

Shackelford's
Surgery of the
Alimentary
Tract

VOLUME

I



SIXTH
EDITION

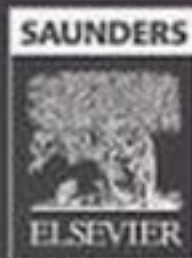
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Shackelford's Surgery of the Alimentary Tract

6th Edition

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*To my wife, Theresa, and my children, William and Katerina;
to my many mentors (alive and deceased) who contributed to the science of surgery;
and to the many colleagues and friends whose work made this sixth edition possible.*

CHARLES J. YEO

To my wife, Barbara, and my son, Patrick.

DANIEL T. DEMPSEY

*To my wife, Julia, and my sons, Jeffrey, David, and Alexander
for their support, their understanding, and their willingness to savor life's adventures.*

ANDREW S. KLEIN

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who each challenged me from the start to aim high;
to my colleagues who supported wordlessly (usually!) these desires,
and to my family, who put up with all of this for so long—my deepest respect, profound thanks, and love.*

JOHN H. PEMBERTON

*To the hard work and dedication of surgeons
struggling with esophageal disease throughout the world
and to those few who have generously shared their wisdom and experience
in my personal education toward the successful management
of diseases of the esophagus.*

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Operations for Morbid Obesity

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New Developments in Chronic Pancreatitis: Before Head Resection, Try Endoscopic Treatment First

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Laparoscopic Esophageal Myotomy: Techniques and Results

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Esophageal Cancer: Current Staging Classifications and Techniques, Endoscopic Ultrasound, and Laparoscopic and Thoracoscopic Staging

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Minimally Invasive Surgical and Image-Guided Interventional Approaches to the Spleen

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Management of Splenic Trauma in Adults

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Human Foregut Anatomy, Prenatal Development and Abnormalities, and Their Relation to Surgical Approaches; Esophageal Mucosal Injury and Duodenal Reflux

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Pseudocysts and Other Complications of Pancreatitis; Operative Management of Strictures and Benign Obstructive Disorders of the Bile Duct

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Carcinoma of the Esophagus and Gastroesophageal Junction

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Surgical Treatment of Constipation

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Techniques of Esophageal Reconstruction

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Splenectomy for Conditions Other Than Trauma; Resection and Ablation of Metastatic Colorectal Cancer to the Liver

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Crohn's Disease: General Considerations, Medical Management, and Surgical Treatment of Small Intestinal Disease

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*Epidemiology, Risk Factors, and Clinical Manifestations of
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*Endoscopic Retrograde Cholangiopancreatography in the Evaluation
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*Management of Malignant Hepatic Neoplasms Other Than
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Management of Hepatobiliary Trauma

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*Perioperative Management and Nutrition in Patients with Liver and
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Cysts and Tumors of the Spleen

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Pseudocysts and Other Complications of Pancreatitis

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Minimally Invasive Surgical and Image-Guided Interventional Approaches to the Spleen

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Cystic Disorders of the Bile Ducts

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Unusual Pancreatic Tumors

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*New Developments in Chronic Pancreatitis: Before Head Resection,
Try Endoscopic Treatment First*

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*Epidemiology, Risk Factors, and Clinical Manifestations of
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*Endoscopic Retrograde Cholangiopancreatography in the Evaluation
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Esophageal Replacement for End-Stage Benign Esophageal Disease

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*Surgical Diseases of the Stomach and Duodenum in Infants and
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Preface

It is with great delight that the section editors and I present the sixth edition of *Shackelford's Surgery of the Alimentary Tract*. This encyclopedic set has served as an invaluable resource for surgeons, internists, gastroenterologists, residents, medical students, and other medical professionals over the past 50 years. I know that you will find this sixth edition educationally fulfilling, nicely illustrated, and up-to-date.

The first edition of *Surgery of the Alimentary Tract* was written by Dr. Richard T. Shackelford and published in 1955. Following the success of that first edition, the W. B. Saunders Company urged Dr. Shackelford to produce a second edition. Between 1978 and 1986 consecutive volumes were released, culminating in a five-volume set that had been expanded substantially from the first edition. Dr. George D. Zuidema was added as a co-editor. It was this second edition that served as my "bible" for alimentary tract diseases during my surgical residency and early faculty appointment.

The third edition, edited by Dr. Zuidema, was published in 1991 and proved to be an important step forward. The field of alimentary tract surgery had advanced, and many emerging techniques and new research findings were included in that edition. For that third edition, Dr. Zuidema enlisted the help of a guest editor for each of the five volumes.

The fourth edition, which was published in 1996, was encyclopedic in scope, breadth, and depth of coverage. This led it to be consulted as the classic reference source for surgeons, internists, gastroenterologists, and others involved in the care of patients with alimentary tract diseases.

In 2002, the fifth edition was published. I was delighted that Dr. Zuidema asked me to join him as a co-editor for that edition. Its publication nicely presented numerous changes in surgical practice, operative techniques, molecular biology, and noninvasive therapies. The world of alimentary tract surgery had continued to change, and the textbook reflected these changes.

This current sixth edition represents even more change, both for the field of alimentary tract surgery and for the textbook itself. All involved listened to the book's many users and have made substantial changes in the look and content of the text. The book has gone from five volumes to two volumes, while adding material and including a four-color production scheme. The authors have emphasized new procedures, including endoscopic and minimally invasive ones, and advances in technology. Dr. Zuidema, who was involved with the second through the fifth editions, has passed the baton, but he remains an inspiration to all those in the field of alimentary tract

surgery. I am delighted to keep this project moving forward and have done so with his blessings and oversight from afar.

This sixth edition has been completed with an enormous amount of help from four colleagues, who have served as section editors for the four major sections of the book. These section editors have worked tirelessly planning, organizing, and developing this massive textbook. They have incorporated numerous changes in surgical practice, operative approaches, and noninvasive therapies within the text. Each area retains extensive sections on anatomy and physiology but then directs attention to both standard and cutting edge innovations. This sixth edition includes the contributions of two new and two retained section editors, in order to provide both innovation and stability.

Section I, "Esophagus and Hernia," is now edited by Dr. Jeffrey H. Peters, the Seymour I. Schwartz Professor and Chairman of the Department of Surgery at the University of Rochester School of Medicine and Dentistry in Rochester, New York. Dr. Peters is a world-renowned expert who brings his detailed knowledge of the esophagus and esophageal diseases to the textbook. He has put together a spectacular section on esophageal diseases, focusing on esophageal pathology and ambulatory diagnostics, gastroesophageal reflux disease, esophageal motility disorders, and esophageal neoplasia. This represents an entirely new presentation of esophageal diseases in *Shackelford's Surgery of the Alimentary Tract*, sixth edition.

For Section II, "Stomach and Small Intestine," Dr. Daniel T. Dempsey has expanded his previous contribution by taking on the jejunioileum as part of his section. Dr. Dempsey is Professor and Chairman of the Department of Surgery at Temple University School of Medicine in Philadelphia, Pennsylvania. He has done a superb job of merging both standard and innovative areas in this field. New to the section are discussions of upper gastrointestinal foreign bodies and bezoars, as well as entirely redone sections dealing with neoplasia, gastrointestinal stromal tumors, and vascular diseases. Dr. Dempsey's section is an outstanding contribution to this area, advancing the field to new heights.

For Section III, "Pancreas, Biliary Tract, Liver, and Spleen," we have a new section editor, Dr. Andrew S. Klein. Dr. Klein is the Esther and Mark Schulman Chair in Surgery and Transplant Medicine and Director of the Cedars-Sinai Comprehensive Transplant Center in Los Angeles. Dr. Klein has put together a tremendous hepatopancreaticobiliary (plus spleen) section, including new contributions about acute pancreatitis, chronic

pancreatitis, cystic neoplasia of the pancreas, and laparoscopic approaches to biliary and liver diseases. Also included are top level discussions of fulminant hepatic failure and the bio-artificial liver, drug-induced liver damage, and extensive operative sections dealing with liver resection and liver transplantation. Dr. Klein has taken a previously very well done section and made it even better.

The last section, Section IV, “Colon, Rectum, and Anus,” has again been supervised by Dr. John H. Pemberton, Professor of Surgery at the Mayo Clinic College of Medicine in Rochester, Minnesota. Dr. Pemberton is a world-renowned figure in his field, and his section has been nicely reworked. Included are new developments in the field, a better understanding of pelvic floor anatomy and physiology, updates regarding diagnosis and interventions for inflammatory bowel disease, as well as the addition of more extensive laparoscopic interventions and their outcomes.

This sixth edition would have been impossible without the hard work of each of these section editors. They have been helped immensely by their colleagues, staff, and all of the chapter contributors. I would like to thank each of these section editors for their hard work, vision, and skill in bringing this project to its fruition.

Very importantly, I would like to express my appreciation to the more than 300 individuals who have contributed chapters to this new, sixth edition. I understand how difficult it is to produce superb chapters, and I wish

to recognize these individuals and thank them for their dedication and commitment. Many of the contributors here are topnotch, world class leaders in their fields, and I am deeply indebted to them for sharing their knowledge and enthusiasm, culminating in an outstanding product.

I would also like to thank the production team at Elsevier/W.B. Saunders, who have been instrumental in making this edition a reality. My thanks go out to Judith Fletcher, Kim Davis, Amy Cannon, and many others, who have been instrumental in overseeing this project. This edition represents an immense amount of new work, redesign, and illustration. These professionals have made it a labor of love to work on this project.

Finally, I must thank individuals who helped me during this process over the past 3 years. The majority of the early correspondence, mailings, and editorial oversight originated in the Department of Surgery at the Johns Hopkins University School of Medicine in Baltimore. My thanks go out to Janet Romanelli and Irma Silkworth for providing me with this support. Additionally, within the past year, Mary Toelke in my office here at the Thomas Jefferson University Hospital and the Jefferson Medical College has been an outstanding assistant and editor, providing me with tremendous support here in Philadelphia.

Charles J. Yeo, MD
Philadelphia, Pennsylvania

The Normal Esophagus

Perspectives on
Esophageal Surgery

Tom R. DeMeester

To write a perspective on a subject is to clearly view a subject through a medium, usually an optical glass such as spectacles or some form of scope. In this instance the scope is history or, if you prefer, the retrospective scope. The accumulation of human experience makes up history and, according to C. S. Lewis, “authority, reason and experience; on these three, mixed in varying proportions all our knowledge depends.”¹ If today’s esophageal surgeon desires to stand on the shoulders of those who went before us and not repeat their mistakes, the knowledge and appreciation of important milestones in esophageal surgery must be appreciated and embraced.

To understand a surgical disease requires the capacity to see and touch the affected tissue. Until the science of surgery was translated to human patients, autopsy reports provided most of our understanding of esophageal disease. They consisted largely of spontaneous perforations (Boerhaave’s syndrome)² and tumors of the esophagus and provided little to the understanding of benign inflammatory disease, such as esophagitis. This is because autolysis of the distal esophageal mucosa by digestive enzymes occurred during the interval between death and autopsy. Any tissue injury around the gastroesophageal junction was assumed to be a postmortem change, much like the organism *Helicobacter pylori* was assumed to not be a pathogen in the stomach. Consequently, the existence and pathologic description of esophagitis and inflammatory strictures were not appreciated until Heinrich

Quincke, a German internist, brought attention to them through his publication on esophageal ulcers in 1879.³

Further, the remote inaccessibility of the esophagus in the posterior mediastinum surrounded by the lungs and heart deterred understanding of diseases that affect the organ until the introduction of rigid esophagoscopy 130 years ago by Bevan in 1868,⁴ Kussmaul in 1868,⁵ and Mikulicz in 1881.⁶ Subsequently, several breakthroughs in technology permitted complete and safe endoscopic examination of the entire esophagus, stomach, and duodenum. First was the invention of the incandescent light bulb by Thomas Edison in the 1870s. Second was the introduction of the rod-lens system by Hopkins in the 1950s. Third was the development of fiberoptic cold-light transmission in the 1960s. Last was the evolution of the computer chip video camera in the 1980s.⁷ Combined, these technologic advancements provided reliable clinical esophagoscopy with the ability to directly examine and biopsy the esophageal mucosa. This ability opened the door to understanding the pathophysiology of esophagitis, stricture, and Barrett’s esophagus with its inherent cancer risk.

ESOPHAGEAL CANCER

Cancer of the esophagus was a unique challenge for the surgeon. For decades, surgical pioneers have struggled with safe removal of the diseased organ. Emslie in

his “Perspectives in the Development of Esophageal Surgery” states, “the history of esophageal surgery is the tale of men repeatedly losing to a stronger adversary yet persisting in this unequal struggle until the nature of the problems became apparent and the war was won.”⁸ The major obstacles were the continuation of respiration with an open thorax and the restoration of alimentary tract continuity after esophageal resection.

The first successful esophagectomy for squamous cell carcinoma was performed by Franz Torek.⁹ General anesthesia was administered by a new technique called insufflation, in which ether was delivered through a woven silk tube used to intubate the patient. The existing technique of a differential pressure chamber was not considered because the rubber cuff around the patient’s neck, used to create subatmospheric pressure about the body, prevented construction of a cervical esophagostomy. The esophagus with a cancer abutting the left main bronchus was removed by a transthoracic transpleural exposure. Dr. Torek avoided injury to the vagi and the possibility of “sudden death due to vagal collapse” by carefully dissecting them off the esophagus. His fear of vagal circulatory collapse is reflected in his statement: “At the site of the tumor the dissection of the vagi was more difficult, and some of the branches crossing over in front of it had to be cut in order to permit liberating the tumor without undue roughness in handling the vagi. To my great satisfaction the pulse never wavered during the procedure, remaining between 93 and 96. The dreaded vagus collapse had, therefore, been safely avoided.”⁹ A pleural infection from an esophageal leak was circumvented by carefully closing the cardia and performing a cervical esophagostomy. The reported existence of extensive adhesions between the left lung and the parietal pleura in all probability prevented collapse of the left lung and contributed as much to the success of the procedure as Torek’s surgery. The patient recovered and survived for another 13 years, with continuity between the cervical esophagostomy and gastrostomy established by an external “rubber tube.”

The fact that 20 barren years intervened between the first and second successful procedure testifies to the challenge that removal of the esophagus posed to surgeons. Wolfgang Denk took up the challenge and developed a totally different approach to resection of the thoracic esophagus.¹⁰ He showed in cadavers that the esophagus could be removed by blunt dissection through the combination of an abdominal transhiatal and a cervical transthoracic inlet approach. This technique, knowingly or unknowingly, was used in the second successful esophagectomy reported by Grey Turner in 1933.¹¹ As suggested by Denk, the procedure was performed without opening the chest by blunt burrowing from the abdomen and neck. The esophagus with a midconstricting neoplasm was successfully removed. Alimentary tract continuity was re-established 7 months after the esophagectomy by a second procedure connecting the cervical esophageal and abdominal gastric stomas by a subcutaneous skin tube.

