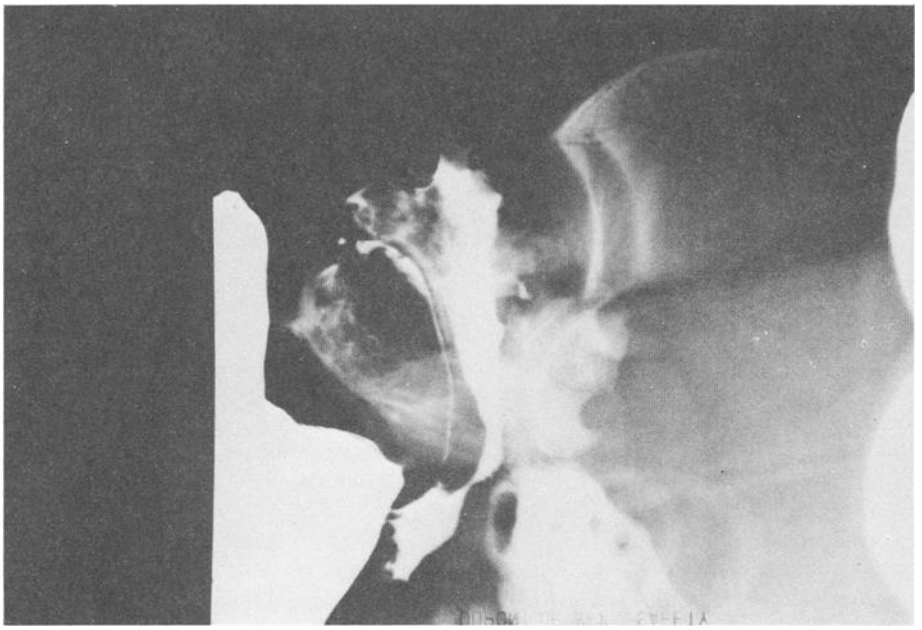

FIG. 2—*Case 1. Light pressure, simulating weight of infant's head, is applied to tip of mandible. Mandible and tongue are displaced posteriorly, forcing soft palate upward and backward to obstruct the nasal airway. Oral passage is simultaneously occluded by tongue.*

edge of the soft palate approximated the posterior wall of the pharynx. Thus, posterior displacement of the mandible caused nearly complete occlusion of the upper airway.

Case 2

The subject was a 4-month-old male found dead in an improvised crib, a car-bed fitted with a small mattress. His head was wedged facedown into the space between the mattress and the soft side of the bed. The back of his head was fixed against a metal bed support. His face was tightly pressed into a blanket. The facial livor mortis showed prominent blanching over the nose and mouth. No pathologic abnormalities, except for petechial hemorrhages in the thymus, were demonstrated. Death was certified as caused by accidental smothering.

Roentgenograms revealed a normal unobstructed airway. Anatomic relationships between the mandible, dorsum of tongue, soft palate, and base of tongue were clearly visualized in lateral projection (Fig. 3). Several attempts were made to displace the mandible posteriorly. Only when a great deal of force was applied could narrowing of the airway at the level of the soft palate be accomplished (Fig. 4). This degree of airway obstruction most probably would not be sufficient to cause death.

Case 3

This one-month old female was found dead seated in a semireclining infant chair. When found, her head was flexed onto her chest. She had been under treatment for a "head cold." Purulent lobular pneumonitis, sufficiently severe to be considered the

