THE SURGICAL TREATMENT OF CONGENITAL TRACHEO-ESOPHAGEAL FISTULA IN THE NEW-BORN

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CONGENITAL malformations of the esophagus are of various types, but fortunately these anomalies are not common. They occur, however, with a greater frequency than is commonly thought, as evidenced by our experience, having seen six cases during 15 months. Brenneman[1] reports the observation of five cases in five years. We believe that as one becomes familiar with the clinical manifestations of these lesions, the incidence will increase. There is no doubt that a large number of cases that supposedly die of birth injuries, especially in those with cyanosis, atelectasis, or postdelivery pneumonia, are cases of undiagnosed congenital tracheo-esophageal fistula.

Although tracheo-esophageal fistulae are the most frequent congenital anomalies of the esophagus, there are many other embryologic defects. Strong and Cummings[2] suggest the following classification of esophageal anomalies:

(A) Involvement of the entire passage
  (1) Stenosis
  (2) Total atresia (reduced throughout to a solid cord)
  (3) Agenesia (no detectable vestige of the structure)
  (4) Doubling of the whole length of the passage.

(B) Regional involvements
  (1) Stenosis (single or multiple stenotic intervals)
  (2) Atresia (either by the narrowing of an interval or by the presence of a barrier fold, or membrane in an esophagus otherwise normal)
  (3) Agenesia (no detectable remnant of a restricted interval)
  (4) Fistula communicating with trachea or a primary bronchus
    (*a*) Esophagus otherwise normal
    (*b*) Esophagus divided into upper and lower segment by a regional stenosis, atresia or agenesia, the fistula being usually single and uniting the lower segment with the trachea
  (5) Doubling in a short extent
  (6) Diverticula
  (7) Cysts

The Group B.4b anomalies are the most frequently encountered. It is these that will be considered in the present discussion. The lesion consists of stenosis, atresia, or agenesia of the esophagus, usually the formation of

a blind pouch at the distal end of its proximal segment and a fistula between the trachea and the distal esophageal segment.

According to Mackenzie,[3] the first recorded case was by Durston, in 1670. He reviewed 62 cases of malformations of the esophagus, of which group 40 (66.1 per cent) had atresia, with the lower end of the esophagus joining the trachea just proximal to the bifurcation. Plass,[4] in 1919, reported a review of 136 collected cases of esophageal atresia with tracheo-esophageal fistulae. In 1926, Hacker and Lotheissen[5] reported 223 cases of malformation of the esophagus, of which 173 (77 per cent) had a communication between the lower segment of the esophagus and the trachea. In 1931, Rosenthal[6] reported 255 cases of congenital esophageal atresia. In 215 (84.4 per cent) there were tracheo-esophageal fistulae. From the above statistics it is evident that the most frequent congenital malformation of the esophagus is an atresia with a tracheo-esophageal fistula. The most common type is that in which there is a segmental agenesia resulting in the formation of a blind esophageal pouch at the distal end of the upper segment and a fistula between the lower end of the trachea and the proximal end of the distal segment of the esophagus which is otherwise normal.

The genesis of this unusual anomaly is explained by a number of theories, some of which are fanciful, while others certainly are based on embryologic dysfunctions that are responsible in part or in whole for the formation of tracheo-esophageal fistulae.

The esophagus and trachea are developed from the primitive foregut. In the young embryo (3 Mm. to 4 Mm.) they are one tube which subsequently becomes divided by the tracheo-esophageal septum derived from a longitudinal projection from each lateral wall, which meet in the midportion of the primitive foregut (Fig. 1). Early in the development the trachea is only partially separated from the esophagus. The anomalous development of this septum that separates the trachea from the esophagus, forming two distinct tubes, is responsible for tracheo-esophageal fistulae. The lung bud anlage is the ventral outgrowth from the primitive foregut at the site of the future tracheal bifurcation, disturbances in the development of which may account for incomplete septal closures in the distal portion.

Luschka[7] and Klebs[8] believe that intra-uterine fetal infection or trauma are responsible for these defects. It has been suggested that normally in the development of the esophagus its lumen is patent up to approximately five weeks. Following this, however, the esophageal lumen becomes obliterated as the result of excessive epithelial proliferation, the lumen subsequently being reestablished by a process of vacuolization. Kreuter[9] suggests esophageal atresia results when vacuolization does not occur. Schridde,[10] however, in the study of 50 human embryos, found a normal lumen in the esophagus at all stages. Forssner[11] demonstrated that the separation of the trachea from the esophagus occurred at an early stage (4 Mm. to 5 Mm. embryo), whereas the epithelial obliteration of the lumen of the esophagus does not occur until the embryo is 20 Mm. in length. Streeter[12] and Rosen-