While surgeons struggled with esophagectomy, advances in anesthesia continued. The description of an intratracheal tube with an inflatable cuff by Theodore

Tuffeir in 1896¹² and its introduction into clinical practice in 1928 by Magill¹³ allowed the development of positive pressure anesthesia and the direct transthoracic approach to the esophagus. Similarly, experimental work on restoration of the alimentary tract after esophageal resection continued. Claude Beck in 1905 showed in animals that a tube constructed along the greater curvature of the stomach could be used to replace a portion of the esophagus.¹⁴ Cesar Roux in 1907 developed the technique of using the jejunum to replace the distal end of the esophagus.¹⁵ G. Kelling devised a method of using an isoperistaltic segment of transverse colon to completely replace the thoracic esophagus.¹⁶

In the wake of these accomplishments, it is not surprising that the final successful step of performing an esophagectomy with an intrathoracic esophagogastric anastomosis was reported by Tatsuo Ohsawa from Japan in 1933.¹⁷ He successfully performed a simultaneous esophagogastrectomy and esophagogastrostomy in eight patients with carcinoma of the lower esophagus and cardia. No follow-up is available on Ohsawa’s patients, and unfortunately his paper did not reach the attention of the Western world for 5 years. Samuel Marshall from the United States reported a similar procedure in one patient in 1938. However, this patient was plagued by persistent esophageal obstruction and esophagitis that required repetitive dilation.¹⁸

With initial success, surgeons realized that performing a dependable intrathoracic esophagogastric anastomosis was a major part of the challenge. Infection of the mediastinum and pleural cavities because of disruption of the anastomosis was the most frequent cause of failure of the operation. Adams and Phemister took the problem to the laboratory, and only when a high degree of success was attained in dogs was a similar anastomotic procedure applied to humans with carcinoma of the thoracic esophagus. Their report in 1938 popularized the one-stage resection for esophageal cancer with an intrathoracic esophagogastrostomy.¹⁹

Today, challenges still remain in the surgical treatment of esophageal cancer. Questions of temporal interest include the following: Does en bloc esophagogastrectomy reduce the incidence of local recurrence of cancer that occurs after more limited resections? Are limited resections for early cancer sufficient to eradicate the disease and are they superior to endoscopic methods of resection? Is a vagal-sparing esophagectomy without lymphadenectomy a less morbid and safer procedure, and is it adequate therapy for early disease?

In the history of surgical practice, therapy for carcinoma of the esophagus carries an aura of pessimism with an attitude that cure is a chance phenomenon. This setting has given rise to two current treatment philosophies. First is that surgical removal of the primary tumor is the goal of therapy and the need for lymph node dissection is of limited benefit. Second, is that surgery alone is insufficient therapy and neoadjuvant or adjuvant radiation therapy or chemotherapy (or both) is necessary to achieve cure. This philosophy persists even though contemporary surgical experience has validated that complete surgical resection of an early tumor and limited nodal disease can cure a patient of esophageal

cancer with an effectiveness better than that achieved by any other single or combined therapy.

ESOPHAGEAL MOTILITY DISORDERS

Surgical therapy for esophageal motility disorders started with the treatment of achalasia. *Megaesophagus*, or *achalasia* as it later came to be known, was first described by Willis in 1674. He advocated the use of a small sponge attached to a long strip of whalebone to force impacted food through the narrow distal esophagus.²⁰ Arthur Hurst showed that an abnormality of the intermuscular nerve plexus was responsible for the disease. He named the disease achalasia of the cardia because the continued tonic contraction of the cardiac sphincter prevented esophageal emptying. Hurst devised rubber tubes of various size with blunt tips filled with mercury to dilate the tonic sphincter. They are now referred to as Hurst dilators and were subsequently modified with tapered tips and called Maloney dilators.²¹

The initial surgical procedures used to relieve a spastic cardia were designed to enlarge the narrowed gastroesophageal junction with various cardioplasties of the Heineke-Mikulicz or Finney pyloroplasty type or to bypass the junction with an esophagogastrostomy. Ernst Heller in 1914 described a simple myotomy for the treatment of achalasia with the suggestion that it replace the more dramatic operation being performed.²² The operation was based on Ramstedt's pyloromyotomy developed in 1912. Ramstedt's operation was immediately accepted by other surgeons for the treatment of congenital pyloric stenosis.²³ In contrast, despite knowledge of Heller's myotomy for achalasia, the procedure was seldom used and largely ignored in Germany, England, and the United States. Part of the problem of acceptance was the unknown etiology of achalasia, the absence of a histologic lesion, and disagreement over the nature of the physiologic abnormality and hence the purpose of the operation. According to Ravitch²⁴ this situation was changed dramatically by a paper from Norman Barrett in 1949 in which he described dismal results after esophagogastrostomy or cardioplasty operations.²⁵ Phillip Allison,²⁶ Barrett, and others were studying reflux esophagitis at the time and pointed out that destroying or bypassing the gastroesophageal junction encouraged esophagitis of such severity that patients suffered heartburn, would not eat, and bled seriously. Barrett proposed Heller's operation as an alternative and reported success with it. Barrett encouraged the use of Groeneveldt's modification of Heller's operation, specifically, performing only one myotomy instead of two. Barrett's paper and the increased awareness and interest in esophagitis led to widespread acceptance of the Heller procedure as the primary mode of operative therapy for achalasia. Dor in 1962²⁷ and Toupet in 1963²⁸ developed antireflux repairs to be used in conjunction with Heller myotomy to provide further protection against the sequelae of esophagitis. Eventually, gastroenterologists were able to rupture the muscle of the cardia with pneumatic dilators and obtain results close to those of surgery. This, along with the fear of surgery and the custom of patients first

contacting the gastroenterologist, led to a decrease in referral of patients for surgical myotomy. The recent introduction of laparoscopic myotomy with its greater safety and minimal morbidity has reversed this trend.

Franz Ingelfinger²⁹ in 1959 and Charles Code³⁰ in 1958 introduced esophageal manometry to clarify the diagnosis of achalasia and identify other esophageal motility disorders such as diffuse spasm and hypertensive lower esophageal sphincter. These latter conditions very rarely require myotomy of the esophageal body or lower esophageal sphincter.

Today, laparoscopic myotomy is the accepted therapy for achalasia. The procedure has been standardized in that most esophageal surgeons perform a myotomy that extends at least 3 cm onto the stomach and add a partial fundoplication to reduce the reflux of gastric juice into the esophagus. The location of the myotomy, either in the anterior quadrant between the "clasp" and "oblique" fibers or in the left lateral quadrant in line with the greater curvature and cutting only the "oblique" fibers, is still debated. The performance of a surgical myotomy is the creation of a defect to correct a defect and, consequently, can never restore the function of the cardia to normal. Therefore, a modified Heller myotomy is a palliative procedure.

ESOPHAGEAL DIVERTICULUM

The first description of a pharyngoesophageal diverticulum is attributed to Abraham Ludlow. He observed the abnormality at an autopsy he performed and reported the finding to William Hunter, John's brother, in 1764. Ludlow eventually published the observation in 1767.³¹ Today, Ludlow's autopsy specimen is registered in the Hunterian Museum. Sir Charles Bell, a surgeon who described Bell's palsy, was the first to define the abnormalities necessary for the development of a pharyngoesophageal diverticulum.³² Before Bell's publication in 1816, the diverticulum was thought to be congenital or traumatic in origin. The two components that Bell identified as necessary for a diverticulum to form were discoordination of the inferior pharyngeal constrictors and the cricopharyngeus muscle and a preexisting anatomic defect between these muscles. These observations predated our modern acceptance of them by 100 years.

The first successful resection of a pharyngoesophageal diverticulum was performed in 1886 by a surgeon with the last name of Wheeler on a patient named Captain E.³³ Diverticulectomy became the standard form of treatment, but the incidence of salivary fistulas and late recurrence was high. This prompted Girard from France in 1896 to treat two patients by invagination of the diverticulum into the lumen of the esophagus and oversewing the resultant dimple.³⁴ This approach was apparently successful, but in subsequent follow-up of the patients, at least one had a complete recurrence. Diverticulopexy was also described during this early period as a means of avoiding contamination of the wound and fistula formation.

The dangers of surgical therapy for pharyngoesophageal diverticula were reported in 1906 by Zesas,³⁵

who collected 42 patients from published reports and noted that primary healing occurred in only 6, fistulization in 26, and death in 8, for a mortality rate of 19%. To avoid the devastating results, Goldmann in 1909³⁶ devised a two-stage method of repair that was later modified by Lahey and Warren in 1954.³⁷ The modified procedure consisted of diverticulopexy and mediastinal packing in the first stage and resection of the diverticulum in the second. In 1945 the one-stage operation was readvocated by Harrington.³⁸ The battle between the protagonists of one-stage and two-stage resection continued for years and diverted attention from identifying the etiology of the diverticulum. Aubin, in 1936, was the first to propose, based on Bell's observations, a rational treatment of a pharyngoesophageal diverticulum that consisted of cricopharyngeal myotomy combined with diverticulectomy.³⁹ His publication refocused attention on the underlying pathology in the skeletal muscle of the cricopharyngeal sphincter and cervical esophagus. His report led to the gradual abandonment of the two-stage operation. In 1966 Ronald Belsey,⁴⁰ in keeping with the desire to avoid contamination of the wound and fistula formation, advocated cricopharyngeal and cervical esophageal myotomy with diverticulopexy for all but very large diverticula.

The story of the pharyngoesophageal diverticulum is an object lesson from the history of surgery. It illustrates that medicine is a science often forced to be practiced before it is understood. It is not uncommon for observations, which form the bases for successful therapy, to be initially ignored or overlooked, rediscovered, and then adopted years later, in this example 2 centuries later!

In 1840 Rokitansky⁴¹ described traction diverticula of the thoracic esophagus but was uncertain about their etiology. He thought that they were due to pressure from ingested food or obstruction of the distal esophagus by a stricture or extrinsic compression. Excision of an intrathoracic diverticulum was rarely reported, probably because of the disastrous results from leakage and fatal mediastinal and pleural sepsis. Moreover, considerable confusion existed during the middle of the 19th century regarding the etiology of the different diverticula affecting the esophagus. The confusion was resolved largely by the pathologist Albert Zenker, who with von Zeimssen in 1877 published "Krankheiten des Oesophagus," the best compendium of information on the esophagus in the latter part of the 19th century.⁴² They introduced for the first time the separation of diverticula into two etiologic categories: traction and pulsion. The former is caused by inflammatory adhesions and the latter by forces within the esophageal muscular tube. The concept was quickly accepted, but confusion persisted with regard to terminology. The concept was further supported when esophageal manometry confirmed that development of a pulsion diverticulum was a complication of a motility disorder rather than a primary anatomic abnormality.⁴³ The major obstacle to accepting the concept was the inconsistency in identifying a motility disorder in all patients with a pulsion diverticulum. This inconsistency led to controversy over the necessity for primary correction of the motility abnormality before any direct attack

was made on the diverticulum. With technical improvements in esophageal manometry, 24-hour ambulatory motility studies became possible and showed, in all patients who had a pulsion diverticulum, a disordered motility pattern distal to the diverticulum. Today, the combination of myotomy of the esophagus distal to the diverticulum, including the lower esophageal sphincter, resection of the diverticulum, and a Dor partial fundoplication has become the standard procedure.²⁷

HIATAL HERNIA AND GASTROESOPHAGEAL REFLUX DISEASE

In 1853 Henry Ingersoll Bowditch commented on hiatal hernia in his published monograph titled *A Treatise on Diaphragmatic Hernia*: "Owing to the ignorance of most of the observers in regard to the true nature of the affection, their modes of treatment have been entirely empirical and generally very absurd, and not a few times absolutely hurtful to the patient."⁴⁴ Even though Heinrich Quinche described esophagitis in 1879,³ symptoms of the abnormality were poorly understood and no consideration was given to reflux of gastric contents up into the esophagus as its cause. In 1928 Harrington reported on 51 patients with a diaphragmatic hernia and concentrated only on describing the anatomic defect and closure of the hiatus for therapy without discussing symptomatology.³⁸ It was not until Philip Allison's publication in 1951²⁶ that the symptoms associated with a hiatal hernia were linked to the reflux of gastric contents into the esophagus. Allison used the term reflux esophagitis to describe the cause of the symptoms and emphasized correction of the defect at the cardia as the proper therapy. The term reflux esophagitis was confusing to gastroenterologists, who emphasized increased gastric acidity as the major problem and advocated reduction of gastric acid and peptic secretion as a means of treating the esophagitis rather than stopping the reflux. This started a lasting controversy between gastroenterologists and surgeons. Gastroenterologists emphasized the use of bougies, antacids, and advice on posture, and surgeons devised operations to restore sphincter competence and sought methods to objectively select patients for the procedure.

Allison described the first logical hiatal hernia repair by emphasizing repositioning of the gastroesophageal junction into its normal intra-abdominal location in the hope of improving its function.²⁶ Recognition of the high incidence of symptomatic and anatomic recurrence after the Allison repair led to the development of procedures designed to place and anchor the lower esophagus in the intra-abdominal position in a more effective manner. A posterior gastropexy in which the phrenoesophageal membrane and the cardioesophageal junction are anchored to the median arcuate ligament of the aortic hiatus was devised, used, and reported by Lucious Hill in 1967.⁴⁵ Two additional operations, the Nissen fundoplication introduced in 1956⁴⁶ and the Belsey Mark IV introduced in 1967,⁴⁷ were designed to augment the lower esophageal sphincter with a cuff of stomach, as well as re-establish an intra-abdominal segment of esophagus.

An important contribution was made in 1957 by Lee Collis in the management of advanced gastroesophageal reflux disease when reflux-induced intramural fibrosis causes esophageal shortening. He worked out a technique to add 4 cm to the length of the esophagus by the creation of a proximal gastropasty tube, around which later surgeons applied a partial or full fundoplication.⁴⁸

Norman Barrett in 1950⁴⁹ opened a whole new era in esophageal disease that ultimately connected benign gastroesophageal reflux disease with esophageal adenocarcinoma, one of the most devastating cancers known to affect humans. He reported his experience on columnar-lined esophagus with accompanying esophagitis and ulceration. He thought that the condition was due to congenital shortening of the esophagus but was subsequently proved wrong by Allison and Johnstone in 1953, who noted normal esophageal musculature and esophageal submucosal glands underneath the columnar epithelium. They reported that the change in epithelium was acquired as a result of erosive injury of the squamous mucosa.⁵⁰ In 1975 Naef and Ozzello⁵¹ cautioned that the acquired columnar epithelium had a predisposition to malignant change. In 1978 Haggitt⁵² suggested and subsequently Skinner⁵³ and Reid⁵⁴ confirmed that only intestinalized columnar mucosa was associated with malignant degeneration.

The Nissen fundoplication, because of its simplicity and effectiveness, was rapidly adopted worldwide as the procedure of choice for gastroesophageal reflux disease. Dorothea Liebermann-Meffert, a personal friend of Nissen, archived the historical development of the Nissen fundoplication.⁵⁵ The first step toward the operation occurred in 1937 when Rudolf Nissen, then in Istanbul, Turkey, operated on a 28-year old man with a chronic bleeding ulcer in the distal esophagus. He resected the cardia and anastomosed the esophageal stump into the gastric fundus. To protect the anastomosis he covered the esophagogastronomy with a cuff of stomach. Sixteen years later Nissen had the opportunity to re-examine the patient, and in contrast to the usual experience after resection of the cardia and esophagogastronomy, the patient was free of symptoms and signs of gastroesophageal reflux. The second step toward fundoplication occurred in 1946 when Nissen, then in New York, performed a transabdominal reduction of a paraesophageal hernia in a patient who refused a thoracotomy. He was surprised by the ease with which the hernia could be reduced and the degree of exposure of the esophageal hiatus through a transabdominal incision. The third and final step occurred in 1954 when Nissen, then in Basel, Switzerland, combined the two previous observations into a planned antireflux procedure in a patient suffering from severe gastroesophageal reflux disease. He formed a fold from the anterior and posterior gastric fundic walls and attached both to each other on the lesser curvature side of the stomach above the gastroesophageal junction. The clinical outcome, a complete success, could be reproduced in a subsequent patient. In the publication of the procedure in *Schweizer Medizinische Wochenschrift* in 1956 he termed the operation “gastroplication” and described it as a “simple and effective operation for reflux esophagitis.”

