CLINICAL ROENTGENOLOGY OF THE DIGESTIVE TRACT

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CLINICAL ROENTGENOLOGY OF THE DIGESTIVE TRACT

Dedicated to my sons

MAURICE FELDMAN, JR., A.B., M.D.

and

CHARLES FELDMAN, A.B.

PREFACE OF THE THIRD EDITION

This book was written to present a clinical roentgenological consideration of the diseases of the gastrointestinal tract. It was written as an aid for the diagnosis of the digestive tract for the roentgenologist, gastroenterologist, student and general practitioner. The need for a book of this type is evidenced by the difficulty one encounters in seeking information on this subject. During the many years in which I have been interested in diagnostic roentgenology of the gastrointestinal tract, I have felt the need of bringing together sources of material which have not heretofore been compiled in a single volume. In collecting the necessary material I have endeavored to cover every phase of the gastrointestinal tract with the object in view of presenting the importance of the diagnostic value of the roentgen examination.

In assembling the material it has not been my intention to mention all of the authors who have contributed to the literature, but only those whose contributions were pertinent to the subject matter. Due credit has been given to each author quoted and to any who have been inadvertently omitted, I wish to express my regrets and credit is here given to them.

In the third edition changes have been made in order to bring the material up to date. Many new chapters, additional information and illustrations have been added.

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CONTENTS

Chapter I

THE ESOPHAGUS

THE PHARYNGO-ESOPHAGUS

In the routine gastrointestinal roentgen examination, the pharyngo-esophageal region is studied. The barium mixture usually passes through this segment rapidly while erect, but may be slowed if the examination is made in the recumbent position. The pharyngo-esophagus is best studied by means of the fluoroscope. If roentgenograms are to be made, the timing with the swallowing action must be carefully observed; otherwise, the segment will be emptied. The patient is given a mouthful of barium and is instructed to swallow, at which time the examination is made. The fluoroscopic examination is begun immediately before the swallowing action.

The caliber of the pharyngo-esophagus, the vallecula and pyriform sinuses, residues, asymmetries and spastic phenomena are noted. Functional disorders affecting the pharyngo-esophagus are common.

ESOPHAGUS

The esophagus is a collapsible, distensible musculo-membranous tube, about 16 inches in length, beginning opposite the upper border of the cricoid cartilage, about 6 inches beyond the incisor teeth and extending from the pharynx to the stomach. It enters the stomach slightly below the diaphragm. The abdominal portion varies in length from 1 to 4 centimeters. The general direction is vertical with three slight curves in its course. The esophagus begins in midline and deviates to the left as far as the root of the neck where it gradually returns toward the midline. In the lower end it again deviates to the left, passing forward to the esophageal orifice in the diaphragm. Its caliber varies slightly; usually it is about 2 centimeters in diameter. It is narrowest at its beginning and where it passes through the diaphragm. The phrenic ampulla is a localized dilatation of the lower end of the esophagus. This is a normal phenomenon, commonly seen in the routine examination. Often it is associated with a cardio-esophageal spasm which narrows the opening and temporarily retards the flow of barium into the stomach.

The esophagus occupies the posterior mediastinum, anterior to the loose tissue of the trachea. It lies to the right and passes in front of the aorta just before it enters the abdomen. The pleura are on either side and the pericardium is in front. Posteriorly, it lies close to the vertebral column.

The esophagus is divided into three portions, the cervical, thoracic and abdominal. Four normal points of narrowing are observed: (1) level of

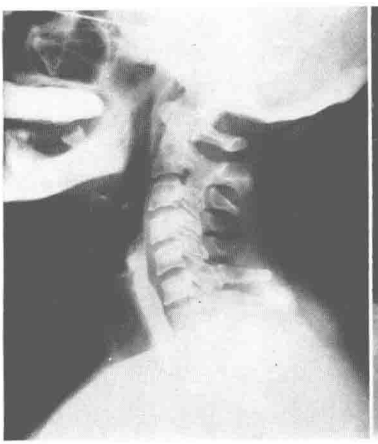

FIG. 1

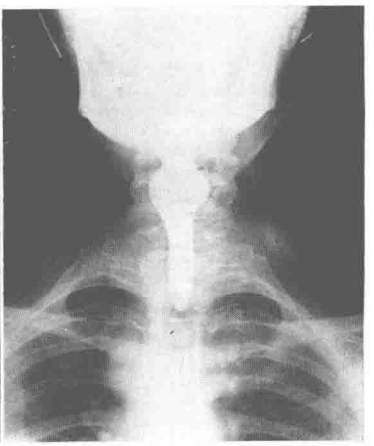

FIG. 2

FIG. 1. Normal filling of the pharyngo-esophagus; lateral view, the normal upper esophagus.