thal[6] suggest that there is a great similarity between the two developmental anomalies, spina bifida and tracheo-esophageal fistula. The latter author states in his conclusions: "The development of the anomaly seems to rest on an early fundamental change in the entodermal cells that are to give rise to the esophagus, and not on primary concomitant abnormalities. This change may be genetic and related to the anterior end of the neurenteric canal." Strong and Cummings[2] state that "the agencies predisposing to the anomaly are to be sought either in the reproductive cells from which the child was produced or in an unfavorable intra-uterine environment obtaining during the critical early period of embryonic life." They believe it is entirely a growth deficiency occurring in the two lateral intralumenal entodermal projections, the anlage of the tracheo-esophageal septum. According to Lateiner,[13] there is in addition to the lateral mesenchymal ridges which divide

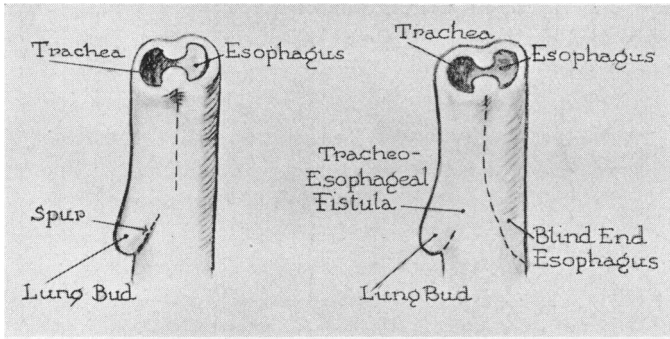

FIG. 1.—Drawing showing the spur formation of the lung bud which normally meets with the two mesenchymal ridges, separating esophagus from trachea. It is failure of this spur to meet the two lateral mesenchymal ridges which causes the spur to attach itself to the posterior wall of the esophagus, thus creating an esophageal atresia and agenesia with tracheo-esophageal fistula. (After Lateiner[13].)

the foregut into the trachea and esophagus a spur formation from the lung bud anlage, which spur grows upward into the foregut, meeting the two lateral ridges. He believes that when there is failure of the spur to join the lateral ridges and a fusion of the septum with the posterior wall of the foregut, the typical lesion results; *i.e.,* atresia of the proximal esophageal segment and fistulous communication between the trachea and the distal esophageal segment (Fig. 1). This abnormality is also dependent upon growth deficiency and the entire abnormal process may be expressed by Rienhoff's[14] law: "There is in the life cycle of a cell or tissue, which is differentiating or growing, a period in which the cell is very sensitive to its environment and any injury of it at this stage will result in the abnormal growth and development of that cell or structure." We ascribe to the theory of Lateiner[13] as being the most plausible. For a more detailed description of the different theories, the reader is referred to the excellent papers of Plass,[4] Hacker and Lotheissen,[5] Rosenthal,[6] and Strong and Cummings.[2]

The gross pathologic picture is remarkably constant in cases seen at

autopsy. In over 80 per cent of the cases there is an atresia with agenesia of the esophagus, dividing the esophagus into two distinct parts with an upper segment which terminates in a blind pouch about 10 to 12 cm. from the alveolar region. The pharyngeal part of the upper segment is normal. The end of the proximal segment is on the average 3.4 cm. below the larynx (Plass[4]). The distal esophageal segment, as a rule, is normal except at its proximal end where it is narrow and forms a fistulous communication with the trachea and rarely with one of the main primary bronchi (usually the right). The average distance of the fistulous opening above the bifurcation of the trachea is 0.5 cm. (Plass[4] and Rosenthal[6]). In most cases the upper blind pouch is entirely separated from the lower segment (agenesia), but occasionally there may be a fibrous cord or muscle band connecting the distal limb of the upper segment to the proximal end of the lower segment (Fig. 5). The tracheo-esophageal fistula is situated just proximal to the bifurcation and consists of a small, slitlike opening, resembling the mouth of a small bronchus (Fig. 8). Bronchoscopically, the fistula appears as the opening of a bronchus. The fistula is so formed that above it the posterior wall of the trachea is depressed, forming a gutter extending down to the fistula, the lower edge of which extends into the lumen of the trachea, producing a valve trap. This results in deviation of secretions and fluids from the trachea into the fistula.