During the 1960s and 1970s gastroesophageal reflux disease was accepted as a distinct disease entity independent of hiatal hernia. With the introduction of water perfusion esophageal manometry in 1956, the lower esophageal sphincter was identified as the major barrier against the reflux of gastric contents, and the physiology of barrier augmentation by a surgical antireflux procedure was clarified. The availability of 24-hour pH monitoring in 1974 allowed gastroesophageal reflux disease to be defined quantitatively and improved the selection of patients for antireflux surgery.⁵⁶ In 1991 Bernard Dallemagne of Liege, Belgium, performed the first known human laparoscopic Nissen fundoplication.⁵⁷ Successful laparoscopic ligation of the short gastric vessels and safe posterior dissection of the abdominal portion of the esophagus were the significant accomplishments at the time. Today, laparoscopic Nissen fundoplication has become commonplace. Its safe, effective, and user-friendly characteristics have positioned surgical therapy for earlier application in the treatment of gastroesophageal reflux disease.

THE ESOPHAGEAL SURGEON

The esophagus has never had a sizable patronage. This is well illustrated in a vignette recorded by Earle Wilkins about Dr. Willy Meyers, who reported successful esophageal resection at the annual meeting of the American Medical Association in 1903. The report was met with indifference and no discussion. The obvious lack of interest among physicians for problems concerning the esophagus was the direct impetus for Dr. Meyer to take the lead, with a small group of “interested” surgeons, and form the American Association for Thoracic Surgery, the founding organization in the clinical specialty of thoracic surgery.⁵⁸ Esophageal surgery, despite being the spark that ignited the first society for thoracic surgery, was soon crowded out by the burgeoning business of coronary bypass surgery. Consequently, over the years the esophagus has been used, sometimes ill-used and sometimes ignorantly used, by gastroenterologists, otolaryngologists, thoracic surgeons, general surgeons, and oncologic surgeons. There have been no specialty hospitals erected to care exclusively for esophageal illnesses. There have been no departments or clinics devoted exclusively to the diagnosis and treatment of esophageal diseases. Many hospitals did not have staff familiar with the postoperative care of esophageal patients. Surprisingly, such clinics are developing today, probably aided by the necessity for an esophageal laboratory to unsnarl complex esophageal disease, an awareness of the relationship of esophageal to pulmonary disease, and the metaplasia-dysplasia-carcinoma sequence in Barrett’s esophagus. Virtual esophageal motility, wireless esophageal pH monitoring, esophageal impedance measurements, endoscopic ultrasound, and a variety of endoscopic diagnostic and therapeutic procedures are now commonplace and have accelerated the status of individual esophageal units. If the anatomic demarcation of the gastrointestinal and cardiothoracic surgeons could give way and the pharynx, esophagus, lungs, and stomach be coalesced, there could

be the advent of a new therapist—a foregut or esophageal surgeon who is competent at endoscopy, as skilled in transthoracic as in transabdominal operations, at home in the esophageal laboratory, and an expert at unsnarling complex foregut problems.

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Human Foregut Anatomy, Prenatal Development and Abnormalities, and Their Relation to Surgical Approaches

Dorothea Liebermann-Meffert ▪ Hubert J. Stein

Anatomy of the Esophagus

MACROSCOPIC FEATURES

General Aspects

The esophagus is a midline structure lying on the anterior surface of the spine. It descends through three compartments: the neck, the chest, and the abdomen. This progression has led to its classic anatomic division into cervical, thoracic, and abdominal segments (Fig. 2-1). Two new subdivisions more useful for clinicians have recently been proposed (see Fig. 2-1). One refers to functional aspects and makes a distinction between the esophageal body and the upper and lower sphincters.¹ The other refers to oncosurgery and distinguishes between the proximal and the distal esophagus, with the tracheal bifurcation used as a partition.² This concept integrates the features of embryologic development, in particular, the differently oriented pathways of lymphatic drainage (see the section “Lymphatic Drainage” later in this chapter).

The topographic relationships of the esophagus to its neighboring structures have been studied extensively by the authors and other experts using different technical approaches. The conclusions are as follows:

Joining the pharynx, the esophagus begins at the cricoid cartilage in front of the sixth cervical vertebra. It passes into the chest at the level of the sternal notch and travels within the chest cavity on the anterior limit of the

posterior mediastinum. Between the thoracic inlet and the diaphragm, the esophagus remains in close relationship with the spine (Fig. 2-2). It ends at the inlet of the stomach, in front of the 12th thoracic vertebra. On radiologic evaluation, the esophageal axis is virtually straight. Unaffected by scoliotic curves of the vertebral column, the esophagus maintains a straight course; in contrast, the large neurovascular structures, because of their origin at the posterior body wall, follow the deformity of the skeleton.³ Vascular anomalies or mediastinal masses, on the other hand, may displace, bow, or indent the esophagus. However, any distortion of its axis strongly suggests mediastinal invasion and retraction, usually by a malignancy.⁴

A healthy esophagus has three minor deviations along its trajectory (see Fig. 2-1). The first is toward the left at the base of the neck (see Fig. 2-2); hence surgical approaches to the esophagus are easier from the left than from the right when performing intestinoesophageal anastomoses after esophagectomy. The second deviation is at the level of the seventh thoracic vertebra, where the esophagus turns slightly to the right of the spine (see Fig. 2-1). Because of the third deviation, the terminal esophagus and the esophagogastric junction are positioned slightly lateral to the xiphoid process of the sternum and to the left of the spine. At this point, the fundus and proximal part of the stomach extend anterolateral to the body of the vertebra (see Fig.

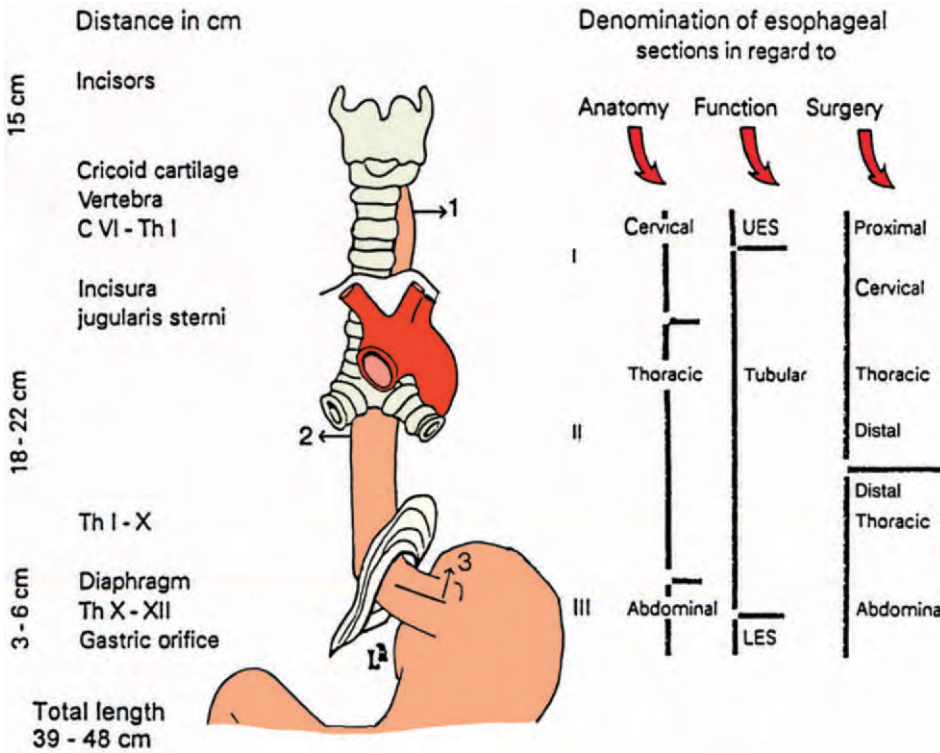


Figure 2-1. Classic anatomic division of the esophagus and its topographic relationship to the cervical (C) and thoracic (Th) vertebrae. The approximate length of each segment is given, and the three narrowings of the esophagus are shown. More recently, the esophagus has been subdivided according to its different functions by Diamant.¹ Based on the embryology and main direction of lymphatic flow, Siewert (1990) proposed a subdivision of the thoracic esophagus at the level of the tracheal bifurcation for determining treatment strategies in patients with esophageal cancer. LES, lower esophageal sphincter; UES, upper esophageal sphincter.

2-2); as a result, the greater curvature faces the posterior subdiaphragmatic space, and the anterior gastric wall faces laterally. This topographic feature is not well displayed in standard anatomy textbooks but is definitely clarified by computed tomographic studies (see Fig. 2-2). A better understanding of the function of the cardia and interpretation of pressure measurement data of the lower esophageal sphincter (LES) are based on this topography.

Measured Dimensions

Length of the Esophagus

The length of the esophagus is defined anatomically as the distance between the cricoid cartilage and the gastric orifice. In adults, it ranges from 22 to 28 cm (24 ± 5 SD), 3 to 6 cm of which is located in the abdomen (see “Suggested Readings”).^{5,6} In contrast to the previous assumption about the incidence of sex differences (see Lerche in “Suggested Readings”), Liebermann-Meffert et al.⁶ found the length of the esophagus to be related to the subject’s height rather than sex.

Identification and marking of the cricoid cartilage are rather difficult in a living individual. For practical reasons, therefore, clinicians measure the length of the esophagus by including the oropharynx and the pharynx and using the incisors as a direct macroscopic landmark during endoscopic procedures (see Savary and Miller in “Suggested Readings”). The distances are shown in Figure 2-1.

Length of the Orthotopic Bypass

Esophagectomy for cancer requires transfer of the substitute to the position formerly occupied by the esophagus. To measure the length required for esophageal replacement, the shortest distance between the cricoid cartilage and the celiac axis was found to be the orthotopic route in the posterior mediastinum (30 cm). The retrosternal location (32 cm) and the subcutaneous route (34 cm) proved to be longer.⁷ There were no differences between men and women.

Diameter of the Esophagus

The esophagus is the narrowest tube in the intestinal tract. It ends by widening into its most voluminous part, the stomach. At rest, the esophagus is collapsed; it forms a soft muscular tube that is flat in its upper and middle parts, with a diameter of 2.5 × 1.6 cm. The lower esophagus is rounded, and its diameter is 2.5 × 2.4 cm.^{6,8}

Compression or constriction by adjacent organs, vessels, or muscles may cause narrowing, which can be visualized by means of fluoroscopy and endoscopy (see “Suggested Readings”). The aortic compression, which is left sided and anterolateral, is caused by crossing of the aortic arch, the left atrium, and the left main bronchus at a location 22 cm from the incisors. Occasionally, a mechanical imprint of the diaphragm exists, but more apparent are two functional muscular constrictions: the upper and the lower esophageal sphincters. They are found manometrically at the esophageal opening, 14 to 16 cm distant from the incisors, and at the entrance into

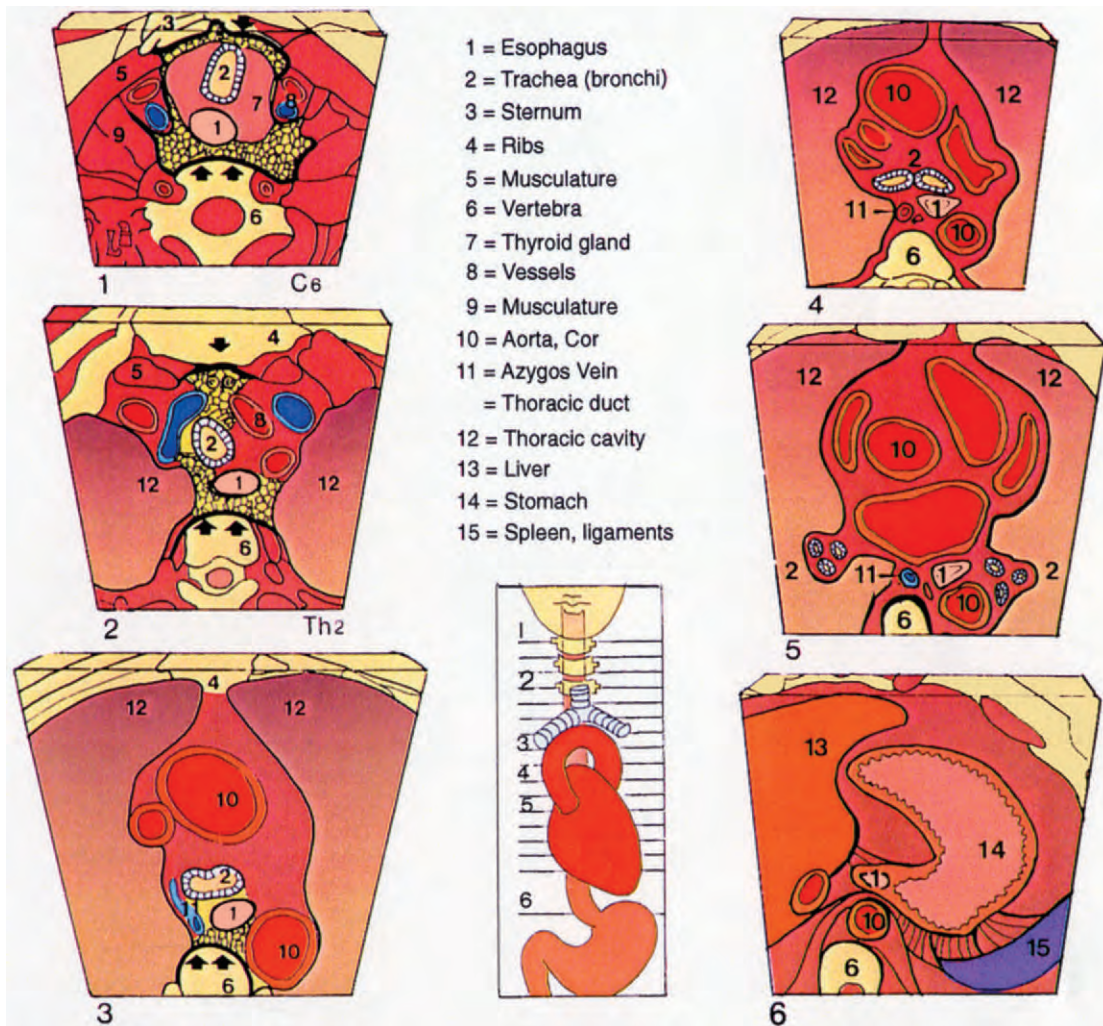


Figure 2-2. Topographic anatomy of the esophagus shown from the cervical level (1) to the esophagogastric junction (6). A transverse section through the mediastinum shows the esophagus and its surrounding structures in a computed tomographic aspect. The close positional relationship among the esophagus, trachea, and vertebrae and the fascial planes is displayed. The *thick dark lines* are the prevertebral and prevertebral fascia (*arrows*); the net-like pattern represents the respective areolar connective tissue. (Modified after Wegener OH: Neuromuscular organization of esophageal and pharyngeal motility. Arch Intern Med 136:524, 1976, with permission.)

the stomach, between 40 and 45 cm from the incisors (see Fig. 2-1) (see the section “Esophageal Sphincters” later in this chapter).