FIG. 2. Antero-posterior view, showing the bulbous appearance of the pharyngo-esophagus.

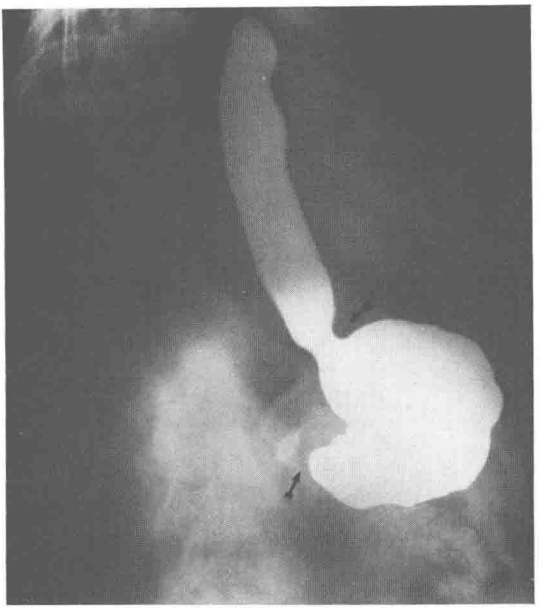

FIG. 3. Complete filling of the esophagus in a case of pyloric obstruction. Arrows point to slight narrowing at the cardioesophageal juncture with an annular filling defect at the pylorus.

the cricoid cartilage, (2) crossing of the aorta, (3) crossing of the left bronchus, (4) at the cardia.

Roentgen diagnosis. The roentgen examination will demonstrate evidence of obstruction or delay in the passage of the opaque meal, defects, contour irregularities, displacement, signs of pressure and spasm. The examination begins with the swallowing of the first intake of the opaque meal and the mechanism is observed under the fluoroscope. Normally one visualizes the rapid transit of barium through the upper portion of the esophagus by gravity and through the lower portion by peristaltic activity. Before entering the stomach, its passage is slowed. Fluids descend in a continuous, quick stream, while semisolids travel slower and the column is usually broken. The detection of peristalsis is difficult because of the

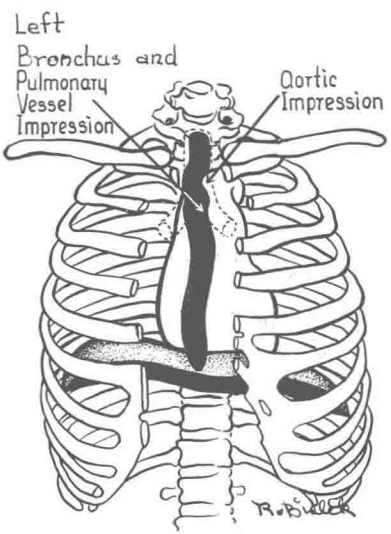

FIG. 4

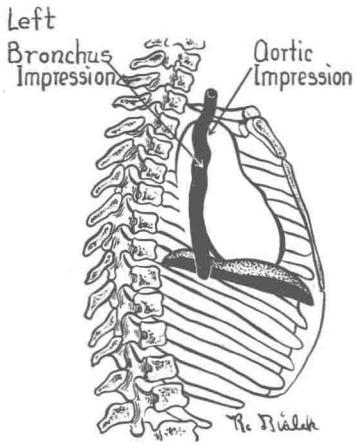

FIG. 5

FIG. 4. The normal esophagus; antero-posterior view, showing the aortic and pulmonary vessel impressions.

FIG. 5. Right oblique view, showing the aortic and the left bronchus impression.

rapidity with which the meal passes down the esophagus. In the roentgen examination, the patient is placed in an erect and recumbent position and is rotated obliquely or laterally. The right recumbent prone position produces a slight upgrade of the esophagus. The advantage of this position is that it slows the passage of the opaque meal and permits a detailed study. Placing the patient in the Trendelenburg position will further retard the meal. The small caliber of the lumen and the rapid transit of the meal affords a relief view of the esophagus. The normal esophagus, especially in the lower third, shows several longitudinal, parallel mucosal folds. Inflammatory changes are evidenced by a marked widening of these mucosal folds. The relief view will often disclose, before any characteristic symptoms are

noted, the early signs of carcinoma, esophagitis, diverticula, peptic ulcer, varicosities and esophageal hernias.