The lungs reveal varying degrees of pneumonia and atelectasis due invariably to the aspiration of fluids and food into the tracheobronchial tree from an esophageal overflow. There is a high incidence of concomitant congenital abnormalities of other organs (Kelly,[15] 60 per cent; Hacker and Lotheissen,[5] 38 per cent; and Plass,[4] 62 per cent). The most common associated anomalies are (1) those in the lower rectum and anus, atresia ani being the most common (Plass,[4] 25.5 per cent), and (2) those in the genito-urinary tract; *i.e.,* horse-shoe kidney, unilateral kidney, double and single ureters with and without stricture. Atresia duodeni, harelip, and other anomalies are rare. However, the majority of associated anomalies are not incompatible with life, and, therefore, they in themselves are no contra-indication to the treatment of tracheo-esophageal fistula.

The symptomatology of esophageal atresia and tracheo-esophageal fistula is so constant that the lesion should be easily recognized. The symptoms can be divided into two main groups: (1) gastro-intestinal, and (2) pulmonary. The infant at birth is well developed and appears normal except for an excessive amount of mucus in the mouth, which is probably due to its accumulation in the blind esophageal pouch and emptying into the pharynx. At times the secretion is so abundant that it interferes with breathing and attacks of cyanosis occur. These symptoms result from filling and emptying of the blind pouch of the proximal segment of the esophagus. The abdomen rapidly becomes distended by the ingress of large quantities of air from the trachea through the fistula into the stomach and small intestine. This is determined both clinically and by roentgenograms (Figs. 3, and 7). The most classic

symptoms are referable to the pulmonary system and occur dramatically at each feeding. At the first attempts at feeding, nothing happens for a few seconds, but soon a definite train of events occur. There is a cough, fluid which is often frothy regurgitates through mouth and nose, and early cyanosis occurs, and more fluid is projected through the nose and mouth as the blind pouch of the esophagus and bronchial tree is emptied of its fluid and food. Almost lifeless relaxation occurs, the cyanosis disappears, and the infant recovers to go through the identical process at the next feeding. It is simply a picture of pulmonary aspiration of fluids which may produce drowning at the first feeding, or, as more often happens, in a few days the infant succumbs to an aspiration pneumonia. These symptoms are due to emptying by reverse peristalsis of the contents of the filled blind esophageal pouch into the hypopharynx and the subsequent spilling into the trachea, some of the fluid passing into the lower segment, then into the stomach by way of the tracheo-esophageal fistula. However, most of the fluid enters the tracheobronchial tree, resulting in suffocation and occasionally death. There naturally follow rapid loss in weight and dehydration due to inability to take fluids and nourishment.

The diagnosis is not difficult if the condition is only considered. However, the majority of cases are never diagnosed until the typical symptoms appear at the first feeding, which is undesirable because the hopelessness of these cases in the past has been due to the early onset of aspiration pneumonia. We believe that the diagnosis should be made at birth. A catheter should be passed gently into the esophagus routinely in all new-borns. In this way a diagnosis can be made before there is any aspiration of the mouth secretions. If a new-born has an excessive flow of mucus from the mouth and the abdomen becomes rapidly distended (a valuable diagnostic sign, according to Brennemann,[1]) the upper esophagus should be immediately investigated. In such cases, we suggest that a catheter be passed into the esophagus and if an obstruction is met one can almost with certainty diagnose the presence of an esophageal atresia. If the catheter meets an obstruction from 10 to 12 cm. from the alveolar ridge then lipiodol should be injected into the esophagus and a roentgenogram taken. This should include the thorax and abdomen. If esophageal atresia and tracheo-esophageal fistula are present, the blind pouch is outlined by the lipiodol. There is usually some spilling of lipiodol into the trachea and lung; and the stomach and small intestine will be filled with gas (Fig. 3). We cannot overemphasize the necessity of using lipiodol instead of barium, as the latter is contraindicated due to its irritating action on the lung parenchyma. If this routine as suggested is followed, then, and not until then, will the surgeon secure the infant free of pulmonary complications.

The treatment of this unfortunate anomaly is entirely surgical. The ideal operation would be the separation of the esophagus from the trachea and an end-to-end anastomosis of the upper to the lower segment. Such an extensive intrathoracic procedure is not justified in a new-born infant, however, and the operation would always be finished as a postmortem procedure. There are two definite surgical indications: (1) closure of the fistula, and

(2) an avenue (gastrostomy) through which the infant can be nourished. Richter,[16] in 1913, suggested and performed the first intrathoracic ligation of the proximal end of the distal segment of the esophagus to close the fistula. He reported two cases that terminated unsuccessfully. We believe that this transpleural ligation is too formidable for an infant two to three days old, and have not used the procedure. We believe that the simplest procedure should be used. Therefore, we recommend intra-abdominal ligation of the cardiac end of the esophagus with umbilical tape to prevent regurgitation into the lung of gastric contents through the tracheo-esophageal fistula, and the formation of a gastrostomy of the Ssabanejew-Frank[17] type.

