

## **Chapter 123: Diagnostic Imaging of the Esophagus**

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### **Techniques**

Various radiologic techniques are currently used to examine the esophagus. These include conventional radiography of the neck and chest, full-column esophagography, air contrast esophagography, mucosal-relief radiography, motion-recording techniques, water-soluble contrast examination, computed tomography (CT), magnetic resonance imaging (MRI), and radionuclide imaging. Except for conventional radiography, CT, MRI, and radionuclide studies, these examinations are performed under fluoroscopic control. The advantages and disadvantages of each technique are discussed below.

#### **Conventional radiographs of the neck and chest**

Plain radiographs of the neck and chest are capable of demonstrating some advanced abnormalities of the esophagus (eg, a large Zenker's diverticulum, a mediastinal air leak, an advanced carcinoma of the esophagus with an air fluid level, or thickening of the azygoesophageal or posterior tracheal soft-tissue stripes). In practice, however, the contribution of plain radiographs to the diagnosis of esophageal disease is minimal because of their insensitivity to early or curable stages of esophageal disease.

#### **Full-column esophagography**

Full-column esophagography is a single contrast technique performed with the patient in the horizontal position. The radiologist observes fluoroscopically as the patient swallows a thin suspension of barium through a straw. Esophageal motility is assessed by observing peristalsis during one or more single swallows of barium. Anatomic integrity of the esophagus is assessed when the esophagus is maximally distended. Maximal distension is best achieved when the patient swallows barium rapidly and then performs the Valsalva maneuver. Spot films are obtained in two views during maximal distension.

Disease processes that alter the contour of the esophagus, such as strictures, webs, circumferential carcinomas, large ulcers, and extrinsic masses, are well delineated by the full-column technique. However, more subtle mucosal abnormalities including small plaque-like neoplasms (Itai et al, 1978), mild-to-moderate cases of esophagitis (Ott et al, 1981), small ulcers and erosions, and many cases of esophageal varices may go undetected by this method. The main advantage of this method, is the speed with which the examination can be performed.

#### **Air contrast esophagography**

The air contrast examination of the esophagus is performed with the patient in the upright position and turned slightly to the left to allow the esophagus to project clear of the spine. The patient ingests an effervescent agent and then drinks a high-density barium suspension as quickly as possible. The repeated swallows interrupt the peristaltic sequence, resulting in esophageal hypotonia. The barium passes through the relaxed lower esophageal

sphincter, leaving the esophageal mucosa coated. At the same time, gas from the distended stomach refluxes back through the lower esophageal sphincter to distend the esophagus.

In some patients adequate double contrast views of the distal esophagus are difficult to obtain. In such cases a small-caliber Foley catheter is passed into the esophagus. As the patient swallows a high-density barium solution, air is insufflated through the catheter, allowing detailed examination of the distal esophageal mucosa (Levine et al, 1984a).

The strength of the air contrast technique is its ability to demonstrate small esophageal tumors (Itai et al, 1978) and subtle mucosal irregularities associated with esophagitis (Koehler et al, 1980; Laufer, 1979; Ott et al, 1981; Skucas, 1978). Although abnormalities such as varices, hiatal hernias, and lower esophageal rings can be seen on double contrast radiographs, full-column esophagography or mucosal-relief radiography may demonstrate them to a better advantage (Gelfand and Ott, 1981).

### **Mucosal-relief radiography**

The mucosal-relief examination is performed by coating the esophagus with a dense barium suspension or a barium paste preparation. After the barium has passed into the stomach, the esophagus is filmed while it is in the collapsed state. This examination is indicated primarily for suspected esophageal varices, although other abnormalities may be demonstrated. Lesions requiring distention for their demonstration, such as strictures, diverticula, webs, and rings, may be invisible with this technique.

### **Motion recording techniques**

Every contrast examination of the esophagus includes ordinary fluoroscopy in which the radiologist observes the sequence of events occurring during swallowing. However, when a subtle abnormality is suspected or when a permanent record is needed, motion-recording techniques allow detailed review of the examination.

Several types of dynamic recording techniques are available including video recording, cineradiography, and rapid-sequence spot film camera recording. Video recording is currently the most useful motion-recording technique because it combines very good spatial resolution with immediate availability of the recording for review. Motion-recording techniques have their greatest use in the evaluation of the cervical esophagus (through which barium passes very rapidly) and the detection of esophageal dysmotility and small esophageal leaks.

### **Water-soluble contrast examination**

Iodinated water-soluble contrast media, such as meglumine and sodium diatrizoate, should be used when perforation of the pharynx or esophagus is suspected because barium in the mediastinum may incite an inflammatory reaction. In addition, barium may be retained in an extra-pharyngeal collection for a long time, making reexamination difficult. However, if an esophageal perforation is suspected but not shown by water-soluble contrast examination, immediate reexamination with barium sometimes demonstrates the abnormality (Dodds et al, 1982a; Foley et al, 1982). On the other hand, patients who are known to aspirate or who are suspected of having a pharyngotracheal or esophagotracheal fistula should not be given water-

soluble contrast media because these agents can cause chemical pneumonitis or pulmonary edema. In cases in which both esophageal leak and aspiration or tracheoesophageal fistula are suspected, nonionic or low-osmolar iodinated water-soluble contrast media may be used because in theory they cause minimal or temporary pulmonary sequelae. However, the usefulness of these agents in such circumstances has not been tested adequately.

### **Computed tomography**

The major use for CT scanning in evaluating the esophagus is in the pretreatment staging of esophageal carcinomas. Although several studies have found CT scanning to be an accurate technique in assessing local tumor extent and metastatic spread (Moss et al, 1981; Picus et al, 1983; Thompson et al, 1983), other studies have been less encouraging (Mannell, 1984; Quint et al, 1985b).

CT scans of the esophagus are performed using approximately 1-cm scan intervals and 1-cm collimation of the x-ray beam. Oral contrast material is routinely administered before the examination. In addition, the patient is asked to sip oral contrast material through a straw while the examination is in progress. Scans are performed to the level of the umbilicus to include the liver and the celiac lymph nodes.

### **Magnetic resonance imaging**

Magnetic resonance imaging (MRI) is another noninvasive cross-sectional imaging technique that has potential for evaluating the esophagus. Experience with MRI in the staging of esophageal carcinoma is limited. Preliminary data indicated that MRI is slightly less accurate than CT in staging esophageal carcinoma (Halvorsen et al, 1987; Quint et al, 1985a). In addition, MRI is more expensive and less readily available than CT. Consequently, CT remains the imaging procedure of choice for staging esophageal carcinoma. The role of MRI in evaluating the esophagus remains to be determined; however, new data predict a greater role in the future (Vellet et al, 1990).

### **Endoscopic ultrasound**

Preliminary reports indicate that endoscopic ultrasound is useful in the diagnosis and local staging of esophageal carcinoma (Hyder, 1987; Murata et al, 1987; Shorvon et al, 1987). However, further data are needed to define the role of ultrasound with respect to other imaging techniques for evaluating the esophagus.

### **Radionuclide imaging**

Radionuclide imaging has been used to evaluate esophageal transit and emptying, gastroesophageal reflux, and response to treatment in patients with achalasia. The studies are performed with orally ingested sulfur colloid or diethylenetriaminepentaacetic acid (DTPA) labeled with technetium-99m. However, because abnormalities of transit and emptying are relatively nonspecific, additional imaging studies are often required to make a more precise diagnosis. Furthermore, radionuclide assessment of gastroesophageal reflux has been criticized because it correlates poorly with the symptoms or severity of reflux esophagitis. Nevertheless, a recent study shows an accuracy of greater than 95% for radionuclide transit studies.

## **Radiographic Anatomy**

### **Normal appearance and anatomic relationships**

The cervical esophagus begins at the level of the sixth cervical vertebra just distal to the cricopharyngeus muscle. This portion of the esophagus is a featureless tube except for a small rounded or weblike indentation on its anterior wall caused by the anterior submucosal venous plexus (Pitman and Fraser, 1965).

