of trauma to the tracheostomy as well as to the trachea is almost inevitable. Our present technique is to insert a red rubber tube through the tracheostomy and anesthetize the patient with halothane and 50 per cent oxygen and air.

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Development of Bronchial Obstruction with Secondary Lobar Emphysema during Anesthesia

Fred J. Spielman, M.D.,* David H. Sprague, M.D.,† Elizabeth S. Mann, M.D.,‡ Valerie S. Mandell, M.D.§

Lobar emphysema secondary to a functioning one-way valve that permits gas to pass in the tracheobronchial tree during inspiration but not during expiration may result in compression atelectasis, a shift of the mediastinum, impairment of venous return, and precipitous hypoxia, hypotension, and cardiac instability.[1] Preoperative awareness of this condition allows for avoidance of anesthetic techniques (*e.g.*, nitrous oxide and positive pressure breathing) that might exacerbate the hyperinflation. In the following case report, we describe a patient who preoperatively had an unrecognized functioning one-way valve which manifested itself as an intraoperative respiratory emergency.

Report of a Case

A 4-month-old, 5.5-kg girl was scheduled for cystoscopy and possible right ureteral reimplantation. Her past medical history was unremarkable except for a history of urinary tract infections and the presence of a dilated right ureter and dilated right calices. The preoperative physical and laboratory evaluations were normal; a chest roentgenogram (fig. 1) was initially interpreted as normal.

One hour after the patient received 5 mg hydroxyzine and 15 mg pentobarbital, intramuscularly, anesthesia was induced with halothane and 60 per cent nitrous oxide in oxygen. Succinylcholine was then administered to facilitate insertion of a 3.5-mm tube in the trachea; immediately thereafter, auscultation of the chest revealed decreased breath sounds over the left upper hemithorax. Because we suspected that a right-sided endobronchial intubation had occurred, the endotracheal tube was pulled back, but no change in breath sounds occurred. As the muscle paralysis disappeared, breathing became spontaneous. Repeat auscultory examination of the chest revealed no change. Therefore, an emergency chest roentgenogram was obtained which showed a marked mediastinal shift to the right, hyperlucency of the left lung, and flattening of the left hemi-diaphragm (fig. 2); the endotracheal tube appeared to be correctly positioned. In addition, when the preoperative chest roentgenogram was compared with the intraoperative roentgenogram, a slight mediastinal shift, which was not originally appreciated by the radiologists, appeared to be present on the initial roentgenogram (fig. 1). Because of these findings, further operative procedures were cancelled, and the trachea was extubated after the child manifested vigorous activity. During the anesthetic, nitrous oxide (60 per cent) had been given for approximately 45 minutes and positive pressure breathing for approximately 15 minutes.

In the recovery room, the patient appeared in no acute distress and a pediatric pulmonary consultant recommended not to immediately investigate the pulmonary problem. However, about two hours later, tachypnea, tachycardia, and sternal retractions were noted. An emergency chest roentgenogram (fig. 3) now showed massive air trapping, herniation of the left lung across the anterior mediastinum, and a further shift of the mediastinum to the right. Therefore, an emergency bronchoscopy was scheduled. Anesthesia was induced and maintained with halothane and oxygen with spontaneous ventilation. A rigid bronchoscope with a side-arm adaptor for ventilation was used. Because bronchoscopy revealed a slit-like left main stem bronchus, a potentially lethal bronchial ball-valve obstruction was thought to exist. Therefore, after substituting an oral endotracheal tube for the bronchoscope, a left thoracotomy was undertaken.

During the thoracotomy, halothane and oxygen were administered via spontaneous respiration until the pleural cavity was entered, and then controlled ventilation was instituted. At this time, the involved

* Fellow, Department of Anesthesiology.
† Associate Professor of Anesthesiology.
‡ Assistant Professor of Anesthesiology.
§ Assistant Professor of Radiology.

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Address reprint requests to Dr. Spielman: Department of Anesthesiology, University of North Carolina, Chapel Hill, North Carolina 27514.