The fluoroscopic examination of the esophagus is the method of choice. Films are made while the patient is drinking the opaque meal so that the filling of the entire esophagus may be demonstrated. Normally, the lumen is well outlined and its contour is usually smooth. The flow of barium is carefully watched, particularly as it approaches the cardia and enters the

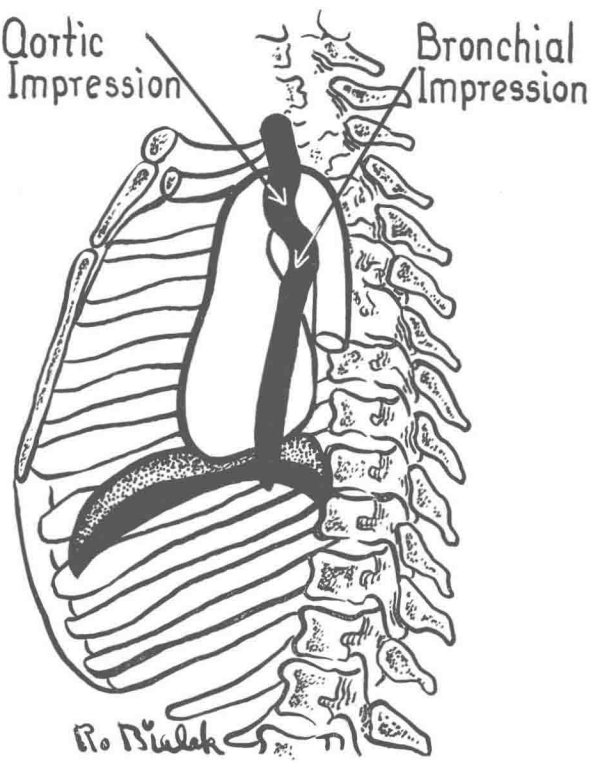

FIG. 6. Left oblique view of esophagus, the aortic and bronchial impression are shown.

stomach. To demonstrate the lower end of the esophagus, the patient is instructed to take deep breaths immediately after swallowing a mouthful of the barium. This procedure temporarily retards the meal at the cardia. The administration of a thick barium paste is often indicated to study the esophagus. A bismuth filled 00-capsule may be given to determine the descent of solids.

The thickness of the barium meal depends upon the type of examination

to be made. For relief views, a thick barium is used; for the demonstration of varices, a thin barium mixture is employed. The rapid descent of the opaque meal makes it necessary to employ short exposures, $\frac{1}{10}$—$\frac{3}{10}$ seconds, in order to obtain a roentgenogram of the upper esophagus.

CLASSIFICATION OF CONGENITAL ANOMALIES OF THE ESOPHAGUS

Congenital anomalies of the esophagus are classified as: (1) absence of the entire esophagus, (2) entire esophagus represented by a solid cord, (3) double esophagus, a. complete doubling of its entire length, b. partial doubling of a short segment, c. bifurcation with reunion at the lower end, (4) atresia with a simple cul-de-sac, (5) atresia with a solid cord between both ends without fistulous communication, (6) atresia with the lower esophagus communicating with the trachea or in rare cases with the bronchus, (7) tracheo-esophageal and tracheo-bronchial fistula, without any other anomaly of the esophagus, (8) congenital stenosis, a. single or multiple stenotic areas, b. strictures caused by folds or membranes, c. pressure from without, (9) congenital dilatation due to spasm, achalasia or paralysis, (10) diverticula, (11) cysts, (12) congenital shortening with thoracic stomach, (13) aberrant tissue.

Congenital absence of the esophagus. Agnesia of the esophagus is an extremely rare condition. Only a few cases have been reported occurring in the human subject. Absence of the entire esophagus occurs only in monsters. Partial absence of a segment of the esophagus is occasionally seen. The esophagus represented by a sold cord is very rare.

Double esophagus. Doubling of the esophagus is exceedingly rare. It occurs as a complete or partial doubling. Partial doubling with bifurcation and reunion at the lower end has been recognized. A rare case of a double esophagus and double stomach was reported by Gjörup.