The technic of the operative procedure is as follows: Through an upper left rectus paramedian incision the peritoneum is opened and the stomach

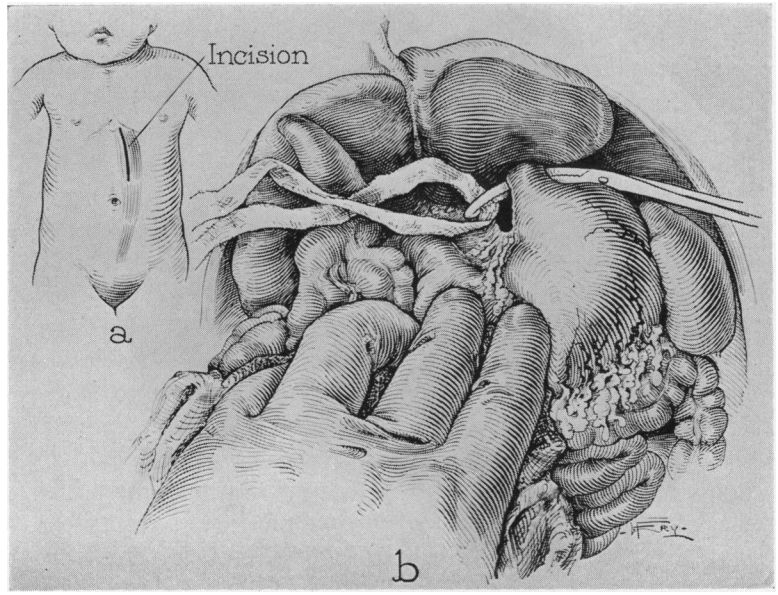

FIG. 2.—Drawing demonstrating the left rectus incision and the method of adjusting the umbilical tape ligature around the cardiac end of the esophagus.

grasped gently by "gauze covered fingers" of the left hand and retracted downward. The left lobe of the liver is elevated upward out of the field by a moist gauze covered retractor. The stomach is then depressed downward and backward. This brings the cardiac end of the esophagus to view just beneath the diaphragm (Fig. 2). A right handed aneurysm needle threaded with catgut is gently passed around the esophagus, the catgut grasped and tied to a ligature of umbilical tape. The needle is left *in situ*; the catgut is removed from right to left, pulling the tape behind the esophagus. This tape ligature is gently but firmly tied, which obliterates the lumen of the esophagus. A curved forcep can be used to pass behind the esophagus and pull the ligature of tape around the esophagus (Fig. 2). However, we recommend the aneurysm needle, as the procedure can be accomplished with

greater rapidity. The vagi are incorporated in this ligature. This cannot be avoided, but we feel that there will result slight if any deviation from the normal physiologic activity of the gastro-intestinal tract.

A gastrostomy is now formed according to the technic of Ssabanejew-Frank as follows: The stomach is grasped with Allis forceps near the greater curvature close to the cardia and by pulling on the Allis forceps a pouch of the anterior gastric wall is formed. This pouch is now pulled under, or over the rectus muscle through a tunnel in the tissues, making its exit through a secondary incision about 3 cm. from the left rectus incision. The pouch is attached to the skin of the secondary incision and the left rectus incision is closed with silk. The tip of the pouch is now cut off and a catheter can be passed easily into the stomach. The advantages of this form of gastrostomy are many: the procedure is rapidly performed, there is a tube lined with epithelium which prevents stricture of opening, and with pressure over the tunnel through which the pouch passes spilling of gastric contents between feedings does not occur. We believe these two procedures are the simplest that can be performed and yet accomplish the desired results, and both should not take over 20 to 25 minutes in their execution.

The postoperative treatment consists of administering fluids and food by the gastrostomy. Fluids should also be given by hypodermoclysis. The upper blind pouch of the esophagus should be aspirated frequently to prevent overflow into the trachea. If this simple procedure is not done aspiration pneumonia may result. The lower blind pouch created by the operation will probably take care of itself for a while, but will ultimately have to be treated by the intratracheal route. We believe that as soon as possible the upper segment should be removed to the outside of the neck as a preliminary to a future plastic operation to form an anterior thoracic esophagus. The lower segment as well as the fistula opening must be obliterated to prevent infection, because this infected material will be aspirated up into the tracheobronchial tree, producing a pneumonitis.

For a successful outcome in these cases the emergency treatment as well as the future formation of a new esophagus depends entirely upon the cooperation of the obstetricians, pediatricians, surgeons, thoracic surgeons, and the bronchoscopists. It is by the combined efforts of the above named specialists that the present mortality of 100 per cent should decline rapidly to a lower level and a few lives may be saved.

