Tracheoesophageal Cleft

A. V. Budris, M.D.,* M. Lippmann, M.D.,* and P. H. Lorhan, M.D.*

The increasing awareness of pediatric congenital anomalies and the early recognition of symptoms are bringing more of these very young patients to surgery. Haight [1] reported that 3 per cent of 500 such cases reviewed had a simple tracheoesophageal fistula. The anesthesiologist is being confronted with these difficult airway problems, the most common of which is a simple tracheoesophageal fistula in over 80 per cent of the cases. He is usually presented with a debilitated newborn with a respiratory infection requiring surgical procedure which will compromise his ventilation. It becomes of prime importance that a near optimal physiologic status would be desirable to decrease the morbidity associated with this repair. A useful index as when to attempt this repair would be evidence of thriving and respiratory improvement. In 1965, Blumberg, *et al.*[2] reviewed the literature on laryngotracheoesophageal clefts and found that 11 of 12 reported cases had died. Four of these patients had undergone surgical repair. Only one child was reported living at an age of 2 years. They noted that these cases were associated with airway problems during surgical management.

Case Report

A 25 day old Caucasian female underwent surgical repair of a complete tracheoesophageal cleft. On July 24, 1966 the child was delivered spontaneously under pudendal block. Newborn physical examination revealed: Left facial paralysis; left hypoplastic ear; and left incomplete external ear canal. A nasogastric tube was passed freely into the stomach and no material was returned with aspiration. It was noted that the child handled its secretions very poorly. An upper respiratory anomaly was suspected.

After several days of oral feedings and close observation it was noted that feeding was associated with respiratory distress. The otolaryngologist called in consultation visualized the glottic area and described a forked epiglottis, normal functioning vocal cords and a small mass between the arytenoids. A perovisional diagnosis of epiglottic malacia was made. On July 28, 1966 radiological evidence of pneumonitis was noted. A feeding gastrostomy was inserted on August 4, 1966 under local anesthesia because of persistent respiratory distress with oral feedings. Reported roentgen examination of the lung fields revealed increasing pneumonitis. Hypaque swallows and cine-esophagram studies on August 11, 1966 failed to reveal a tracheoesophageal fistula, but the cine-esophagram suggested the presence of achalasia. Feedings via the gastrostomy were tolerated well and the chest was noted to clear clinically and radiographically with only residual evidence of pneumonia in the right upper lobe.

Between August 16 and August 17 the infant had three apneic episodes, the last being accompanied by cardiac arrest, which was successfully treated with external cardiac massage. The otolaryngologist re-examined the entire airway and noted that a complete communication existed between the trachea and the esophagus from the arytenoid cartilages to the carina. A tracheostomy was performed for ventilation and tracheal toilet.

On August 18, the infant was taken to surgery, classified as physical status E-4, with a hemoglobin of 10.7 g. and white blood count 29,000. No preoperative medications were given and an intravenous cutdown was inserted into the left greater saphenous vein. The infant was monitored by cardioscope, rectal thermistor and precordial stethoscope. Normal body temperature was maintained with a Gaymor water mattress. Induction was performed by allowing a 5 liter flow of 1.5 per cent halothane in oxygen to flow over the tracheal opening while surgical preparation was in process.

At the onset of the surgical procedure, the surgeons removed the tracheostomy tube and inserted a no. 18 Cole endotracheal tube into the tracheostomy. The endotracheal tube was then connected to sterile infant corrugated tubes of a Bloomquist apparatus. An anesthesiologist was scrubbed and gowned in the surgical field. In this position, he was able to maintain the proper position of the Cole tube and regulate the relief valve of the Bloomquist circle. This required close observation of the breathing bag and rapid responses to the commands of the anesthesiologist controlling the gas flow.

The surgical procedure consisted of two stages. The first stage was a right lateral neck approach through which closure of the communication was performed as far as possible into the mediastinum.

* Department of Anesthesiology, Harbor General Hospital, Torrance, California and the University of California Medical Center, Los Angeles, California.

The second stage was a right lateral thoracic approach for completion of the separation and repair. Throughout the surgical procedure the patient was maintained in a light plane of anesthesia with intermittent flows of 0.5 to 1 per cent halothane. Depth of anesthesia was determined by cardiac rate, muscle movement, respiratory rate and resistance. This required constant communication between the two anesthesiologists for ensuring manipulation of the gas flow rate, venting and adjustment of anesthetic concentrations. On several occasions, airway control was poorly maintained because of surgical manipulation. Attempts were made to anticipate airway pressure loss with prior hyperventilation and secondarily with high gas flow rates. At times, maximal gas flow rates of eleven liters per minute were necessary to compensate for gas stream divergence. Through such management the vital signs were stable and the infant tolerated the surgical procedure well. At the end of the operation, which lasted 9½ hours, the patient was doing well. When the Cole tube was replaced with a tracheostomy tube by the surgeon it was noted that the patient developed respiratory difficulty from which she very rapidly expired. The cause of death was attributed to cardiorespiratory failure. Post-mortem examination revealed a flap of tissue between the trachea and the esophagus which was not sutured. The Cole tube had splinted this area thereby allowing assisted ventilation but with insertion of the tracheostomy tube the air stream was diverted and accounted for the respiratory distress.

Comment

The importance of an anesthesiologist in the surgical field was well demonstrated by the successful completion of the surgical repair and the close attention afforded by the presence of two anesthesiologists allowed the surgical team to proceed with expediency. Because of the nature of the congenital defect, a prolonged surgical procedure was anticipated and the factors increasing the morbidity and mortality were carefully considered. Dehydration was avoided by maintaining a high flow rate of balanced electrolytes in dextrose and water. This was necessary to compensate for the drying effect of the high flow rates of gases during ventilation and the operative exposure. Blood was replaced as the loss occurred. The anesthesia was controlled to maintain the patient in light planes of surgical anesthsesia in order to avoid cardiovascular depression and prolonged post-anesthetic recovery. The body core temperature was kept at physiological levels to hasten recovery. The success of this management was demonstrated in the stability of the vital signs during the protracted course of anesthesia and by the rapid vigorous arousal on termination of the anesthetic.

The cause of death was explained by the post-mortem findings. During the course of surgical repair, the Cole tube was used as a splint to repair the posterior wall of the trachea. A small flap of tissue was not sutured. Because of the splinting, an airway leak was not noted. Upon extubation the unsutured flap permitted communication with the mediastinum and thoracic cavity. In retrospect this might have been recognized by an increased flow of gas into the water-seal bottles. Awareness of this possibility and a slow withdrawal of the Cole tube may have demonstrated this defect at the time of insertion of the tracheostomy tube.

References

1. Haight, C.: Congenital tracheoesophageal fistula without esophageal atresia, J. Thor. Surg. 17: 600, 1948.
2. Blumberg, J. B., Stevenson, J. K., Lemere, R. J., and Boyden, E. A.: Laryngotrachealesophageal cleft, the embryologic implications, Review of the literature, Surgery 57: 559, 1965.