Periesophageal Tissue, Compartments, and Fascial Planes

Unlike the general structure of the digestive tract, the esophageal tube has neither mesentery nor serosal coating. Its position within the mediastinum and a complete envelope of loose connective tissue allow the esophagus extensive transverse and longitudinal mobility.^{9,10} Respiration may induce craniocaudal movement over a few millimeters, and swallows may result in excursion over as much as the height of one vertebral body. This mobility is also the reason why the esophagus may be sub-

jected to easy blunt stripping from the mediastinum. Invasion by malignant tumor and fixation to the surroundings, however, strictly contraindicate the use of this technique.^{4,6,11}

Another anatomic peculiarity is of clinical relevance: the connective tissues in which the esophagus and trachea are embedded are bounded by fascial planes, the pretracheal fascia anteriorly and the prevertebral fascia posteriorly. In the upper part of the chest, both fascias unite to form the carotid sheath, and the anterior and posterior spaces between these fascias form a communicating compartment between the neck and the chest that provides a plane for rapid spread of infection through the mediastinum (see Fig. 2-2).

The anterior space coincides with the prevertebral (i.e., pretracheal) space. Infections spreading from anterior lesions of the esophagus may follow this route, but they

are limited distally by the strong fibrous tissue of the pericardium. The posterior space, which is the retrovisceral (i.e., prevertebral) space, extends from the base of the skull to the diaphragm. It is formed by the buccopharyngeal fascia spreading downward via a sheath that separates the esophageal tissue bed from the prevertebral fascia. This space is clinically of greater importance than the previsceral space. The reason is that most instrument perforations with subsequent outflow of esophageal contents occur above the narrowing of the cricopharyngeal sphincter in the posterior hypopharynx (see Savary and Miller in “Suggested Readings”). At this level, as in the chest, there is no barrier to the spread of infection into the mediastinum. Rupture of the esophagus or leakage of an esophageal anastomosis may result in descending mediastinitis along these planes as well. Prompt diagnosis is vital for the patient because the prognosis for esophageal perforation depends on the rapidity with which treatment is initiated!

Stabilizing and Anchoring Structures

The esophagus is stabilized by bony, cartilaginous, and membranous structures (Fig. 2–3).

Anchorage in the Neck

Through the exterior longitudinal layer of its muscle coat the cranial end of the esophagus fastens at the posterior ridge of the cricoid cartilage via the cricoesophageal tendon (Fig. 2–4).

Anchorage of the Body of the Esophagus

The tubular esophagus lies in the loose areolar tissue bed of the mediastinum (see Fig. 2–2). The claim that broad fibrous tissue or muscle strings connect the trachea and esophagus, as depicted by Laimer¹⁰ and later adopted in Netter’s atlas,¹³ could not be substantiated by the authors’ studies.^{8,10} Instead, there were numerous delicate, slightly undulated membranes mostly 170 μm in thickness and approximately 3 to 5 mm in length (Fig. 2–5A and B). They connected the esophagus with the trachea (see Fig. 2–5A) and the surrounding tissue (see Fig. 2–5B). Consisting of collagen and elastic fiber elements and occasional interpositioned sparse muscle fibers, the membranes are stretchable to some extent and accumulate around the tracheal bifurcation.¹⁰ A few individuals possess membranes up to 700 μm in thickness, together with firm intramural insertion (see Fig. 2–5A).

Anchorage of the Cardia

When the distal end of the esophagus traverses the diaphragm through the esophageal hiatus, it is bounded by the two diaphragmatic crura and the phrenoesophageal membrane (Figs. 2–6 and 2–7; see also Fig. 2–3).

The muscular portion of the diaphragm is inserted on the lumbar vertebrae, the ribs, and the sternum. The central membranous portion is frequently larger than that described in the literature, and the left crus of the

ANCHORING STRUCTURES OF THE ESOPHAGUS

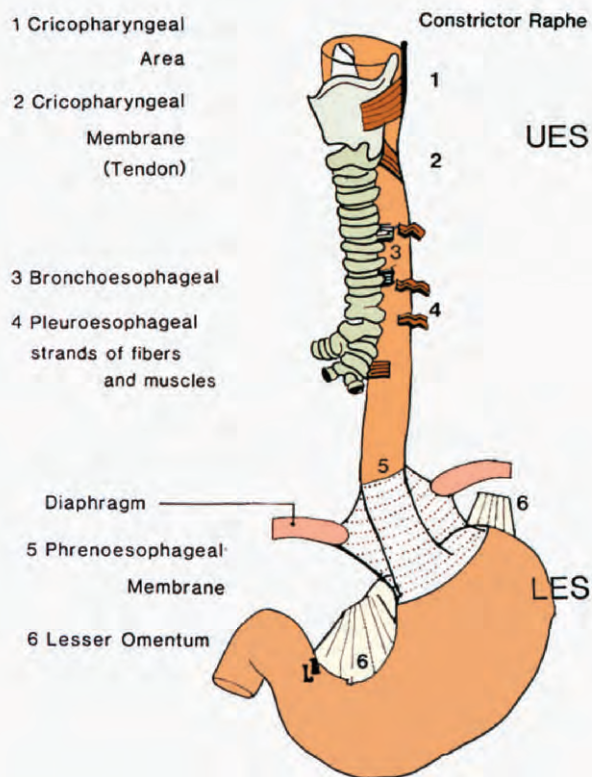
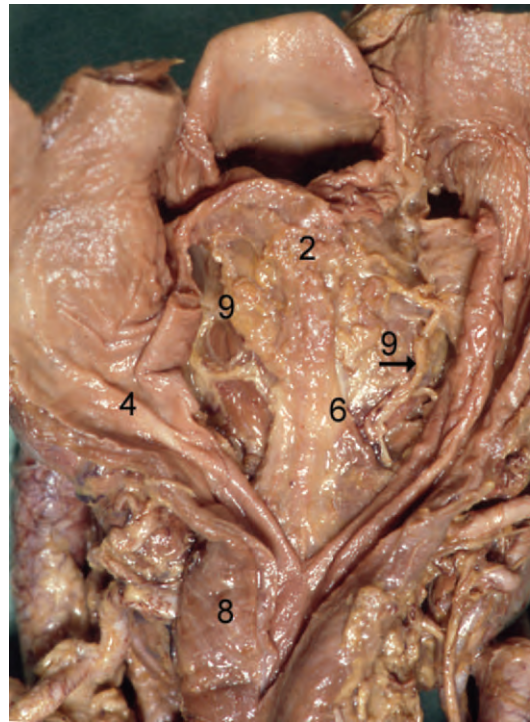


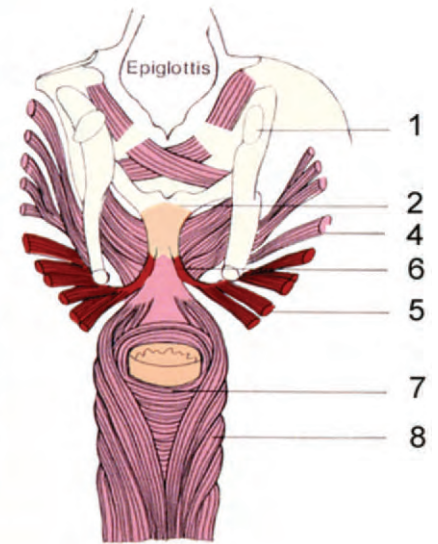
Figure 2–3. Attachments of the esophagus. The upper end of the esophagus obtains firm anchorage by the insertion of its longitudinal muscle into the cartilaginous structures of the hypopharynx (1) via the cricoesophageal tendon (2). The circular muscle is stabilized by its continuity with the inferior laryngeal constrictor muscles (1), which insert via the raphe to the sphenoid bone. Tiny membranes connect the esophagus with the trachea, bronchi, pleura, and prevertebral fascia (3 and 4). The attachment at the lower end by the phrenoesophageal membrane (5) is rather mobile, whereas the posterior gastric ligaments, such as the gastrosplenic, phrenocolic, and phrenogastric ligaments (6), and the lesser omentum (6) yield a tight adherence. LES, lower esophageal sphincter; UES, upper esophageal sphincter.

diaphragm may consist of membranous tissue rather than a significant muscular mass (see Fig. 2–6) (see also Williams and Warwick in “Suggested Readings”). The subdiaphragmatic and endothoracic aponeuroses blend at the central margin of the diaphragm to constitute the phrenoesophageal membrane (PEM), also known as Laimer’s ligament or Allison’s membrane. Intraoperatively, the PEM can be recognized by its well-defined lower edge (Fig. 2–7) and its slightly yellow color, even in the presence of severe periesophagitis. The PEM is composed of elastic and collagenous fiber elements, which guarantee sufficient pliability. Because of its origin

Figure 2–4. The posterior walls of the pharynx (4) and the esophagus (7 and 8) have been cut open in the midline, as shown in a specimen (A) and half-schematically (B). The structures of the hypopharynx are exposed by retracting the overlying incised tissue and removing the mucosa. In the center lies the cricoesophageal tendon (6), which attaches the longitudinal muscle layer of the esophagus (8) to the cricoid cartilage (2). The terminal branches of the left laryngeal recurrent nerve (9) are dissected and are seen lateral to the cricoesophageal tendon. 1, Thyroid cartilage. (Specimen and photo courtesy of Liebermann-Meffert, Munich.)

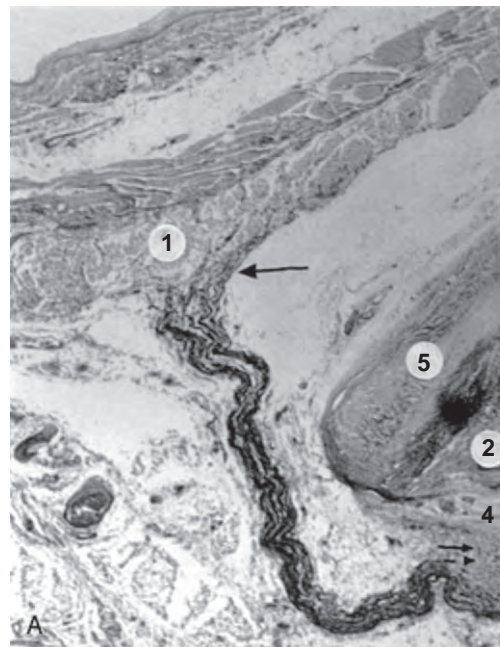


A



B

Figure 2–5. A and B, Example of the tiny fiber membranes that connect the esophagus (1), trachea (2), pleura (3), tracheal membrane (4), and cartilaginous structures (5). At their insertions, the fiber elements fan out to deep finger-shaped extensions between the muscular bundles of the esophagus (arrow) and into the membranous part of the trachea (double arrows). This texture, in conjunction with the elasticity of the membranes, certainly provides adequate adjustment during movement of the esophagus. In case of rapid pull, the fibers eventually tear off the tissues in which they are anchored (human esophagus, transverse section, hematoxylin and eosin stain). (Courtesy of Huber, Haeberle, and Liebermann-Meffert, Munich.)



A



B

from a fascia, the PEM in general is relatively strong. It splits into two sheets (Fig. 2–8). One sheet extends 2 to 4 cm upward through the hiatus, where its fibers traverse the esophageal musculature to insert on the submucosa.^{10,14} The other sheet passes down across the cardia up to the level of the gastric fundus, where it blends into the gastric serosa, the gastrohepatic ligament, and the dorsal gastric mesentery (see Figs. 2–3 and 2–7).

Although there are sparse attachments via elastic cords in the pattern shown in Figure 2–8, the PEM is clearly only some distance away and separated by loose connective tissue and fat accumulation from the musculature of the gastroesophageal junction (see Fig. 2–7). This structural arrangement allows the terminal esophagus and the junction to move in relation to the diaphragm and to “slip through the hiatus like in a tendon sheath.”¹⁵ With

advancing age, the elastic fibers are replaced by inelastic collagenous tissue, and the adhesion of the PEM to the lower portion of the esophagus loosens,¹⁴ which leads to loss of pliability. Disruption of the anchoring structures

of the cardia and the proximal part of the stomach in conjunction with a wide hiatus may result in herniation of the gastroesophageal junction and the cardia, or even parts of the stomach, into the mediastinum. Abnormal anchoring of the PEM in youth and pathologic accumulation of adipose tissue in the separating connective tissue space between the PEM and the cardia musculature are thought to contribute to the development of a hiatal hernia.¹⁴

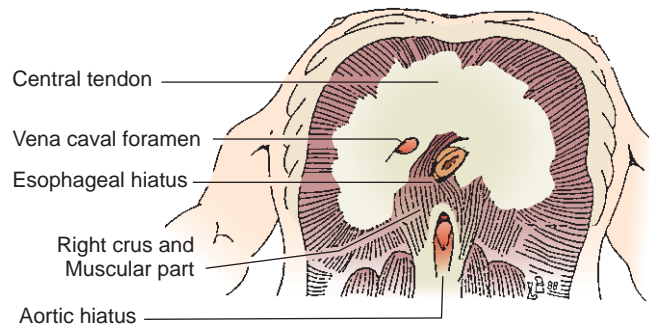


Figure 2-6. Diaphragm and esophageal hiatus viewed from the abdominal aspect.

Selected Topographic Relationships and Surgical Risk Areas

Neck

Ventral to the cervical esophagus lie the fibrous membranes that unite the adjacent hoops of the tracheal cartilage (Fig. 2-9). Note that only an inconspicuous amount of areolar connective tissue—if any—separates the two structures (see Figs. 2-5 and 2-9). Malignant

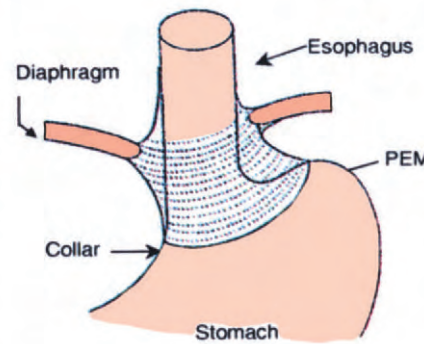


Figure 2-7. The phrenoesophageal membrane (PEM). The lower component of the membrane inserts on the gastric fundus. On the *left*, the diaphragm is held up with forceps. Diaphragmatic decussating fibers (*long arrow*) and a submembranous inlay of adipose tissue (*short arrow*) are seen. The PEM wraps the esophago-gastric junction with a wide membranous collar. (Specimen and photo courtesy of Liebermann-Meffert, Munich.)