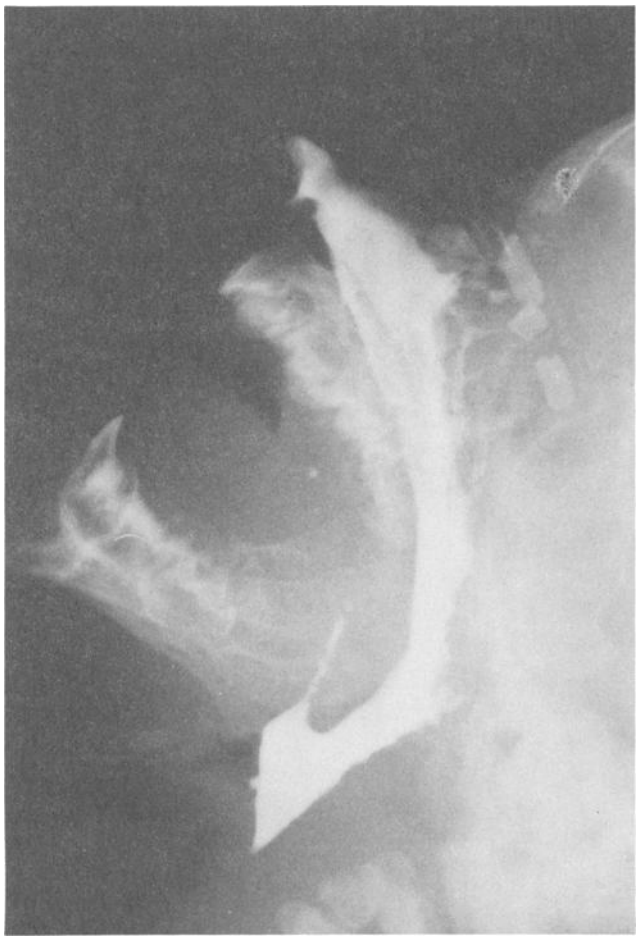

FIG. 3—*Case 2. The infant is supine with the neck extended. Normal patency of the nasal airway is illustrated.*

cause of death, was demonstrated at autopsy. Intrathoracic petechial hemorrhages were not found.

Roentgenograms obtained with the head extended revealed a normal unobstructed airway (Fig. 5). Pressure applied to displace the mandible posteriorly produced only narrowing of the airway similar to that seen in Case 2 (Fig. 4). However, when the head was gently flexed forward upon the chest, simulating the posture at the time the infant was found dead, mechanical obstruction of the airway was clearly apparent in the lateral projection (Fig. 6).

Discussion

Investigations of mechanical obstruction as a cause of SIDS have generally focused on the nasal passages and nasopharynx. Since these spaces are exceedingly difficult to evaluate at autopsy, a number of workers have used roentgenography to evaluate the upper airway [*7,8*]. Their findings have tended to exclude mechanical obstruction of the upper airway as a causative factor. Previous studies, however, have examined either

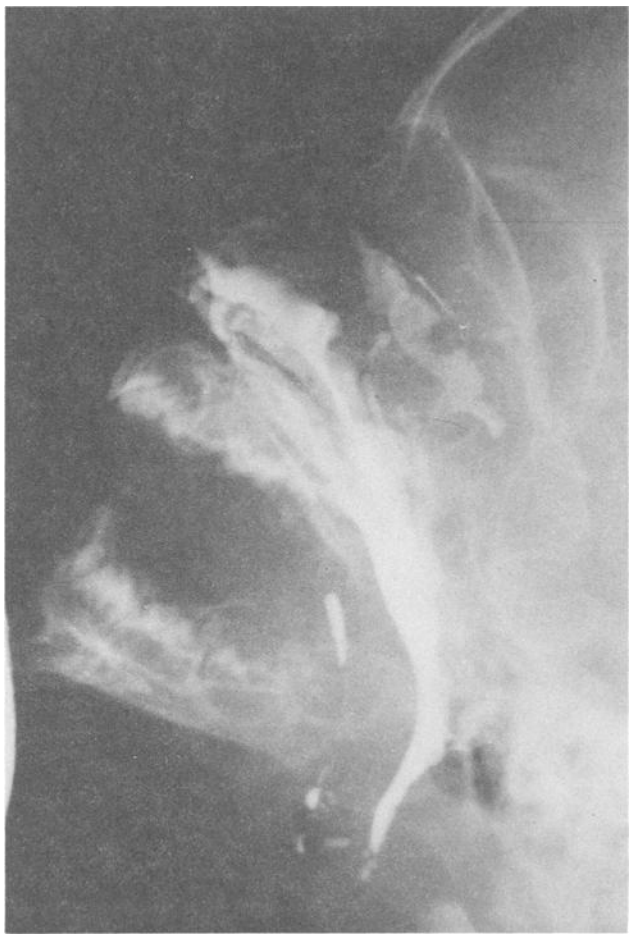

FIG. 4—*Case 2. Excessive force is applied on the mandible tip to achieve maximum posterior jaw displacement. Although the airway is noticeably narrowed, the soft palate does not occlude the nasal passage. Remaining airway is probably adequate for respiration.*