Important anatomic relationships of the cervical esophagus include the trachea anteriorly, the cervical spine posteriorly, and the thyroid gland, parathyroid glands, and cervical lymph nodes laterally. Enlargement of these structures may deviate or compress the cervical esophagus. Tracheoesophageal fistulas may be acquired by trauma, surgery, or infection, and large anterior cervical osteophytes may compromise the cervical esophageal lumen, causing dysphagia. In addition, abscesses involving the prevertebral soft tissues may deviate the cervical esophagus anteriorly.

The thoracic esophagus lies anterior and slightly to the left of the spine. Three indentations can generally be seen deforming the left anterolateral aspect of the esophagus. These indentations are produced by the aortic arch, left main bronchus, and left atrium (Fig. 123-1). The distal third of the esophagus curves gently to the left as it approaches the diaphragmatic hiatus. The tubular lumen enlarges just proximal to the esophagogastric junction to form the vestibule, or phrenic ampulla.

The esophageal mucosa has a smooth, featureless appearance. When the esophagus is collapsed, smooth longitudinal folds traverse its entire length. Occasionally, delicate transverse folds may be observed. These are believed to be caused by contraction of the muscularis mucosae (Laufer, 1982).

The relationship of the thoracic esophagus to other intrathoracic structures is complex. Aortic tortuosity and ectasia may deviate the distal esophagus anteriorly (Fig. 123-2). Aneurysms in the region of the aortic arch produce an exaggeration of the normal aortic impression and deviate the proximal thoracic aorta to the right. Mediastinal lymph nodes lie in intimate contact with the esophagus, particularly in the subcarinal region and, when enlarged, may deviate or produce discrete impressions on the esophagus (Fig. 123-3). Cardiac enlargement, particularly left atrial enlargement, deviates the distal esophagus in a posterior and lateral direction. Acquired fistulous connections between the esophagus and tracheobronchial tree may be caused by invasion from bronchogenic carcinoma, by extension of esophageal carcinoma anteriorly to the trachea or anterolaterally to the bronchi, and occasionally by severe mediastinal infections such as histoplasmosis.

## **Congenital Anomalies**

The most important congenital malformations of the esophagus are esophageal atresia and tracheoesophageal (TE) fistula. Atresia of the esophagus is believed to result from incomplete canalization or defective unfolding of the walls of the foregut; fistulas between the trachea and esophagus are thought to result from incomplete separation of the tracheal bud from the esophagus (Zaino and Beneventano, 1977). Approximately 86% of esophageal atresias are accompanied by a distal TE fistula. In about 8% of cases atresia is present without a TE fistula, and in about 4% an H-type fistula occurs without esophageal atresia.

Infants with proximal esophageal atresia and a TE fistula to the distal esophageal segment usually have gaseous abdominal distension, whereas those with atresia alone or with a TE fistula to the proximal esophageal segment have a gasless abdomen. Other plain-film findings include aspiration pneumonitis or atelectasis and dilatation of the gas-filled proximal esophageal pouch. Inability to pass a nasogastric tube into the stomach with coiling of the tube in the pouch is a reliable sign of esophageal atresia. If necessary, a small amount of thin barium can be injected into the pouch to confirm the diagnosis. The barium should then be aspirated.

An H-type TE fistula may not be detected until late in infancy or early childhood following recurrent unexplained pneumonia. The diagnosis can be made by intubating the esophagus with a single-hole catheter, placing the child in the lateral position, and repeatedly injecting barium into the esophagus under fluoroscopic control, beginning with the catheter tip near the esophagogastric junction and gradually withdrawing it cephalad. Care must be taken to prevent the child from regurgitating and aspirating barium because distinguishing tracheal filling by this route from tracheal filling via a TE fistula may be difficult.

Esophageal duplication is a rare congenital anomaly that is usually discovered incidentally. It is classified into two general types: (1) intramural cyst, representing a true duplication arising from persistent vacuoles that normally reconstitute the esophageal lumen, and (2) neurenteric cysts, representing a foregut anomaly that is a remnant of the dorsal part of the notochord (Kirwan et al, 1973). The latter type is commonly associated with cervical and thoracic spinal abnormalities. Esophageal duplication is usually segmental and located in the lower posterior mediastinum. On chest radiography esophageal duplication appears as a round or oval posterior mediastinal mass (Fig. 123-4, A). The mass usually causes compression or deviation of the esophagus (Fig. 123-4, B). In general, it does not communicate with the esophageal lumen. When communication is demonstrated, the barium esophagogram may reveal a double-barrel appearance, similar to that seen with iatrogenic esophageal dissection (Fig. 123-5). CT scanning demonstrates a cystic mass closely applied to the esophagus (Fig. 123-4, C).

Other congenital anomalies of the esophagus, some of which are very rare, include webs, rings or diaphragms, diverticula, strictures of congenital origin, esophageal cartilaginous rings, brachyoesophagus, idiopathic muscular hypertrophy, bronchoesophageal fistulas, laryngotracheal esophageal clefts, and esophageal bronchus. Discussion of these congenital anomalies is beyond the scope of this chapter.

## Motility Disorders

The main function of the esophagus is to transport ingested food from the pharynx to the stomach. This function is accomplished primarily by esophageal peristalsis, although gravity aids in transporting the bolus when an individual is upright. A discussion of the neuromuscular events controlling esophageal peristalsis is beyond the scope of this chapter. However, for simplicity, peristalsis may be envisioned as a rapid wave of inhibition followed by a slower wave of contraction, traversing the esophagus in an aboral direction (Ingelfinger, 1958). When the inhibitory wave reaches the lower esophageal sphincter (LES), the sphincter relaxes. Relaxation is terminated when the peristaltic contraction reaches the LES.

Although many consider manometry to be the method of choice for the detection and evaluation of esophageal motility disorders, fluoroscopy (Dodds et al, 1976), cineradiography (Clements et al, 1979), and radionuclide studies (Taillefer et al, 1991) have been shown to be as useful as manometry for qualitative assessment of esophageal peristaltic activity. Radiographic techniques have the advantage of demonstrating structural defects. The patient is examined in the recumbent position to eliminate gravity as a factor in bolus transport. As the contraction wave passes through the esophagus, the proximal end, or tail, of the barium column takes on an inverted V configuration. The tail of the bolus is observed following several single swallows because a second swallow shortly after the first interferes with the peristaltic wave produced by the first swallow. Three basic types of esophageal contractions are recognized. Primary peristaltic contractions are initiated by swallowing. Secondary peristaltic contractions occur in response to local esophageal stimulation such as distension produced by gastroesophageal reflux or by material left in the esophagus after primary peristalsis. Once initiated, primary and secondary peristalsis have similar features. Tertiary contractions are nonperistaltic contractions, that is, they do not propagate aborally along the length of the esophagus.

Abnormalities of peristalsis that may be observed during fluoroscopy include decreased incidence of peristalsis in response to swallowing, failure of the peristaltic wave to progress to the esophagogastric junction, and complete absence of peristalsis (aperistalsis). Sphincter abnormalities include absent or incomplete relaxation of the LES and continuous LES relaxation (low resting LES pressure). Peristaltic and sphincter function may be affected either singly or in combination. Some of the diseases that cause disordered esophageal motility are discussed below.

### Achalasia

Achalasia is a neuromuscular disorder associated with degeneration of ganglion cells of Auerbach's plexus (Ellis and Olsen, 1969). Radiologic hallmarks of achalasia include aperistalsis, esophageal dilatation, and failure of the LES to relax for long periods of time, resulting in esophageal retention of ingested material (Fig. 123-6, A). Often the distal esophagus has a conical narrowing with a beaklike appearance at the esophagogastric junction (Fig. 123-6, B). In earlier stages of the disease the esophagus may be only minimally dilated and may demonstrate nonperistaltic, irregular contraction waves resembling diffuse esophageal spasm. This variation has been termed *vigorous achalasia* (Bondi et al, 1972) (Fig. 123-7).