CASE REPORTS

Case 1.—Premature white male, born October 22, 1935, at 1:45 A.M. The infant cried immediately after delivery. However, respirations soon became difficult and cyanosis occurred. Cyanosis and difficult respirations were promptly relieved by aspirating mucus from the hypopharynx. These attacks recurred at frequent intervals and were relieved by removal of the excessive mucous secretions. The infant was put to the breast every two hours beginning at 10:00 A.M. the date of birth. It was noted that all attempts at nursing resulted in regurgitation of food through both the mouth and the nose. The infant struggled for air, cyanosis rapidly appeared, and it seemed that death would supervene. However, the

infant finally started breathing normally and the cyanosis disappeared. Due to the repeated attacks of cyanosis the infant was put in the Drinker respirator.

A catheter was passed into the esophagus where it met with an obstruction about 12 cm. from the alveolar margin. Lipiodol was introduced into the upper end of the esophagus and a roentgenogram was made. This revealed a blind esophageal pouch of the upper segment of the esophagus. The stomach and the small intestine were filled with gas (Fig. 3). A diagnosis of esophageal atresia with agenesia and tracheo-esophageal fistula was made.

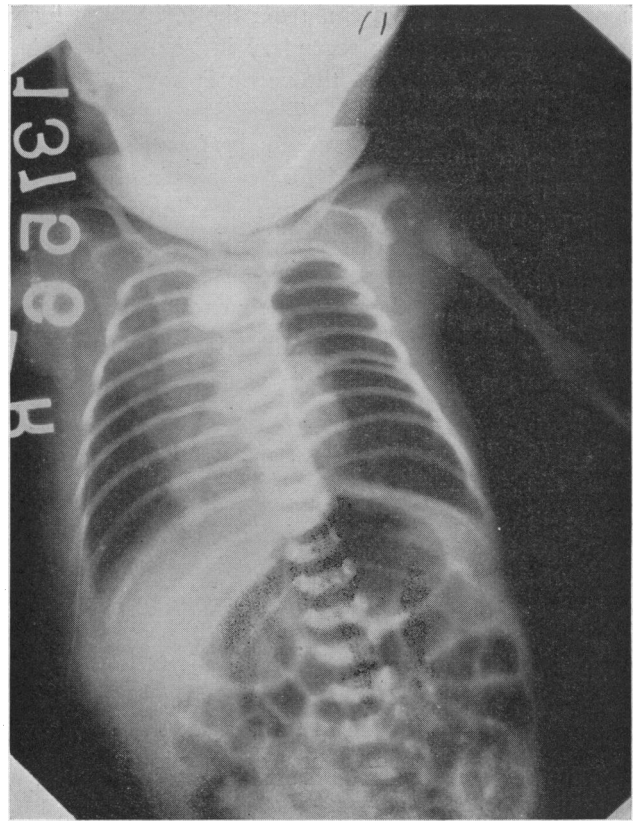

FIG. 3.—Roentgenogram of thorax and abdomen after the esophagus had been injected with lipiodol. The blind pouch of the esophagus is well defined, and the typical gaseous distention of the stomach and small intestine is well depicted (Case 1).

Operation.—Through a high paramedian left rectus incision the cardiac end of the esophagus was ligated with umbilical tape. A simple gastrostomy was performed by the technic of Kaader (Fig. 5). The infant was returned to the Drinker respirator where it remained until October 25 when it died from an aspiration pneumonia.

Autopsy Findings.—Esophageal atresia with agenesia and tracheo-esophageal fistula; bilateral aspiration pneumonia. The following associated congenital anomalies were present: Right kidney replaced by a large cyst; right ureter had a stricture at its lower end (Figs. 4, 5 and 6).

Case 2.—White female infant, born July 6, 1935, full term, weight 6.5 lbs. Physical examination revealed a normal female infant without any external evidence of congenital malformations. The infant continued to react normally until the first feeding, at which