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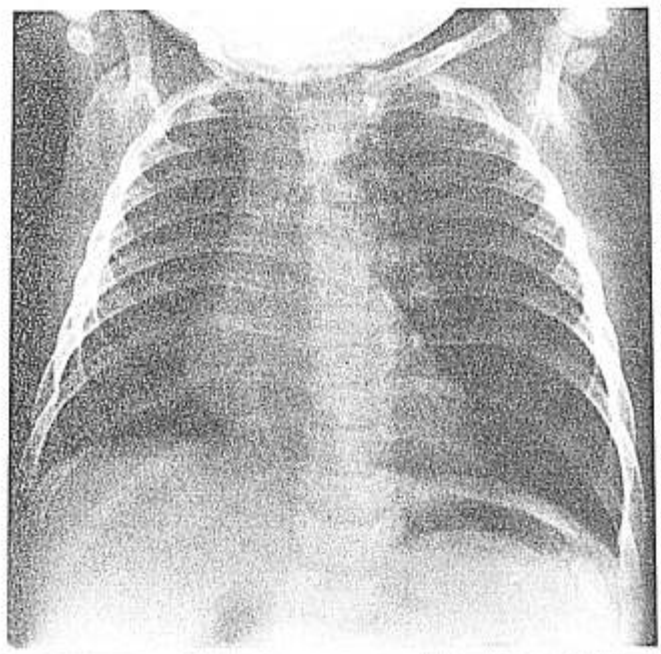

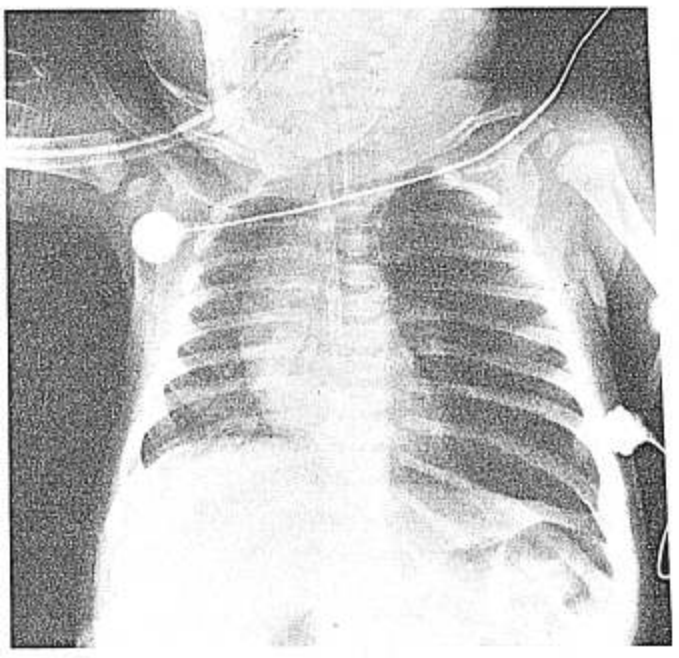

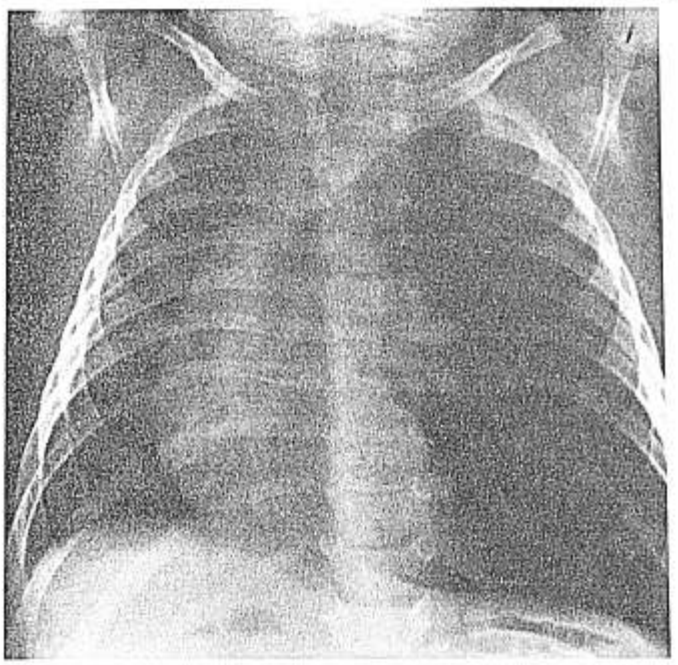

FIG. 1 (*top left*). The preoperative roentgenogram which was originally interpreted as normal shows a slight mediastinal shift to the right due to early air trapping. Early hyperlucency, diminished vascularity, and widened rib spaces of the left thorax are more subtle findings. FIG. 2 (*top right*). The intraoperative roentgenogram reveals a marked mediastinal shift, obvious hyperlucency of the left lung, and flattening of the left hemi-diaphragm due to rapid progressive air trapping during ventilation. The endotracheal tube is in the correct position. FIG. 3 (*bottom left*). This emergency roentgenogram taken three hours after the first operation shows massive air trapping with herniation of the left lung across the anterior mediastinum and further mediastinal shift.