living infants breathing normally or dead infants on their backs with their necks in extension. To the best of our knowledge radiopaque contrast media have not been used in such studies nor have attempts been made to explore the anatomic relationships which might have existed in the airway at the time of an infant's death.

Tonkin [*9*], in 1974, suggested the possibility that SIDS might be precipitated by airway occlusion at the level between the palate and the base of the skull. She drew attention to the anatomic and functional relationships between the palate, tongue, posterior wall of the oropharynx, and the mobility of the infant mandible at the tempero-mandibular joints. She attempted to demonstrate these relationships roentgenographically in a stillborn infant. We consider these present observations to be a logical extension of her work.

Anatomy of the Infantile Airway

Anatomic relationships of upper airway structures of infants are considerably different

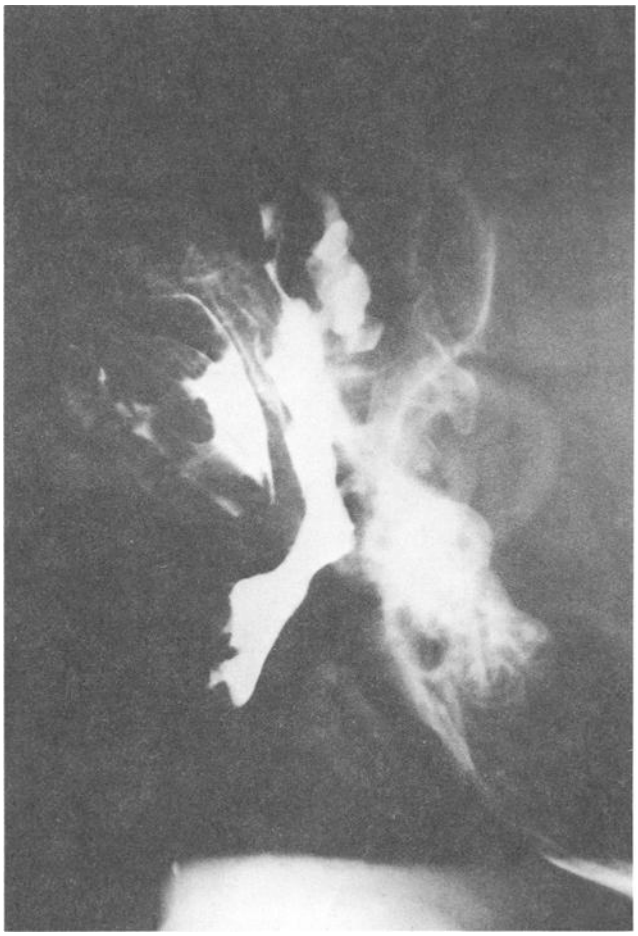

FIG. 5—*Case 3. A normal airway is shown with the neck extended.*

from those of adults. The hard palate is relatively short and, in the horizontal plane, is opposite the base of the skull rather than the first cervical vertebra. The soft palate, however, is long, being nearly as large as in the adult [*10*]. Since the cervical viscera occupy a relatively higher position in the neck, the posterior margin of the soft palate approximates the tip of the epiglottis. Functional continuity between palate and epiglottis is thought to provide the anatomic basis for obligate nose breathing in the neonate [*11*].