Although impairment of the LES opening is a constant feature of achalasia, it is not pathognomonic. Occasional patients with diffuse esophageal spasm, presbyesophagus, and connective tissue disease exhibit failure of LES relaxation in response to swallowing (Creamer et al, 1956, 1958; Kaye, 1973; Soergel et al, 1964). Carcinoma involving the distal esophagus can also produce radiographic and manometric findings that are indistinguishable from primary achalasia (Lawson and Dodds, 1976; Seaman et al, 1963; Tucker et al, 1978). Because most tumors responsible for secondary achalasia arise from the gastric fundus, careful attention to this region is essential when evaluating patients for suspected achalasia. The mechanism responsible for producing this abnormality is unknown, but destruction of the myenteric plexus and altered neuromuscular sensitivity secondary to esophageal obstruction and dilatation have been suggested (Seaman, 1981). Esophageal involvement in Chagas' disease, a systemic disorder caused by the parasite *Trypanosoma cruzi*, may also closely resemble achalasia. Histologic examination of the esophagus in Chagas' disease demonstrates destruction of ganglion cells (Owen and Brandborh, 1983). Finally, failure of relaxation of the lower esophageal sphincter may be observed in some neuropathies (such as those seen after cerebral vascular accident, in post-vagotomy syndrome, or in diabetes mellitus) and in association with stricture secondary to reflux esophagitis (Eisenberg, 1990).

In the past, pharmaceutical aids such as methacholine (Mecholyl), amylnitrite, or a carbon dioxide-releasing source (eg, Seidleitz powder) have been used to help to establish the diagnosis of achalasia. However, these tests have been superseded by manometry and endoscopy with biopsy when required.

Patients with longstanding achalasia have an increased incidence of esophageal carcinoma, ranging from 0.5% to 29% (Camara-Lopez, 1961; Hankins and McLaughlin, 1975; Just-Viera and Haight, 1969; Seaman, 1983; Seaman et al, 1963; Wychulis et al, 1971) and averaging 4% (Seaman, 1983). The prognosis appears to be poorer than for the usual esophageal cancer, possibly as a result of delayed diagnosis because early symptoms are attributed to the achalasia (Seaman, 1981). In addition, tumor is difficult to identify radiographically or endoscopically in an esophagus filled with fluid and food.

### **Diffuse esophageal spasm**

In patients with diffuse esophageal spasm (DES), peristalsis in the proximal one third of the esophagus is normal, but contractions in the smooth muscle portion of the esophagus are uncoordinated and nonperistaltic. The radiographic patterns produced by this disorder include a spiral or cork-screw appearance (Fig. 123-8), a series of pouches separated by lumen-obliterating contractions (rosary or shish-kebab appearance), or diffuse narrowing of the esophagus. The proximal esophagus is often dilated, and a hiatus hernia is invariably present, probably secondary to spasm of the longitudinal as well as the circular muscles (Seaman, 1981). Because the radiographic characteristics of DES can be seen in asymptomatic patients and in patients with other esophageal motility disorders (Bennett and Hendrix, 1970), the diagnosis of DES is limited to patients with substernal chest pain or dysphagia who manifest the above radiographic findings and in whom coronary artery disease or other causes of chest pain have been excluded by appropriate investigations (Laufer, 1989).

The cause of DES is still unknown. Opinions differ as to whether DES is part of the same disease spectrum as achalasia or is a completely separate entity (Dodds, 1983).

## **Scleroderma**

Involvement of the esophagus in scleroderma is classically described as being characterized by diminished or absent peristalsis in the distal two thirds of the esophagus. On radiographic examination, peristaltic waves are decreased in incidence, and those that occur are weak. The esophagus is generally mildly to moderately dilated. However, in severe cases the esophagus may be aperistaltic with significant dilatation. The LES is often patulous, predisposing to reflux esophagitis and stricture (Fig. 123-9). A hiatus hernia is commonly present (Garrett et al, 1971). The primary histologic abnormality is atrophy of smooth muscle (D'Angelo et al, 1969; Treacy et al, 1963). Scleroderma may be complicated by *Candida* esophagitis (Geftter et al, 1981), aspiration pneumonia, and Barrett's esophagus as a result of severe reflux esophagitis (Recht et al, 1988). Thus, patients with scleroderma have a predisposition to esophageal adenocarcinoma (Halpert et al, 1983).

## **Other connective tissue diseases**

Esophageal dysmotility may occur with the connective tissue diseases, including systemic lupus erythematosus, Raynaud's disease, and dermatomyositis (Ramirez-Mata et al, 1974; Stevens et al, 1964). Lupus and Raynaud's disease produce a radiographic pattern similar to that of scleroderma. Dermatomyositis differs in that dysmotility is observed primarily in the pharynx and proximal striated muscle portion of the esophagus (O'Hara et al, 1967), although smooth muscle dysfunction does occur in this group of patients (Jacob et al, 1983). It should be noted, however, that patients with rheumatoid arthritis usually do not have radiographically demonstrable esophageal dysmotility.

## **Presbyesophagus**

Presbyesophagus refers to abnormal esophageal motor function associated with aging (Soergel et al, 1964). Although an occasional patient with presbyesophagus may have dysphagia when eating solid foods, these patients usually do not have esophageal symptoms. The histologic abnormality associated with presbyesophagus is a significant decrease in ganglion cells in Auerbach's plexus (Eckhardt and LeCompte, 1978).

Many patients described in early reports of presbyesophagus had neurologic disorders or diabetes mellitus, which by themselves can cause a decreased incidence of normal peristalsis, and increased frequency of tertiary contractions, and LES dysfunction. In fact, the vast majority of healthy elderly individuals show relatively minor changes of esophageal function that are only minimally progressive with aging (Ott, 1988). Therefore the existence of presbyesophagus as a separate entity has been challenged recently. Several authors have proposed the general term *nonspecific esophageal motor disorder* (NEMD) to describe symptomatic patients with motility disturbances that defy specific classification (Ott, 1988).

Common radiologic findings include failure of the primary peristaltic wave to traverse the entire esophagus and nonperistaltic (tertiary) contractions (Zboralske et al, 1964). In some patients the LES may fail to relax and the esophagus may be mildly to moderately dilated.



## **Esophageal atresia and tracheoesophageal fistula**

Children who have undergone successful repair of esophageal atresia and tracheoesophageal fistula nearly always have esophageal dysmotility (Burgess et al, 1968). Radiologic and manometric studies demonstrate discontinuity of the peristaltic stripping wave in the esophageal segment containing the surgical anastomosis. In addition, many patients have gastroesophageal reflux (Orringer et al, 1977; Parker et al, 1979). These abnormalities may result in dysphagia and aspiration (Chrispin et al, 1966). Children with an H-type tracheoesophageal fistula commonly have similar esophageal motor abnormalities (Thomas and Chrispin, 1969).

### **Miscellaneous disease**

A large number of diseases, both local and systemic, have been associated with esophageal dysmotility. Severe esophagitis of any cause (peptic disease, caustic agents, alcohol, infection, radiation) may result in absence of peristalsis in the involved segment or in the entire esophagus (Simeone et al, 1977).

Following truncal vagotomy up to 10% of patients may experience dysphagia (Dagradi et al, 1962). Radiologic examination of these patients shows mild esophageal dilatation and narrowing of the distal esophagus. These findings usually result from postoperative edema, hematoma, or vagal denervation and remit spontaneously within several weeks to months (Dodds, 1983).

Abnormal peristalsis can occur in association with neuropathies resulting from diabetes mellitus (Hollis et al, 1977; Mandelstam et al, 1969) or alcoholism (Winship et al, 1968) and in patients with endocrine diseases such as myxedema (Christensen, 1967) or hyperthyroidism (Meshkinpour et al, 1979). Primary diseases of the central nervous system (Fischer et al, 1965; Silbiger et al, 1967) and primary muscle diseases may also be responsible for esophageal dysmotility. Although most neuromuscular diseases primarily affect the pharynx and proximal striated muscle portion of the esophagus, motor dysfunction may occur in the distal smooth muscle portion as well (Dodds, 1983).

The term *nutcracker esophagus* has been used to describe the association of chest pain with high-amplitude peristaltic waves (Benjamin et al, 1979). Because the peristaltic wave appears normal fluoroscopically, this condition, also variously described as *symptomatic esophageal spasm* and *hypertensive peristaltic esophagus*, is not diagnosed radiologically and requires manometry for confirmation.