lung was not hyperinflated but actually was somewhat atelectatic. The lung also did not hyperinflate with controlled ventilation. Gross inspection of the left main bronchus revealed an infiltrating mass through the cartilagenous wall of the bronchus with approximately two thirds of the mass outside the wall and one third inside. The surgeons deemed the tumor to be locally unresectable and therefore performed a pneumonectomy. Subsequent microscopic examination of the tumor led to the diagnosis of zygomycosis for which the patient received a 30-day course of amphotericin. The child was discharged in good health two months after surgery.

DISCUSSION

Causes of bronchial obstruction with secondary lobar emphysema from a functioning ball-valve type of obstruction include: 1) foreign bodies; 2) mucosal flaps; 3) local inflammation; 4) mucous plugs; 5) luminal adenomas; 6) softened bronchial cartilages; 7) anatomically narrowed bronchi; 8) kinking of the bronchus of a herniated lobe; and 9) external compression by vessels or tissue masses.[2-4] Although the latter mechanism appeared to play the major role in our patient, common to all these causes is the development of a lesion that allows gas to flow by it during inspiration but inhibits gas flow by it during expiration. As a result, the pulmonary tissue distal to the obstruction gradually becomes hyperinflated and encroaches on adjacent chest structures.

In our patient, the development of obstruction and

lobar emphysema probably was more apt to occur because of the presence of age-related anatomic factors. In the infant, the soft, pliable, bronchial cartilages are more readily deformed and the pores of Kohn are less developed and provide less opportunity for collateral ventilation than in the adult.[5] Also, because flow of gas through the airways is directly proportional to the fourth power of the radius, only slight degrees of narrowing of the small bronchi of infants are necessary for obstruction to occur.

The intraoperative signs of airway obstruction in our patient may have been related to the combined effects of anesthetic-induced relaxation of airway-supporting muscles, paralysis of skeletal muscles, use of controlled ventilation and nitrous oxide. In the normal tracheobronchial tree, smooth muscles in the airway contract during expiration and thereby narrow the bronchioles and stiffen the large bronchi. If these muscles were relaxed, increased compressibility of the large airways would occur. Similarly, skeletal muscle paralysis would enhance the development of a more positive intrapleural pressure and thereby increase compressibility of the airways. In such a situation, controlled ventilation might enhance air trapping by forcing gas by the obstruction and by removing forces promoting the escape of the gas during expiration. Because of differences in the solubilities of nitrous oxide and nitrogen, the use of nitrous oxide also would increase hyperinflation as long as perfusion to the obstructed lung tissue remained intact and blood-alveolar gradients for nitrous oxide and nitrogen existed.[6]

During the second anesthetic, controlled ventilation and nitrous oxide were avoided because of the fear of inflating the lung even further. Therefore, we were surprised, once the lung was exposed, to find the involved lung somewhat atelectatic and unaffected by the institution of controlled ventilation. Apparently, the bronchus had become totally obstructed and absorption atelectasis had started to occur.

In addition to the clinical signs of obstructive emphysema, roentgenologic examination often offers the best means for making the diagnosis. In such a situation, the roentgenographic findings may include: 1) air trapping with hyperlucency of the involved lung; 2) atelectasis of adjacent lung; 3) a shift of the mediastinum to the opposite side; 4) herniation of the affected lobe to the opposite side; 5) compression of the contralateral lung; and 6) depression of the diaphragm on the affected side.[7] If all of these roentgenographic findings are marked, the diagnosis is usually quite evident; however, when subtle, the condition may not be appreciated. Such was the case with our patient, since the rib space widening and hyperlucency were not appreciated (fig. 1) and the mediastinal shift was attributed to the patient's slight rotation. Had the possibility of air trapping been entertained on the basis of the plain films, it could have been confirmed with fluoroscopic evaluation. This would have allowed for a more adequate preoperative diagnosis.

In conclusion, the case described emphasizes the importance of anesthetic techniques in furthering the development of obstructive emphysema. When a functioning one-way valve mechanism is unsuspected preoperatively, prompt diagnosis and therapy are necessary to avoid severe respiratory and cardiovascular deterioration.

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