Esophageal duplication. Congenital esophageal duplications have been described by Ladd and others, are cystic structures arising in the posterior mediastinum. They are usually intimately attached to the esophagus, but in a few reported cases there was no attachment. These duplications are lined with esophageal or gastric mucosa. Ordinarily there is no communication between the esophagus and the cystic structure.

They are usually observed in infancy and childhood. The symptoms are those of compression on the adjacent organs. Dysphagia, cough, dyspnea and cyanosis are the predominant symptoms encountered. The roentgen examination yields evidence of a mediastinal mass simulating a tumor. It displaces the esophagus, mediastinum and heart. The cystic structure is smooth in contour and usually spherical. Occasionally it may erode the vertebra or adjacent ribs. Barium administered on fluoroscopic examination reveal the mass adherent to the esophageal wall.

Aberrant tissue in the esophagus. Aberrant tissue in the esophagus is seldom observed clinically. The incidence of aberrant gastric mucosa in the esophagus varies. Rector and Connerley in 1,000 autopsies found 7.8 per cent of infants and children with aberrant tissue in the esophagus. This congenital condition is not demonstrable in the roentgen examination. The prevalence of complications occurring in this condition has not been fully established. Peptic ulcer of the esophagus may originate in aberrant gastric mucosa.

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ATRESIA OF THE ESOPHAGUS

Atresia of the esophagus is not a frequent congenital anomaly. There are less than 2,000 cases recorded. The incidence varies. The autopsy incidence is approximately 0.03 per cent. Shukowsky and Baron found one case in 50,000 new-born infants. In 3,630 deliveries, Keener and Hickey found 2 cases.

Etiology. Atresia of the esophagus is due to a developmental error of the segment in which the pharynx, trachea and upper portion of the esophagus have a common origin. The esophagus and trachea are represented by a single tube until the second month of intra-uterine life. The normal anlage of the respiratory system is a longitudinal groove on the ventral portion of the anterior archenteron. This is separated from the dorsal portion by two lateral and longitudinal ridges which gradually fuse from below upward, until the ventral respiratory anlage is separated from the dorsal portion, the esophagus. Failure to pinch off completely at any point would leave the pulmonary diverticulum still in communication with the esophagus with the formation of a fistula. In this condition the middle portion of the gullet fails to develop and the pharynx and upper esophagus lead down a short distance into a blind pouch. Then follows a hiatus where no esophagus can be demonstrated, or it is represented by a fibrous cord. Congenital atresia of the esophagus is usually complete.

Atresia of the esophagus may be classified into five forms: (1) simple cul-de-sac, (2) solid cord connecting both ends without fistulous communication with the trachea, (3) the upper end terminating in a simple cul-de-sac, the lower part communicating with the trachea or rarely with the bronchus, (4) simple fistula due to malformation of the tracheo-esophageal septum, the esophagus and trachea being normal except for the fistula, (5) partial atresia.

Atresia with fistula formation are the commonest, occurring in approximately 85 per cent of cases. The position of the atresia end of the upper esophagus is always at the level of the lower portion of the trachea. There is a classic similarity in all cases of atresia of the esophagus with tracheal fistula varying only in minor details. The esophagus ends blindly in a pouch, varying from 3 to 5 centimeters in length. The cul-de-sac is