### **Esophagitis**

Inflammation of the esophagus can be caused by a host of physical, chemical, and infectious agents. By far the most common cause of esophagitis is gastroesophageal reflux (GER). With the increasing use of immunosuppressive medications, opportunistic infection of the esophagus is becoming a more frequent occurrence. Caustic agents, foreign bodies, irradiation, oral medications, and a variety of systemic diseases are all capable of inciting an inflammatory response in the esophagus.

The use of double-contrast esophagography allows detection of radiologic abnormalities in earlier stages of esophagitis than with single-contrast studies (Creteur et al, 1983b; Koehler et al, 1980; Kressel et al, 1981; Ott et al, 1981). Although the use of double-contrast views alone may not significantly increase the radiographic sensitivity in detecting esophagitis, the combined use of single- and double-contrast techniques increases radiographic sensitivity from approximately 75% to approximately 90% when compared to use of the single-contrast technique alone (Creteur et al, 1983b; Koehler et al, 1980).

### **Reflux esophagitis**

Radiologic evaluation for esophagitis always includes testing for GER. In addition to searching fluoroscopically for spontaneous reflux when the patient is turning from the prone to the supine position, various provocative maneuvers can be used. These include the Valsalva maneuver, coughing, leg-raising, placing the examination table in the Trendelenburg position, and compression. Despite the use of these stress maneuvers, GER has been reported as demonstrable in no more than 50% of patients who have endoscopic evidence of reflux esophagitis (Dodds et al, 1976; Kantrowitz et al, 1968). The incidence of GER is increased with the use of the water siphonage test in which the patient is observed while taking several swallows of water (Linsman, 1965). However, this test yields positive results in a significant percentage of normal individuals because with swallowing the LES undergoes physiologic relaxation. The most likely explanation for the failure to demonstrate barium reflux in a large percentage of patients with known esophagitis is that the predominant mechanism for GER in these patients is transient complete relaxation of the LES rather than sustained low resting LES pressure (Dodds et al, 1982b). Several recent studies have confirmed the relatively low sensitivity of fluoroscopy in the demonstration of GER (Breen and Whelan, 1978; Kaul et al, 1985). However, these studies used endoscopy as a standard. It is now recognized that many patients with acid reflux have no endoscopic abnormalities (DeMeester et al, 1980). When 24-hour esophageal pH monitoring is used as a standard, and fluoroscopy is combined with abdominal compression and measurement of the internal diameter of the cardiac esophagus, barium radiology has a sensitivity of 87% and an accuracy of 81% whereas endoscopy is only 64% accurate (Sellars et al, 1987).

The earliest radiographic findings in active reflux esophagitis include abnormal motility, mucosal granularity, and superficial mucosal erosion, which appears as small round dots or streaks of barium in the distal esophagus. Frank ulceration occurs in more severe cases and can be either linear (Fig. 123-10) or round in configuration (Figs. 123-9 and 123-11). Thickening of the esophageal folds and limited distensibility of the esophagus resulting from edema are nonspecific findings that can occur in esophagitis of any cause. Although transient, delicate, transverse folds can be seen in patients without esophageal pathology, fixed, coarse, transverse folds are a sign of moderate or severe esophagitis (Levine and Goldstein, 1984). Aperistalsis has been reported to occur in patients with severe esophagitis (Simeone et al, 1977). Normal peristalsis may return when the esophagitis resolves. A hiatus hernia is often present, but its significance and its relationship to GER remain controversial. A radiographic finding that appears to be specific for reflux esophagitis is the inflammatory esophagogastric polyp and fold, which consists of an enlarged gastric fundal fold that crosses the esophagogastric junction and ends in a polypoid protuberance in the distal esophagus (Bleshman et al, 1978; Gharemani et al, 1984; Styles et al, 1985) (Fig. 123-12). One or more discrete plaque-like defects in the distal esophagus have also been described as a sign of

reflux esophagitis (Levine et al, 1986).

Healing of esophageal ulcers results in a variable degree of scarring, ranging from mild irregularity and asymmetry of the distal esophagus to a stricture. Although the resulting strictures may be symmetric (Fig. 123-13, A), they are often asymmetric (Figs. 123-13, B and C), and endoscopy with biopsy may be necessary to exclude a carcinoma. Other sequelae occasionally seen with healed esophagitis are prominent sacculations in a region of esophageal scarring (Fig. 123-14) and distal esophageal webs (Weaver et al, 1984).

### **Barrett's esophagus**

Partial lining of the esophagus by columnar epithelium, once thought to be a congenital abnormality, is now generally considered to be an acquired mucosal alteration representing migration of columnar epithelium from the stomach proximally into the esophagus as a result of severe chronic reflux esophagitis (Spechler and Goyal, 1986). Formerly considered an uncommon condition, the prevalence of Barrett's esophagus in patients with reflux esophagitis has been estimated to be approximately 10% or possibly more (Sarr et al, 1985). In most patients a stricture is present at the junction of the squamous and columnar epithelium. Although a specific radiologic diagnosis of Barrett's esophagus is difficult to make, it should be suspected in patients with esophageal ulceration or stricture (Robbins et al, 1977, 1978) (Fig. 123-15), particularly if these findings are present in the mid- or proximal esophagus (Fig. 123-16). Levine et al (1984b) have described a fine, reticular, mucosal pattern in patients with Barrett's esophagus. In most cases this pattern was observed just distal to a stricture on air contrast esophagograms. The diagnosis of Barrett's esophagus is important because the risk of developing esophageal cancer has been estimated to be up to 40 times greater than that seen in the absence of Barrett's esophagus (Cameron et al, 1985). Therefore, it has been suggested that periodic endoscopy with biopsy should be performed at 6- to 12-month intervals after the diagnosis of Barrett's esophagus has been established (Harle et al, 1985; Haggitt et al, 1978; Naef et al, 1975; Sarr et al, 1985), although the efficacy and cost effectiveness of such a surveillance program remain to be demonstrated.

### **Infectious esophagitis**

Esophagitis caused by infection occurs predominantly in immunocompromised patients (those with AIDS, cancer, or other debilitating illnesses, including severe diabetes mellitus; or those receiving immunosuppressive medications such as cancer or organ transplant patients), although cases occurring in otherwise healthy individuals have been reported (Owensby and Stammer, 1978; Springer et al, 1979). Long segments of the esophagus are usually involved. Most patients complain of odynophagia.

### ***Candidiasis***

The most common organism infecting the esophagus is *Candida albicans*. Although immunosuppression is the most common factor predisposing to *Candida* esophagitis, a second important factor is esophageal stasis, accounting for up to 25% of cases (Levine et al, 1985). Stasis is seen most commonly with obstruction caused by achalasia or strictures. In the earliest stages, radiologic examination demonstrates abnormal esophageal motility (Lewicki and Moore, 1975). Other early radiologic findings are small plaque-like filling defects on the

mucosal surface, which are often longitudinal in orientation (Athey et al, 1977; Laufer, 1982; Lewicki and Moore, 1975). In more advanced cases extensive plaque formation produces a shaggy or cobblestone appearance (Goldberg and Dodds, 1968) (Fig. 123-17). Although the shaggy form of candidiasis was considered infrequent until recently, the AIDS epidemic has increased the number of patients with this appearance (Levine et al, 1987). Uncommon radiologic appearances of *Candida* esophagitis include a fungus ball, nodular masses, a focal constrictive lesion, or even complete esophageal obstruction. These forms may be mistaken for neoplasia (Farman et al, 1986; Roberts et al, 1987).

Chronic *Candida* esophagitis occurs in patients with mucocutaneous candidiasis (Rohrman and Kidd, 1978) or esophageal stasis (Geftter et al, 1981). Radiographic characteristics include a reticular mucosal pattern and large nodular filling defects. Some chronic forms of *Candida* esophagitis are predominantly characterized by stricture rather than other mucosal findings. Intramural pseudodiverticulosis occasionally occurs in association with *Candida* infection of the esophagus (Rohrman and Kidd, 1978). The abnormalities of chronic and acute *Candida* esophagitis are reversible with antifungal treatment.