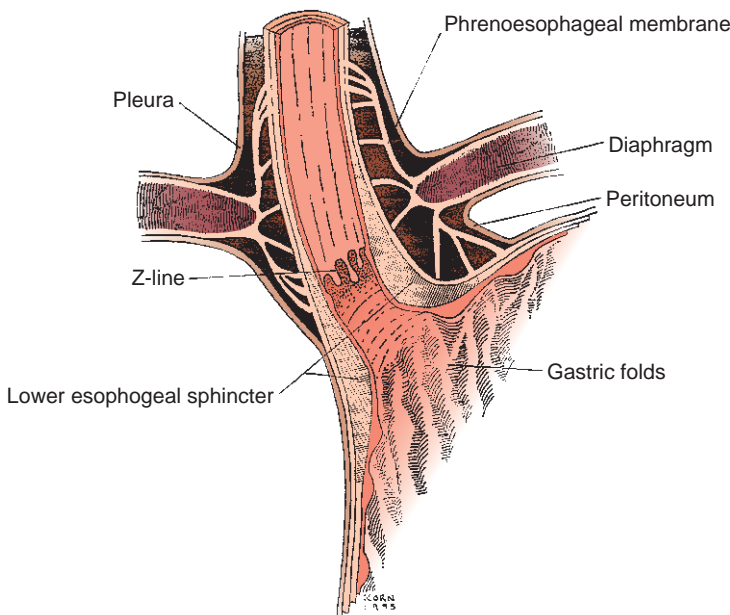


Figure 2-8. Diagram of the tissue organization and supporting structures at the esophago-gastric junction. The esophagus is opened alongside the greater and lesser curvatures. The luminal aspect is displayed from the left side. The fiber elements that attach the phrenoesophageal membrane to the muscle wall of the terminal esophagus are shown. The fibers equal those shown in Figure 2-5. (Courtesy of Dr. Owen Korn, Munich and Santiago di Chile.)

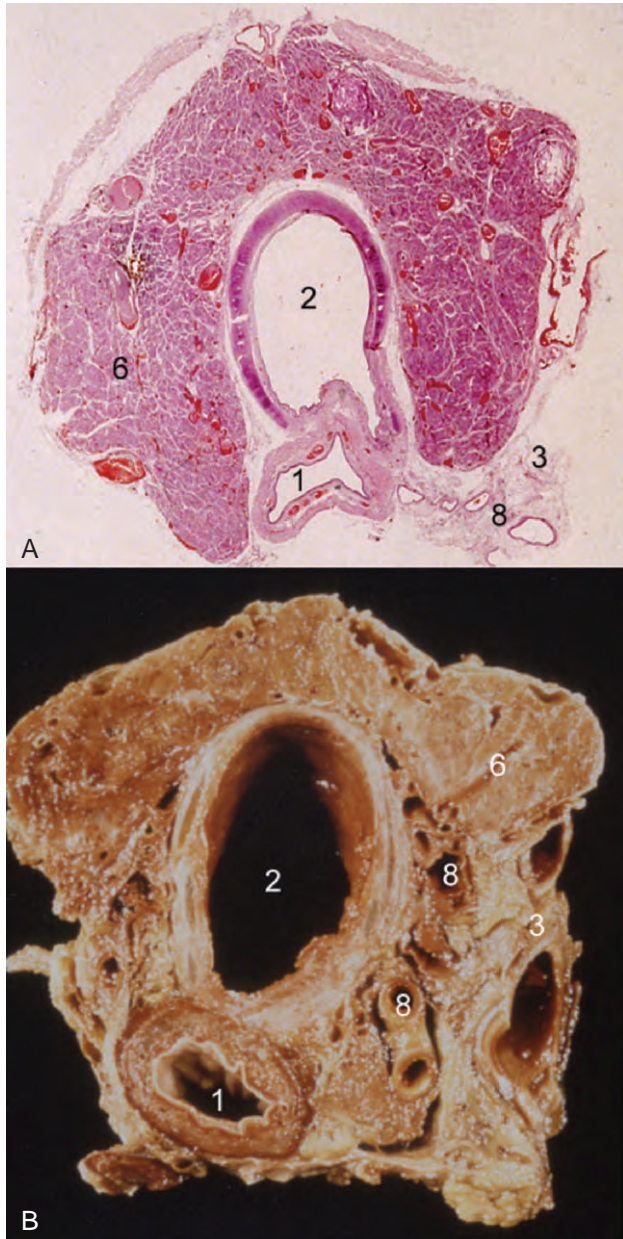


Figure 2-9. Transverse section through the neck and upper part of the chest of a human autopsy specimen viewed from a cranial aspect. 1, Esophagus; 2, trachea; 3, pleura; 6, thyroid gland; and 8, vessels. The histologic section shows the esophagus still in the midline posterior position (A), whereas on the more distal level of the macroscopic cut surface (B), the esophagus has shifted toward the left. Note the intimate local relationship between the esophagus and the trachea. (From Liebermann-Meffert D: Funktionsstörungen des pharyngo-ösophagealen Übergangs: Funktionelle und chirurgisch orientierte Anatomie. In Fuchs KH, Stein HJ, Thiede A [eds]: Gastrointestinale Funktionsstörungen. Berlin, Springer, 1997, with permission.)

THE AZYGOS VEIN

From Lateral =
RIGHT THORACIC APPROACH

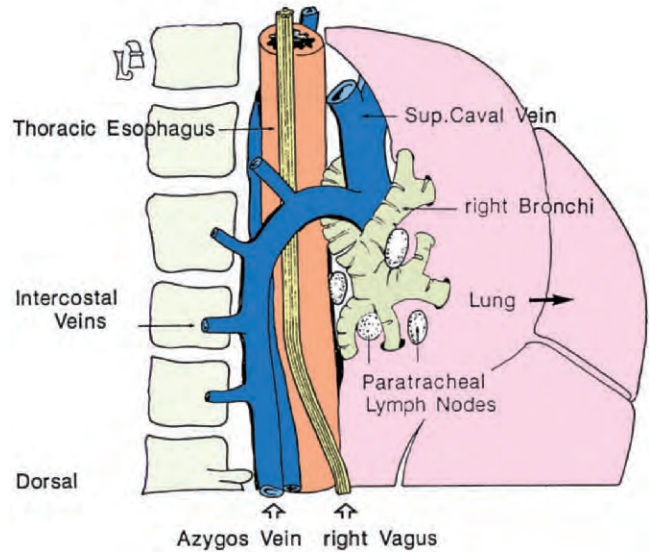


Figure 2-10. The position and relationships of the azygos vein, the thoracic duct, and the vagus nerve are shown from a right lateral aspect.

tumors are known to spread from the trachea to the esophagus and vice versa. Clinically, such spread results in an “acquired fistula.”¹⁶⁻¹⁸ Unfortunately, it appears that the lack of interposed connective tissue between the two organs predisposes to this unlucky event. Remember that a tracheoesophageal fistula after either an instrumental perforation, esophagectomy, or chemotherapy and irradiation in this inherently weak area is a catastrophic problem for both the patient and physician.¹⁶⁻¹⁹

Chest

Between the thoracic inlet and the tracheal bifurcation (which lies at the level of the fifth thoracic vertebra), the esophagus retains its intimate relationship to the trachea ventrally and to the prevertebral fascia posteriorly (see Fig. 2-2). The mediastinal pleura, the lungs, and their hila are positioned on both sides. On the right lies the subclavian artery and the azygos vein, which arches over the right main bronchus to end in the superior vena cava (Fig. 2-10). When performing transthoracic esophagectomy, surgical access for safe removal of the esophagus is preferably through the right side of the chest, and the azygos vein must usually be divided before the esophagus can be dissected free (see “Suggested Readings”). The primarily right side–positioned thoracic duct crosses behind the esophagus just above the arch of the azygos vein at the level of T4 to T5. Structures on the left of the esophagus are the aortic arch and the aorta, which subsequently turns to the midline and travels in a posterior course behind the esophagus (see Fig. 2-2). In front of

TISSUE TEXTURE OF THE ESOPHAGOGASTRIC JUNCTION

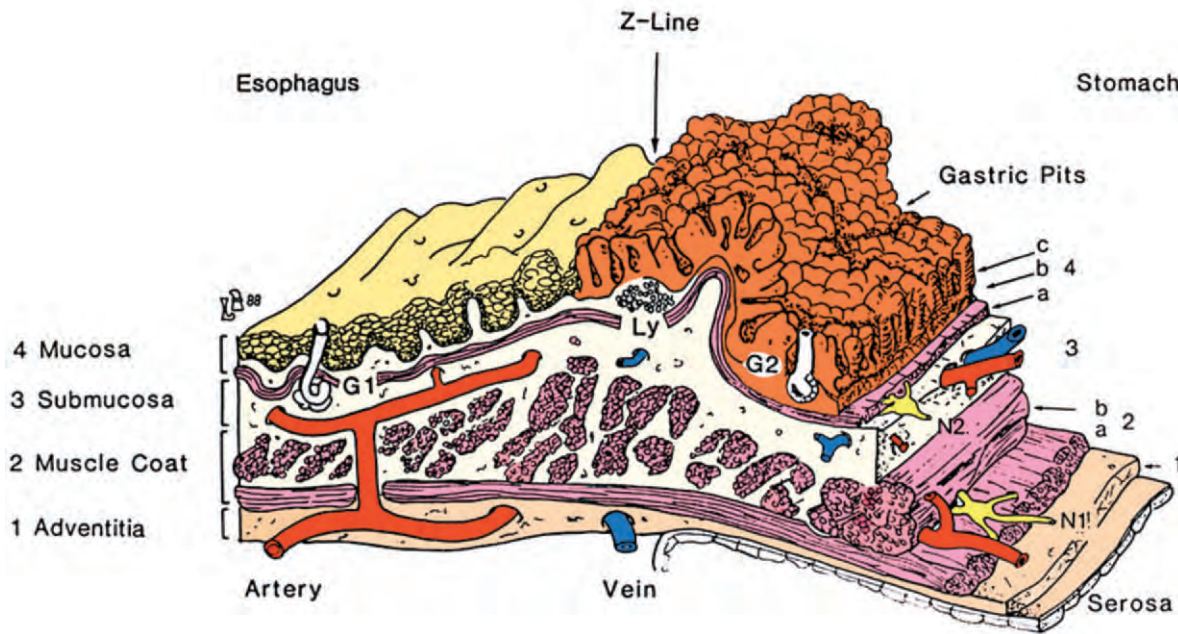


Figure 2-11. Wall structure at the esophagogastric junction. The tunica muscularis is composed of both a longitudinal (2a) and a circular layer (2b). a, muscularis mucosae; b, lamina propria; c, epithelium; G1, esophageal glands; G2, gastric glands; Ly, lymph vessels; N1, myenteric plexus; N2, submucous nerve plexus.

the esophagus are the lung hilum and the heart. The pleura on the left side of the mediastinum may occasionally extend behind the esophagus. Both vagi accompany the esophagus as it passes through the hiatus at the level of the 10th thoracic vertebra.

Abdomen

In the abdomen, part of the left lobe of the liver lies ventral to the esophagus. The two diaphragmatic crura are lateral and posterior. The inferior vena cava is lateral to the right crus, whereas the aorta is posterior to the left crus. The cranial pole of the spleen is in close relationship to the terminal esophagus (see Fig. 2-2). Other vessels and nerves that supply the esophagus and the adjacent organs are discussed later in this chapter.

Constituents and Tissue Organization of the Foregut

The basic tissue organization of the esophagus and cardia is shown in Figure 2-11.

TISSUES

Tunica Adventitia

This thin coat of loose connective tissue envelops the esophagus, connects it to adjacent structures, and

contains small vessels, lymphatic channels, and nerves (see Fig. 2-11).

Tunica Muscularis

Esophageal Body

Muscular Arrangement The tunica muscularis coats the lumen of the esophagus in two layers, the fibers of which follow a diametric course: the external muscle layer parallels the longitudinal axis of the tube, whereas the muscle fibers of the inner layer are arranged in the horizontal axis (Fig. 2-12). For this reason, these muscle layers are classically called longitudinal and circular, respectively.

The *longitudinal layer* originates from the dorsal plane of the cricoid cartilage as shown earlier in Figure 2-4. Its muscular bundles fan out in a posterior direction, with an area of circular muscle left vacant—Laimer's triangle—before they wrap the esophagus entirely (Figs. 2-13 and 2-14; see also Fig. 2-11).^{8,20} As long bundles, they run in a straight course down the esophageal tube and cross the gastric inlet (see Fig. 2-12).

The *circular layer* begins at the level of the cricoid cartilage. In their descent, the short fibers of the inner muscular layer form imperfect circles with overlapping ends, as illustrated in Figures 2-12 and 2-13.^{8,20}

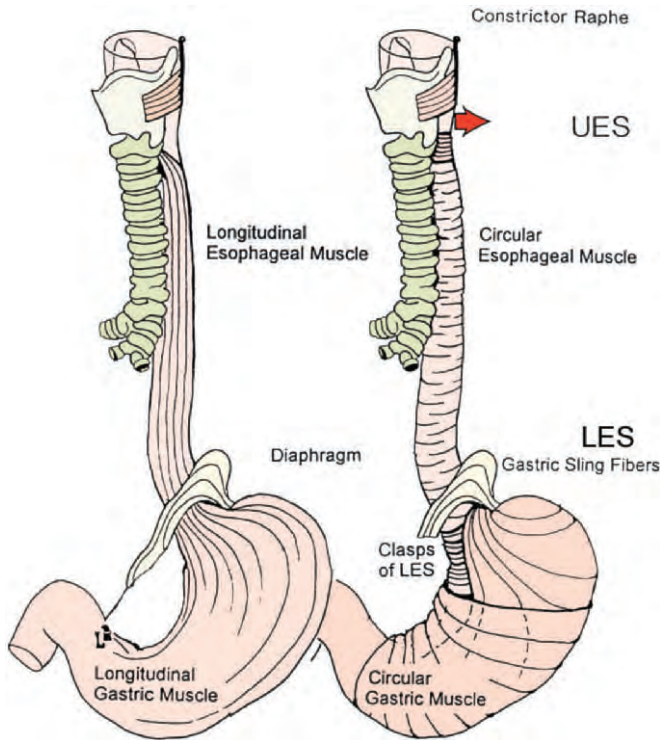


Figure 2-12. Architecture of the longitudinal and circular muscle layers across the esophagus, stomach, and respective junctions. LES, lower esophageal sphincter; UES, upper esophageal sphincter.

Muscle Types: Striated Versus Smooth It is generally accepted that the striated musculature behaves different from smooth muscle. Both types of muscle are present in the esophagus. The question has been raised of how the striated and smooth muscle is distributed in the wall of the esophagus. When systematically examining serial histologic sections of the esophagus from 15 individuals,²¹ the authors found exclusively striated musculature in the pharynx and particularly in the cricopharyngeal muscle, which of course is the upper esophageal sphincter muscle (UES). The first sparse smooth muscle fascicles appear 2 to 3 mm caudal to the UES. Farther caudally, progressively more and more smooth muscle bundles replace the striated muscle in both the external and internal layers. The transition between both types is neither abrupt nor confined to individual muscle bundles and lacks any distinct anatomic border (Fig. 2-16).^{10,21} Caudal to the tracheal bifurcation, no striated muscle elements are seen any more. With regard to sphincter function, it might be of interest to be aware that the muscle type of the UES differs completely from that of the LES!

The muscularis mucosa is composed uniquely of smooth muscle fibers throughout the entire esophagus.

Esophageal Sphincters

Zones of increased pressure in the esophagus have been verified, one at the upper and the other at the lower end.