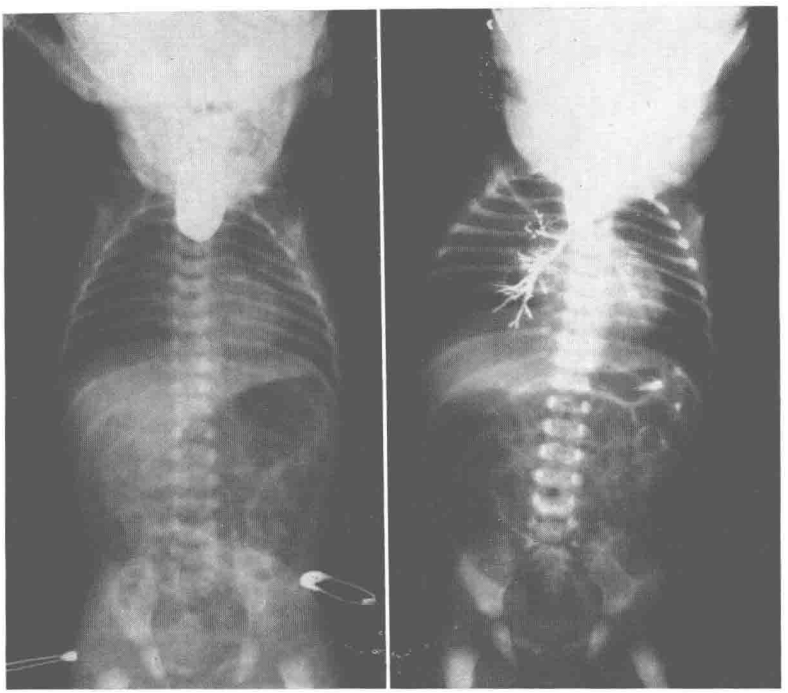

FIG. 7 FIG. 8

FIG. 7. Atresia of the esophagus with tracheo-esophageal fistula. Note the smooth contour of the upper esophagus with the smooth rounded end. Also note the large amount of air in the gastrointestinal tract.

FIG. 8. Same case demonstrating an overflow of the esophagus filling the bronchi. Note the small amount of opaque media in the stomach which entered by way of the fistula.

uniformly dilated. The fistula usually occurs between the trachea and lower esophagus. A fistulous communication with the left bronchus may occur. Rarely, the upper end of the esophagus may communicate with the trachea. Multiple tracheo-esophageal fistulae are exceedingly rare. The fistulous opening may be a small slit-like aperture or a large round opening. Atresia of the esophagus in a simple cul-de-sac, with no vestige of esophageal structure below, is a rare type. The lower part of the esophagus may be

completely absent or represented by a cord. It usually arises as a direct continuation from the posterior aspect of the trachea as a narrow tube, gradually increasing to its normal caliber and opens into the stomach in a normal fashion. In many cases the middle third is represented by a solid cord. The two ends of the esophagus are often united by a fibrous cord. The varying length of the fibrous cord depends on the distance between the ends of the esophagus, which is usually not more than 1 centimeter. The lower portion of the esophagus is usually connected with the trachea about 1 to 2 centimeters above the bifurcation.

Simple tracheo-esophageal fistula without atresia, due to a malformation of the esophago-tracheal septum, is very rare. The formation of a tracheo-esophageal fistula without atresia occurs as a result of an incomplete development of the septum between the trachea and esophagus separating the respiratory from the alimentary tracts.

Partial atresia is very rare. O'Bannon reported a case associated with congenital diverticulum.

Associated anomalies. Multiple congenital defects in the same subject occur in 30 to 50 per cent of cases. Plass in 94 collected cases of atresia found an incidence of associated anomalies in 62.8 per cent; of these, 40.7 per cent showed an atresia of the anus. Meckel's diverticulum and duodenal atresia may be associated with esophageal atresia. In 114 cases of atresia, Ladd and Swenson found the following associated anomalies in 91 cases: atresia or stenosis of small intestine in 7, Meckel's diverticulum 8, malrotation 4, imperforate anus 11, fistula of rectum 4, anomalies of heart and aorta 20, anomalies of urinary system 13 and miscellaneous 24 cases.

Symptoms. The onset of symptoms occur immediately following birth, with spells of choking, cyanosis, especially after feeding. The infant spits up everything taken by mouth. There is progressive loss of weight and emaciation.

Age and sex. Most cases are observed within the first few days of life. The sexes are equally affected.

Heredity. There is usually no history of heredity. Grieve and McDermott reported two male births with atresia in the same family.

Roentgen diagnosis. Roentgen examination will reveal the blind end of the upper esophagus and signs of a fistulous communication between the esophagus and respiratory organs. The smooth blind end is cone shaped, smooth in contour, sacculated and often slightly dilated. The lower esophagus is not demonstrable. It may be fusiform and pointed at its upper end as it enters the posterior aspect of the trachea. There may be narrowing and displacement of the adjacent trachea.

If there is no communication between the upper esophagus and trachea, there will be an overflow of opaque medium into the trachea, outlining the

bronchial tree. If the upper segment of the esophagus communicates with the trachea, the opaque mixture passes directly into the tracheobronchial tract.