### ***Herpes esophagitis***

Herpes esophagitis occurs less commonly than *Candida* esophagitis but is another important cause of esophagitis in immunosuppressed patients. It is often diagnosed when severe dysphagia thought to be caused by candidiasis fails to respond to appropriate antifungal treatment (Donner et al, 1981). Although seen predominantly in immunosuppressed patients with or without active oral herpetic lesions, its occurrence in otherwise healthy young adults has been reported (Owensby and Stammer, 1978; Springer et al, 1979).

The earliest endoscopic finding of vesicle formation cannot be identified radiographically. It is only the vesicular rupture that leads to formation of discrete superficial ulcers that is radiologically visible (Agha et al, 1986a; Levine et al, 1988). Although herpes esophagitis may be radiographically indistinguishable from *Candida* esophagitis, esophagography in some patients demonstrates ulcerated mucosal plaques and discrete ulcers on an otherwise normal mucosal background. This pattern, when present, is highly suggestive of viral esophagitis (Levine et al, 1981) (Fig. 123-18). Less common presentations of herpetic esophagitis include strictures or a single giant ulcer (Levine et al, 1988). Herpes esophagitis is usually self-limiting. The diagnosis is established by the cytologic demonstration of multinucleated cells containing intranuclear inclusion bodies.

### **Caustic esophagitis**

The radiographic findings in esophagitis that are produced by ingestion of caustic agents depend on the type of agent ingested and the stage at which the patient is examined. In general ingested acids cause coagulation necrosis, which limits their penetration. Alkalis, in contrast, dissolve tissue and thus penetrate more deeply. Caustic esophagitis caused by concentrated sodium hydroxide is the most common caustic injury to the esophagus seen in the USA (Goldman and Weigert, 1984). In the acute stage, ingestion of caustic agents results in mucosal irregularity, ulceration, fold thickening, and weakened peristalsis (Martel, 1972). The esophageal wall may be thickened and the lumen narrowed (Franken, 1973). Filling defects caused by sloughing of the mucosa may be seen (Donner et al, 1981; Franken, 1973).

Examination of the esophagus during this stage should be initiated with a water-soluble contrast medium to exclude esophageal or gastric perforation (Dodds, 1983). When perforation has been excluded, barium may be used. Radiologic examination in later stages (more than 3 weeks after the injury) usually shows a long, smooth stricture (Fig. 123-19), but local areas of submucosal fibrosis may cause nodular defects or scalloping. Esophagograms performed to evaluate caustic ingestion should be supplemented by an upper GI examination to exclude an associated gastric injury because up to 25% of patients with alkaline corrosive esophagitis may have gastric involvement (Franken, 1973). This is especially important because endoscopy is often terminated when an injured area of the esophagus is reached, leading to an incomplete evaluation of the potentially injured areas (Neimark and Rogers, 1985).

Patients with longstanding lye strictures are at risk for developing esophageal carcinoma approximately 30 years after the initial caustic injury (Appelquist and Salmo, 1980; Hopkins and Postlethwait, 1981; Lansing et al, 1969). This complication may be recognized by the development of nodularity or ulceration within a previously smooth stricture.

### **Radiation esophagitis**

Radiation esophagitis occurs in a small percentage of patients receiving mediastinal irradiation. In general, such patients experience symptoms after receiving a dose of 4500 to 6000 rad over 6 to 8 weeks (Goldstein et al, 1975). However, symptomatic radiation esophagitis may occur with much smaller radiation doses in patients who are also receiving doxorubicin hydrochloride (Adriamycin), a chemotherapeutic agent that interferes with tissue repair (Boal et al, 1979; Newberger et al, 1978). Furthermore, doses between 2000 and 4500 rad can cause self-limited esophagitis. Esophagography is of limited use in the acute stage of the disease process, but it is well-suited to identify chronic changes.

The most common finding on radiologic examination is esophageal motor dysfunction without alteration of morphology, seen 1 to 2 months following radiotherapy (Lepke and Lischitz, 1983). Morphologic abnormalities, when present, consist of diffuse ulceration in the acute stage and smooth tapered strictures in later stages (Goldstein et al, 1975; Lepke and Lischitz, 1983).

### **Drug-induced esophagitis**

Several types of oral medications are capable of producing localized esophagitis when the contact time between the drug and the mucosa is prolonged. Drug-induced esophagitis occurs most commonly in patients who ingest tablets just before retiring (Creteur et al, 1983a; Teplick et al, 1980), especially when the medication is taken with little or no water (Fisher et al, 1982). The occurrence of drug-induced esophagitis in these patients may be related in part to the decreased salivation and swallowing that occurs during sleep (Dent et al, 1980). Any functional obstruction of the esophagus such as that resulting from compression by an enlarged heart or a tortuous aorta further increases the chance of prolonged drug-mucosa contact. Most patients with drug-induced esophagitis have no underlying esophageal disorder (Kikendall et al, 1983). The medications most frequently responsible for this type of esophagitis are antibiotics (such as tetracycline and doxycycline), quinidine, slow-release potassium chloride tablets, and vitamin C, although many other agents have been implicated.

Radiologic examination demonstrates ulceration that may be superficial (eg, caused by tetracyclines) or deep (eg, caused by potassium chloride or quinine tablets). The findings are best identified on double-contrast examination (Agha et al, 1986b). Ulceration is usually focal and located in the mid-esophagus (Fig. 123-20). The ulcerations are self-limited with normal mucosa demonstrable within 2 weeks after withdrawal of the offending agent. The deep ulcerations associated with potassium chloride or quinidine may produce an inflammatory mass seen radiographically as a localized esophageal narrowing, most commonly above an enlarged left atrium. Such a stricture is constant on fluoroscopy, distinguishing it from an area of gradually resolving spasm, also commonly seen with drug-induced esophageal injury.

### **Nasogastric intubation**

Nasogastric intubation renders the lower esophageal sphincter incompetent and interferes with esophageal peristalsis. As a result, patients requiring prolonged nasogastric intubation, particularly those with bile reflux secondary to gastric surgery, may develop severe peptic esophagitis. The severe esophagitis produced by prolonged nasogastric intubation characteristically results in a long tapered stricture (Graham et al, 1959). Although most strictures develop after repeated or prolonged nasogastric intubation, such complications have been observed to develop after intubation lasting only 2 days or in some cases only after removal of the tube (Levine et al, 1989b). Strictures occur less frequently with the use of narrow-gauge tubes such as the Dobhoff catheter.

### **Granulomatous disease**

Crohn's disease may rarely involve the esophagus (Cynn et al, 1977; Gharemani et al, 1982), invariably after the diagnosis has been established in the small or large bowel. In the early stages, aphthous ulcers can occur (Laufer, 1982). In advanced cases larger undermining ulcers, strictures, and intramural tracking may be present. Superficial endoscopic biopsies may fail to provide histologic confirmation of the disease because Crohn's disease is patchy in distribution. Therefore, negative biopsies do not preclude the diagnosis of Crohn's esophagitis (Gharemani et al, 1982).

Tuberculous esophagitis is rare and is caused most often by direct extension from caseous mediastinal lymph nodes. Several cases of complicated tuberculous esophagitis have been reported in patients suffering from AIDS (DeSilva et al, 1990). Swallowed infected sputum generally produces radiographically demonstrable esophagitis only when a preexisting lesion such as a stricture or carcinoma is present (Schneider, 1976; Williford et al, 1983). Rarely, the esophagus may be involved by direct extension from an active focus in the larynx or the thoracic spine. Radiographic findings in tuberculous esophagitis are nonspecific and include contour irregularity, strictures, sinus tracts, or fistulas (DeSilva et al, 1990).

Histoplasmosis is another granulomatous disease that may affect the esophagus secondarily from mediastinal lymphadenopathy or fibrosing mediastinitis. Thus histoplasmosis may present with mediastinal lymphadenopathy causing extrinsic compression of the esophagus. Esophagobronchial fistulas are also occasionally observed as a complication of this disease. Another manifestation of histoplasmosis is nonspecific esophageal ulceration.

Behçet's disease is a multisystem disorder that usually involves the colon but may rarely involve the esophagus in the form of discrete shallow ulcers. The ulcers tend to spare the upper esophagus and may respond to steroid therapy (Lebwohl et al, 1977).