Diverse factors and mechanisms are suggested as the cause of the sphincter pressure, but ultimately, all remain disputable. This has prompted us to reinvestigate the human muscle morphology of the esophagus and both sphincters with special techniques. The results have been published previously in detail,^{8,9,20,22} and the findings are presented in abridged version in the following two sections.

Structural Counterpart of the High-Pressure Zone: The Upper Esophageal Sphincter The UES is manometrically a 2- to 4-cm-long zone of elevated pressure²³ and marks the entrance into the esophagus. The high pressure results from contraction of the cricopharyngeus muscle. This semicircular muscle originates from the lateral cricoid processes (see Figs. 2-13 and 2-14) and closes the esophageal opening by exerting pressure toward the posterior plane of the cricoid cartilage. This arrangement accounts for the asymmetric pressure profile in manometric measurements.²³⁻²⁵ The position of the cricopharyngeal muscle at the end of the pharynx implies that the structure is a “lower pharyngeal” rather than an “upper esophageal” sphincter.

Muscular Counterpart of the High-Pressure Zone: The Lower Esophageal Sphincter The LES is manometrically a 3- to 5-cm-long zone of elevated pressure and marks the end of the esophagus and the entry into the stomach.²⁶ Biochemically, the muscle of this area behaves differently from the muscle above and below it.^{2,24,27} Markers applied surgically to these muscles in a simultaneous radiomorphologic study have shown that this high-pressure zone correlates with the thickened musculature at this site.^{20,22} The high pressure results from contraction of the special muscle organization at this location.

It is unfortunate that fresh muscle tissue inevitably retracts when cut through, in particular, hollow organs such as the intestinal tube. Distorted muscle architecture escapes critical examination; sphincters can neither be palpated nor compared with the neighboring muscle wall. To circumvent this dilemma, the authors used en bloc fixation of the chest and upper abdominal organs to study this anatomic situation in autopsy specimens.^{20,22} Such study allowed macroscopic measurement of the muscle thickness of the LES in order to compare it with that of the esophageal body and stomach. Another group of specimens was used to study the respective muscle arrangement.^{20,22} The results indicated that the muscular sphincter was the equivalent of the physiologic high-pressure zone (Fig. 2-15).

We described in this paper and depicted in detail²⁰ that approximately 3 cm cranial to the junction with the stomach, the imperfect muscle circles of the circular layer (see Fig. 2-12) increase in number and result in a stepwise, significant thickening ($P < .001$) of the terminal esophageal musculature.^{10,20} This transition is consistent with conspicuous remodeling of the muscle architecture, specifically, asymmetric rearrangement of the muscle fibers of the inner layer (see Figs. 2-12 and 2-15). The bundles on the side of the lesser curvature retain their orientation and form short muscle clasps, whereas those on the greater curvature change to

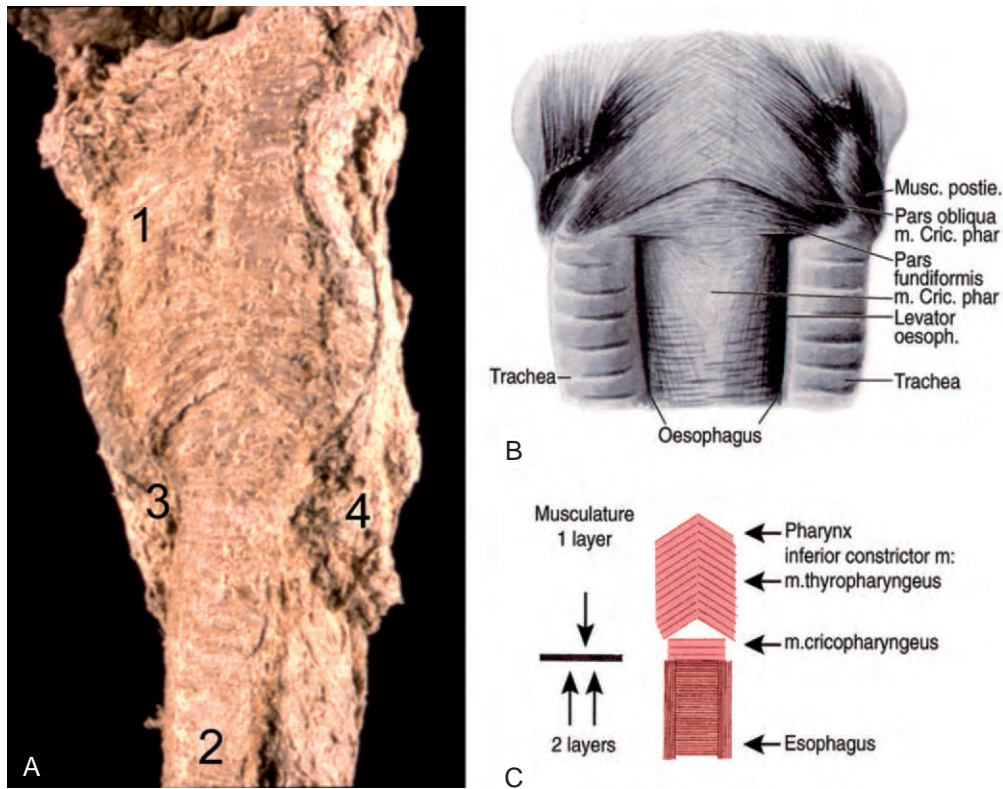


Figure 2-13. Structures at the pharyngo-esophageal junction viewed from a posterior aspect. They are shown in a human dried-fiber specimen (A) (by Liebermann-Meffert), of a schematic drawing of an anteriorly opened and unfolded specimen (B) (by Killian), and in a simplified diagram of the muscle organization (C). The muscular arrangement of the inferior constrictor of the pharynx (1) confirms Killian's observation of the tile-shaped arrangement of the bundles of the inferior constrictor muscle (Killian G: Z Ohrenheilk 55:1, 1908). With respect to the junction, two features should be emphasized: (a) the change of one muscle layer at the pharynx (1) into two at the esophagus (2) just caudal to the cricopharyngeal muscle (3) (upper esophageal sphincter); (b) the cricopharyngeal muscle is part of the pharynx by position and anatomic characteristics. 4, Residual tissue from the removed thyroid gland. (From Liebermann-Meffert D: Funktionsstörungen des pharyngo-ösophagealen Übergangs: Funktionelle und chirurgisch orientierte Anatomie. In Fuchs KH, Stein HJ, Thiede A [eds]: Gastrointestinale Funktionsstörungen, Berlin, Springer, 1997, with permission.)

become the oblique gastric sling fibers. It has been suggested that myotomy for achalasia should preferably be performed between the muscle clasps and gastric sling fibers to preserve the complete strength of the sling (i.e., maintain sphincter competence).²⁸

The specific arrangement of the musculature, which we have shown in Figures 2-12 and 2-15, also accounts for sphincter asymmetry.^{9,20,22,29} Asymmetry of the high-pressure zone at this position has likewise been proved manometrically.²⁶ The manometric pressure image of the lower esophageal high-pressure zone, obtained by a three-dimensional computerized vector diagram, matches the muscular asymmetry at the human cardia perfectly (see Fig. 2-15).³⁰⁻³² Surgical removal of these structures by partial or total myectomy was shown to significantly reduce the specific sphincter pressure values of this muscle arrangement as recorded on manometry.^{2,28,33} Displacement of the LES into the chest through the diaphragm or dissection of the PEM produced no effect on the pressure values of the sphincter in long-lasting animal experiments.²²

Tela Submucosa

The submucosa is the connective tissue layer that lies between the muscular coat and the mucosa. It contains a meshwork of small blood and lymph vessels, nerves, and mucous glands. The deep esophageal glands are small branching glands of a mixed type, and their ducts pierce the muscularis mucosae (see Fig. 2-11).

Tunica Mucosa

The mucous layer is composed of three components: the muscularis mucosae, the tunica propria, and the inner lining of nonkeratinizing stratified squamous epithelium (see Fig. 2-11). The muscularis mucosae forms the long mucosal folds that run in the longitudinal axis of the tube and shapes the small transverse ripple folds at the cardia.^{20,34} All these folds disappear on distention of the esophageal lumen. The tunica propria contains areolar connective tissue, blood vessels, and lymph channels derived from the lower level of the mucosa. At

MUSCULAR ARCHITECTURE OF UPPER ESOPHAGEAL SPHINCTER

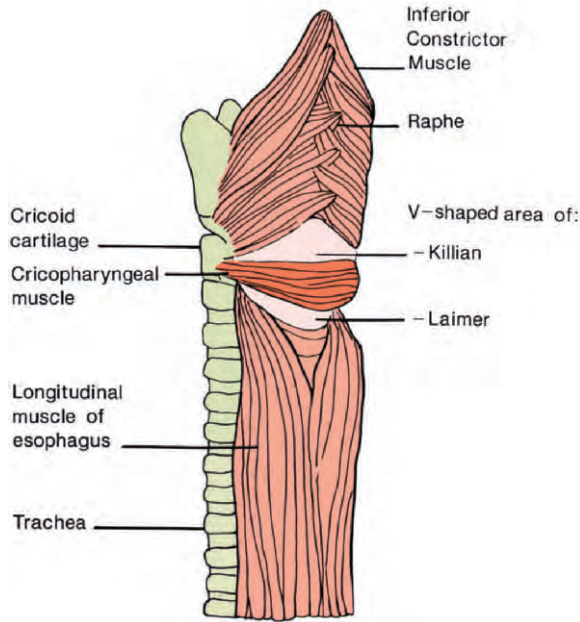


Figure 2-14. Schematic drawing of the structures at the pharyngoesophageal junction seen from the posterior aspect. The location of Killian's and Laimer's triangles is indicated; Zenker's diverticula develop cranial to the cricopharyngeal muscle, and the upper esophageal sphincter is located caudal to the V-shaped area of Killian.

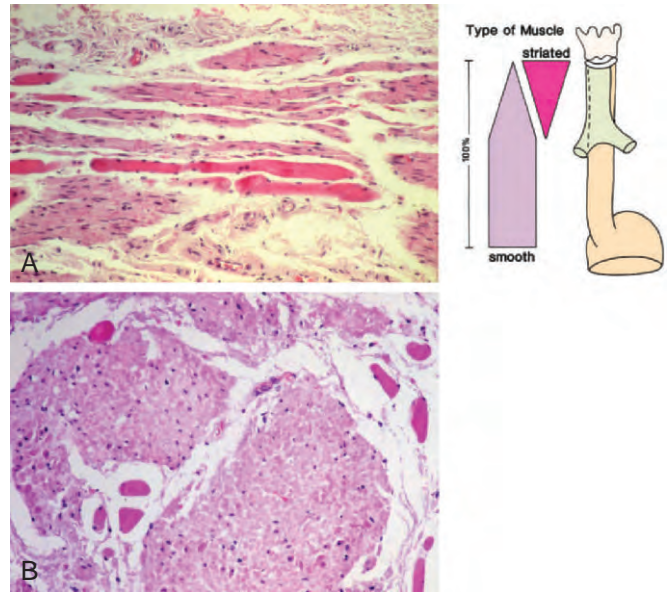


Figure 2-16. Histologic specimens of the human esophagus taken in transverse (A) and longitudinal (B) sections 4 cm above the tracheal bifurcation cranial to the transition between striated and smooth muscle. Individual striated muscle fibers are interspersed among smooth muscle strands. The diagram shows the distribution of striated and smooth muscle in adult esophagus as evaluated from consecutive serial histologic sections of 13 esophagi. (Specimen and photo courtesy of Liebermann-Meffert, Geissdörfer, and Winter, Munich.)

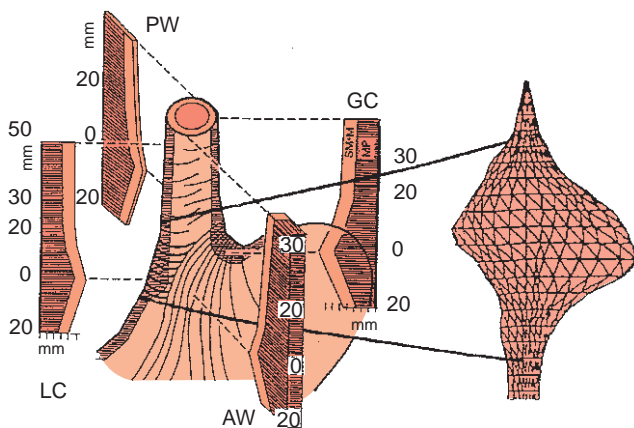


Figure 2-15. Schematic drawing showing the correlation between radial muscle thickness (left) and a three-dimensional manometric pressure image (right) at the gastroesophageal junction. Muscle thickness across the gastroesophageal junction at the posterior gastric wall (PW), greater curvature (GC), anterior gastric wall (AW), and lesser curvature (LC) is shown in millimeters. Radial pressure at the gastroesophageal junction (in mm Hg) is plotted around an axis representing atmospheric pressure. Note the marked radial and axial asymmetry of both the muscular thickness coinciding with the manometric pressure profile.

the esophagogastric junction, a short 0.5- to 1.0-cm area of superficial (mucous) glands that resemble cardiac glands is a consistent finding.^{35,36} Heterotopic gastric mucosa may occasionally also be found at the upper end of the esophagus.³⁷

Clinically, the surface of the esophageal mucosa is reddish but becomes paler toward the lower third of the esophagus. The smooth esophageal mucosa can easily be distinguished from the dark mammillated gastric mucosa. The mucosal transition at the squamocolumnar junction is an objectively recognizable reference point for the endoscopist (see Savary and Miller in "Suggested Readings"). On fresh anatomic specimens, the transition is characterized by a serrated, but abrupt demarcation line. The so-called Z-line is located at or immediately above the gastric orifice. Any proximal extension of gastric- or intestinal-type columnar epithelium is considered pathologic and attributed to long-standing reflux of gastric contents causing chronic, severe esophageal mucosal damage.³⁸ The transition between the two types of mucosa is a "mucosal junction" wherever it is positioned. By no means should it be considered a "sphincter" (as the mucosal transition at the cardia is occasionally termed by gastroenterologists). The term *sphincter* by traditional anatomic definition is restricted to the presence of muscular constrictor structures.