Concomitant with the relatively high position of the cervical structures, the infant's tongue is nearly horizontal within the oral cavity, unlike the adult in whom the posterior third of the tongue is in the vertical plane. An infant's tongue almost completely fills the oral cavity, and its superior surface closely follows the contour of the palate. This is, in part, a function of poorly developed alveolar ridges and the relatively short mandibular rami.

The infant mandible has short rami with small cartilaginous heads which articulate with nearly flat glenoid fossae at the temporo-mandibular joints. Movement is limited by the joint capsules and the temporo-mandibular ligaments. Thus, the articulation of the infant mandible allows considerable displacement in the horizontal plane [*12*].

FIG. 6—*Case 3. The neck is flexed, elevating floor of mouth and tongue as well as telescoping epiglottis and soft palate to obstruct the airway.*

As the mandible moves posteriorly it carries with it the tongue which, in turn, elevates the soft palate.

These anatomic features of the upper airway are common to all normal infants. However, about 3 in 1000 apparently normal infants become victims of SIDS. There are several mechanisms by which an infant with an anatomically "normal" airway can become a victim of postural asphyxia.

Mechanisms of Airway Obstruction

Many, but by no means all, SIDS victims are found lying facedown. Some of the weight of an infant's head can be transmitted in a force vector through the mandible toward the temporo-mandibular joints. The mandible and tongue, as an anatomical unit, can move posteriorly, forcing the tongue against the posterior margin of the soft palate which, in turn, will rotate toward the posterior pharyngeal wall. That gentle pressure applied to the tip of the chin will, in some infants, occlude the airway by this

mechanism is vividly demonstrated by the lateral projection roentgenogram of Case 1 (Fig. 2).

Direct pressure applied through the floor of the mouth to the base of the tongue can elevate the posterior third of the tongue against the soft palate and cause airway obstruction by a similar mechanism.

Neck flexion can produce airway obstruction by a somewhat more complex mechanism. Radiologists have long recognized that flexion of the neck is accompanied by foreshortening of the cervical tissues, buckling or redundancy of the cervical trachea just above the thoracic inlet, and elevation of the laryngeal and hypopharyngeal structures relative to the mandible and palate. As illustrated in Case 3 (Fig. 6), flexion of the neck so that the chin approaches the sternum can result in telescoping of the epiglottis and soft palate, the tip of the latter passing into the vallecula.

As the head is flexed forward the soft tissues of the floor of the mouth are compressed and the overlying skin is thrown into folds. This is especially prominent in healthy and well-nourished infants with abundant subcutaneous tissue. In addition to the visible rolls of tissue beneath the floor of the mouth there is also upward displacement of entrapped tissues, with a resultant force directed through the floor of the mouth, pushing the tongue upward and backward against the palate and into the hypopharynx.

Thus, when the head is sharply flexed forward the nasal airway can be obstructed by the epiglottis impinging on the posterior pharyngeal wall, and the pharyngeal airway can be blocked by overlap of the epiglottis and uvula. Encroachment by the tongue further obstructs the airway.

Airway obstruction caused by posterior displacement of the mandible and by elevation of the floor of the mouth has been observed in living children. Cross and Lewis [*13*], in 1971, while doing trunk plethysmography, noted that if the plethysmograph cuff either pushed the jaw backward or elevated the floor of the mouth the infant experienced respiratory distress. They further observed that "that baby's sleep is in no way disturbed by this obstruction."

As seen in Case 2 (Fig. 4), not every infant will experience severe airway obstruction even if the pressure applied to displace the mandible posteriorly is of far greater magnitude than could be produced by the weight of the infant's head. Similarly, not all infants suffer airway obstruction when the head is flexed forward onto the chest. We submit, however, that some infants, as illustrated by Cases 1 and 3, have a constellation of anatomical relationships, including mandible size and mobility, tongue size, palate length, laryngeal mobility, and pharyngeal contour, so that airway obstruction can occur under certain postural circumstances. One of the anatomical variables, pharyngeal contour, appears to be especially significant.

In two of our three cases the lateral projection roentgenograms reveal a soft tissue prominence in the posterior pharyngeal wall. This soft tissue mass, presumably adenoid, contributes to airway obstruction. In Case 2, where nasopharyngeal adenoidal tissue is absent, the airway is seen to be narrowed but not significantly obstructed.