Air in the gastrointestinal tract is a definite sign of the presence of a fistula. Solis-Cohen and Levine described a "pencil-line" airway leading from the bifurcation of the trachea to the stomach, which they believe represents air in the lower esophagus. If opaque medium reaches the stomach

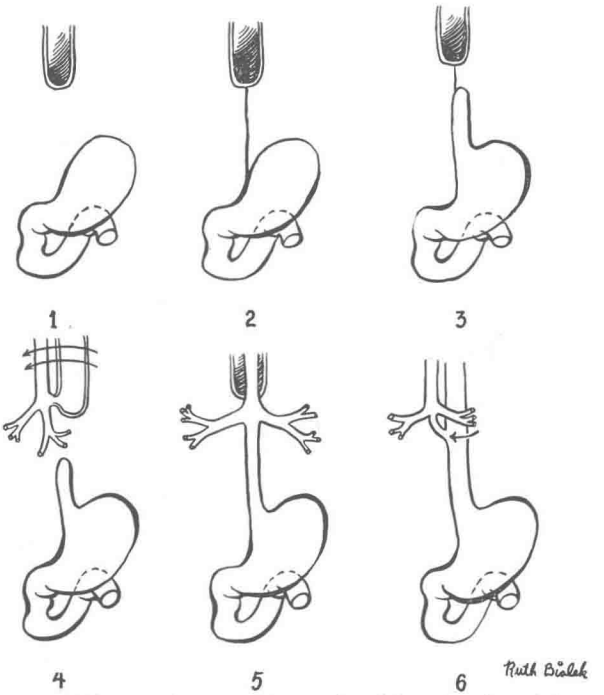

FIG. 9. Atresia of the esophagus; (*1*) atresia with a simple cul-de-sac, no vestige of esophageal structure below, (*2*) atresia with the lower esophagus represented by solid cord, (*3*) atresia with short solid cord connecting both ends of esophagus, (*4*) atresia with upper end communicating with the trachea, (*5*) upper esophagus shown as a simple cul-de-sac. The lower esophagus communicating with the trachea, (*6*) tracheo-esophageal fistula without any other anomaly of the esophagus.

via the respiratory tract, one can be certain of the presence of a fistula. The use of barium as an opaque medium is not advisable. Lipiodol or air may be given through a catheter placed into the cul-de-sac. Usually a small amount of lipiodol, 1 to 2 cc. is required to demonstrate the anomaly.

In the roentgen examination the infant is placed in the prone position. In this position the characteristic sac of the filled upper end of the esophagus is best portrayed. In the lateral position there is often an anterior dis-

placement and narrowing of the trachea, with a pressure defect on the trachea posteriorly.

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CONGENITAL STENOSIS OF THE ESOPHAGUS

Congenital stenosis of the esophagus are classified as: (1) congenital narrowing or stricture, (2) membrane, web, or diaphragm, (3) fold or valve in the mucous membrane, (4) spasm.

Congenital stenosis of the esophagus is an abnormal localized narrowing of the lumen of the esophagus without organic pathology. Congenital strictures are rare. In congenital stenosis there is a history of dysphagia and regurgitation from birth. When there is little narrowing of the esophagus, the majority of infants show no symptoms as long as the diet is fluid. When a semisolid diet is given, the symptoms begin.

Stenosis may occur at any level of the esophagus. The congenital condition is a true fibrous stenosis and not an hypertrophy. There is usually some dilatation above the stricture. The roentgen examination shows the narrowing of the lumen and its smooth contour with varying degrees of obstruction.

STENOSIS DUE TO MEMBRANE, BAND, WEB, FOLD, VALVE OR LIP

Simple stenosis at the entrance of the esophagus and congenital narrowing of the lower part of the hypopharynx or upper part of the esophagus may be produced by a membranous diaphragm, fold or ledge.

Congenital webs or membranes are uncommon. They usually occur in the upper esophagus and produce a dysphagia with varying degrees of stenosis. The x-ray shows an abrupt narrowing of the lumen at the site of the web. According to Paterson, the common site for these webs is behind the cricoid cartilage.

Guisez described cases of incompletely valvular stenosis of the esophagus in which the roentgen examination showed nothing abnormal, but esophagoscopy revealed a semilunar valve partially closing the lumen of the esophagus. This condition occurs at a level with the opening in the diaphragm.

Roentgen diagnosis. The passage of the opaque meal is observed under the fluoroscope and the impediment to the flow is noted. As an aid in the