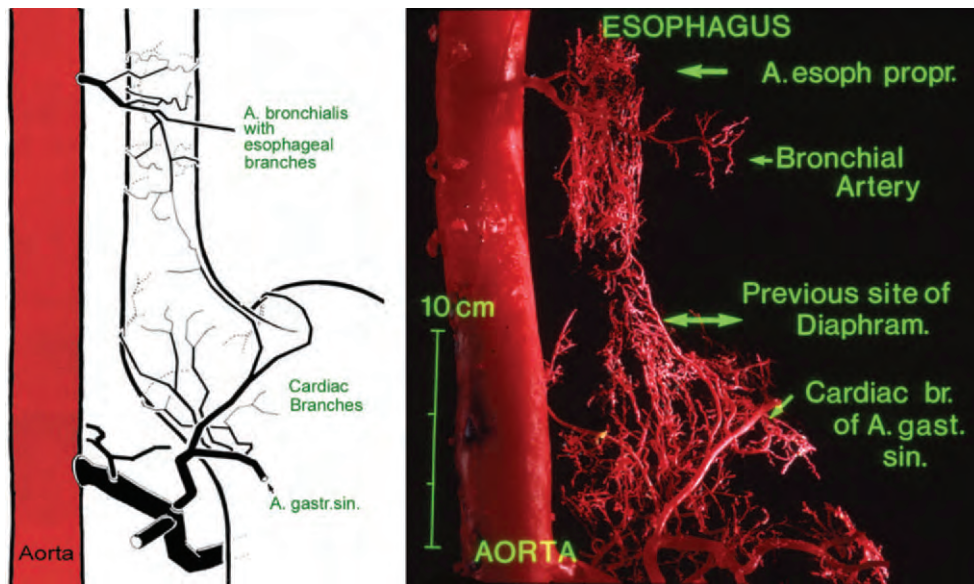


Figure 2-17. Arterial cast showing the vascular supply to the middle and lower portions of the esophagus. Note that the esophageal branch derives from the bronchial artery. During esophageal resection, it should be ligated close to the esophageal wall so that the blood supply of the left main bronchus is not jeopardized. In this context, it should be mentioned that the esophagus shares its blood supply with other organs: the thyroid gland, the trachea, the stomach, and the spleen.

STRUCTURES SUPPLYING THE ESOPHAGUS

Arterial Supply

Extraparietal Sources

Knowledge of the blood supply of the foregut assumes increasing importance. Adequate display of the esophageal vessels is technical difficult, and inadequate technique has caused errors in evaluation and description. Angiograms do not outline the arterial pattern well because of the overlying arteries associated with other structures. Large en bloc corrosion casts, however, produce realistic three-dimensional replicas of the macrovascular and microvascular systems, as seen in Figures 2-17 and 2-18. Such casts establish that the esophagus is an organ of “shared vasculature” because it receives its blood through vessels feeding mainly other organs such as the thyroid gland, trachea, and stomach.⁶ There are three principal extraparietal arterial sources for the esophagus (Fig. 2-19). In the neck, the upper superior and inferior thyroid arteries send small descending arteries to the cervical esophagus. At the level of the aortic arch, a group of three to five tracheo-bronchial arteries arise from the concavity of the arch and give rise to several tracheoesophageal tributaries. Small proper esophageal arteries most often arise from the anterior wall of the thoracic aorta via a larger bronchial artery (see Fig. 2-17). At the cardia, the left gastric artery gives off up to 11 branches that ascend and supply the anterior and right aspects of the lower part of the esophagus (see Fig. 2-17).^{6,39} Vessels arising from the splenic artery supply the esophageal wall and parts of the greater curvature from the posterior aspect as seen in Figures 2-17 and 2-19. Two facts became obvious through Liebermann-Meffert and colleagues’ studies⁶ that had not been appreciated before: all the major arterial vessels divide into minute branches at some distance

from the esophageal wall (see Fig. 2-17), and it appears that such small esophageal tributaries, when torn from the esophagus, have the benefit of contractile periesophageal hemostasis.

Intraparietal Vasculature

Previous claims that essential nutritional vessels arise from the intercostal or phrenic arteries or directly from the aorta could not be confirmed.⁶ The minute extra-esophageal branches enter the esophageal wall, pass through the tunica muscularis, and give off branches to the muscle before they form the wide vascular plexus within the submucosa and mucosa as seen in Figure 2-18. The clear continuity of the vessels and the rich anastomosing intramural vascularity^{6,40,41} explain why a mobilized esophagus retains an excellent blood supply over a long distance⁴²; on the other hand, the extremely small caliber of the nutritional vessels also explains leaks after esophagointestinal anastomosis in the event of mechanical damage to the microvascular circulation.

Blunt pull-through stripping of the esophagus without thoracotomy for cancer of the cardia has found an increasing number of advocates.^{4,6,11,41} It is described as a relatively safe procedure^{6,11,41} that involves minor blood loss, provided that dissection is undertaken close to the esophagus. When hemorrhage has occurred after stripping of the esophagus, it was most often from the site of malignant tumor fixation and, in particular, from injury to the azygos vein.

Venous Drainage

Intraparietal Veins and Plexuses

The most comprehensive macroscopic description of esophageal venous drainage was presumably presented by Butler⁴³ in 1951. He classified the esophageal veins

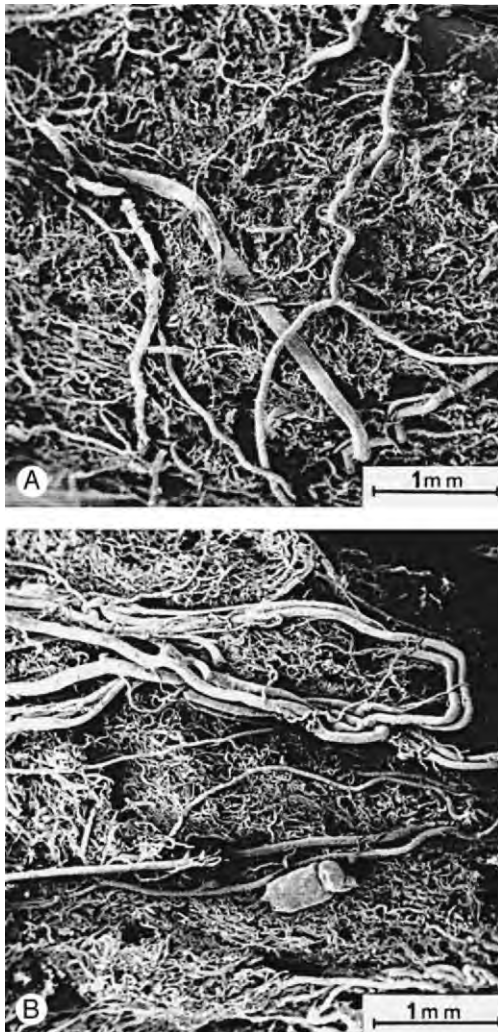


Figure 2-18. Scanning electron micrographs of complete vascular casts using a specially created resin without particles. The microvascular supply in the esophageal submucosa in the midesophagus (A) and in the cardia (B) is displayed. The vessels form a polygonal meshwork overlying the mucosa. (Courtesy of Duggelin and Liebermann-Meffert, Basel.)

into *intrinsic* and *extrinsic* veins, referring to intra-esophageal and extraesophageal wall veins. The intra-esophageal veins include a subepithelial plexus in the lamina propria mucosa that receives blood from the adjacent capillaries. Aharinejad et al.⁴⁰ described two small veins that usually accompany the arteries in the lamina submucosa, pierce the muscular wall of the esophagus together with the perforating arteries, and then form the extramural veins at the surface of the esophagus.⁴⁰ No valves were found within the esophageal venous circulatory system.^{40,43}

It is clinically noteworthy that two clearly delineated venous plexuses are present beneath the mucosa of the hypopharynx. These plexuses had been described in 1918 by Elze and Beck,⁸ but their report had not hitherto been well appreciated (Fig. 2-20). One plexus lies

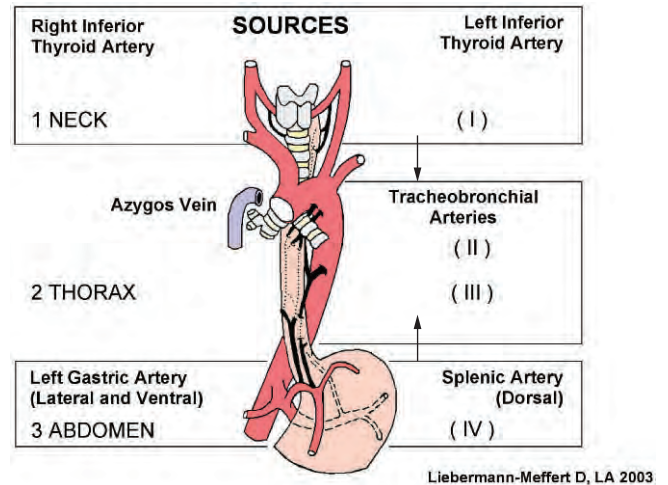


Figure 2-19. Extravisceral sources of arterial blood supply to the esophagus, intramural anastomoses (dotted line), and topographic relationship of the azygos vein to the esophagus and tracheal bifurcation. The arrows indicate the direction of flow.

on the dorsal aspect of the inferior constrictor muscle, and the other is in the midline posterior to the cricoid cartilage. This is exactly at the level of the pharyngo-esophageal junction. In the 10 specimens restudied by Liebermann-Meffert,⁸ the plexuses were located within an extremely thin submucosa; both were 2 to 3 cm broad and 4 cm long. The veins were up to 4 mm thick and of mostly longitudinal orientation, similar to Figure 2-20. The plexuses receive blood from the mucosa of the laryngopharynx and esophagus and drain into the thyroid and jugular veins. Considered to account for the postcricoid impression on the esophagus (for reference, see legend for Fig. 2-20), they may be involved in the “globus sensation” in patients with venous stasis and tissue swelling. It is tempting to postulate that the plexuses also contribute to some extent to the competence and action of the UES.

It may be of further clinical interest that a specialized venous arrangement, clearly documented by Vianna et al., is present at the terminal esophagus (Fig. 2-21). It has been suggested that these venous anastomoses possibly constitute a communication between the azygos and the portal systems. The intermediate “palisade zone” (see Fig. 2-21) is thought to act as a high-resistance watershed between both systems that provides bidirectional flow. Anastomoses between the systemic and the portal systems are found in the submucosa and lamina propria of the lower end of the esophagus and may enlarge in patients with portal venous obstruction to form varices.

Extraparietal Veins

The extrinsic veins drain into the locally corresponding large vessels: the inferior and superior thyroid veins, the azygos and hemiazygos veins, and the gastric and splenic veins. One point of surgical interest is that because of the

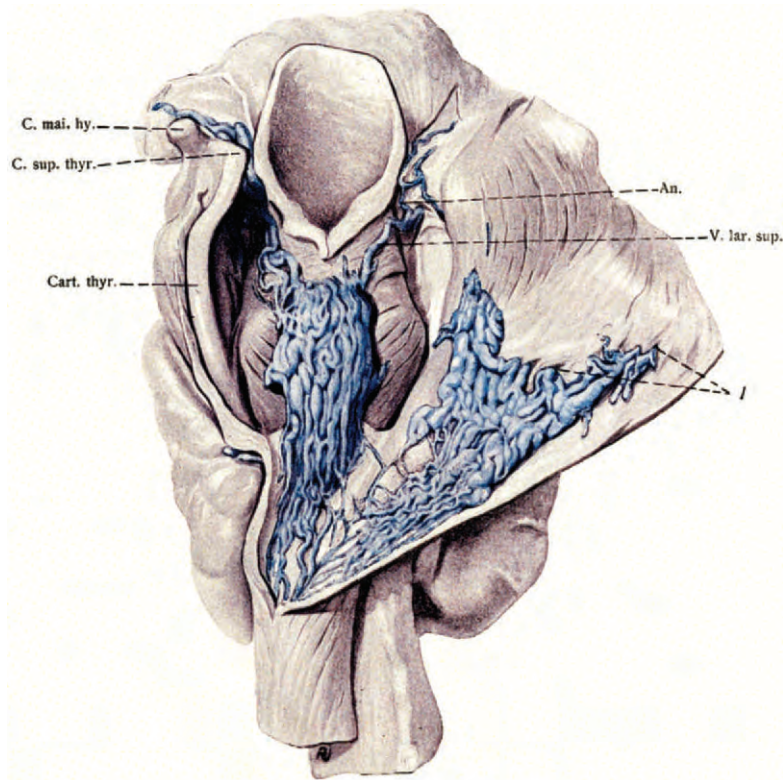


Figure 2-20. The hypopharyngeal-endoesophageal venous plexuses, which are located just underneath the mucosa. Original drawing. (From Elze C, Beck K: Die venösen Wundernetze des Hypopharynx. Z Ohrenheilk 77:185, 1918.)

proximity to the hilum of the lung and its lymph nodes, the azygos vein is one of the initial structures to become involved by extramural spread of tumors of the midesophagus (see Fig. 2-10). In this situation, the azygos vein may easily be injured during esophageal resection. In particular, during blunt pull-through stripping, injury to this vein is a high-risk factor for fatal hemorrhage. Collateral circulation between the azygos vein and the hemiazygos vein is well known. However, the hemiazygos, the accessory hemiazygos, and the superior intercostal trunks may also form a vessel that does not connect with the azygos vein. The hemiazygos vein, if not ligated out, can be a source of severe bleeding when the esophagus is resected through a right thoracotomy.

Lymphatic Drainage

Initial Lymphatic Pathways

The lymphatic drainage in healthy individuals has been sparsely investigated. At present, the authors are conducting a study to demonstrate the pathways of the lymphatic drainage of the esophagus. The histologic picture of the initial lymphatics (as demonstrated by electron microscopy) resembles that elegantly shown by Lehnert in Figure 2-22 concerning the stomach.

Lymph capillaries may commence in the tissue spaces of the mucosa and then unite to form blind endothelial sacculations or channels (see Fig. 2-22). These initial lymphatics appear to originate exclusively in the region between the mucosa and the submucosa and form a

network of collecting channels within the submucosa that run parallel to the organ axis (Fig. 2-23). Eventually, the plexuses give off branches that pass the muscle layers and empty into the collecting subadventitial and surface trunks. In contrast to the esophageal veins, all these channels possess valves (see Fig. 2-23).

Clinical Implication The concept that lymph flows in the submucosal channels more readily longitudinally than through the few transverse connections in the muscle (see Fig. 2-23) and that only finally does lymph flow through the subadventitial lymphatics and small ducts into the mediastinal lymph nodes is supported by the clinical observation that initial tumor spread follows the longitudinal axis of the esophagus within the submucosa rather than extending in a circular manner. A paucity of lymphatics within the lamina mucosa and the abundance of submucosal lymphatic channels¹⁰ may explain why intramural cancer spreads predominantly within this layer. Unappreciated malignant mucosal lesions may be accompanied by extensive tumor spread underneath an intact mucosa, and tumor cells may follow the lymphatic channels for a considerable distance before they pass the muscular coat to empty into the lymph nodes. A tumor-free margin at the resection line, as confirmed from the anatomic point of view, does not guarantee radical tumor removal. This may be consistent with the relatively high postoperative recurrence rate at the resection line, including satellite tumors and metastases in the submucosa far distant from the primary tumor,⁴ even if the margins at the resection line were previously tumor-free.