There is great variation in the size and bulk of nasopharyngeal adenoids. In the newborn adenoidal tissue is very sparse. By three months of age this lymphoid mass is often 0.5 cm thick. However, there is considerable variation from one infant to another [*14*]. Nevertheless, nasopharyngeal adenoidal tissue is present in approximately two thirds of infants between one and six months of age [*15*].

Another significant variable is the ease with which the anatomy of the airway can be deranged. The above cases illustrate mechanical obstruction by postural manipulation of dead infants. Similar derangements can readily occur in the living.

Many unexpected infant deaths occur while the infant is in his crib, after a meal, and while he is presumably asleep. During sleep muscle tone is lost. The laxity of the sleeping infant's jaw and neck can be readily demonstrated in the nursery. When the infant

awakes muscle tone is quickly restored, and further attempts to displace the mandible or forcibly flex the neck are actively resisted. Sustained pressure will, however, cause loss of resistance through muscle fatigue.

It is apparent, then, that some infants with anatomically normal airways can experience significant airway obstruction from simple postural mechanisms which posteriorly displace the mandible, elevate the floor of the mouth, or sharply flex the neck. If the airway becomes sufficiently compromised asphyxial death may occur.

Asphyxial Death During Infancy

Pathologists have long been impressed by the similarity between autopsy findings in SIDS victims and in infants who are known to have died of asphyxia. Although scant, the pathological features are remarkably constant and include intrathoracic petechial hemorrhages, pulmonary congestion, and pulmonary edema.

Both clinical and experimental observations document the vulnerability of infants to airway obstruction. Infants with congenital choanal atresia often die quickly if they are unable to establish a pattern of mouth breathing [*16*]. Shaw [*2*] suggested that SIDS occurs among the approximately 30% of infants who are obligate nose breathers. He postulated that the nasal airway might become obstructed by congestion and edema from seemingly trivial upper respiratory tract infections. Subsequent investigators, however, have been unable to document nasal airway obstruction in any significant percentage of SIDS cases [*3*].

Unlike adults, infants may exhibit reduced pulmonary tidal volume with no increase in respiratory rate as the degree of upper airway obstruction is increased [*13*]. Hypoxia may depress respiratory drive, leading to periodic breathing and apnea [*17*]. Even sleeping adults have reduced ventilatory responses to changes in PO_2 and PCO_2 [*18*].

Short-term mechanical occlusion of the nasal airway in infant monkeys has been shown to produce apnea. On occasion this apnea continues after the obstruction is released, and resuscitation is necessary to prevent the animal's death [*19*]. Whether this phenomenon occurs in human infants is yet unknown.

Conclusions

The preponderance of evidence supports the view that asphyxia is a significant causative factor in sudden infant death syndrome. The problem has been to demonstrate anatomic or pathophysiologic mechanisms, or both, that are capable of causing lethal airway obstruction in seemingly healthy infants and yet completely elude detection at autopsy. The three cases illustrate that there are several mechanisms by which mechanical obstruction of an anatomically normal infantile airway can occur and that this mechanical obstruction is, in fact, demonstrable by appropriate roentgenographic techniques.

References

[*1*] Valdes-Depena, M. A., "Sudden and Unexpected Death in Infancy: A Review of the World Literature 1954-1966," *Pediatrics,* Vol. 39, No. 1, 1967, pp. 123-138.

[*2*] Shaw, E. B., "Sudden Unexpected Death in Infancy Syndrome," *American Journal of Diseases of Children,* Vol. 119, No. 5, 1970, pp. 416-418.

[*3*] Valdes-Depena, M. A., "The Sudden Infant Death Syndrome—1975. An Update for Pathologists," *Bulletin, International Academy of Pathology,* Vol. 16, No. 1, 1975, pp. 15-25.