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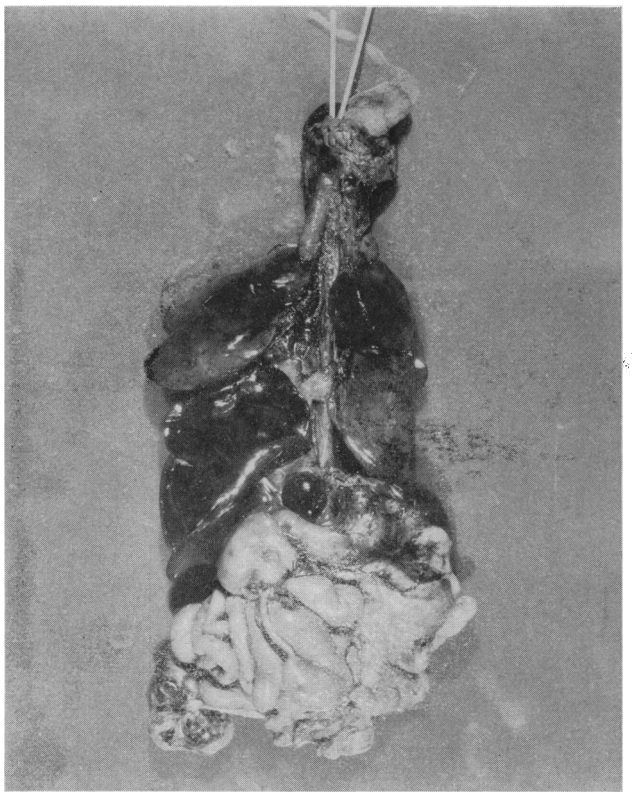

FIG. 4.—Photograph showing the entire viscera (Case 1). The blind pouch of the upper segment of the esophagus is well shown. The lower segment of the esophagus with ligature attached is seen beneath the liver. The gastrostomy tube is shown protruding from the anterior stomach wall.

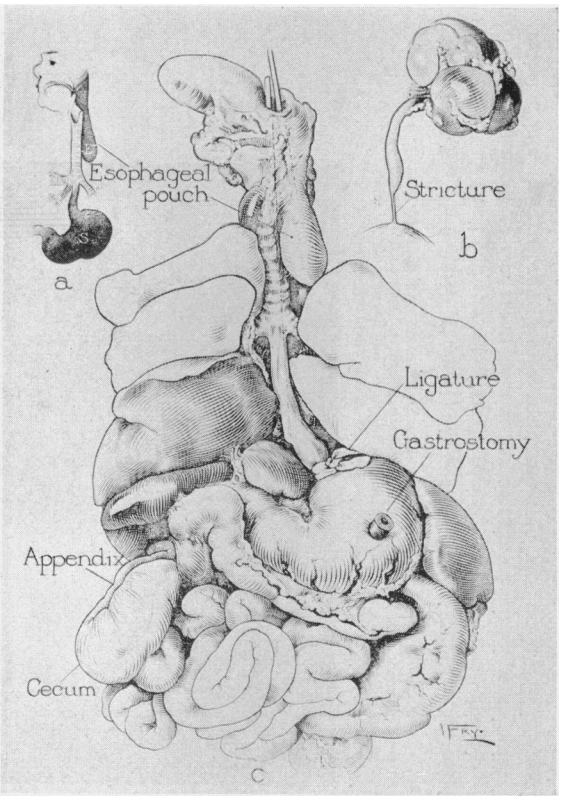

FIG. 5.—Drawing of the gross specimen shown in Fig. 4. The drawing shows: (a) the blind pouch of the esophagus and the tracheo-esophageal fistula; (b) the cyst of the right kidney and the stricture of the lower part of the right ureter; and (c) the anatomic relationships of the entire congenital anomaly with the ligature of umbilical tape around the cardiac end of the lower segment of the esophagus. The gastrostomy tube is seen projecting from the anterior wall of the stomach.

time she took fluid for a few seconds, which was soon regurgitated through both the mouth and the nose. Cyanosis occurred, the infant gasped for breath, the fluid ceased flowing from the nose and the mouth, the cyanosis disappeared, and the infant again appeared normal. This train of clinical signs and symptoms occurred at each feeding.

The infant was admitted to the Touro Infirmary on July 31, 1935, at 12:35 A.M. Examination at this time revealed an infant four days old who was apparently normal with the exception of a slight dehydration and elevation of the rectal temperature to 102° F.