### **Intramural pseudodiverticulosis**

Intramural pseudodiverticulosis is a condition in which the ducts of mucous glands of the esophagus are ectatic. This has a very specific appearance on esophagography. It is a condition that occurs as a sequela of any type of severe esophagitis, most commonly reflux esophagitis (Bruhlmann et al, 1981). In 90% of patients with this disease a stricture is present (Bruhlmann et al, 1981; Muhletaler et al, 1980) and dilatation of the stricture results in amelioration or disappearance of the clinical symptoms. *Candida albicans* has been isolated from the esophagus in about 40% of cases (Sabanathan et al, 1985). Most authorities feel that *Candida albicans* is a secondary colonizer of the pseudodiverticula rather than a cause of the condition. The characteristic radiologic appearance consists of multiple, small, flasklike collections of barium seen outside the esophageal lumen (Fig. 123-21).

### **Bullous dermatoses**

Bullous dermatoses involving the squamous epithelium of the mucous membranes may involve the esophagus. These conditions include pemphigus (Raque et al, 1970), pemphigoid (Agha and Raji, 1982; Al-Kutoubi and Eliot, 1984; Person and Rogers, 1977), epidermolysis bullosa dystrophica (Agha et al, 1983; Orlando et al, 1974), toxic epidermal necrolysis, and Stevens-Johnson syndrome (Calcaterra and Strahan, 1971).

#### ***Pemphigoid***

Pemphigoid is a chronic blistering disease of mucosal epithelium occurring in the middle aged and elderly. The esophagus is involved in approximately 5% of cases (Person and Rogers, 1977). The radiographic features of the esophageal involvement vary with the stage of the disease (Agha and Raji, 1982; Al-Kutoubi and Eliot, 1984). The earliest findings consist of nonspecific inflammatory changes including mucosal edema and spasm. As the bullae rupture and healing takes place, scar formation leads to adhesions, webs, and strictures, primarily involving the upper esophagus. The bullae themselves may be difficult, if not impossible, to demonstrate. Early recognition of esophageal involvement is important because early adhesions can be dislodged and severe stenosis prevented (Agha and Raji, 1982; Al-Kutoubi and Eliot, 1984). Therefore, patients with pemphigoid who complain of dysphagia or who present with upper gastrointestinal bleeding should have careful radiologic evaluation of the esophagus.

#### ***Epidermolysis bullosa***

Epidermolysis bullosa comprises a group of rare hereditary skin disorders in which the basic defect is loss of cohesion between the epidermis and dermis, resulting in recurrent formation of blisters that rupture, ulcerate, and heal with scarring (Seaman, 1983). Only patients with epidermolysis bullosa dystrophica, which is inherited as a recessive trait, develop clinically significant esophageal involvement (Agha et al, 1983). Esophageal involvement usually begins in early childhood. Radiographic features vary with stage of the disease (Agha

et al, 1983; Orlando et al, 1974). Early findings include edema, spasm, dysmotility, and superficial erosions or ulcerations secondary to eruption of bullae. Small nodular filling defects representing bullae may occur. The bullae rupture and heal with scarring, leading to the development of webs and strictures, most commonly involving the upper esophagus (Fig. 123-22). If esophageal scarring is severe, a traction hiatus hernia may result from esophageal shortening. Complete esophageal occlusion occurs rarely.

Esophagography is the preferred method of studying the esophagus in epidermolysis bullosa because of the risks of endoscopy. Esophagoscopy and bougienage should be done only when absolutely necessary because the trauma of these procedures may create new bullae or cause ulceration or perforation of the esophagus (Bauer and Cooper, 1981). In addition, the benefit from bougienage may be short-lived (Tishler et al, 1983). Balloon dilatation is an alternative procedure for managing esophageal stenoses, which avoids the harmful shearing forces of bougienage (Feurle et al, 1984). Colonic interposition has been used in patients with esophageal strictures that no longer respond to dilatation procedures (Bauer and Cooper, 1981).

### **Diverticula**

Diverticula tend to occur in three locations within the esophagus: at the pharyngoesophageal junction (Zenker's diverticula), in the subcarinal region of the midesophagus, and in the distal one third of the esophagus (epiphrenic diverticula). In general, they are false diverticula, consisting of mucosa that has herniated through the muscularis propria (Bruggeman and Seaman, 1973). With rare exceptions esophageal diverticula are acquired abnormalities. Fluoroscopic observations suggest that pulsion forces cause nearly all esophageal diverticula, regardless of location (Dodds, 1977). Although most esophageal diverticula are not associated with symptoms, a majority are associated with esophageal motility disorders, some of which may be symptomatic. An association of midesophageal and epiphrenic diverticula with dysphagia, reflux, vomiting, and weight loss has been reported (Rifkin et al, 1984).

Zenker's diverticula are actually pharyngeal diverticula occurring just proximal to the cricopharyngeus muscle. They are believed to result from increased intraluminal pressure caused by premature closure of the cricopharyngeus muscle. Zenker's diverticula are discussed fully in Chapter 130.

Traditionally, midesophageal diverticula have been classified as traction diverticula resulting from adhesions to inflamed mediastinal lymph nodes. In fact, few data support this mechanism of occurrence (Dodds, 1983; Kaye, 1974; Rifkin et al, 1984). Most midesophageal diverticula are smooth and oval without any evidence of traction (Fig. 123-23), are often seen to expand and contract concentrically during peristalsis, and exhibit longitudinal peristaltic excursions without evidence of mediastinal fixation (Dodds, 1977). In addition, manometric evidence of motor dysfunction has been demonstrated in patients with midesophageal diverticula (Kaye, 1974; Rifkin et al, 1984).

Small epiphrenic diverticula are asymptomatic unless they are associated with other esophageal disease. Large epiphrenic diverticula may be symptomatic and are almost always associated with esophageal dysmotility (Bruggeman and Seaman, 1973; Rifkin et al, 1984).



They usually project anterolaterally to the right side of the esophagus (Fig. 123-24).

### Varices

Esophageal varices are dilated veins located in the submucosa of the esophagus. They generally occur in patients with portal hypertension as a result of increased blood flow in the coronary vein. This increased flow is caused by shunting of mesenteric venous blood away from the portal system to collateral pathways that communicate with the azygous system and superior vena cava. The direction of blood flow in esophageal varices caused by portal hypertension is cephalad. These "uphill" varices are seen primarily in the lower esophagus. "Downhill" varices occasionally occur when the superior vena cava is obstructed distal to the entry of the azygous vein (Johnson et al, 1978; Mikkelsen, 1963). The latter varices predominate in the upper esophagus. A third variety of esophageal varices is not associated with portal hypertension or superior vena caval obstruction and is referred to as "idiopathic" (Kelsen and Burbige, 1982).

On barium swallow examination esophageal varices may appear as thickened longitudinal esophageal folds, nodular filling defects, or serpentine lower esophageal filling defects that give the esophagus a scalloped contour (Fig. 123-25) (Cockerill et al, 1976). These defects generally undergo a change in configuration with respiration and peristalsis.

Optimal radiological visualization of varices requires adequate mucosal coating and esophageal relaxation and/or collapse. Appropriate mucosal coating is achieved with high-density barium or a barium paste preparation. Esophageal relaxation and/or collapse is achieved by recumbent positioning of the patient (Cockerill et al, 1976). However, in some patients varices are best demonstrated in the upright position (Dodds, 1983). After the barium has been swallowed, it is important that the patient avoid further swallowing to allow the varices to fill. Anticholinergic agents may also be helpful in demonstrating varices (Cockerill et al, 1976; Ghahremani et al, 1972).

Radiologic interventional techniques have been introduced for treating esophageal varices (Funaro et al, 1979; Keller et al, 1983; Lunderquist and Vang, 1974; Uflacker, 1983; Viamonte et al, 1977; Yune et al, 1982). Following percutaneous catheterization of the portal vein and the venous collaterals supplying the paraesophageal plexus, particulate embolic material or a sclerosing substance such as absolute ethanol is injected to occlude the varices. These methods have been successful in controlling acute variceal hemorrhage. However, the recurrence rate of bleeding and recanalization of varices is high (Keller et al, 1983). Thus, percutaneous transcatheter occlusion of varices is best considered a temporizing measure rather than permanent treatment.