[*4*] Johnson, P., "Laryngeal Induced Apnea," in *SIDS 1974, Proceedings of the Francis E. Camps International Symposium on Sudden and Unexpected Death in Infancy,* R. Robinson, Ed., The Canadian Foundation For The Study of Infant Deaths, Toronto, 1974, pp. 231-242.

[5] Wolf, S., "Sudden Death and the Oxygen-Conserving Reflex," *American Heart Journal,* Vol. 71, No. 6, 1966, pp. 840–841.

[6] Bergman, A. B., Beckwith, J. B., and Ray, C. G., Eds., *Sudden Infant Death Syndrome: Proceedings of the Second International Conference on Causes of Sudden Death in Infants,* University of Washington Press, Seattle, 1970, p. 248.

[7] Ardran, G. M. and Kemp, F. H., "The Nasal and Cervical Airway in Sleep in the Neonatal Period," *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine,* Vol. 108, No. 3, 1970, pp. 537–542.

[8] French, J. W., Beckwith, J. B., Graham, C. B., et al, "Lack of Postmortem Radiographic Evidence of Nasopharyngeal Obstruction in the Sudden Infant Death Syndrome," *Journal of Pediatrics,* Vol. 81, No. 6, 1972, pp. 1145–1148.

[9] Tonkin, S., "Airway Occlusion as a Possible Cause of SIDS," in *SIDS 1974, Proceedings of the Francis E. Camps International Symposium on Sudden and Unexpected Death in Infancy,* R. Robinson, Ed., The Canadian Foundation for the Study of Infant Deaths, Toronto, 1974, pp. 73–75.

[10] Tulley, W. J., "The Development and Growth of the Head, Jaws and Associated Parts," in *Current Orthodontics,* D. P. Walthren, Ed., John Wright and Sons Ltd., Bristol, 1966, pp. 1–22.

[11] Moss, M. L., "The Veloepiglottic Sphincter and Obligate Nose Breathing in the Neonate," *Journal of Pediatrics,* Vol. 67, No. 2, 1965, pp. 330–331.

[12] Weinman, J. B. and Sicher, H., *Bone and Bones: Fundamentals of Bone Biology,* Harvey Kimpton, London, 1947, pp. 86–92.

[13] Cross, K. W. and Lewis, S. R., "Upper Respiratory Obstruction and Cot Death," *Archives of Disease in Childhood,* Vol. 46, No. 4, 1971, pp. 211–213.

[14] Swichuk, L. K., Smith, P. C., and Fagan, C. J., "Abnormalities of the Pharynx and Larynx in Childhood," *Seminars in Roentgenology,* Vol. 9, No. 4, 1974, pp. 283–300.

[15] Capitanio, M. A. and Kirkpatrick, J. A., "Nasopharyngeal Lymphoid Tissue," *Radiology,* Vol. 96, No. 2, 1970, pp. 389–391.

[16] Beinfield, H. H., "Ways and Means to Reduce Infant Mortality Due to Suffocation," *Journal of the American Medical Association,* Vol. 170, No. 6, 1959, pp. 647–650.

[17] Rigatto, H., "Respiratory Response to Hypercapnia and Hypoxia in Preterm Infants," in *SIDS 1974, Proceedings of the Francis E. Camps International Symposium on Sudden and Unexpected Death In Infancy,* R. Robinson, Ed., The Canadian Foundation For The Study of Infant Deaths, Toronto, 1974, pp. 257–260.

[18] Robin, E. D., Whaley, R. D., Crump, C. H., et al, "Alveolar Gas Tensions, Pulmonary Ventilation, and Blood pH During Physiologic Sleep in Normal Subjects," *Journal of Clinical Investigation,* Vol. 37, No. 7, 1958, pp. 981–989.

[19] French, J. W., Morgan, B. C., and Guntheroth, W. G., "Infant Monkeys—A Model for Crib Death," *American Journal of Diseases of Children,* Vol. 123, No. 5, 1972, pp. 480–484.

Department of Pathology
Edward W. Sparrow Hospital
1215 East Michigan Ave.
Lansing, Mich. 48902