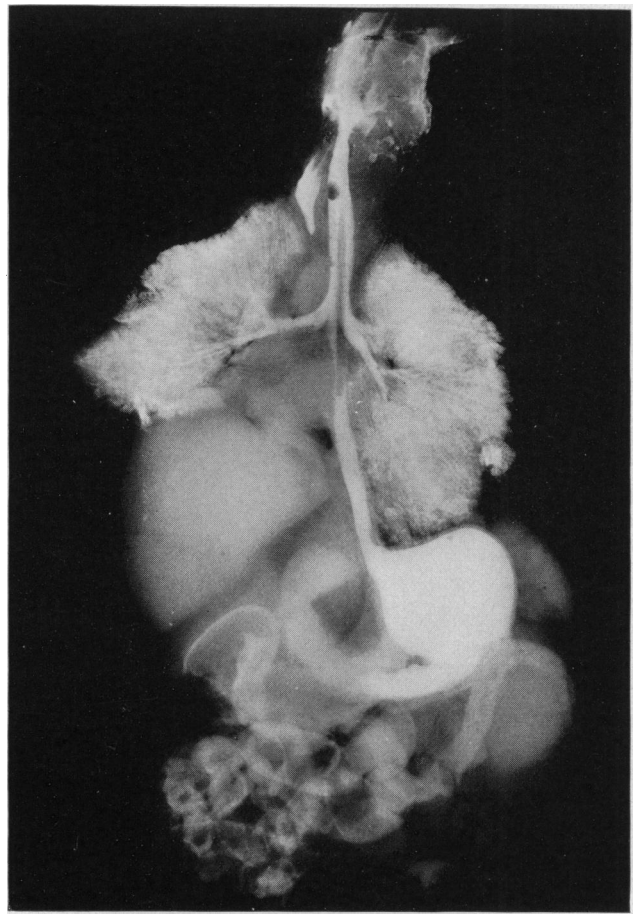

FIG. 6.—Roentgenogram of thoracic and abdominal viscera removed from Case 1, after the injection of barium into the trachea. The lungs, tracheo-esophageal fistula, trachea, esophagus, upper and lower segments, and stomach are visualized.

From the history a tentative diagnosis of esophageal atresia and tracheo-esophageal fistula was made. A small catheter was introduced into the esophagus where it encountered an obstruction about 12 cm. from the alveolar margin. Lipiodol was instilled through the catheter into the esophagus and a roentgenogram was made. This revealed a typical complete esophageal atresia and agenesia of the upper segment. Tracheo-esophageal fistula was also inferred because the stomach and small intestine were filled with gas (Fig. 7).

Operation.—July 31, 1935, under ether anesthesia the cardiac end of the esophagus

was ligated through a high left rectus paramedian incision. A gastrostomy was also performed by the technic of Ssabanejew-Frank.

The infant reacted well from the operation and was given fluids at regular intervals through the gastrostomy tube. On August 1, the infant suddenly became cyanotic and had great difficulty in breathing. The temperature was elevated to 102.8° F. Cyanosis and difficult respirations continued and the infant succumbed at 3:00 A.M. August 2, 1935.

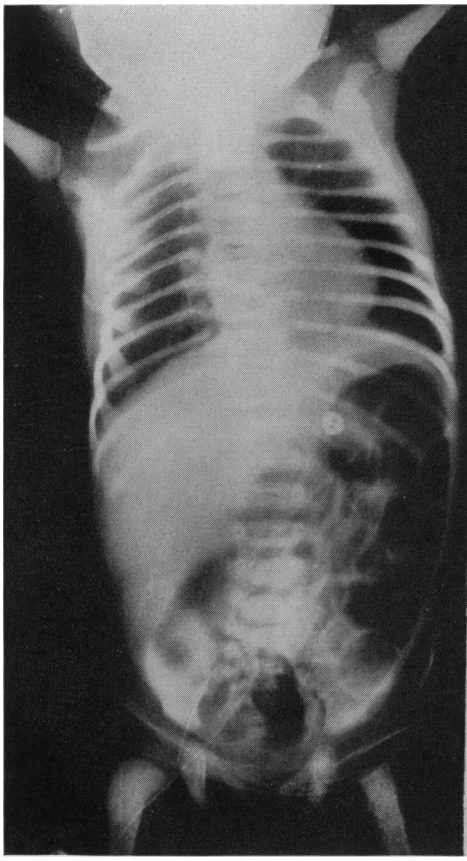

FIG. 7.—Roentgenogram of the thorax and abdomen (Case 2), showing the typical blind pouch of the upper segment of the esophagus in the upper thorax. The typical gaseous distention of both the stomach and the small intestine, so characteristic of tracheo-esophageal fistula, is well outlined.

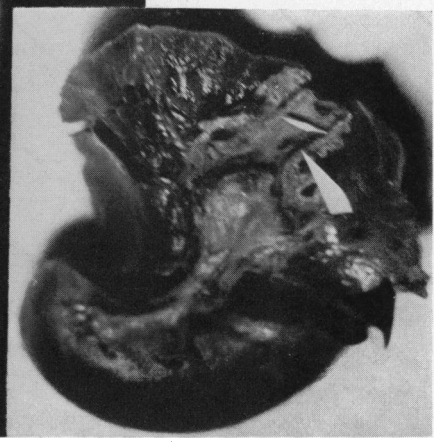

FIG. 8.—Photograph of viscera removed from Case 2, showing tracheo-esophageal fistula opening just above the bifurcation of the trachea. The opening is opposite the point of the white marker.