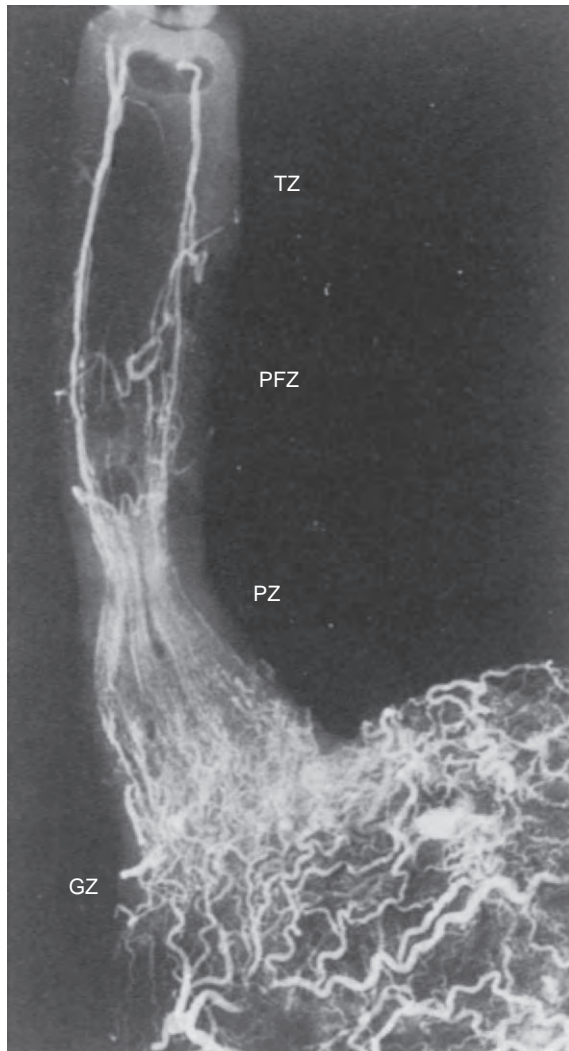


Figure 2-21. Radiograph of the venous circulation at the esophagogastric junction and the esophagus after injection with barium gelatin. This example shows the various zones of different venous architecture, such as the gastric zone (GZ), the palisade zone (PZ), the perforating zone (PFZ), and the truncal zone (TZ), as well as the irregular polygonal network of the proper gastric veins. (From Vianna A, Hayes PC, Moscoso G, et al: Normal venous circulation of the gastroesophageal junction: A route of understanding varices. *Gastroenterology* 93:876, 1987, with permission.)

From clinical observations in cancer patients,⁴ one may deduce (see Fig. 2-24) that lymph from above the carina flows in a cranial direction into the thoracic duct or subclavian lymph trunks whereas lymph from below the carina flows mainly toward the cisterna chyli via the lower mediastinal, left gastric, and celiac lymph nodes. Flow may, however, change under pathologic conditions. When lymph vessels become blocked and dilated because of tumor invasion, the valves become incompetent and the flow reverses (see Figs. 2-22 and 2-23). This explains the retrograde and unexpected spread of some malig-

nant tumors but limits the value of establishing pathways of normal flow.

Lymphatic Ducts and Lymph Nodes

The lymphatic ducts at the surface of a healthy esophagus are thought to empty into the regional lymph nodes. As has been postulated,¹³ the thoracic esophagus drains into the paratracheal, tracheobronchial, carinal, juxtaesophageal, and intra-aorticoesophageal lymph nodes, and the abdominal esophagus empties into the superior gastric, pericardiac, and inferior diaphragmatic lymph nodes. Large, often dark lymph nodes normally accumulate around the tracheal bifurcation (Fig. 2-24). However, the author's study failed to display the classic chain of lymph nodes surrounding the esophagus as described in textbooks and illustrated by Netter.¹³ Instead, 17 noncancerous autopsy specimens revealed only a small number of lymph nodes being prominent in the periesophageal tissue. This observation coincides with the report of Wirth and Frommhold,⁴⁴ who found mediastinal lymph nodes in only 5% of 500 normal lymphograms. Moreover, the authors microscopically identified multiple tiny lymph nodes with a diameter less than 1 mm located in the entire tracheoesophageal sulcus. It is conceivable that such small lymph nodes could increase in size when involved in inflammatory processes or tumor disease, thus augmenting the number of visible nodes. Furthermore, regional differences may potentially prevail.

Thoracic Duct

The thoracic duct begins at the proximal end of the cisterna chyli, at the level of the 12th thoracic vertebra, and passes up through the diaphragm via the aortic foramen. It then ascends through the posterior mediastinum, between the aorta on its left and the azygos vein on its right aspect, and continues left dorsal to the esophagus (Fig. 2-25; see also Fig. 2-10). At the level of the fifth thoracic vertebra and just above the arch of the azygos vein, the thoracic duct inclines to the left to become left side positioned with regard to the esophagus and spine.⁴⁴ Then it ascends lateroposteriorly parallel to the trachea and esophagus to convey the lymph into the bloodstream and terminates at the confluence between the left subclavian and jugular veins. There are, however, numerous anatomic variations.^{13,44} The close local relationship of the delicate thoracic duct to the esophagus and trachea accounts for the occasional injury causing chylothorax during esophagectomy and cervical anastomosis.¹¹

Innervation

Innervation of the esophagus is through the visceral (splanchnic) component of the autonomic nervous system. It consists of two parts, the sympathetic and the parasympathetic systems, that exert antagonistic influences on the viscera. The various pathways have been described in detail elsewhere.²⁴ The nerve trunks and the

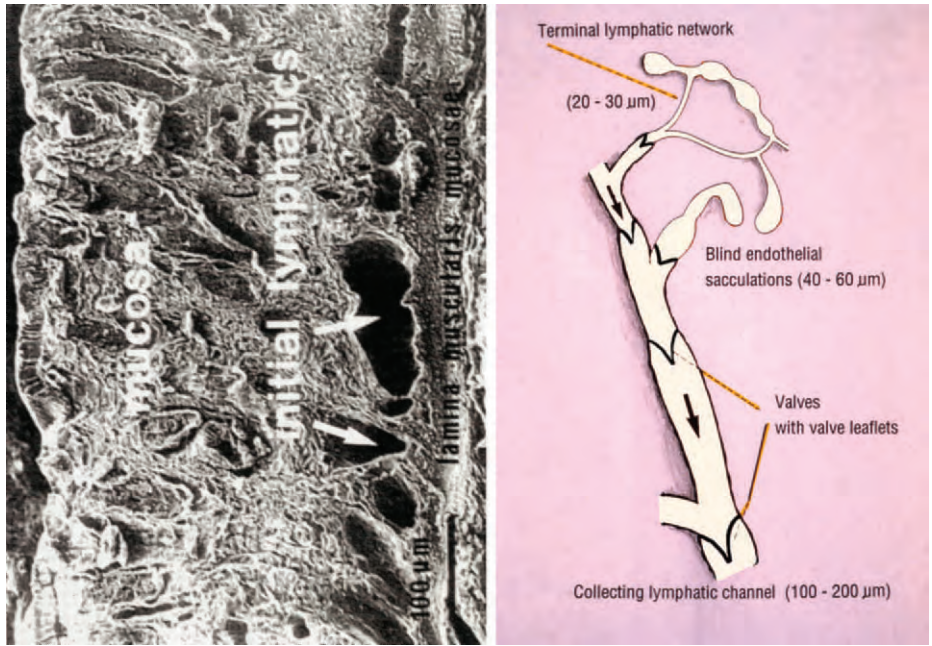


Figure 2-22. Initial lymphatics (arrows) between the lower border of the tunica mucosa and the tela submucosa seen on a histologic photomicrograph and in a schematic drawing. This view is taken from the gastric wall, but it also seems to be of relevance for the esophagus. (Left, from Lehnert T, Erlandson RA, Decosse JJ, et al: Lymph and blood capillaries of the human gastric mucosa. *Gastroenterology* 89:939, 1985.)

LOCAL LYMPHATIC DRAINAGE OF ESOPHAGEAL WALL

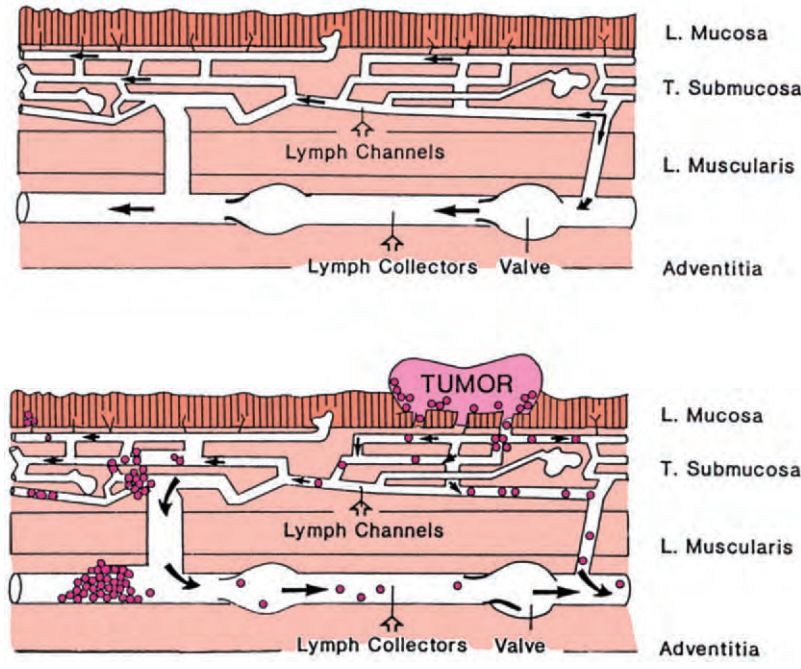


Figure 2-23. Lymphatic pathways in the esophageal wall. The suggested pattern of lymph flow is shown to explain the possible local and distal spread of tumor cells, including the block of distal lymphatics. The embryologic development and the presence and alignment of valves suggest this pattern of lymph flow, although it has never been substantiated experimentally up to now.

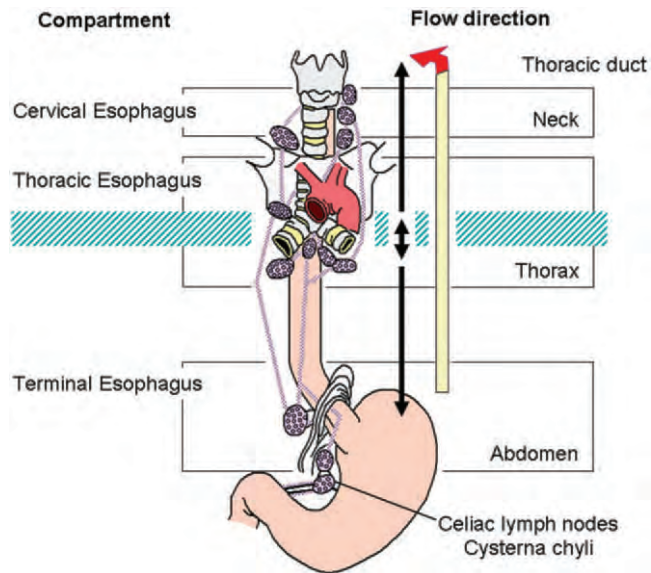


Figure 2-24. Knowledge of the direction of lymph flow and the position of major lymph nodes is essential for understanding the potential spread of an esophageal malignancy. Lymph from areas above the tracheal bifurcation drains mostly toward the neck, and that below the tracheal bifurcation flows preferentially toward the celiac axis. Lymph flow at the bifurcation appears to be bidirectional. The dimensions of the lymph nodes are out of scale. In the normal, nonmalignant condition, esophageal and mediastinal lymph nodes are difficult to discern because of their small diameter of only 3 to 7 mm. Lymph nodes that drain the lung are usually bigger and can be easily visualized by their carbon particle content.

principal branches are composed of parallel nerve bundles that contain efferent or afferent axons. The epineurium, a dense connective tissue sheath, surrounds the nerve trunk.

Extramural Innervation

The *sympathetic nerve* supply, according to the classic description, is via the cervical and thoracic sympathetic chain, which runs downward lateral to the spine (Fig. 2-26). The other sources of sympathetic supply to the middle and lower portions of the esophagus are the cardiobronchial and periesophageal splanchnic nerves, which derive from the celiac plexus.¹³ Interconnecting with fibers of the parasympathetic cervical and thoracic plexus, the sympathetic nervous system also uses the vagus nerve as a carrier for some of its fibers.^{13,24}

The *vagus nerve* is the 10th cranial nerve and is derived from the dorsal vagal nucleus. The fibers that supply the striated musculature in the pharynx and esophagus, however, derive from the nucleus ambiguus. The vagus is a mixed nerve that also carries sensory fibers from the superior ganglion and inferior ganglion (nodose ganglion). As thick trunks, the right and left vagus nerves descend bilaterally (see Fig. 2-26); they reduce their

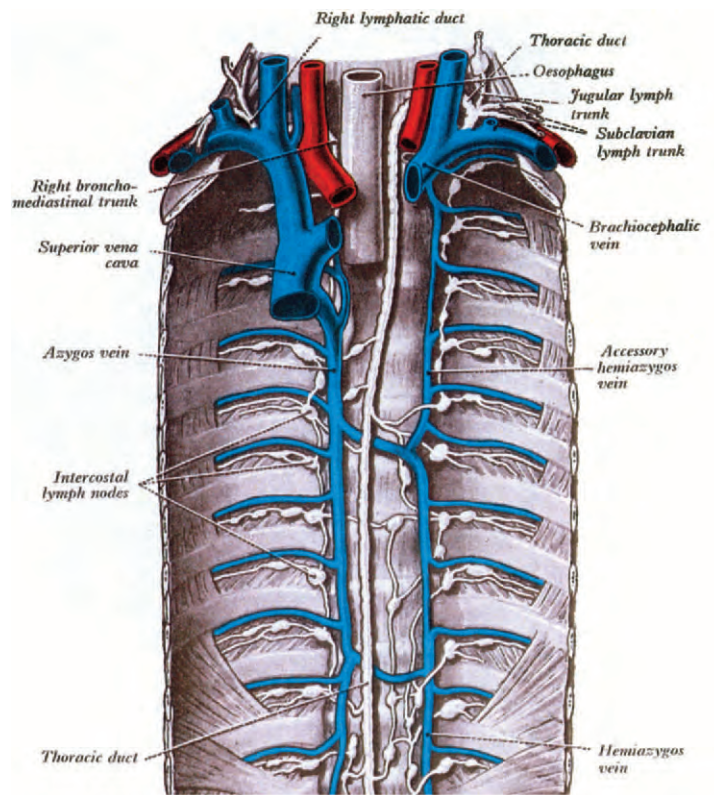


Figure 2-25. The upper thoracic and right lymphatic ducts. (From Warwick R, Williams RL [eds]: Gray's Anatomy, 35th ed. Edinburgh, Longman, 1973, p 727.)

diameters by giving off fibers in the neck to the superior laryngeal nerves (SLNs), which innervate the pharynx and larynx musculature. The inferior (recurrent) laryngeal nerves (RLNs) originate within the chest. The right RLN leaves the vagus and turns dorsally around the subclavian artery (see Fig. 2-26). The left RLN leaves the vagus and circles around the aortic arch. On both sides, the RLNs ascend as slack cords that sinuously pass upward within the lateral peritracheal loose connective tissue, the left being closer to the tracheal groove than the right (Figs. 2-27 and 2-28).¹² The left RLN lies closer to the esophagus than the right does. Both RLNs give off 8 to 14 branches to the esophagus and trachea in equal distribution.¹² When stretched, they are 2.5 mm to 1 cm long. Toward the cranial aspect, the RLNs “disappear” beneath the thyroid glands, where the thyroid vessels, in an unpredictable manner, encircle the RLNs in the fashion displayed in Figure 2-29. They enter the larynx laterocaudal to the cricopharyngeal muscle (see Figs. 2-28 and 2-29; see also Fig. 2-4A). Except for the cricothyroids, they innervate all the muscles of the larynx via small branches.^{12,45} Injury to the RLN is an unwelcome and not infrequent complication of operations on or near the upper thoracic and cervical esophagus. Because the RLN and SLN supply the same laryngeal