## Neoplasms

### Malignant neoplasms

A carcinoma of the esophagus is usually in an advanced stage at the time of diagnosis because it spreads rapidly and often does not produce significant symptoms until it has caused considerable luminal narrowing. Lack of a serosal layer and the existence of a rich periesophageal lymphatic network account for the rapid spread of an esophageal carcinoma to mediastinal lymph nodes and to contiguous structures such as the trachea, bronchi, aorta, and pericardium (Cederquist et al, 1978; Daffner et al, 1979; Drucker et al, 1979). Infradiaphragmatic spread to the liver, adrenal glands, and left gastric lymph nodes is also common.

Approximately 90% of esophageal carcinomas are of squamous cell origin. Of the remaining 10% nearly all are adenocarcinomas. Carcinosarcomas, spindle cell carcinomas, oat cell carcinomas, adenoid cystic carcinomas, lymphomas, and secondary esophageal cancer occur rarely. Individuals using tobacco and alcohol are at high risk for developing esophageal cancer (Wynder and Brass, 1961). Others at higher risk than normal include patients with cancer of the head and neck (Bundrick and Cho, 1983; Gluckman et al, 1980; Goldstein and Zornoza, 1978; McGuirt, 1982), Barrett's esophagus (Haggitt et al, 1978; Levine et al, 1984b; Naef et al, 1975), lye stricture (Lansing et al, 1969), achalasia (Wychulis et al, 1971), sprue (Harris et al, 1967), and tylosis (Harper et al, 1970).

Diagnostic radiology plays two important roles in patients with esophageal carcinomas. The barium swallow examination is the primary method for detecting esophageal tumors. Once the presence of an esophageal tumor has been established, cross-sectional imaging such as CT or MRI is the method of choice for pretherapy staging.

Although the chest radiographs of patients with esophageal carcinomas usually do not provide information specifically relating to the esophageal lesion, certain abnormalities can occasionally be seen. These include a soft tissue mass, an air-fluid level in the esophagus, widening of the mediastinum, anterior bowing of the trachea, and thickening of the retrotracheal stripe (Daffner et al, 1978; Lindell et al, 1979).

On contrast examination an esophageal carcinoma has a variety of morphologic appearances. In early stages it appears as a plaque or a flat sessile polyp (Itai et al, 1978; Koehler et al, 1976). A more advanced tumor appears as an exophytic mass (Fig. 123-26), an annular constricting mass (Fig. 123-27), an irregular segment of esophageal narrowing, or a large ulceration. A carcinoma that diffusely infiltrates the submucosa rarely may have an appearance simulating that of varices (Silver and Goldstein, 1974; Yates et al, 1977) (Fig. 123-28). Such a varicoid tumor can be differentiated from varices by its lack of change in configuration with respiration and peristalsis. A verrucous pattern has been described in tumors associated with lye strictures and achalasia (Sudhor et al, 1980). On CT scanning an esophageal tumor appears as a soft tissue mass or focal wall thickening with an eccentric lumen (Moss et al, 1981) (Figs. 123-29 and 123-30).

The esophagogram is capable of providing some information about the vertical extent of tumor within the esophagus. However, to assess extraesophageal spread of disease, cross-sectional imaging such as CT or MRI is necessary. CT scanning has been reported to be an accurate method for detecting local tumor extension into the aorta and tracheobronchial tree (Figs. 123-30 and 123-31), distant lymphatic spread into the left gastric and celiac lymph nodes, and widespread metastases to the liver and adrenal glands (Moss et al, 1981; Picus et al, 1983; Thompson et al, 1983). Some authors feel that CT underestimates the extent of spread (Mannell et al, 1984; Quint et al, 1985b). However, use of high resolution (ie, 2-mm section) CT scanning may improve detection of periesophageal invasion (Fig. 123-31). CT scanning is not accurate in assessing spread to the periesophageal lymph nodes because these nodes are frequently involved without being enlarged (Picus et al, 1983). However, involvement of these nodes does not necessarily alter the prognosis. CT scanning has also been shown to be useful in following patients with known tumors after radiation therapy or esophagogastrectomy (Heiken et al, 1984) (Fig. 123-32). CT and MRI have a similar accuracy in detecting mediastinal invasion (Halvorsen et al, 1987). A potential advantage of MRI over CT is its superior tissue contrast, which may allow greater sensitivity in the detection of early invasion of the mediastinal fat (Vellet et al, 1990) (Fig. 123-33). The precise role of MRI in both staging and follow-up of patients with esophageal cancer is yet to be determined.

### **Adenocarcinomas**

Esophageal adenocarcinomas occur predominantly in the distal esophagus. Because most adenocarcinomas discovered in the distal esophagus involve the gastric cardia (Fig. 123-34), they are usually interpreted as gastric carcinomas that have secondarily invaded the esophagus (Balthazar et al, 1980; Fierst, 1972; Raphael et al, 1966; Scicchitano and Camishion, 1962; Turnbull and Goodner, 1968). However, Levine et al (1984b) have presented data indicating that a large portion of distal esophageal adenocarcinomas arise primarily in the esophagus on a background of Barrett's epithelium. Regardless of the origin, patients with adenocarcinoma of the gastroesophageal junction have a lower mean survival than their counterparts with more proximal tumors (Papachristov and Fortner, 1980). Furthermore, the gastroesophageal junction is difficult to assess on both CT (Halvorsen and Thompson, 1984) and MRI. Adenocarcinomas and squamous cell carcinomas of the esophagus are indistinguishable radiographically.

### **Miscellaneous tumors**

Carcinomasarcomas and spindle cell sarcomas are rare tumors of the esophagus that have both epithelial and connective tissue elements (McCort, 1972; Moore et al, 1963; Olmstead et al, 1983). They grow as bulky, intraluminal, polypoid masses and often expand the esophageal lumen. Although this appearance is characteristic of these tumors, squamous cell carcinomas may rarely have a similar appearance (Fig. 123-35). Esophageal leiomyosarcomas account for less than 1% of esophageal malignancies. They are slow-growing, late metastasizing tumors occurring chiefly in the distal two thirds of the esophagus. Other sarcomas of the esophagus are exceedingly rare (Camishion et al, 1961). Oat cell carcinoma is another rare primary malignancy of the esophagus that is histologically indistinguishable from pulmonary oat cell carcinoma, but possesses no adrenocortical hypersecretory side effects. It may have a bulky, polypoid configuration (Cook et al, 1976; Olmstead et al, 1983; Rivera et al, 1981) and tends to metastasize early.

Primary adenoid cystic carcinoma (cylindroma) is a rare esophageal tumor that arises from the ducts of esophageal mucous glands (Sweeny and Cooney, 1980). It is indistinguishable histologically from the salivary gland cylindroma but differs by being highly malignant. Adenoid cystic carcinoma cannot be distinguished radiologically from other esophageal carcinomas.

Esophageal melanoma was until recently thought to represent a metastasis from an occult melanoma elsewhere (Goldstein et al, 1977). However, it is likely that in many cases it is a primary malignant tumor of the esophagus (Levine et al, 1989b).

The esophagus is the least common part of the alimentary tract to be involved by a lymphoma. Esophageal lymphoma invariably presents as a late manifestation of a widespread lymphoma elsewhere, reaching the esophagus by localized invasion from the stomach or mediastinal lymph nodes (Agha and Schnitzer, 1985; Carnovale et al, 1977; Goldstein et al, 1981). It is uncertain to what extent the appearance of esophageal lymphoma influences the prognosis of the patient. The radiographic appearance is indistinguishable from that of a carcinoma (Dodds, 1983).

Although metastases to the esophagus are rarely observed radiographically, they are present at autopsy in 3% of patients dying of cancer (Anderson and Harell, 1980). Secondary esophageal tumors may involve the esophagus by direct extension from an adjacent tumor, by extension from mediastinal lymph node metastases, or rarely by hematogenous spread. The tumors that most frequently involve the esophagus are gastric, lung, breast, and pancreatic carcinoma. Less common but important sites for the otolaryngologist are neck cancers, particularly laryngeal, pharyngeal, and thyroid carcinomas. On esophagography the appearance of secondary esophageal involvement depends on the mechanism of spread to the esophagus. Thus, the appearance may be that of an eccentric asymmetric mass, a short, segmental narrowing, or less frequently, a long stricture or intraluminal filling defect (Anderson and Harell, 1980).