Autopsy Findings.—Esophageal atresia with agenesia and a tracheo-esophageal fistula with the opening just above the bifurcation of the trachea (Fig. 8). The lungs revealed a bilateral aspiration pneumonia of both upper lobes. A horse-shoe kidney was an associated congenital anomaly.

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DISCUSSION.—DR. URBAN MAES (New Orleans, La.).—I think I can add a small bit of encouragement to the very depressing report Doctor Gage has made, and at least point out a mistake that should not be made. These children usually come to the surgeon only after they have been given barium for roentgenologic examination, which is a serious and usually fatal error. I was discussing the subject a few weeks ago with Doctor Scott of the University of Rochester, and his experience parallels my own in the three cases I have seen personally—that all the children have pneumonia, and that the lung picture always shows a tiny fleck of barium in the center of each patch of pneumonia.

The diagnosis ought to be made in some other way. Usually the clinical history and physical findings are clear, and if they are not, and some visualization is necessary, the use of lipodol is less harmful than the use of barium.

Doctor Scott informed me that one of his patients lived six weeks and one three months after operation, which shows that something can be accomplished.

DR. CHARLES G. MIXTER (Boston, Mass.).—Doctor Gage's paper has been of great interest to me as for many years much of my work was devoted to the surgery of infants and children. He is to be congratulated on developing a technic that bids fair to offer a chance of success in this disastrous anomaly that up to the present time has invariably proved fatal.

He has spoken of the frequency of its occurrence. Our experience at the Children's Hospital in Boston entirely corroborates his statement. In the past ten years 23 instances of tracheo-esophageal fistula have been observed. We have classified esophageal malformations into two main groups, *i.e.*, (1) those in which the esophagus ends blindly and, (2) those in which there is a tracheal fistula. The second group may be subdivided into three classes.

Sixty per cent belong to the class in which the distal segment has a fistulous opening into the trachea, the lower end of the proximal segment ending blindly.

Up to 1929, a palliative gastrostomy was done. This did not accomplish its purpose as all cases invariably died of pneumonia, caused directly by the fistula or indirectly by aspiration of the contents of the blind esophageal segment. The site of the fistula has always been found just above the bifurcation of the trachea. In 1929, I made my first operative attack on the tracheo-esophageal fistula by a posterior approach through the posterior mediastinum on the right side. A considerable extent of two ribs at the level of the bifurcation were excised; by pushing the pleura laterally the region of the fistula was readily exposed. The right azygos vein usually crosses over the fistulous communication and this is dislocated and the opening severed and sutured. The separated distal segment of the esophagus is brought out and implanted in the incision in the back or in a stab wound. A catheter is placed through the esophagus to the stomach for feeding purposes. The first infant lived 57 hours following operation before succumbing to the pneumonia which was present before operation. After several futile attempts, it was found that distal esophagostomy was insufficient, as overflow of secretion from the blind proximal end was followed by a fatal pneumonia. Therefore, proximal esophagostomy through the neck, either at the primary operation or within a few days, was added. Nine operative attempts have been made in the last six years, with and without proximal esophagostomy. All have ended fatally, but in two instances the infants have lived for 15 and 30 days, respectively. It is, of course, a palliative procedure, but it is hoped that, if the infant survives, an esophageal reconstruction can be done later.

DR. VILRAY P. BLAIR (St. Louis, Mo.).—I do not wish to encourage surgical exercises, but Doctor Mixter said something that I think has some value. If you meditate a radical operative attempt, do it at the earliest possible moment and you will have a better chance than on the older child, if you have any chance at all.

DR. MIMS GAGE (closing).—The object of presenting the subject of congenital tracheo-esophageal fistula was to stimulate discussion which may result in a more direct surgical attack on the lesion and thus lower the mortality rate and secure better end results.

Doctor Mixter's experience with these cases is of interest from the standpoint of his operative "transmediastinal approach." However, I do not believe that these patients can withstand any extreme dissection or manipulation. The surgical attack of the fistulous opening at its source is the ideal procedure, as it does not leave behind an esophageal pouch that will become infected and from which regurgitation into the tracheal tree will cause an acute pneumonitis. I agree with Doctor Blair's admonition that an infant can withstand more surgical trauma immediately after birth than it can several days later.

I should like to stress again the importance of keeping the upper esophageal pouch clean, in order to prevent filling and overflow with subsequent aspiration pneumonia. The upper esophagus should be mobilized to the outside as soon as possible. The lower segment must be drained by postural treatment with the head down until intratracheal manipulation, aspiration, and obliteration of the pouch can be accomplished.