## **Benign masses**

### *Neoplasms*

Benign tumors of the esophagus account for close to 20% of all esophageal neoplasms and are found in approximately 0.5% of autopsies (Ming, 1973; Plachta, 1962). The great majority of them are asymptomatic and are found only incidentally.

Leiomyoma is the most common benign esophageal tumor. On radiologic examination it appears as a smooth rounded filling defect that forms a right or obtuse angle with the esophageal wall (Fig. 123-36). Radiographically a leiomyoma is indistinguishable from other less common submucosal lesions such as lipoma, neurofibroma, fibroma, angioma, and granular cell tumor.

The most common mucosal lesion is squamous papilloma, previously thought to be a rare benign epithelial polyp accounting for less than 5% of benign esophageal tumors (Plachta, 1962). However, it is recognized on double-contrast esophagography more commonly than in the past (Montesi et al, 1983). A papilloma appears most commonly as a

solitary small sessile filling defect in the distal third of the esophagus, although multiple papillomas have been described (esophageal papillomatosis). Such papillomas can extend to the larynx (Nuwayhid et al, 1977; Parnell et al, 1978).

Adenomas are rare, accounting for less than 1% of benign esophageal tumors. Most arise in Barrett's mucosa (Levine et al, 1984b; McDonald et al, 1977). Because they are premalignant, removal is indicated (Levine et al, 1984b; McDonald et al, 1977).

### ***Polyps and cysts***

Fibrovascular polyps are nonneoplastic tumors containing varying amounts of fibrous, vascular, and adipose tissue. They occur as pedunculate masses projecting into the esophageal lumen (Jang et al, 1969), arising invariably in the cervical esophagus and often attaining substantial size (Patel et al, 1984).

An inflammatory esophageal polyp is actually the proximal part of a thickened gastric fold extending into the distal esophagus. It is composed of inflammatory cells and granulation tissue and represents a radiologic and pathologic sign of chronic reflux esophagitis. This type of polyp has no malignant potential.

Acquired esophageal cysts (retention cysts) are caused by ductal obstruction of an esophageal mucous gland. They tend to occur in the distal esophagus and are usually multiple but may be solitary. Radiographically they resemble submucosal tumors (Nehme and Robiah, 1977).

## **Trauma**

### **Foreign bodies**

Foreign bodies that lodge in the esophagus usually do so at areas of pathologic narrowing (Fig. 123-37) or anatomic narrowing such as the thoracic inlet, aortic arch, or diaphragmatic hiatus. The most commonly encountered esophageal foreign body is an unchewed bolus of meat that usually lodges just above the gastroesophageal junction. Sharp foreign bodies may lodge anywhere; bones specifically tend to lodge in the proximal third of the esophagus. The great majority of foreign bodies in the esophagus do not require any treatment, but up to 20% require intervention (Webb et al, 1984). Regardless of the method of removal of the foreign body, subsequent studies are required to exclude an underlying pathologic cause of luminal narrowing, especially in an adult.

Signs of perforation such as gas in the mediastinum or in the neck should be sought on the plain radiograph of the chest and neck. Swelling of the prevertebral space may also be observed in cases of perforation by a sharp object. The initial contrast examination should be performed with water-soluble contrast material to exclude esophageal perforation. If evidence of perforation is not found, barium may be used for subsequent swallows. Although contrast esophagography often demonstrates the foreign body, small foreign bodies may go undetected. Once an esophageal foreign body is detected, several methods may be used to remove it. Blunt foreign bodies can be removed endoscopically or under fluoroscopy; the latter method should be performed with a balloon catheter (Nixon, 1979) or a basket (Shaffer

et al, 1986). Food impaction may be relieved with intravenous glucagon, which decreases lower esophageal sphincter pressure (Ferruci and Long, 1977; Trenkner et al, 1983), or with gas-forming agents that distend the esophagus and propel the food into the stomach (Rice et al, 1983). Proteolytic enzymes like papain are discouraged because although they can relieve food impaction, they may digest an adjacent ischemic esophagus and cause perforation (Anderson et al, 1959; Holsinger et al, 1968). If noninvasive attempts to relieve foreign body obstruction are unsuccessful, endoscopic or surgical intervention may be required.

### **Esophageal perforation**

Most esophageal perforations are caused by trauma. Most commonly the trauma is iatrogenic, occurring during intubation, endoscopy, or dilatation procedures. Blunt and penetrating injuries may also result in esophageal perforation. So-called spontaneous esophageal rupture (Boerhaave's syndrome) in fact represents a traumatic esophageal disruption caused by severe vomiting, straining, or a blow to the abdomen or thorax (Rogers et al, 1972). Regardless of the cause, perforation of the thoracic esophagus may be rapidly fatal because of a fulminant mediastinitis. Prompt treatment is therefore mandatory.

Radiographic evaluation of suspected esophageal perforation begins with a radiograph of the chest and neck. Abnormal findings include pneumomediastinum, mediastinal widening, cervical emphysema, or widening of the prevertebral space. A left pleural effusion and pulmonary infiltrates may develop within 12 to 24 hours (Love and Berkow, 1978). Contrast esophagography should be performed initially with a water-soluble agent. If a perforation is not demonstrated in this manner, immediate reexamination with barium sometimes demonstrates a small perforation or mucosal tear (Dodds et al, 1982a; Foley et al, 1982). An esophageal perforation may be confined to a segment of the mediastinum or may communicate with other anatomic spaces such as the pleural cavity. Because an esophageal tear may seal temporarily, a repeat contrast examination may be required for diagnosis (Dodds, 1983).

### **Mallory-Weiss syndrome**

A Mallory-Weiss tear is a mucosal tear at the lower end of the esophagus caused by forceful vomiting (Hastings et al, 1981). Although such an injury may result in brisk bleeding, most tears heal spontaneously within 3 days (Levine, 1989d).

Esophagography is insensitive for the detection of Mallory-Weiss tears and therefore endoscopy is the procedure of choice when this condition is suspected. Selective left gastric angiography can often demonstrate the site of bleeding. In such an instance, infusion of vasopressin through the angiographic catheter is an excellent method for controlling the bleeding. If bleeding is massive or fails to decrease significantly with conservative therapy, transcatheter embolization or endoscopic coagulation of a bleeding vessel may be used (Carsen et al, 1978; Clark, 1979).

## **Esophageal hematomas**

Esophageal hematoma may be spontaneous or secondary to a mucosal laceration, either iatrogenic or spontaneous. Spontaneous hematomas are usually caused by an underlying bleeding disorder.

The majority of traumatic hematomas occur as a single lesion in the distal esophagus. Hematomas caused by underlying compromised hemostasis tend to predominate in the upper and middle esophagus (Shay et al, 1981). Most hematomas have smooth borders and appear as submucosal masses. Less commonly, they have an irregular surface or produce symmetric narrowing of the esophagus. Occasionally a "double-barrel" appearance may be seen (Pellicano et al, 1987).

## **Fistulas**

Esophagotracheal-tracheal fistula, the most common type of traumatic esophageal fistula, is usually caused by radiation treatment of an esophageal cancer or, less commonly, occurs secondary to trauma. Other nontraumatic causes include neoplasia without radiation or various mediastinal infections (DeSilva et al, 1990). Esophagopericardial fistulas can be caused by trauma (such as swallowed foreign bodies or surgery) or they can develop secondary to esophageal inflammation or cancer (Cyriak et al, 1983). An esophagoaortic fistula is sometimes seen as an iatrogenic complication of aortic prosthetic graft surgery (Seymour, 1978) or as being caused by a swallowed foreign body. Nontraumatic causes include ruptured aortic aneurysm or esophageal carcinoma. Esophagopleural fistulas are usually iatrogenic (Wechsler et al, 1982), but can also be secondary to invasive esophageal cancer.